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Electron-microscopic Studies on the Cytopathology of Maize Plants Infected with the Maize Rough Dwarf Virus (MRDV)

By

N. KISLEV, I. HARPAZ and M. KLEIN

Hebrew University, Faculty of Agriculture, Rehovot, Israel

Electron micrographs of sections of maize leaves experimentally infected with MRDV revealed many virus-like particles. Two kinds of particles were distinguished in the cytoplasm of infected cells, which do not appear in cells of uninfected plants:

1. "Immature" dense particles, about 50 mμ in diameter; these are found mainly in big masses of "viroplasm" and are composed of many fibrils.

2. "Mature" particles, about 70 mμ in diameter, consisting of a dense inner core, 50 mμ in diameter, and a less dense envelope. These particles appear free in the cytoplasm or within tubules of various lengths. Both kinds of particles sometimes form pseudocrystals.

Chloroplasts, mitochondria and nuclei of the infected cells are only rarely affected by the virus infection.

Crystals that normally occur in the cytoplasm of maize plants, whether surrounded by membranes or not, seem to be found in greater numbers in infected plants.

Introduction

The rough dwarf disease of maize (MRDV), known in the Mediterranean Basin for nearly 20 years, is caused by a planthopper-borne virus (HARPAZ, 1966). The characteristic symptoms of the disease are: *a)* much darker green coloration of the infected plant, as compared to non-infected ones; *b)* severe dwarfing of the affected plant due to extreme shortening of its internodes; and *c)* the development of numerous minute swellings along the veins on the lower leaf-surface, giving it a rough texture. Hence the name "rough dwarf" given to the disease.

The first studies on the histopathology of this disease were made, under the light microscope, by BIRAGHI (1952), who suggested that the leaf vein galls (swellings), which he termed "hyperplasia", are caused by abnormal developments in the phloem tissues of the affected plants.

In more recent studies, carried out in Italy, GEROLA and BASSI (1966), GEROLA et al. (1966), LOVISOLO and CONTI (1966), and VIDANO (1967), using electron-microscope techniques observed virus-like particles in the vein galls of maize and wheat plants experimentally infected with the virus, as well as in the fat-body, salivary glands, mycetome cells, gut, hypodermis, muscles and tracheae of the inoculator insect vector *Laodelphax (= Delphacodes) striatellus* (Fallén).

In the present work a number of ultrastructural changes induced in maize leaf cells by the MRDV infection will be described and compared to those reported from Italy by the above-mentioned authors. This was deemed particularly worthwhile in view of the doubts expressed by BLATTNY, POZDENA and PROCHAZKOVA (1965) as to whether the maize rough dwarf disease described from Israel is produced by the same virus causing rough dwarf in Italy and Czechoslovakia.

Materials and Methods

Very young maize seedlings of the hybrid varieties "Nevé Yaar 22" and "Nevé Yaar 182" were inoculated with the virus at their coleoptile stage by means of the planthopper vector *L. striatellus* according to a method described by HARPAZ et al. (1965). The inoculative planthoppers had previously acquired the virus from MRDV-infected wheat source plants. The inoculated seedlings, along with uninoculated control plants, were placed in a temperature-controlled greenhouse at 24 ± 1 °C, which is the optimum temperature for symptom development in the maize test plant (HARPAZ, 1964).

Two months after inoculation, small leaf strips were cut out from the infected and non-infected plants, and fixed for 1 hour in cold 5% glutaraldehyde in 0.1 M phosphate buffer pH 7.4. Further samples were taken from plants 4 months after inoculation, but then the leaves were sliced while submerged in the cold fixative in order to avoid damage to cells.

After fixation the tissues were rinsed three times in the same buffer and post-fixed for 1 hour in cold osmium tetroxide (1 or 2% in the same buffer). The samples were then rinsed again, dehydrated in ethanol and embedded in Epon 812 (LUFT, 1961). Ultrathin sections were cut in a microtome, stained with uranyl acetate and examined under JEM – 7 and RCA MU3 electron microscopes.

A quicker method, which we used for observing the virus particles under the electron microscope, was the one described by LOVISOLO (1967), though with less success as compared to that reported by him.

Results

Examination of cross and tangential sections of leaves from diseased plants revealed the presence of large numbers of virus-like particles not found in the non-infected control plants. They are distributed mainly in the cells of the vein galls. These cells in the neoplasia are more or less oval or circular in cross section, and elongate in tangential sections. They have thick walls, and their cytoplasm is full with virus particles (Figs 1, 2, 6).

Two months after inoculation these virus particles can already be seen in mesophyll cells. There are some vacuoles in chloroplast edges of both mesophyll and parenchymatous sheath cells, as GEROLA and BASSI (1966) have already noticed. However, the same kind of vacuoles occur also in non-infected plants (Fig. 3)

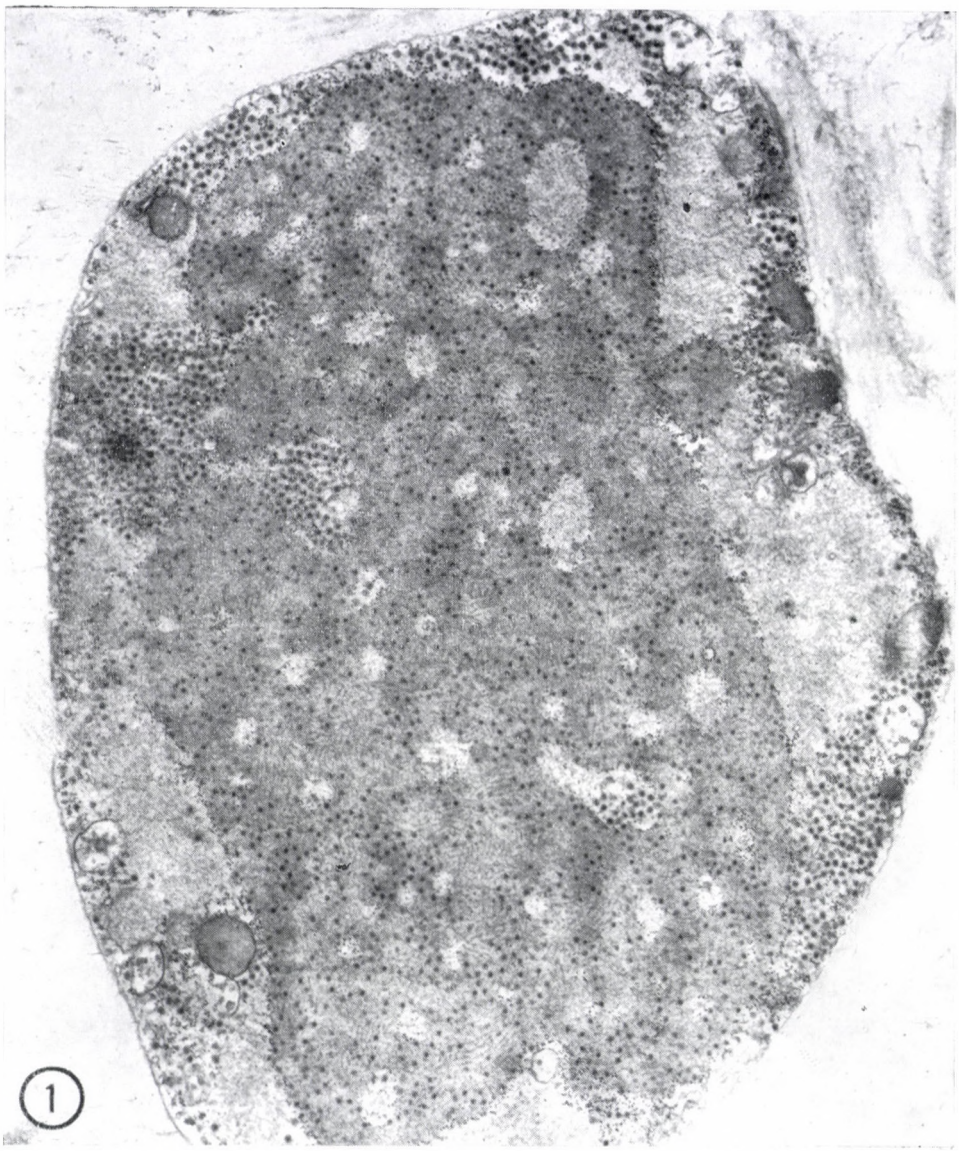


Fig. 1. Cross section of a neoplastic cell showing dense viroplasm rich in membraneous structure and many "core" virus particles. "Mature" virus particles are seen as larger black dots free in the cytoplasm and in areas of lesser density within the viroplasm. Ribosomes are seen at viroplasm edges. × 10 800

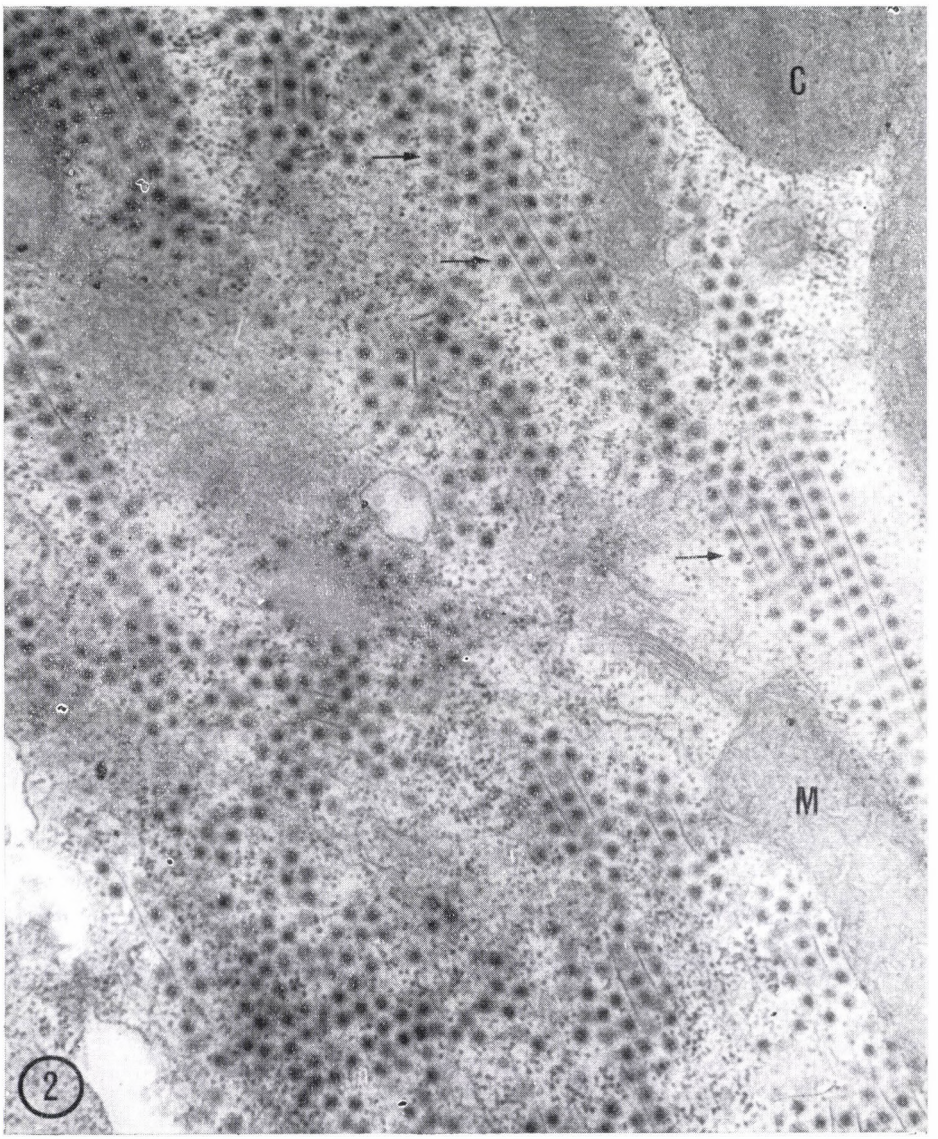


Fig. 2. Tangential section of a cell filled with "mature" virus particles, some within tubules. The particles consist of a dense-staining "core" and a coat of lesser density (arrows). Chloroplasts (C) and mitochondria (M) are unchanged. × 39 000

and therefore cannot be safely attributed to the effect of the virus infection (see also Discussion).

Crystals, non-enclosed in membranes, were observed in the cytoplasm in both healthy and infected plants (Fig. 4), they were however more abundant in

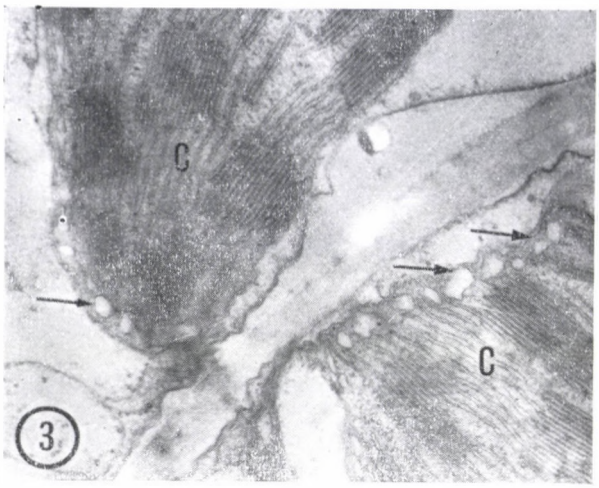


Fig. 3. Edges of two chloroplasts (C) with many vacuoles (arrows) from two cells of an uninfected plant. ×34 000

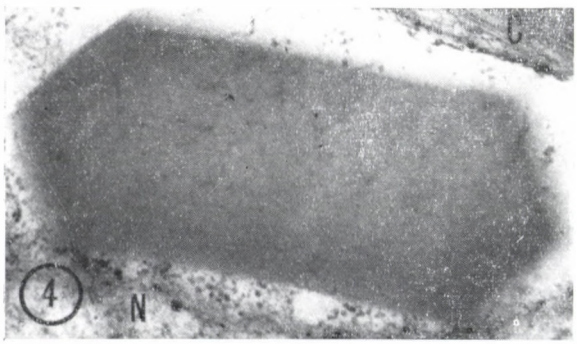


Fig. 4. Crystal in cytoplasm of mesophyll cell from uninfected plant. Chloroplast (C) edges and nucleus (N) are seen. ×45 000

diseased plants. Similar crystalline bodies were also observed by GEROLA and BASSI (1966). These crystals though varying in size and shape resemble protein crystals known in other plants (LANCE-NOUGARÈDE, 1965, 1966).

A different type of crystal occurs in the veins. These are enveloped within a double membrane and may be found free in the cytoplasm after its rupture (Fig. 5). Probably the same type of crystals were recently observed in the xylem of maize root tips by ARNOTT and DAUWALDER (1966).

Ultrastructural changes induced by the virus infection are more or less restricted to those tissues where virus replication actually takes place. Part of these changes were already described by GEROLA and BASSI (1966) and by GEROLA

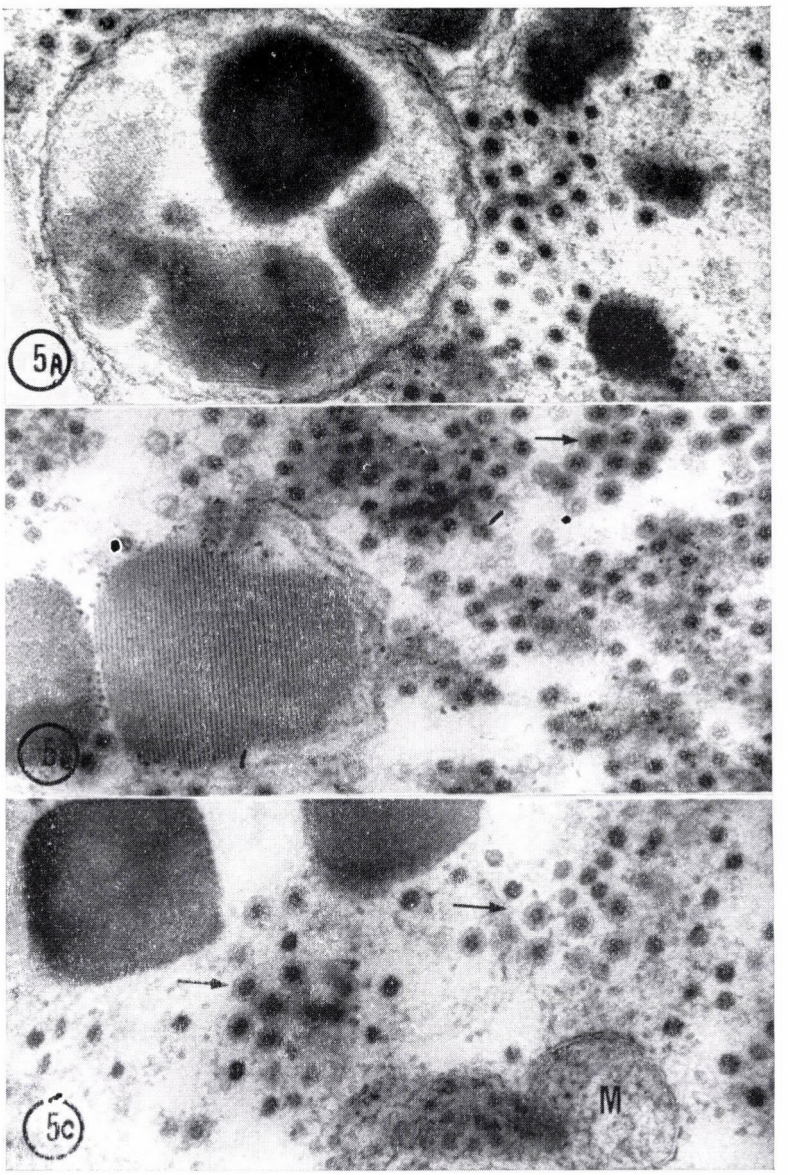


Fig. 5. Crystals within complete (5A), and ruptured (5B) membranes and free in cytoplasm (5C, 5A). Many "mature" virus particles are seen, with dense-staining core and less dense envelope (arrows). Some virus particles are seen within a membraneous body which might be mitochondria (M). Fig. 5A ×54 000; Fig. 5B ×50 000; Fig. 5C ×68 000

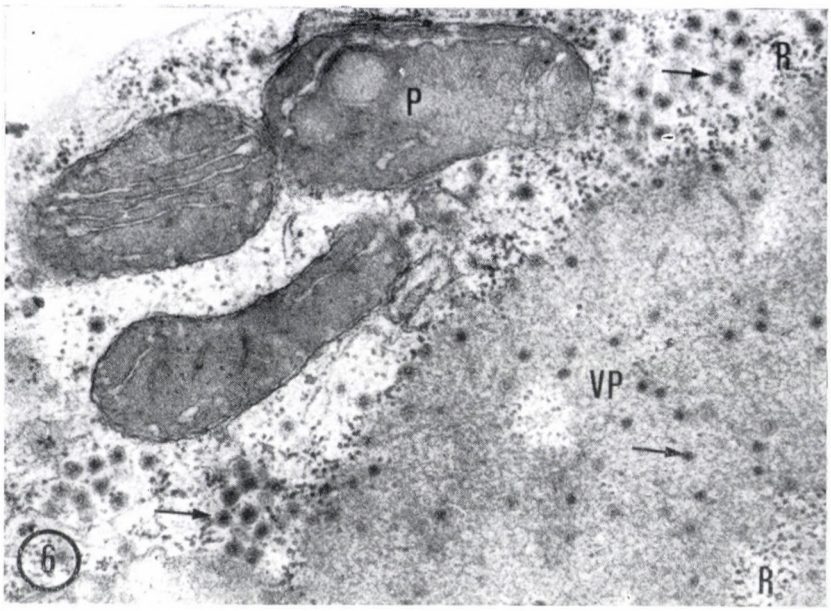


Fig. 6. Part of neoplastic cell showing viroplasm (VP), ribosomes (R), virus particles (arrows) and three plastids (P) with few thylakoids. × 39 000

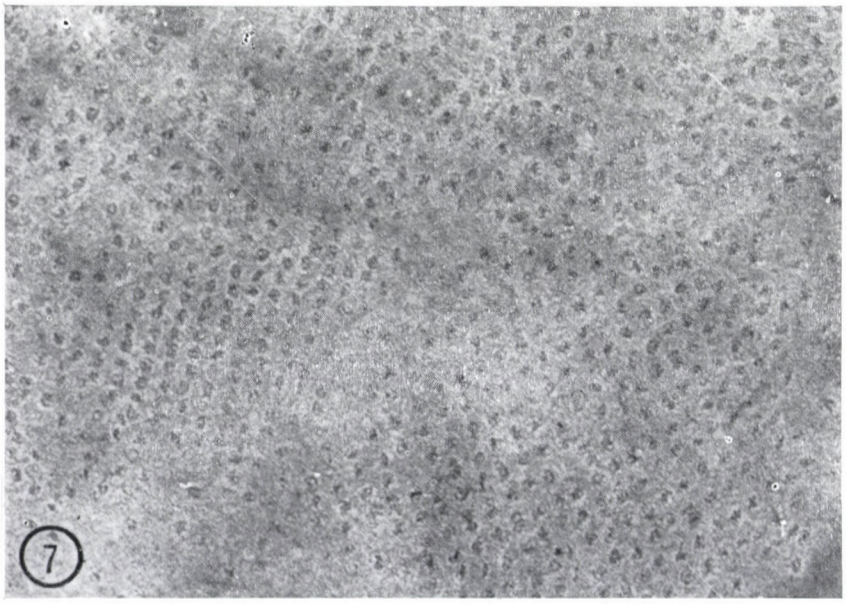


Fig. 7. Pseudo-crystal arrangement of "core" virus particles within viroplasm. × 42 000

et al. (1966), namely the development of "viroplasm" masses that contain many fine fibrils and "immature" or "core" virus particles, about 50 mμ in diameter. These masses may occupy most of the cytoplasmic space, and are surrounded by many ribosomes at their edges (Figs 1, 2, 6). Close to these masses, and sometimes also in areas of lesser density in the viroplasm, many "mature" virus particles may be seen (Figs 1, 2, 5, 6). They have a diameter of about 70 mμ, and consist of a dense-staining core, 50 mμ in diameter (probably RNA), surrounded by an envelope of the same stainability as the viroplasm. Part of the "mature" viruses appear within tubules of various lengths (Fig. 2). Sometimes the viruses seem to form pseudo-crystals. At least in one instance a pseudo-crystal of "immature" particles was observed (Fig. 7). Virus particles were mostly found in the cytoplasm, though on rare occasions similarly looking particles were observed also in lamellar bodies, namely in mitochondria (Fig. 5C) and chloroplasts.

Changes in cell organelles were noticed mainly in the chloroplasts of the neoplastic cells, which contain only few long thylakoids (Fig. 6) that are sometimes grouped together. In the mesophyll and parenchymatous sheath cells the chloroplasts are unchanged even when the cytoplasm is filled with virus particles.

Discussion

The ultrastructural changes found in MRDV-infected maize leaves are similar to those found by GEROLA and BASSI (1966). However, evidence published to date does not render a complete picture of the stages of development of this virus. It is clear, however, that the massive appearance of fibrils and tubules in the cytoplasm is connected with the virus development, since they are not found in the non-infected plants. The fine fibrils in the viroplasm (Fig. 1) resemble x-bodies, or fibrils, found in cells of petunia plants infected with petunia ringspot virus (RUBIO-HUERTOS, 1966).

Pseudo-crystals of "core" particles (Fig. 7) may represent early stages of development, as found in the case of bacteriophages (KELLENBERGER, 1962).

Changes in cell organelles, mainly changes of plastids in neoplastic cells (Fig. 6) may be regarded as an arrested development of the proplastids to "mature" chloroplasts. This kind of plastids, with only few thylakoids and some inclusions are anticipated in translucent or albicate leaf tissues, as described in variegated leaves of other plants (MURAKAMI, 1962), or in chlorotic leaves of iron-deficient plants (BOGORAD et al., 1958; LAMPRECHT, 1961; KISLEV, SWIFT and BOGORAD, 1965).

It is not clear yet whether this abnormal plastid is the direct effect of virus development within these cells, or the result of post-infectional division of cells that originally contained this type of plastids. This development of neoplastic tissue could be equated to the development of parenchymatous sheath cells in maize leaves which also have one type of chloroplasts with a few grana (HODGE, MCLEAN and MERCER, 1955; WEIER, STOCKING and SHUMWAY, 1967). Another instance is the development of chloroplasts with long thylakoids observed in the

palisade layer of *Cannabis sativa* leaves following thiouracil treatment (HESLOP-HARRISON, 1963).

In contrast to the cells of the leaf galls, there are almost no changes in chloroplasts of parenchymatous sheath and mesophyll cells even four months after infection, when the cytoplasm is already loaded with virus particles. Rows of vesicles in chloroplasts, described by GEROLA and BASSI (1966) as MRDV-induced manifestations were found in our study also in uninfected control plants. Their size, however, did not increase in course of time following infection. In fact the same kind of vesicles were also observed in electron micrographs of sections of maize leaves taken by Vatter (RABINOWITCH and GOVINDJEE, 1965) and by WEIER et al. (1967), both from plants grown in the United States, where MRDV is not known. The vesicles appear to be swellings of the outer thylakoids, or their edges.

As regards the relationship between the Israel maize rough dwarf virus and the Italian one (see Introduction), it now appears from the viewpoint of morphology of the virus as well that we are dealing with the same entity in both countries. In fact, the identity of the three maize rough dwarf viruses, described from Italy, Israel and Czechoslovakia respectively has already been claimed earlier on the strength of symptom expression and vector relations (HARPAZ, 1966).

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Influence of Host-exudates on Conidial Germination in *Colletotrichum falcatum*[1]

By

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The present host-exudate studies have shown that some chemical nutrients exude out from most of the aerial parts of sugarcane plant, i.e. nodal region of the stalk, leaf sheath and leaf mid-rib, through passive ex-osmosis and which help considerably in increasing the germination of conidia of *Colletotrichum falcatum* Went. Thus, it indicates a chance of greater infection on these plant parts. No such exudate, however, appears from leaf lamina. Exudates from older and mature plant parts seem to stimulate more than the younger ones. This might be one of the reasons as to why the former are more commonly infected with red rot than the latter. The response of the light type isolates is more pronounced than the dark type ones.

Introduction

The importance of host-exudates in the germination of spores was first emphasized by DE BARY as early as 1886 in his studies with *Sclerotinia libertiana* where he remarked that certain amount of feeding was necessary on the surface of the host and he termed this as "saprophytic nourishment". In his views, this nourishment exuded from the host through passive ex-osmosis and helped considerably in increasing the parasitic vigour of the pathogen. Subsequent works by BROWN (1916, 1922 a, 1922 b, 1936, 1946), NOBLE (1924), RILEY (1949), BALD (1952) and FLENTJE (1959) have suggested that such nutrients, present in the host-exudates, provide some sort of chemotropic stimulus for the initiation and growth of the germ tube or the infection hyphae.

In red rot disease of sugarcane, caused by *Colletotrichum falcatum* Went, the nodal region of the cane stalk with its components, like leaf scar, root primordia, growth rings and buds, provide the chief avenues for the entry of the fungus. Leaf infection, however, is restricted to mid-rib lesions only. Present investigations were initiated to find out if any nutrient exudation is taking place from the leaf mid-rib, leaf lamina, leaf sheath and the nodal region of the cane plant. If so, then how far does this exudation help the growth and entry of the

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pathogen? The importance of the present work is greatly enhanced in view of the earlier reports (VASUDEVA, CHONA and SRIVASTAVA, 1953; BAJAJ, GANJU and CHATRATH, 1959; SINGH, 1962, 1965, 1966) which record that the conidia of the fungus, particularly of the light type, highly sporing and virulent isolates show poor germination in distilled water under optimum conditions. The results obtained on the response of six isolates of *C. falcatum* to any such stimulus are being presented in this paper.

Materials and Methods

Single spored, actively growing cultures (12—16 days old) of three typically light type, highly sporing and virulent isolates (Nos.* 244, 301 and strain 'I') and three typically dark type, sparsely sporing and weakly pathogenic ones (Nos.* 7, 78 and 300) of *C. falcatum* were used in the present investigations.

Exudates from leaf mid-rib, leaf lamina, leaf sheath and nodes of sugarcane stalk (variety Co. 647) were collected as follows: Fifty millilitres of distilled water was placed in the form of drops or thin film on the surface of the bits of thoroughly washed leaf mid-rib, leaf lamina, and leaf sheath, 25 g each, for 24 hours at 8 °C** in a refrigerator. In case of nodes, small circular bits were made out of 25 g of the plant part and their ends sealed with wax. These were soaked in 50 ml of distilled water for 24 hours at 8 °C,** thus permitting exudation only through leaf scars and root primordia. In all these cases, the 'water' containing the exudates was collected separately and filtered through Whatman filter paper No. 1; thus freed it off any solid particles.

Germination for each isolate was studied in standard conidial*** suspension (15—20 spores per microscopic field under low power) in moist chambers made out of pairs of sterilized Petri plates after 18 hours' incubation**** at 27 °C.**** The criterion for germination of a spore was the emergence of a germ tube to a length exceeding its width. Each experiment was repeated three times and the average data for each experiment were based on about 3000 spore counts/germ tube measurements.

Results

Effect of the 'water' collected from the leaf sheath pocket. CHONA (1950) and STEIB and CHILTON (1951) reported that spores lodged in the pocket or cavity formed between leaf sheath and cane stalk germinate readily and the

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** The solutions, being unautoclaved, were kept at 8 °C in a refrigerator to avoid any bacterial or fungal growth.

*** The harvested conidia were freed off the adhering external nutrients and mycelial fragments through washing them twice in distilled water by centrifugation (2800 rpm for 5 minutes, each).

**** Optimum incubation period and temperature (SINGH, 1962).

infection hyphae, thus formed, penetrate the stalk easily at the nodal region through root primordia or leaf scars. In view of this, 'water'* was collected from these pockets (somewhere approximately in the middle of the cane length) and conidial germination was studied in it to find out if there is any stimulus being provided by this 'water'. Two controls for each isolate were run, one in distilled water and the other in rain water, collected in a clean glass trough directly as it falls.

Table 1

Effect of 'water' on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage			Germ tube length (μ)		
	'Water'	Control A	Control B	'Water'	Control A	Control B
244	48.9	15.0	16.9	190.5	80.1	79.6
'I'	56.0	24.1	25.0	80.1	34.6	34.6
301	40.6	17.6	16.9	112.6	82.3	82.3
78	55.0	39.0	41.2	201.5	101.8	96.2
300	62.9	47.5	49.3	229.6	88.8	84.4
7	59.9	36.8	40.3	91.0	54.1	67.3

(A = distilled water; B = rain water.)

The presence of a distinct stimulus in 'water' is quite evident by an increase in germination as compared to both the controls. The influence being more pronounced on the light type isolates (Table 1). There was practically no or very insignificant difference between the two controls, run in distilled water and rain water.

Effect of exudate from nodal region of a cane stalk. To investigate the source of the stimulus in the 'water', exudates were obtained from young (3rd or 4th from the top) and mature (4th or 5th from the base) nodal regions of a cane stalk and the germination studied. A control for each isolate was run in distilled water; the one in rain water was avoided as no significant difference was observed between the results obtained in distilled water and rain water, evident in the previous experiment.

The results (Table 2) show the presence of a stimulatory effect in both the mature and the young cane tissue exudates, to a greater or lesser extent. Stimulation, though very little, does show that some nutritional exudation is taking place from certain weak points on the rind, like leaf scars, growth rings and root primordia at the nodes, or some fine cracks on the adjoining portion of the internode. The light type isolates exhibit greater response than the dark type ones. Also, the exudates obtained from the mature portions of the cane stalk seem to induce greater stimulation than the younger portions.

* Its source being rain, which falls on the cane stalk and leaves, and which is washed down and gets collected in these pockets.

Table 2

Effect of exudate from nodal region on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage			Germ tube length (μ)		
	Young	Mature	Control	Young	Mature	Control
244	19.6	27.0	16.6	82.3	145.1	84.5
'I'	29.3	31.7	26.5	49.8	123.5	41.1
301	24.6	28.4	16.3	75.8	173.3	71.5
78	42.4	48.7	40.0	101.8	110.5	93.1
300	48.2	51.0	46.0	91.0	91.0	82.3
7	41.5	44.5	37.5	60.6	56.3	49.8

Effect of exudate from leaf sheath, mid-rib and lamina. The results of the previous experiment indicate that some stimulatory nutrients are exuded from the nodal region. In view of the fact that in nature, leaf sheaths are quite frequently infected with red rot, an experiment was set up to study if any such exudation is taking place from the leaf sheath.

Table 3

Effect of exudate from leaf sheath on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage			Germ tube length (μ)		
	Young	Mature	Control	Young	Mature	Control
244	28.6	36.4	13.9	164.6	190.6	71.5
'I'	39.0	45.2	21.6	75.8	88.8	36.8
301	25.1	28.9	18.0	117.0	123.5	67.1
78	43.5	39.8	37.4	166.8	184.1	86.6
300	49.4	43.1	42.9	121.3	136.5	80.1
7	40.2	44.4	39.4	84.5	93.1	52.0

The results (Table 3) show that the leaf sheaths also exude some nutrients or chemicals, which stimulate the germination of conidia to a considerable extent.

In nature, mid-rib is seen to be the most common target of red rot infection and the lamina is invariably free of infection. Not much is known to explain this. However, it has been assumed to be due to the fact that mid-rib tissues are comparatively softer and easily vulnerable than that of lamina, the latter having 1 – 3 layers of thick walled epi- and hypodermis, overladen with calcium silicate and very little or no spongy tissue.

Exudates from young as well as mature leaf mid-ribs and leaf lamina were used to study the germination. The results obtained are given in Tables 4 and 5.

Table 4

Effect of exudate from leaf mid-rib on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage			Germ tube length (μ)		
	Young	Mature	Control	Young	Mature	Control
244	22.1	32.2	15.2	93.1	171.1	71.5
'I'	34.6	42.0	24.1	80.1	130.0	41.1
301	26.5	31.3	18.2	106.1	182.0	67.1
78	45.0	50.3	40.5	164.6	223.1	86.3
300	44.5	53.4	43.9	188.5	231.8	84.5
7	39.3	45.7	36.5	106.1	114.8	52.0

Table 5

Effect of exudate from leaf lamina on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage			Germ tube length (μ)		
	Young	Mature	Control	Young	Mature	Control
244	14.1	13.9	13.8	67.1	71.5	69.3
'I'	25.5	24.9	24.9	34.6	34.6	43.3
301	17.1	16.9	17.0	62.8	65.0	69.3
78	42.1	42.3	41.4	91.0	82.3	91.0
300	46.5	47.2	45.6	69.3	78.0	73.6
7	37.1	35.2	35.0	47.6	52.0	54.1

The results in Table 4 show enhanced germination. However, no such stimulation is observed in Table 5, which probably is due to the fact that no such stimulus is being provided by leaf lamina. Mature tissues are seen to induce greater response in all the isolates. The stimulatory effect is exhibited both on the percentage of germination and the length of the germ tube. The response of all the six isolates is similar.

Comparative study with the exudates obtained from sugarcane variety Co. 647 and Co. 312. Earlier studies in the text were carried out with sugarcane variety Co. 647, moderately resistant to red rot. To compare the results with those of a susceptible variety, the investigations were extended to Co. 312, a moderately susceptible variety. Exudates were obtained from leaf mid-ribs and nodal regions of the mature canes of varieties Co. 647 and Co. 312 as described in Materials and Methods. The germination was studied and the results are given in Table 6.

Enhanced germination of conidia was observed in all the four treatments. Lesser stimulation was, however, observed in the nodal exudates, both from the point of view of the germination percentage and the length of the germ tube.

Table 6

Effect of exudate from leaf mid-rib and nodal region of sugarcane varieties Co. 647 and Co. 312 on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage					Germ tube length (μ)				
	Co. 647		Co. 312		C	Co. 647		Co. 312		C
	LE	CE	LE	CE		LE	CE	LE	CE	
244	36.4	25.3	33.5	20.9	14.5	168.5	124.4	127.1	116.0	78.0
'I'	39.3	28.7	36.6	23.2	23.3	152.0	80.3	104.1	71.5	36.8
301	32.5	27.1	28.4	27.2	17.5	189.5	137.1	177.1	98.5	73.6
78	58.9	47.0	57.2	43.3	41.6	181.6	161.6	160.1	163.2	91.0
300	55.5	52.0	58.4	50.2	47.3	178.2	128.2	162.5	126.4	80.5
7	46.2	41.7	38.4	38.4	38.7	98.5	88.5	76.5	74.0	54.1

(LE = leaf exudate; CE = cane nodal region exudate; C = control.)

There is practically no difference between the effects of the exudates from Co. 647 and Co. 312, obtained either from the nodal region or the leaf mid-rib.

Effect of autoclaving and boiling on the exudate. Exudates were obtained as usual from mature leaf mid-ribs and nodal regions of cane variety Co. 647 and divided in each case, into three portions (A, B, C). Each of these portions was treated separately.

Part A – autoclaved to 10 lb pressure for 10 minutes.
Part B – autoclaved to 15 lb pressure for 20 minutes.
Part C – boiled for 20 minutes.

The germination of conidia of the six isolates was studied in these three solutions (A, B, C) and the results are given in Table 7. The control was run in distilled water.

It is seen (Table 7) that the exudates, which have been autoclaved at 10 lb pressure for 10 minutes (A), did retain the stimulatory factor almost to the same strength as in the unautoclaved one (in the earlier experiments). However, autoclaving to 15 lb pressure for 20 minutes (B) nullified the stimulatory effect or reduced it considerably both from the point of view of percentage of germination and the length of the germ tube. It appears as if the stimulatory factor has been destroyed by 20 minutes' autoclaving. Boiling, on the other hand, adversely affected germination percentage as well as germ tube length. In some cases, even no germination was observed at all.

Germination in distilled water on the surface of leaf mid-rib and leaf lamina. The presence of the stimulatory factor in the exudates from young and mature leaf mid-ribs, and its absence in leaf laminar exudate has been confirmed by

Table 7

Effect of autoclaved and boiled exudates from leaf mid-rib and nodal region of sugarcane variety Co. 647 on conidial germination of the six *C. falcatum* isolates

Isolate No.	Germination percentage						
	Leaf exudate			Cane exudate			'C'
	A	B	C	A	B	C	
244	30.0	14.6	0	19.4	14.0	1.6	12.7
'I'	37.3	24.6	0	28.9	22.3	0	21.8
301	29.8	22.0	2.3	22.4	20.2	6.0	19.0
78	56.7	45.6	7.6	49.1	45.7	0	43.0
300	61.0	47.8	5.4	52.4	38.8	2.0	45.6
7	51.4	39.4	0	42.4	29.2	9.1	36.5
244	189.4	69.8	—	148.9	58.3	9.7	69.0
'I'	104.1	34.6	—	87.6	45.1	—	34.8
301	243.0	67.3	11.4	184.0	73.4	8.6	69.3
78	234.6	67.6	4.3	196.7	67.8	—	91.0
300	202.4	84.6	21.2	166.6	80.2	4.6	73.6
7	124.1	58.9	—	78.9	61.3	7.3	54.1

('C' = control in distilled water.)

another experiment. Large number of drops of standard conidial suspension in distilled water were placed on the surface of clean (thoroughly washed under running water) mid-ribs as well as lamina, and spread into a thin film. Care was taken to avoid the suspension film from running off the surface of the mid-rib or the lamina. Germination of the conidia was studied and the results obtained are given in Table 8. The controls were run in distilled water on glass slides.

Table 8

Germination of the conidia of the six *C. falcatum* in distilled water on the surface of leaf mid-rib and leaf lamina

Isolate No.	Germination percentage					Germ tube length (μ)				
	Mid-rib		Lamina		C	Mid-rib		Lamina		C
	A	B	A	B		A	B	A	B	
244	24.1	34.8	14.4	14.8	13.9	117.5	144.0	80.1	84.9	76.4
'I'	31.2	39.6	21.6	22.6	21.8	88.8	138.2	56.2	58.1	47.2
301	27.0	34.6	16.5	16.8	16.7	116.8	146.8	65.2	68.9	65.6
78	45.0	53.1	37.1	37.2	36.4	124.3	202.4	85.6	87.1	84.2
300	46.2	56.6	41.8	44.3	42.7	161.2	226.0	92.1	93.1	90.0
7	42.0	49.6	35.8	36.4	35.0	101.6	121.2	56.0	56.8	55.6

(A= Young; B= Mature; C= Control.)

The results (Table 8) show that the conidia on the surface of leaf mid-ribs have germinated better as compared to those lying on the lamina or on the glass slides, both from the point of view of percentage of germination and the length of the germ tube. The stimulation being in the same range, thus supporting the results from the previous experiments – indicating some exudation of nutrients or chemicals taking place from the leaf mid-rib and not from the leaf lamina.

Discussion

The host-exudate investigations form an integral part of the study of interaction between a host and a pathogen in a way that influences pre-penetration and penetration phase (FLENTJE, 1959). The entry of a pathogen is invariably helped by certain chemicals or nutrients secreted by the host through passive exosmosis.

Studies were conducted with the exudates obtained from the aerial parts of sugarcane plant, i.e. stalk and leaves. Investigations with 'water' collected from the cavity or pocket formed between leaf sheath and cane stalk indicated the presence of a stimulus, which probably would give explanation for the observation of CHONA (1950), and STEIB and CHILTON (1951), i.e. why the spores lodged in this cavity germinate readily. The stimulus has been assumed to be some nutrient exuded out from the nodal region as well as leaf sheath. TURNER (1957) expressed that increased vigour of germination leads to greater chance of successful parasitic invasion. In view of this increased germination in the exudates obtained from the nodal region and leaf sheath, it could probably explain as to why these plant parts are usually better substrates for infection. Furthermore, the younger nodes show lesser stimulation than the mature ones, which probably could be due to greater amount of exudation from the latter. However, this aspect needs further investigations. No doubt, the older plant parts have considerable accumulation of secondary tissue elements all over, yet the weak points, like leaf scar, root primordia and growth rings normally continue to have comparatively thin and soft epidermal coverings (EDGERTON, 1958; and MARTIN, ABBOT and HUGHES, 1961). These weak points seem to act as good outlets for exudation in older and mature cane parts.

Leaf sheath is seen to contribute more toward this stimulation in the 'water', as evidenced by greater stimulation of germination. The former could be due to the fact that the leaf sheath tissue is soft and spongy (being constituted of thin walled parenchyma with intercellular spaces), with highly permeable outer surface (EDGERTON, 1958; and MARTIN, ABBOT and HUGHES, 1961). MAYEUX and COLMER (1960) reported that leaf sheath contains glucose and certain amino acids, like aspartic acid, glutamic acid and alanine, and thus has the ability to support the growth of several micro-organisms. It is now considered that the leaf sheath pocket provides a very suitable site for the development of nodal infection of red rot, i.e. by offering suitably soft substrate of leaf sheath and easy avenues on the nodal region (leaf scars, root primordia and growth rings) for the infection; high humidity and comparatively lower temperature because of the 'water' present in

the pocket; and nutrients exuded out from the leaf sheath and the nodal region (leaf scars, root primordia and growth rings), which exert a stimulatory effect on the germination of conidia and early development of the fungus.

Leaf mid-rib is the most common target of red rot infection and very rarely the leaf lamina is seen infected. Mid-rib tissue is comparatively thicker, softer and spongy than the leaf lamina, which is thinner and hard. The latter has 1—3 layers of thick walled (lignified) upper epi- and hypodermis overladen with calcium silicate and very little parenchyma (EDGERTON, 1958; and MARTIN, ABBOT and HUGHES, 1961). Present studies have indicated that some nutrients do exude from the leaf mid-ribs which stimulate the germination of conidia lying over these. This results in greater chance of successful infection (BROWN, 1916; 1922; 1936; and BROWN, 1946). However, no such exudation appears to take place from leaf lamina, which is quite evident from the fact that there is no such stimulation of the germination of conidia. In view of this, it is presumed that besides anatomical resistance of laminar tissue and its impervious covering, lack of laminar exudates is also a reason for the lamina not getting infected. Perhaps, the conidia do not germinate as frequently as they do on the mid-rib. This assumption is amply substantiated by the results obtained from the germination of conidia on the surface of leaf mid-rib and leaf lamina.

Mature leaf mid-ribs appear to exude more than the younger ones. This is just presumed on the basis of greater stimulation by the former. This probably is supported by the fact that the mid-rib lesions usually occur on the old and mature leaves, 3rd, 4th, 5th or still lower leaves from the top, and not on the young and the uppermost ones. These findings agree with those of RILEY (1949), who reported better germination percentage and longer germ tubes of spores of *Alternaria longipes* germinating on mature leaves than those germinating on seedlings and plant tops. The 3rd, 4th, 5th or still lower leaves from the top of the cane have been observed to serve also as better substrate, perhaps, because they are shaded and hidden from direct sunlight (EDGERTON, 1958) and hence with comparatively lower temperature in hot summer season, which is more favourable for the growth and development of the fungus.

The light and the dark type isolates exhibited similar response, isolate No. 244 and strain 'I' exhibiting the most pronounced effect. In view of TURNER's hypothesis (1957), it can be presumed that better germination of light type isolates indicated greater chance of successful infection and hence greater virulence; a factor of considerable importance in the analysis of parasitic vigour.

No detailed attempt has been made to analyze the stimulus or to characterize it in one way or the other. However, it is indicated that the stimulus is a nutrient and is not volatile. Also, it is capable of withstanding mild autoclaving of 10 lb pressure for 10 minutes. However, it cannot withstand longer autoclaving of 15 lb pressure for 20 minutes, or boiling. In view of this, it is considered that the stimulatory factor in the host exudate is some sort of organic nutrient. MAYEUX and COLMER (1960) have already reported the presence of glucose and certain amino acids, like aspartic acid, glutamic acid and alanine in the leaf sheath of sugarcane.

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Some New Statements on Distribution and Harmfulness of Black Locust Mosaic in Hungary

By

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Black locust mosaic has been found in Hungary in all places with a comparatively warm climate where there was the opportunity to examine a large number of black locust trees. In the mountains near Sopron and in Bulgarian mountains the disease could not be detected. The absence or rarity in cooler regions of large colonies of *Aphis craccivora*, obviously the most important vector of true black locust mosaic virus under natural conditions, seems to be the cause for the uneven distribution of the disease. Grafts of black locust clones on seedlings showed that mosaic may induce heavy losses. After three years of growth healthy trees had produced five to six times more wood than diseased ones. Additional losses were caused by the fact that high percentages of diseased plants died during winter time.

Introduction

The black locust (*Robinia pseudo-acacia* L.) covers about 17 per cent of the woodland in Hungary. Due to this Hungary is one of the European countries with the largest production of black locust wood (KERESZTESI, 1965). Therefore, the statement that black locust mosaic – first described as a suspected virus symptom by ATANASOFF (1935) in Bulgaria – is caused by virus (MILINKÓ and SCHMELZER, 1961) has special importance for Hungary. These and other papers (SCHMELZER, 1962/63, SCHMELZER and MILINKÓ, 1963; 1964, SCHMELZER and MILIČIČ, 1965) gave some information on the geographical distribution of the disease which has been found in Bulgaria, Hungary, Czechoslovakia, Roumania, Yugoslavia, and West Berlin.

From mosaic-affected black locusts a number of sap-transmissible virus isolates were obtained and cultured in herbaceous hosts. It was shown that they belong to two entirely different viruses, each of them having several strains in black locust. One virus is the nematode-transmissible tomato black-ring virus, the other one proved to be aphid-transmissible and obviously had not been described before. It was named "True Black Locust Mosaic Virus" (Echtes Robinienmosaik-Virus) and characterized in detail (SCHMELZER, 1967). Either of the two viruses is able to induce the symptoms of black locust mosaic when inoculated singly into black locust seedlings.

Our paper gives some new data on habitats and epidemiology of black locust mosaic and on the losses caused by this disease in Hungary.

Distribution of Black Locust Mosaic

Everywhere in the plains of Hungary, where we had the opportunity to examine a large number of black locust trees, we recorded the presence of black locust mosaic. Not only the normal form of the tree but also the vegetatively propagated form with a sphere-like head often proved to be mosaic infected. Obviously in most cases the aphid-transmissible virus is the cause of the disease in Hungary. Habitats of black locust mosaic near Kópháza, Nagycenk, Tőzegmajor, and Hegyeshalom, in several cases only some hundred meters from the border, show that the disease is probably also present in Austria.

However, it could not be found in several other regions. In vain we searched for black locust mosaic in the mountains near Sopron. In Bulgarian mountains, especially in the southern part of the Shipka pass in the Balkan mountains, where there are numerous black locust trees, we did not detect virus diseased ones. In all these higher situated regions the black aphid species *Aphis craccivora* Koch did not appear in large colonies on black locusts as they are to be found in the warm Hungarian plains. This aphid species is almost certainly the most important vector of true black locust mosaic virus under natural conditions. Apparently, the aphid needs rather warm weather conditions so that it is understandable why large colonies rarely occur on black locust in comparatively cool mountainous regions and in northern countries. In experiments *Aphis craccivora*, like *Myzus persicae* Sulz., proved to be a rather inefficient vector of true black locust mosaic virus (SCHMELZER, 1967). Therefore rather large numbers of aphids are probably needed to infect a tree, especially an old one. Only occasionally a few alatae may drift by wind from plains into higher regions and may induce an infection. According to this hypothesis we predicted that in the higher mountainous regions of Yugoslavia there will be no infection with black locust mosaic such as occurs in the neighbourhood of Belgrade, although the mountains are situated more to the south (SCHMELZER and MILIČIČ, 1965). Further investigations are needed to test the validity of our suppositions.

Damage Induced by Black Locust Mosaic

Earlier observations and measurements showed that older trees of black locust as well as younger ones seem to be severely affected by mosaic in growth and wood production (MILINKÓ and SCHMELZER, 1961). However, possible differences in genetical constitution and in environmental situations made definite statements difficult (MILINKÓ, 1967).

At the end of July 1964 we saw a trial of Dr. B. KERESZTESI at the Experimental Station Sárvár of the Institute for Forest Sciences in Budapest. In this trial twigs of some black locust trees were grafted into stools of two-year old seedlings in the spring of 1964. About ten per cent of the grafts of the variety "Fagytűrő" ("Frost Resistant") from the village Jászkisér were mosaic infected. Because the scions of this variety and also those of others were taken only from

one tree each, it is probable that the infections followed grafting. So we had rather uniform material for further investigations infected within a short period.

We measured the shoot length of twenty pairs of diseased and healthy grafts of the variety "Fagytűrő" at July 31st 1964. The plants of each pair grew in close proximity. At this time the grafts had only one shoot with no branches. The diseased plants reached only two thirds of the height of the healthy ones (Table 1). The loss in weight was estimated to one to two thirds of the controls.

Table 1

Comparison of the shoot length (in centimeters) of mosaic infected and healthy grafts of the black locust variety "Fagytűrő" at the end of July 1964 in Sárvár

Diseased	Healthy	Difference
115	160	45
108	144	36
95	184	89
108	161	53
76	143	67
120	183	63
158	187	29
121	165	44
115	196	81
123	168	45
130	160	30
118	169	51
65	164	99
115	155	40
108	180	72
74	133	59
100	152	52
78	166	88
86	167	81
103	135	32
Average 105.8	163.6	57.8
		$s_d =$ 4.76

By the kindness of Dr. KERESZTESI we had the opportunity to select 16 healthy and 16 diseased grafts. They belonged to the already mentioned frost resistant Hungarian variety and to the American variety "H. C. 4138". Care was taken to obtain as uniform material as possible. The trees were planted in the experimental field of the Plant Protection Department of the Agricultural College Mosonmagyaróvár. After planting the trees were never pruned unless they were damaged by frost. Then the killed parts were removed. Each tree had a space of 2×2 meters. The experimental field consisted of loamy sand. At yearly intervals

we measured the height of the trees and – after the second year – their stem circumference 10 centimeters above the ground (Tables 2 and 3).

Table 2

Comparison of the shoot length (in centimeters) of mosaic infected and healthy grafts of two black locust varieties after 1 to 3 vegetation periods (in Mosonmagyaróvár)

Time of measurement	Diseased			Healthy		
	20.4.65	20.4.66	28.3.67	20.4.65	20.4.66	28.3.67
Variety "Fagytűrő"	93	103	275	210	260	680
	168	199	344	228	166*	555
	115+	—	—	210	259	430
	193	198	430	247	272	500
	133	109*	330	278	268*	580
	110	88*	280	280	362	618
	187	241	540	246	278	635
	168	215	465	280	255*	590
	200	216	445	292	367	600
	128	20+	—	265	359	655
Average	149	154	389	254	285	584
s_x	12.49	25.41	33.43	8.91	19.39	23.96
Variety "H. C. 4138"	68+	—	—	212	291	552
	125+	—	—	231	290	565
	84	120	270	202	210	535
	162	185	320	210	258	470
	100	114+	—	240	310	607
	168	191	232	230	269	490
Average	118	153	274	221	271	537
s_x	16.85	19.94	25.48	5.43	14.96	19.14

+ Plants killed by frost.
* Plants heavily damaged by frost in preceding winter time.

Healthy plants, as well as diseased ones, showed after the first and after the second winter some degree of frost damage. After the winter 1966/67 there was no frost damage. Whereas none of the 16 healthy trees died, half the number of the diseased trees of "H. C. 4138" and 20 per cent of the frost resistant variety were killed apparently by the combined action of the virus and of the low temperatures in winter time. One year after grafting the average difference in height between healthy and mosaic affected plants was 1 meter. At the end of the third year the differences were 2 to $2^1/_2$ meters and the stem circumference of diseased trees amounted to only 50 to 60 per cent of the controls. At this time it was estimated

Table 3

Comparison of the stem circumference (in centimeters) of mosaic and healthy grafts of two black locust varieties after 2 and 3 vegetation periods (in Mosonmagyaróvár)

Time of measurement	Diseased		Healthy	
	20.4.66	24.3.67	20.4.66	24.3.67
Variety "Fagytűrő"	4.0	8.50	10.5	20.50
	8.0	7.00	10.0*	15.25
	+—	—	9.0	16.50
	6.5	12.50	10.0	21.50
	4.0*	11.00	12.0*	21.00
	4.0*	6.50	13.0	17.50
	8.5	12.50	11.0	16.50
	6.5	10.50	11.5*	17.50
	6.0	9.00	12.5	15.00
	0.4*	—	12.0	19.50
Average	5.32	9.69	11.15	18.08
s_x	0.83	0.82	0.40	0.75
	+ —	—	10.0	16.00
Variety	+ —	—	11.0	16.50
"H. C. 4138"	5.0	8.00	6.5	16.50
	7.0	11.00	9.0	11.00
	4.0*	—	12.0	18.50
	7.0	8.00	9.5	16.50
Average	5.8	9.00	9.7	15.83
s_x	0.68	1.00	0.73	1.03

+ * for explanation see table 2.

that a diseased tree produced only 15 to 20 per cent of wood in comparison to a healthy one (Fig. 1). This large loss exceeds our previous estimations which were based on very young seedlings less than 18 months old, or on old trees obviously not infected at a very young stage. However, because of the death of infected trees there was an even greater loss with mosaic affected plants which was not previously recorded. It is probable that the real loss may amount to an average of 85 to 90 per cent of the wood production of healthy trees.

Discussion

There is no doubt that black locust mosaic is widespread in South-East Europe and that true black locust mosaic virus is its most frequent cause. From the grafts used for the determination of growth reductions unfortunately we had



Fig. 1. Healthy (right and left) and mosaic diseased (in the middle) black locust grafts of the frost resistant variety after the third vegetation period

the oppurtunity to identify the true black locust virus only in the American variety, but by the mentioned virus obviously both were infected.

It seems that black locust mosaic is one of the most important virus diseases of forest trees in Europe. So far as we know, out of the European forest viroses only poplar mosaic has been investigated exactly concerning its effect on wood production. VAN DER MEIDEN (1964) stated that this disease caused no significant growth reduction in the varieties "Robusta" and "Gelrica". Unpublished results of the junior author confirmed the little effect of poplar mosaic on hybrid varieties of black poplar.

In contrast to poplar mosaic, black locust mosaic considerably diminishes the frost resistance of infected trees. The mechanism of the increased frost susceptibility should be investigated in more detail. A late start of the winter rest period and an insufficient ripening of wood seem to be involved in this problem.

Fortunately, the heavy losses found in comparisons between healthy and mosaic infected black locust trees are often reduced in forest areas. In general, young infected seedlings are suppressed by their neighbours which will fill up the gap. When the trees are planted in single rows along a street or along a road then the losses can be very obvious. However, in such cases mostly older seedlings

are planted selected for normal and uniform appearance and growth. Therefore they may be healthy. With trees infected in a later stage the losses in growth and vigour seem to be lower.

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Preparation of Antiserum against Stolbur Virus

By

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The paper presents a fast and simple method for antiserum preparation against stolbur virus, which provides the practice with a serum of diagnostical value. In course of the serological investigations stolbur virus isolates of Hungarian origin were studied. The method presented proved to be very reliable in detecting virus infections in potato and tomato plants.

Introduction

The use of serological methods in plant virology has become generally accepted. Serology has an outstanding significance in potato breeding and production of virus-free seed potatoes, as it proved to be very suitable for serial determination of latent and early infections.

In Hungary SZIRMAI prepared the first time serum against potato virus X (1947); then SÁRVÁRI and co-workers have been producing antisera against potato viruses X, Y, S, M, K in Kecskemét, later on at the College of Agriculture, Keszthely. The quantities prepared by the latter are sufficient to cover the needs of potato breeding in Hungary.

In recent years an increasing importance has been attributed to the stolbur virus (*Chlorogenus australiensis var. stolbur* KOVACHEWSKY) in potato degeneration (MILINKÓ et al. 1966, GÁBORJÁNYI, 1966, GÁBORJÁNYI and LÖNHARD, 1967). So far only POZDENA (1954), POZDENA and CECH (1958) succeeded in preparing antiserum against stolbur virus.

In Hungary the production of antiserum against stolbur virus failed so far; we aimed in our experiments to work out a method, which would solve the problem of preparation of efficient serum for diagnostic purposes.

Materials and Methods

For antigen production "Kecskeméti 42" tomato plants were used containing a natural stolbur infection, but free of any other viruses (isolates S7/66 and S11/66). From the median one-third of the plants leaves showing symptoms were collected and after removing the petioles their sap was expressed.

The crude plant sap was filtered through cheesecloth and freed of larger particulate material by low-speed centrifugation (5000 r. p. m. for 15 minutes); then to the supernatant ammonium sulphate was added until half-saturation (0.377

g/ml$(NH_4)_2SO_4$). In half an hour the proteins of the plant sap precipitated and were collected by centrifugation (1000 r. p. m. for 15 minutes). The precipitate was then solved in physiological saline amounting to the 1/5 of volume of plant sap measured subsequently to the first centrifugation. The solution prepared thus was injected immediately intravenously into ears of two rabbits, using dosages of 0.5, 1.0 and 2.0 ml in 3-day intervals. One week after the third injection blood sample was taken for testing the efficiency of the inoculum used, then an additional 2 ml injection was carried out to increase the titre of antisera. Seven days after the final injection the rabbits were bled by cutting their arteria carotis. The blood was stored at room temperature for 3 hours and the fibrine fibers clotted to the glass container were carefully removed by using glass capillary. After 12 hours of storage at 4 °C the blood cells became completely separated from the serum; the translucent, non-haemolyzed serum was then decanted and centrifuged for 5 minutes at 5000 r. p. m.

After centrifugation, to the supernatant centrifuged sap of healthy tomato plants of "Kecskeméti 42" variety was added until the samples ceased to give positive serological reaction with the plant sap. After this absorption the serum was centrifuged again for 5 minutes (5000 r. p. m.). One part of the antiserum was conserved by adding to each 9 ml serum 1 ml phenol solution (phenol solved in physiological saline) which gave for phenol a final concentration of 0.5%. The other part of the antiserum was lyophilized.

Results and Discussion

The rabbits inoculated with stolbur-infected plant sap were bled after the fourth injection and the serum precipitated with healthy plant sap was collected for effectivity. Both isolates (S7/66 and S11/66) gave strong precipitin reaction. The suitability of plant sap was controlled by normal rabbit serum and by antisera against potato X, Y, K and tobacco mosaic viruses; in each case negative results were recorded.

In further experiments the dilution series of antisera conserved by use of phenol were tested with centrifuged plant sap of infected tomato plants (isolate S7/66). From the latter also a dilution series was prepared. The results are shown in Table 1.

It may be concluded that the precipitin reaction gave reliable results even under such high dilutions which do not occur in the practice.

In addition to tomato, other plants were investigated as well for testing the effectiveness of antiserum in demonstrating virus infection. Plants exhibiting severe stolbur symptoms were used as control, inoculated previously with known virus isolates, by using grafting technique. Similarly, also some stolbur-infected host plants were investigated, collected under field conditions in 1966. The results showed that infections can be readily demonstrated in potato and tomato, however in the case of pepper and *Datura* the precipitin reaction does not give reliable results.

Table 1

Dilution end-point test

Tomato plant sap extract[1]	Antiserum[2]						
	1/1	1/2	1/4	1/8	1/16	1/32	1/64
1/1	++++	++++	++++	+++	++	+	+
1/2	++++	+++	+++	+++	++	+	—
1/4	+++	++	++	++	+	+	—
1/8	++	++	+	+	+	—	—
1/16	+	+	+	—	—	—	—
1/32	+	+	—	—	—	—	—
1/64	—	—	—	—	—	—	—

[1] plant sap/water.
[2] antiserum/normal saline.

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Susceptibility and Hypersensitivity to Tobacco Mosaic Virus in Wild Species of Potatoes

By

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In our work 35 derivates of 28 plant species belonging to 9 series (*Etuberosa* Juz., *Cardiophylla* Buk., *Pinnatisecta* Rydb., *Commersoniana* Buk., *Acaulia* Juz., *Demissa* Buk., *Longipedicellata* Buk., *Megistacroloba* Card. et Hawkes and *Tuberosa* Rydb.) were tested for susceptibility and resistance to mechanical inoculation with tobacco mosaic virus (TMV) U1 strain (TMV-U1). Among the examined *Solanum* species only *Solanum acaule* Bitt., *S. cardiophyllum* Lindl. and *S. vernei* Bitt. et Wittm. have shown hypersensitive reaction to virus infection. Further 25 *Solanum* species were diseased both locally and systemically. Among the latter plants two species (*Solanum acroscopicum* Ochoa and *S. etuberosum* Lindl.) although containing the virus showed neither local nor systemic symptoms, while total necrosis was exhibited by six derivates of four species [*Solanum ehrenbergii* (Bitt.) Rydb., *S. gourlayi* Hawkes, *S. hougasii* Corr., and *S. megistacrolobum* Bitt.].

Local symptoms became manifest as black necrotic ring-like spots and leaf abscission, while the systemic symptoms in the form of mosaic and leaf distortion.

Introduction

The fact that tobacco mosaic virus (TMV) is pathogenic to potato has been known for more than four decades (JOHNSON, 1925; FERNOW, 1925; BLODGETT, 1927). Among wild *Solanum* species a case of spontaneous infection of *Solanum commersonii* Dun. by TMV has recently been reported by HANSEN (1960). To our knowledge – except this work of HANSEN (1960) – no paper has been published yet on the resistance of wild potato species to TMV, then PHATAK and VERMA (1967) reported from India TMV infection in a potato variety already in general farming. It may be assumed that a TMV strain adapted to potato was concerned here. Earlier literary data but particularly the report of HANSEN (1960) prompted us to examine the reactions of a few wild *Solanum* species to inoculations with TMV.

Materials and Methods

Solanum species belonging to *Etuberosa* Juz., *Cardiophylla* Buk., *Pinnatisecta* Rydb., *Commersoniana* Buk., *Acaulia* Juz., *Demissa* Buk., *Longipedicellata* Buk., *Megistacroloba* Card. et Hawkes and *Tuberosa* Rydb. series were used in the experiments (Table 1). The collection of wild potato species was kindly supplied

Table 1

Susceptibility and hypersensitivity of Solanum species to tobacco mosaic virus

Plant introduction number	Solanum species	Collector or donor number[1]	Plant reaction[2]
P. I. 208856	S. acaule Bitt.	Bru 33—Argentina	L: BlNRi, LeAb S: —
P. I. 230495	S. acroscopicum Ochoa	Och 2043—Peru	L: Latent S: Latent
P. I. 255490	S. ajanhuiri Juz. et Buk.	CEEA 17—Bolivia	L: BlNRi, LeAb S: Mo, Ld
P. I. 265857	S. berthaultii Hawkes	EBS 1799—Bolivia	L: BlNRi, LeAb S: Mo
P. I. 265858	S. berthaultii Hawkes	EBS 1846—Bolivia	L: BlNRi, LeAb S: Vc, Mo
P. I. 265860	S. boliviense Dun.	EBS 1795—Bolivia	L: BlNRi, LeAb S: Vc, DifMo
P. I. 275179	S. brachycarpum Corr.	Haw 1547—Mexico	L: BlNRi, LeAb S: DifMo
P. I. 275180	S. brachycarpum Corr.	Haw 1577—Mexico	L: Latent S: DifMo, Ld
P. I. 265871	S. brevicaule Bitt.	EBS 1812—Bolivia	L: BlNRi, LeAb S: Mo
P. I. 265864	S. canasense Hawkes	EBS 1831—Bolivia	L: BlNRi S: Mo, Ld
P. I. 283063	S. cardiophyllum Lindl.	(218×298)× ×(218×297)	L: BlNRi, LeAb S: —
P. I. 275136	S. chacoense Bitt.	Hje 349—Argentina	L: BlNRi, LeAb S: Mo
P. I. 255520	S. ehrenbergii (Bitt.) Rydb.	CPC 371—Mexico	TN
P. I. 275214	S. ehrenbergii (Bitt.) Rydb.	Haw 1429—Mexico	TN
P. I. 245939	S. etuberosum Lindl.	Cor C. 134—Chile	L: Latent S: Latent
P. I. 275143	S. famatinae Bitt. et Wittm.	Hje 1893—Argentina	L: BlNRi, LeAb S: DifMo, Ld
P. I. 265883	S. gourlayi Hawkes	EBS 1850—Bolivia	TN
P. I. 161727	S. guerreroense Corr.	Cor 14 342—Mexico	L: BlNRi S: Mo, TN
P. I. 161730	S. guerreroense Corr.	Cor 14 410—Mexico	L: BlNRi, Vn, LeAb S: Vc, Mo
P. I. 275174	S. hjertingii Hawkes	Haw 1356—Mexico	L: BlNRi S: Mo
P. I. 161174	S. hougasii Corr.	Cor 14 253—Mexico	TN
P. I. 161726	S. hougasii Corr.	Cor 14 340—Mexico	TN
P. I. 275264	S. jamesii Torr.	CPC 386×388—USA	L: Latent S: DifMo, Ld
P. I. 175435	S. kurtzianum Bitt. et Wittm.	EBS 42—Bolivia	L: BlNRi, LeAb S: Mo
P. I. 205390	S. kurtzianum Bitt. et Wittm.	Bru—Argentina	L: BlNRi, LeAb S: Mo

Plant introduction number	Solanum species	Collector or donor number[1]	Plant reaction[2]
P. I. 210056	S. leptophyes Bitt.	Hje 1592—Peru	L: BlNRi, LeAb S: Mo
P. I. 210045	S. medians Bitt.	Hje 1162—Peru	L: BlNRi, LeAb S: Mo
P. I. 275147	S. megistacrolobum Bitt.	Hje 366—Argentina	TN
P. I. 218221	S. sanctae-rosae Hawkes	EBS 438—Argentina	L: BlNRi, LeAb S: Mo
P. I. 218224	S. simplicifolium Bitt.	EBS 447—Argentina	L: TN S: DifMo, StN
P. I. 265882	S. sucrense Hawkes	EBS 1797—Argentina	L: BlNRi, LeAb S: Mo, Ld
P. I. 217457	S. tarijense Hawkes	Sleu 4002—Argentina	L: BlNRi, LeAb S: YRi, Ld
P. I. 230468	S. vernei Bitt. et Wittm.	CPC 2413—Argentina	L: BlNRi, LeAb S: —
P. I. 275155	S. vernei Bitt. et Wittm.	Hje 351—Argentina	L: BlNRi, LeAb S: —
P. I. 275257	S. verrucosum Schlechtd.	Haw 1532—Mexico	L: BlNRi, LeAb S: Vc, Mo

by Prof. Dr. R. W. Ross, Potato Introduction Station, Sturgeon Bay, Wisconsin, USA.[3] In the tested plants of species grown from the true seed all leaves were inoculated when the plants were 3 to 4 inches in height. Inoculation experiments were performed with the U1 strain of TMV (SIEGEL and WILDMAN, 1954). This U1 strain of TMV was kindly supplied by Prof. Dr. M. ZAITLIN, University of Arizona, Department of Agricultural Biochemistry, Tucson, Arizona, USA.

[1] A key to the prefixes is listed below: Bru — Brücher, E. H., Universidad Nacional de Cuyo, Mendoza, Argentina; CCC — Coleccion Central Colombiana, Centro Nacional de Investigaciones Agricolas Tibaitatá, Bogotá, Colombia; CEEA — Coleccion Estación Experimental Altiplanó, Servició Agricola Interamericanó, La Paz, Bolivia; Cor — Correll, D. S., Texas Research Foundation, Renner, Texas; CPC — Commonwealth Potato Collection, John Innes Institute, Bayfordbury, Hertford, England; EBS — Erwin Bauer Sortiment, Max-Planck-Institut für Züchtungsforschung, Köln-Vogelsang, Germany; Haw — Hawkes, J. G., University of Birmingham, Edgbaston, Birmingham, England; Hje — Hjerting, J. P., Copenhagen Botanical Gardens, Copenhagen, Denmark; Och — Ochoa, C. M., Universidad Nacional Agraria, Lima, Peru; Sleu — Sleumer, H., Universidad Nacional de Tucuman, Tucuman, Argentina.

[2] The abbreviations used below are as follows: L = local symptoms, S = systemic symptoms, BlNRi = black necrotic ring-like spots, Dif = diffuse, Ld = leaf distortion, LeAb = leaf abscission, Mo = mosaic, StN = stem necrosis, TN = total necrosis, Vc = vein clearing, Vn = vein necrosis, YRi = yellow ring-like spots and — = no symptoms and virus was not recovered.

[3] Thanks are due to Prof. Dr. R. W. Ross for sending the seeds of these *Solanum* species.

All experimental plants used in the inoculation studies with TMV were kept in the greenhouse and the mechanical inoculations were made with raw undiluted juice extracted from infected tobacco plants *(Nicotiana tabacum* L. cv. *Samsun)* with a mortar and pestle, and applied to carborundum (500 mesh) dusted leaf surfaces with a glass spatula. After the mechanical inoculation all infected leaves were washed immediately with a fine stream of water. Plant symptoms and/or subinoculations made from inoculated plants to the appropriate host were used as criteria to evaluate the susceptibility of selections to TMV infection. *Nicotiana glutinosa* L., *Nicotiana tabacum* L. cv. *Xanthi*, *Nicotiana tabacum* L. cv. *Hicks Fixed A2—426*, *Nicotina tabacum* L. cv. *Samsun* and *Datura stramonium* L. were the diagnostic hosts used for TMV.

Results and Discussion

The experimental results are summarized in Table 1. Some of the wild potato species gave local reactions to TMV, whereas others systemic symptoms to TMV. Following the infection by TMV—U1 most of the inoculated *Solanum* species [*S. ajanhuiri* Juz. et Buk., *S. berthaultii* Hawkes, *S. boliviense* Dun., *S. brachycarpum* Corr. (P. I. 275179), *S. brevicaule* Bitt., *S. canasense* Hawkes, *S. chacoense* Bitt., *S. famatinae* Bitt. et Wittm., *S. guerreroense* Corr., *S. hjertingii* Hawkes, *S. kurtzianum* Bitt. et Wittm., *S. leptophyes* Bitt., *S. medians* Bitt., *S. sanctae-rosae* Hawkes, *S. sucrense* Hawkes, *S. tarijense* Hawkes and *S. verrucosum* Schlechtd.] — appertaining to the *Tuberosa*, *Demissa*, *Longipedicellata*, *Megistacroloba* and *Commersoniana* series — displayed local (black necrotic ring-like spots, leaf abscission) and systemic (mosaic, leaf distortion) symptoms (Figs. 1 to 4).

The systemic infections of *Solanum* species were verified on test plants as described in Materials and Methods (Fig. 5). HANSEN (1960) has already reported the susceptibility of *S. chacoense* Bitt. and *S. famatinae* Bitt. et Wittm. to TMV. According to his data on inoculated leaves of *Solanum chacoense* Bitt., "local lesions and dropping" and on other parts "apparently healthy", "etched small rings and spots" and "mosaic and malformation" arose depending on the clone tested. On infected leaves of *Solanum famatinae* Bitt. et Wittm. on one clone "apparently healthy" and on the other clone "local lesions and dropping" and on other parts "apparently healthy" as also "necrotic spots and malformation" could be observed. On species belonging to the *Acaulia* series *(Solanum acaule* Bitt.), to the *Cardiophylla* series *(S. cardiophyllum* Lindl.) and to the *Tuberosa* series *(S. vernei* Bitt. et Wittm.) only local symptoms (black necrotic ring-like spots, leaf abscission) were observed by us. No systemic symptoms were present and we did not succeed to isolate the TMV—U1 from the non-infected plant parts to diagnostical test plants either. During the inoculation of *Solanum acaule* Bitt. local ("local lesions and dropping") and systemic ("necrotic spots", "mosaic and malformation") symptoms were found by HANSEN (1960), while in one clone of *Solanum vernei* Bitt. et Wittm. subsp. *ballsi* he noted "systemic acropetal necrosis and severe dwarfing" symptoms after infection. A recent type of symptoms

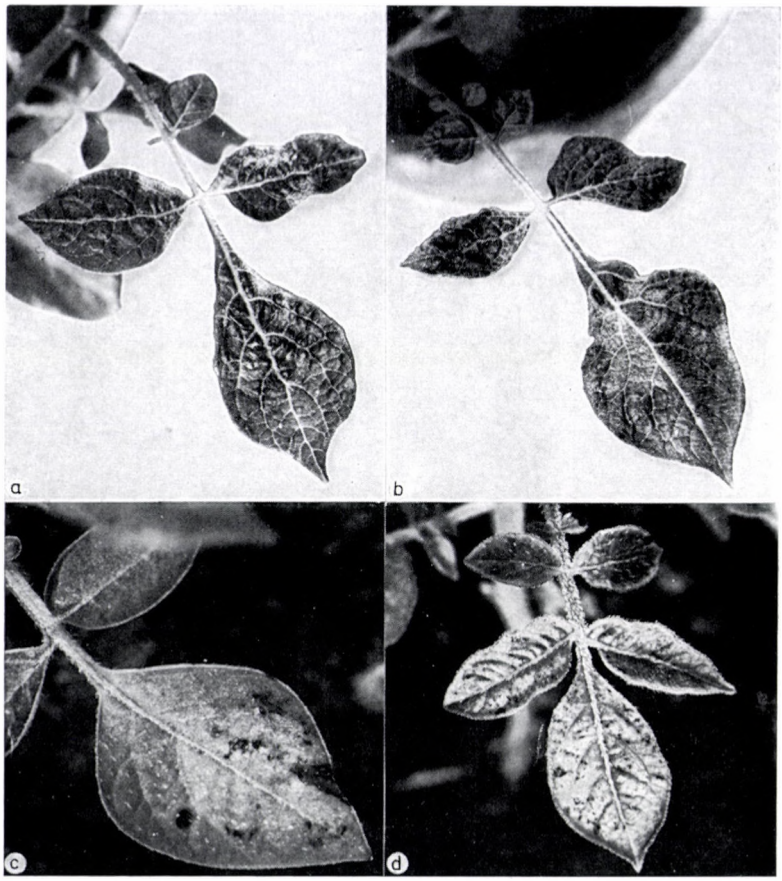


Fig. 1. Reaction of *Solanum brachycarpum* Corr. to tobacco mosaic virus. *a* and *b:* Systemic symptoms on *S. brachycarpum* Corr. (P. I. 275180), *c* and *d:* Local symptoms on *S. brachycarpum* Corr. (P. I. 275179)

is characterized by local symptoms leading to total necrosis and systemic symptoms resulting in the formation of a diffuse mosaic pattern and stem necrosis. This symptom-type was typical for *Solanum simplicifolium* Bitt. belonging to the *Tuberosa* series. Latent and systemic reactions appearing as diffuse mosaic pattern and leaf deformations were obtained when *Solanum brachycarpum* Corr. (P. I. 275180) plants belonging to the *Demissa* series and *Solanum jamesii* Torr. (P. I. 275264) belonging to the *Pinnatisecta* series were inoculated with TMV–U1. Another derivate of the same plant species (P. I. 275179) exhibited characteristic local black necrotic ring-like spots and leaf abscission symptoms. A complete (local and systemic) latency to virus infection was displayed by *Solanum etuberosum* Lindl. belonging to the *Etuberosa* series and by *S. acroscopicum* Ochoa belong-

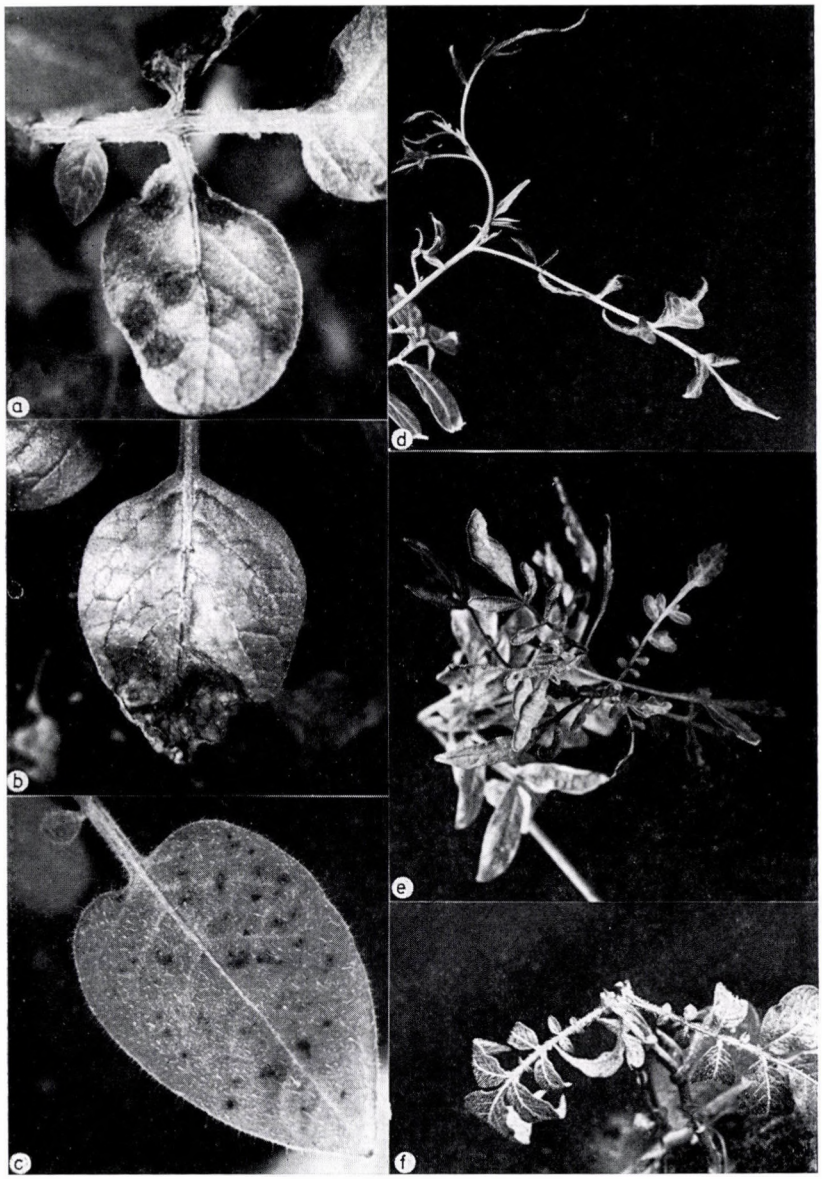


Fig. 2. Symptom reaction of tobacco mosaic virus on wild species of potatoes. *a: Solanum sanctae-rosae* Hawkes (P. I. 218221), *b: Solanum boliviense* Dun. (P. I. 265860), *c: Solanum canasense* Hawkes (P. I. 265864), *d* and *e: Solanum jamesii* Torr. (P. I. 275264) and *f: Solanum guerreroense* Corr. (P. I. 161727)

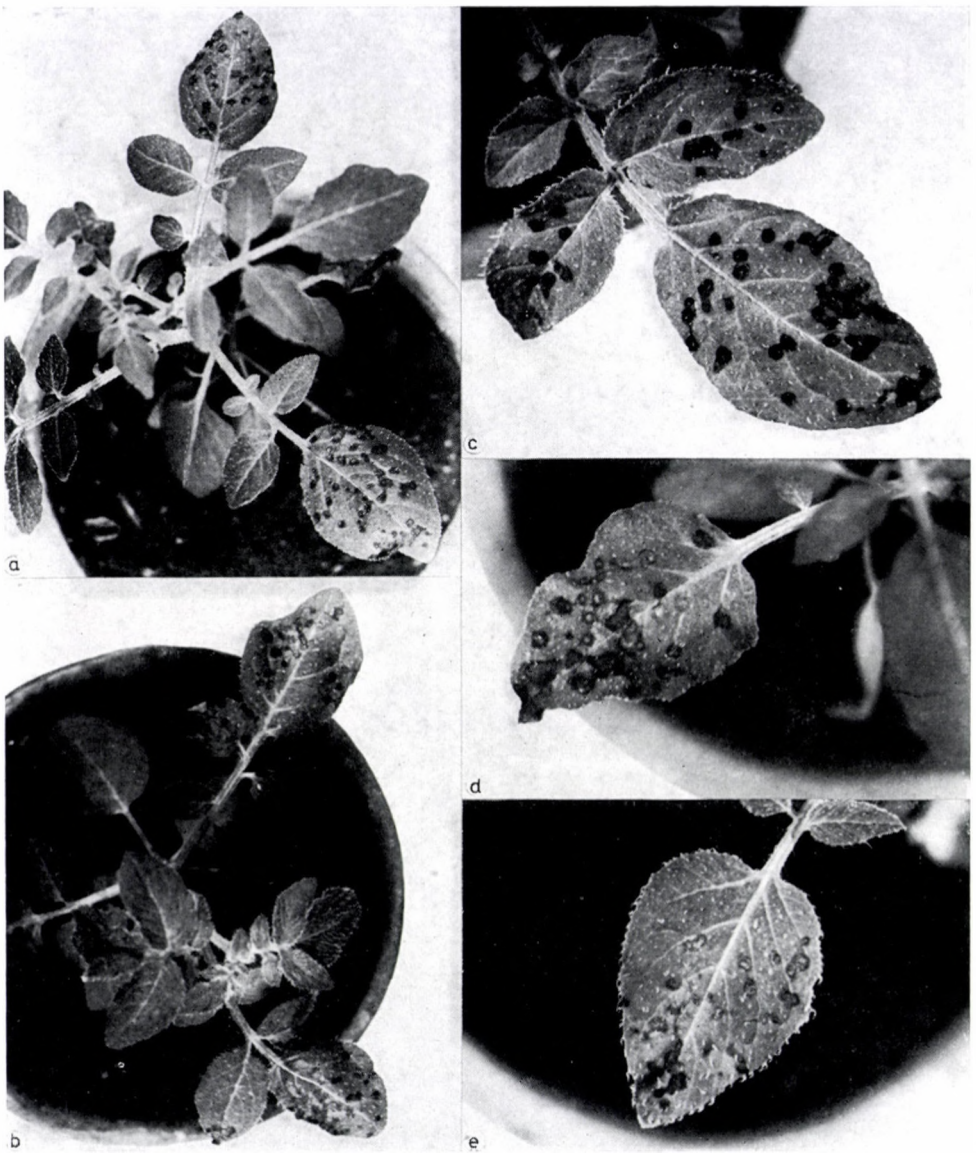


Fig. 3. Symptom reaction of tobacco mosaic virus on *Solanum kurtzianum* Bitt. et Wittm. *a*, *b*, *c* and *d:* P. I. 205390, *e:* P. I. 175435

ing to the *Tuberosa* series. A total necrosis to TMV–U1 inoculation was shown by species belonging to the *Cardiophylla* [*Solanum ehrenbergii* (Bitt.) Rydb.], to the *Tuberosa* (*Solanum gourlayi* Hawkes), to the *Demissa* (*Solanum hougasii*

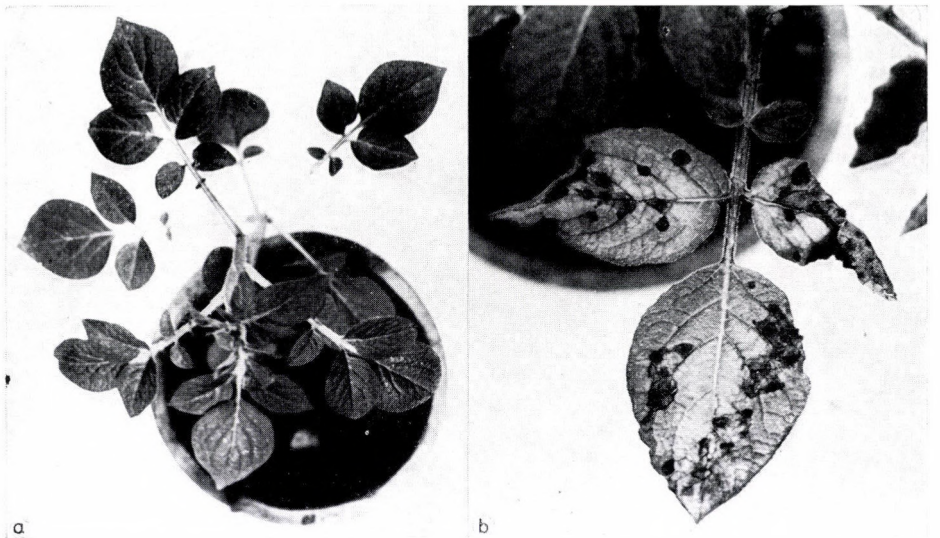


Fig. 4. Reaction of *Solanum* species to tobacco mosaic virus. *a: Solanum acaule* Bitt. (P. I. 208856) and *b: Solanum vernei* Bitt. et Wittm. (P. I. 230468)

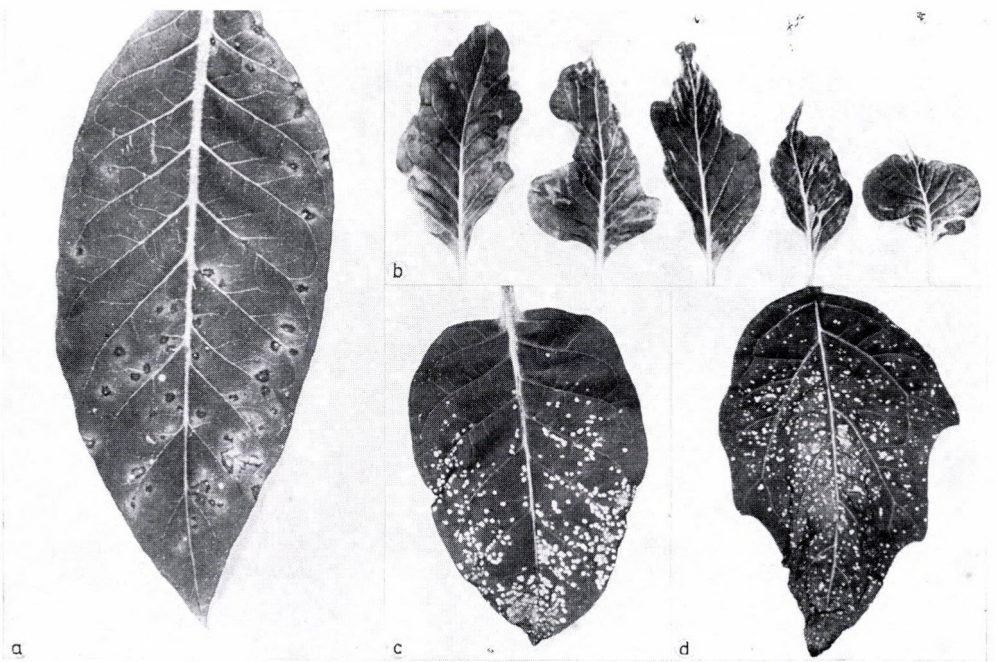


Fig. 5. Symptom reaction of tobacco mosaic virus on test plants. *a: Nicotiana tabacum* L. cv. Hicks Fixed A2—426, *b: Nicotiana tabacum* L. cv. Samsun, *c: Nicotiana tabacum* L. cv. Xanthi and *d: Datura stramonium* L.

Corr.) and to the *Megistacroloba* series (*Solanum megistacrolobum* Bitt.). The data referring to *Solanum megistacrolobum* Bitt. in the paper of HANSEN (1960) are in agreement with our experimental results. In the paper of HANSEN (1960) data can be found also on the artificial TMV infection of *Solanum microdontum* Bitt. and natural TMV infection of *S. commersonii* Dun. not examined by us.

Among the plant species *Solanum acaule* Bitt., *Solanum cardiopyllum* Lindl. and *Solanum vernei* Bitt. et Wittm. showing hypersensitive reactions to strain U1 of TMV. *Solanum acaule* Bitt. and *S. vernei* Bitt. et Wittm. may be worthy of attention from a point of view of plant breeding since it is known that plant species belonging to the *Acaulia* and *Tuberosa* series have valuable resistance qualities and can be successfully crossed with plant species appertaining to other series. *Solanum cardiophyllum* Lindl. belonging to the *Cardiophylla* series, has little significance from the point of view of plant breeding though it displays a hypersensitive reaction, because it cannot be crossed with *Solanum tuberosum* L. According to data of SWAMINATHAN (1955) plants belonging to the *Cardiophylla* series may be crossed with plants appertaining to the *Pinnatisecta* series. In our experiments only *Solanum jamesii* Torr. was included from the *Pinnatisecta* series. This plant species displayed however a total necrosis to strain U1 of TMV.

Acknowledgements

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The First Occurrence of Disease of the Carnation Caused by *Pseudomonas caryophylli* (Burkholder) Starr et Burkholder in Hungary

By

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The occurrence of the *Pseudomonas caryophylli* (Burkholder) Starr et Burkholder as the causative agent of the disease in the carnation *(Dianthus caryophyllus)* was detected for the first time by us in Hungary. The most conspicuous symptoms of the typical vascular disease were the longitudinal cracks appearing on the internodes of the stem.

The leaves became twisted and deformed and yellow discolouration showed on the midrib in some cases. After the isolation of the pathogen, artificial inoculation and reisolation, the biochemical properties of the bacteria were investigated.

Introduction

A disease occurred on the carnation *(Dianthus caryophyllus* L.) grown in the glasshouse in 1967, which was hitherto unknown in Hungary.* According to the symptoms it could be supposed that the disease was of bacterial origin. On the basis of the investigations of the bacterial strains isolated from the diseased plants, it was proved that the disease was caused by *Pseudomonas caryophylli* (Burkholder) Starr et Burkholder. It first appeared in the USA (Jones, 1941) but it is occurring in many countries of Europe (e.g. England, Denmark, the Netherlands, Germany, Switzerland and Sweden). The pathogen was described as *Phytomonas caryophylli* n. sp. by Burkholder (1942). This name was changed to *Pseudomonas caryophylli* (Burkholder) Starr et Burkholder by Starr and Burkholder (1942).

This paper presents the first description of the pathogen in Hungary.

Symptoms

The symptoms of the disease were first described by Jones (1941). The leaves of the infected carnation at first show a grey-green discolouration and then turn yellow. The plant wilts and dies. The infection is accompanied by the yellowing of the vascular bundle. According to Hellmers (1958) the typical vascular disease attacks the vessels of the stem and roots of the plant. Longitudinal cracks form

* The experimental material was collected by Mrs. Sz. Nagy.



Fig. 1. The carnations infected by *P. caryophylli*

on the internodes of the stem. Other symptoms of the disease are wilting and rotting of the roots.

HOLTZMANN and THOMAS (1953) demonstrated that the infection spread to the upper part of the shoots and the leaves too. STAHL (1955) observed small, glassy, transparent swellings on the midrib of the leaves.

Depending on the degree of the disease, the age of the plant and the environmental factors and especially on the temperature, the development of the disease may drag on from a few weeks to some years. The typical symptoms of the disease may disappear especially in older plants (HELLMERS, 1958).

DICKEY and NELSON (1963) established a correlation between soil temperature and wilting and stem crack. The wilting symptoms become conspicuous with increase of the temperature. Between 13.9—17.7 °C the stem cracks showed to the greatest extent and with the additional rise in temperature decreased.



Fig. 2. Typical stem cracks caused by *P. caryophylli*



Fig. 3. The appearance of the crack on the stem and root rotting of the carnation diseased by *P. caryophylli*

Most of the symptoms known in the literature were detected on the diseased plants examined by us (Figs 1, 2, 3), but the longitudinal cracks of the stem were the most striking symptoms of the disease. The cracks showed on the internodes, then tumours developed from them. The leaves were twisted and deformed and there was a yellowish discolouration of the midrib in certain cases, without an appearance of small glassy swellings of the midrib of the leaves.

Isolation of the Pathogen

The sterile tap water mash of the infected tissues were plated onto nutrient agar medium. After 48 hrs incubation 11 bacterial strains were isolated.

Artificial Inoculation Experiments

Healthy young carnation plants were infected with the isolated strains in the glasshouse. About 10^8 cell/ml suspension of the bacterial strains which had been incubated for 48 hours at 28 °C was prepared in sterile tap water. The plants were inoculated with a lancet, which had been dipped in this suspension and after pricked in many places of the internodes.

The typical symptoms of the disease were noticed after two weeks (Figs 4, 5). Longitudinal cracks of the stem, twisted, deformed leaves, growth depression appeared on the plants which were infected successfully and flowering did not occur.

The bacterial strains used for the infection could be reisolated from the plant indicating the typical symptoms. Four of the 11 strains used for the infection proved to be pathogenic (Sz. 1, Sz. 2, Sz. 3, Sz. 4).

Biochemical Properties of the Pathogen

The reisolated bacterial strains were investigated with the generally applied methods (DOWSON, 1957). The four strains with the exception of one test gave a uniform reaction.

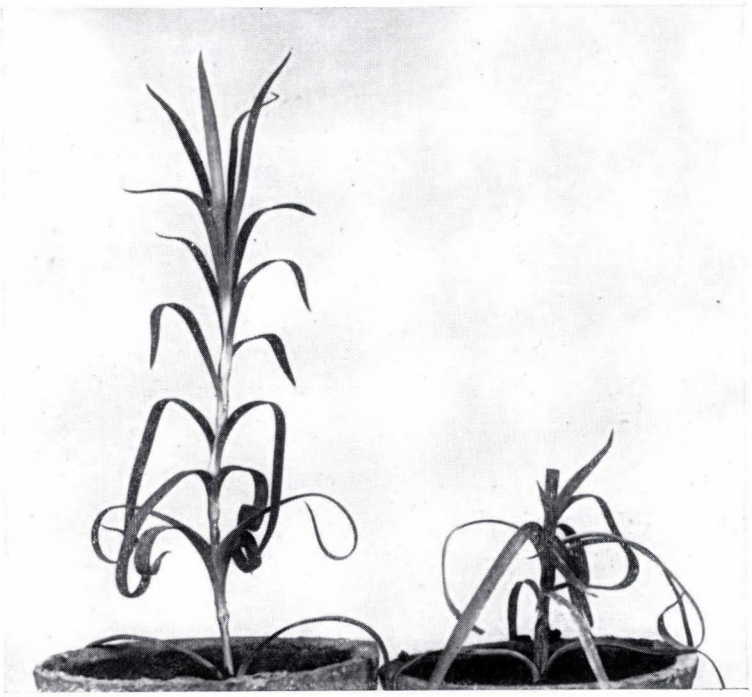


Fig. 4. The carnation artificially inoculated by *P. caryophylli* (right) and the healthy control (left)

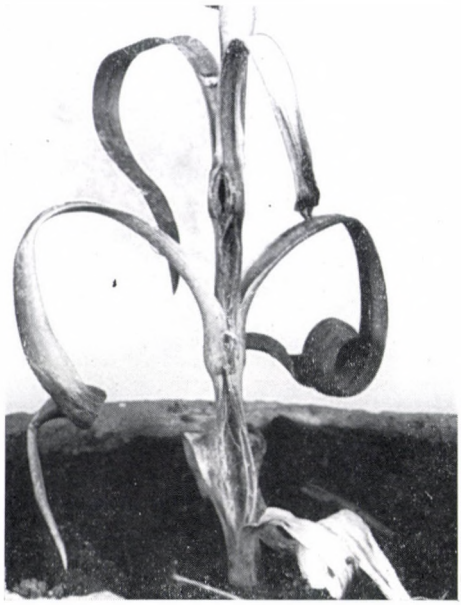


Fig. 5. Stem crack and wilting on the carnation artificially inoculated by *P. caryophylli*

The gelatin was not liquefied by the cultures of strains. Among the carbohydrates added to the synthetic medium the following compounds were decomposed with acid production after eight days incubation: D-xylose, DL-arabinose, D-glucose, D-mannose, galactose, saccharose. The very weak utilization of the maltose and lactose was indicated by a hardly visible decrease of the pH degree after 22 days. Glycerol was utilized very weakly after 30 days too, but mannitol was utilized well within 8 days. Acid production was not recorded from salicin. Starch was not hydrolized, and nitrate was reduced to nitrite. Hydrogen sulphide and indol were not produced. Alkaline reaction and in the case of the Sz. 1 strain, slight peptonization were observed in litmus milk. After three weeks there was a dirty-white precipitation in the bottom of the tubes.

Discussion

The biochemical properties of *P. caryophylli* were studied intensively by BURKHOLDER (1942) and HELLMERS (1958). The authors came to diverging results in a few reactions. BURKHOLDER (1942) established after three to four weeks a weak gelatin liquefaction. According to HELLMERS (1958) the gelatin was not liquefied by the strains. BURKHOLDER (1942) considered that maltose and lactose were utilizable C-sources. HELLMERS (1957) however received only a very weak reaction after three-four weeks. According to BURKHOLDER's (1942) original description the salicin was decomposed by the *P. caryophylli* strains. HELLMERS

(1958) did not detect acid production from salicin. HELLMERS' (1958) investigations were supported by our examinations in every respect.

There was a slight peptonization in litmus milk after four weeks in the case of the Sz. 1 strain isolated by us. The other three strains did not peptonize the milk.

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The Susceptibility of *Hypera postica* (Gyll.) to *Beauveria densa* (Link) Picard*

By

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An entomogenous fungus *Beauveria densa* (Link) Picard isolated from the larvae of *Chilo zonellus* (Swinhoe) was found to be pathogenic when tested in the laboratory against the grubs of the alfalfa weevil, *Hypera postica* (Gyll.). The pathogenicity was tested by two techniques viz. feeding and crawling. The data were analysed statistically. The histological studies of the infected grub revealed that the hyphae penetrated through the cuticle disintegrating the hypodermis.

Lucerne (*Medicago sativa* L.) is an important fodder crop extensively cultivated all over the world. This crop has as its chief insect enemy, *Hypera postica* (Gyll.),** which causes considerable damage in the Northern parts of India as well as in other countries. The chemical control of this pest has not been very satisfactory and attention is being increasingly paid to explore means of controlling it biologically. This paper presents the observations made in the laboratory during a study of the fungal diseases affecting this pest. Such studies may, subsequently, prove useful in the control of many noxious pests.

Mackie (1942) reported the parasitism of *Beauveria globulifera* (Speg.) Picard and *Entomophthora sphaerosperma* Fresenius on *Hypera punctata*. Prillieux and Delacroix (1891) successfully infected the larvae of *Cetonia aurata*, *Rhizotrogus solstitialis* and caterpillars of *Euproctis chrysorrhoea* by *Beauveria densa* (Link) Picard. Giard (1891) concluded from his studies that *B. densa* can attack Coleoptera and Lepidoptera but not Orthoptera. A *Beauveria* sp. was previously reported from Hawaii on sweet potato weevil, *Cyclas formicarius* F. (Sherman, 1952, Sherman and Tamashiro, 1954).

An entomogenous fungus, *Beauveria densa* (Link) Picard isolated from *Chilo zonellus* Swinhoe (Mathur et al. 1966) was found to be pathogenic when tested in the laboratory against alfalfa weevil grub, *Hypera postica* (Gyll.).

Material and Method

Alfalfa weevil grubs were collected in sterilized glass tubes and pathogenicity was tested by two techniques viz.: 1. feeding and 2. crawling.

* This research has been financed in part by a grant made by the United States Department of Agriculture under P. L.-480.

** In the European literature the species is mentioned more often as *Hypera variabilis* Hbst.

1. *Feeding:* Healthy grubs of *Hypera postica* were starved for 10 hours. The spore suspension containing an undetermined number of spores was made in a medium of sterile distilled water. Dry milk powder was used as a sticker. The spore suspension was sprayed on the lucerne leaves and healthy grubs were kept in the glass jars containing the sprayed leaves. Six replications of ten grubs were used in both the experiments. Sterile distilled water mixed with milk powder was used in the case of control. The experiment was carried out in 75% relative humidity and at a temperature of 25 ± 1 °C.

2. *Crawling:* This experiment was carried out in the same way and under similar conditions of temperature and relative humidity except that the grubs were allowed to crawl over the fungus in the test tubes for 4 hours and then placed in glass jars containing the fresh lucerne leaves. In case of control healthy grubs were kept as such on the lucerne leaves.

For histological studies microtome sections of the diseased grub were cut, stained, using heamatoxylin-eosin stain and photomicrographs were taken.

Results

Within 48 hours after treatment, the grubs were sometimes observed to be ill at ease, becoming lethargic and failed to respond to external stimuli. They also stopped feeding with the change in body colour from green to light yellow. The grubs died within 3–8 days, slightly contracted and the colour changed to light pink. Microscopic examination revealed the presence of spores and mycelia in abundance inside the body. The fungus was reisolated on potato dextrose agar (P. D. A.) medium. In case of the second technique i.e. crawling, some grubs became sluggish after 36 hours showing the symptoms of the disease as described in the



Fig. 1. Diseased mummified grubs of *Hypera postica* (Gyll.) infected with *Beauveria densa*

feeding technique. Death occurred after 2–7 days. About 3–4 days after death, the fungus emerged and the white mycelium covered part or the whole body surface of the grub (Fig. 1).

Table 1

Per cent corrected mortality, per cent recovery of the pathogen and the difference between the two

Method of testing the pathogenicity	Number of replications	Corrected mortality		Recovery of the pathogen		Difference between per cent corrected mortality and per cent recovery of the pathogen
		%	Transformed angular values	%	Transformed angular values	Transformed angular values
Feeding	1	62.5	52.5	60.0	51.1	1.4
	2	55.5	48.4	50.0	45.3	3.1
	3	66.4	55.0	60.0	51.1	3.9
	4	71.4	58.0	70.0	57.1	0.9
	5	57.1	49.4	50.0	45.3	4.1
	6	62.5	52.5	60.0	51.1	1.4
			52.63		50.16	2.46
Crawling	1	75.0	60.3	70.0	57.1	3.2
	2	66.6	55.0	60.0	51.1	3.9
	3	71.4	58.0	70.0	57.1	0.9
	4	70.0	57.1	60.0	51.1	6.0
	5	77.7	62.2	70.0	57.1	5.1
	6	66.6	55.0	60.0	51.1	3.9
			57.93		54.10	3.83

Table 2

Analysis of variance for corrected mortality

Source of variance	*D. F.*	*SS*	*MS*	*F*
M	1	84.27	84.27	8.061*
E	10	104.53	10.45	
Total	11	188.80		

S. E. = 1.86

C. D. at 5% level = 4.1478

* Significant

Table 3
Analysis of variance for recovery of the pathogen

Source of variance	*D. F.*	*SS*	*MS*	*F*
M	1	46.41	46.41	3.052 n. s.
E	10	152.06	15.20	
Total	11	198.47		

S. E. = 2.25

The data regarding the per cent corrected mortality, per cent recovery of the pathogen from the dead larvae and the difference in the two among the two techniques used for testing the pathogenicity were analysed statistically and results are summarized in Table 1. Tables 2, 3 and 4 show the analysis of variance. The observed mortality in treatment was adjusted with the mortality occurring in control by applying ABBOTT's formula (ABBOTT, 1925).

Discussion

It is evident from the data in Table 1 that there is no significant difference between the per cent corrected mortality and per cent recovery of the pathogen

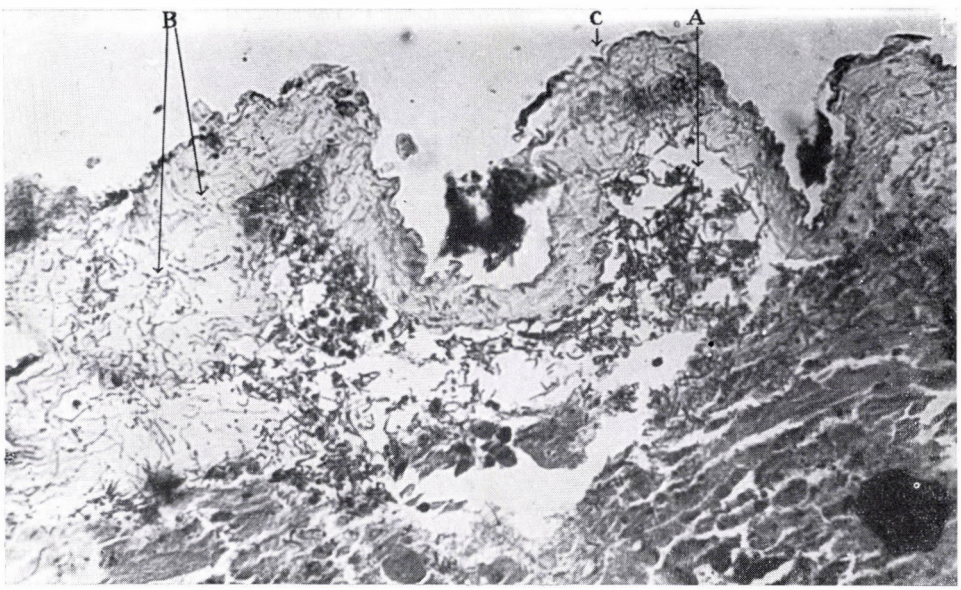


Fig. 2. A portion of the T. S. of the diseased grub, showing the germinating hyphae penetrating through the cuticle. (Photograph taken under low power). (A) Hypodermis filled with mycelia and conidia. (B) Developing hyphae. (C) Cuticle

Table 4

Analysis of variance for difference between corrected mortality and recovery of the pathogen

Source of variance	*DF*	*SS*	*MS*	*F*
M	1	5.60	5.60	2.224 n. s.
E	10	25.17	2.51	
Total	11	30.77		

S. E. = 0.91

n. s. Not significant

from the dead infected grubs when the data of the two techniques were analysed statistically.The difference between the means of the two techniques viz. feeding and crawlin g for corrected mortality (transformed angular values) was significant

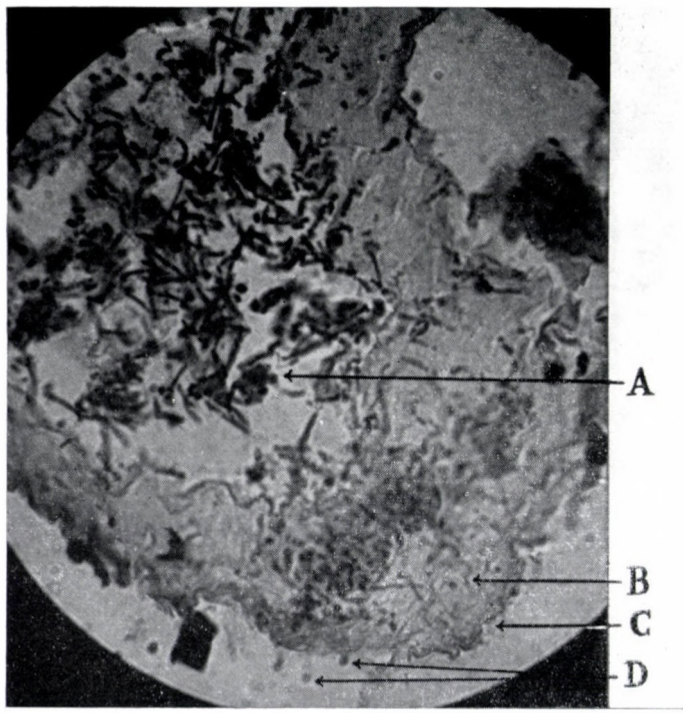


Fig. 3. A portion of the T. S. of the diseased grub. (Photograph taken under high power)· (A) Hypodermis filled with mycelia and conidia. (B) Developing hyphae. (C) Cuticle. (D) Germinating conidia

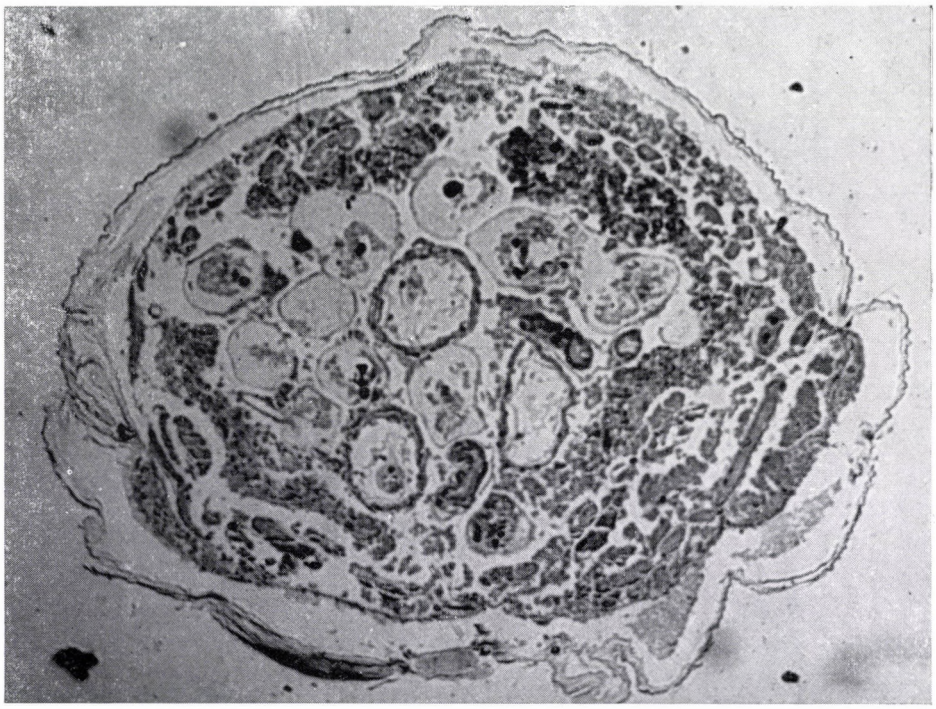


Fig. 4. A T. S. of a healthy grub of *Hypera postica*

at 5% level, but found to be insignificant for the per cent recovery of the pathogen (transformed angular values). LEFEBVRE (1934) reported that in case of *B. bassiana*, there is good evidence to show that infection may take place by way of the alimentary canal. PILAT (1938) made histological observations for *B. bassiana* and observed the infection through the digestive tract. GIARD (1892) studied the parasitism of *B. densa* on Cockchafers and inferred that the cuticle is penetrated with the aid of an enzyme which is secreted at the apex of the hyphae. Microtome transverse sections of the infected grubs when studied under the microscope, revealed that germ tubes emerging from the conidia were seen penetrating through the cuticle and in the act disintegrating the hypodermis (Figs 2 and 3). The fungus sometimes penetrates into the lumen of the digestive tract and fills it with the mycelia. The hyphae were also seen developing in the fat bodies and muscles. In stained slides, the hyphal-bodies which are greater in diameter than the hyphae were generally ovoid with small, very darkly stained nuclei. Similar observations were made by TANADA (1955) on *Pieris rapae* (L.).

Thanks are due to Dr. S. PRADHAN for providing the necessary facilities.

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The Flight Period of Wild Bees *(Hymenoptera, Apoidea)* Pollinating Lucerne, and its Plant Protection Aspects

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On the basis of their flight period, wild bees might be divided into three main groups: species with short, medium, and long flight period. Among the species with a short flight period, spring, early summer, and high summer animals are known. The long flying group contains bivoltine, solitary, and continuously developing social insects. The main participants in the pollination of lucerne are the short flying summer species, as well as some medium and long flying taxa. The short flying pollinators of lucerne build their nests in the lucerne fields but since they fly from the end of June to the end of August, the application of insecticides administrated up to the middle of June and later than the middle of August does not affect them. The other species do not nest in lucerne and visit the fields only for food, i.e., in the period of blossoming. Assuming weedless fields, these species are not poisoned by treatments applied prior and subsequent to flowering time. However, the period lasting from the middle of June to the middle of August is dangerous for all pollinating insects. According to our investigations Melipax and Thiodan applied in this period are toxic to the wild bee species in the field. Treatments before and after the dangerous period defined above are also advisable to be made by spraying or wet dusting, because a certain rate of poisoning of the nesting sites and of food providing areas is unavoidable when done by simple dusting. Weed control decreases the feeding possibilities of the wild bees, causing on the one hand a diminishing of their numbers and, on the other, a decrease of their offsprings owing to the shortening of the flight period. This situation is especially striking in cultivated areas, therefore also in the vicinity of the lucerne fields. Feeding conditions must therefore be assured by artificial means in these areas.

Lucerne in Hungary is not pollinated by the honeybee but by wild bee species (MÓCZÁR, 1959), indeed it seems, at least according to recent studies, that in areas showing a high number of wild bees the role of the honeybee is to a certain extent negative (MÓCZÁR, 1961a). More than 600 species of wild bees live in Hungary; of these, however, merely about 100 have been found on lucerne (MÓCZÁR, 1961c, 1961d). Of the wild bee species visiting lucerne, 86.41 per cent of the mean nationwide material was composed of 15 most frequent species (MÓCZÁR, 1961c). According to recent studies (MÓCZÁR, 1961b, 1961c), the preponderant majority of the wild bees visiting lucerne was represented by these 15 species in every region of the country. Since the role of the other, incidental species is negligible from the point of view of applied entomology, only these 15 species will hereafter be studied.

S. MANNINGER is recently conducting experiments in Hungary with artificially bred individuals of *Megachile rotundata* FABRICIUS in using them for the pollination of lucerne; the flight period of this species, and that of one of its near allies, *Megachile ericetorum* LEPELETIER, will therefore also be studied.

In Hungary, lucerne seed is usually harvested from the second and third lucerne crops, with the flowering period therefore being restricted to the phase extending from the beginning of July to the middle of August.

Method

The flight period is examined by the "museum method" (Soós, 1958), using the data of collection specimens. The data deriving from specimens collected in the same time in the identical locality are regarded as a unit. Thus specimen or specimens collected in different days in a given locality, or those gathered on the same day in different localities, submit diverse data. Data gained by this method are subgrouped into decades (1 – 10, 11 – 20, 21 – 30), and the data values per decades are plotted on the perpendicular axis of the flight diagrams. The reliability of the method is discussed and justified, in a still unpublished manuscript (BENEDEK and JÁSZAI, 1968), by recourse to light trap data. We have also pointed out in that paper that the method reflects average conditions. The flight is usually 2 – 3 weeks shorter than that readable from the diagrams, since the beginning and end of the flight period are determined on the diagrams by the warmest years.

Flying types of Hungarian Wild Bee Species

On the basis of their flight period, the wild bee species can be divided into three main groups: species of short, medium, and long flight periods may be distinguished. Species with a short flight period are to be found for 2 – 3 months, the medium one for 3 – 4 months, the long ones for 4 – 6 months. The species showing short and medium flight periods are univoltine, those of the long one bivoltine or continuously breeding. It is generally characteristic of the wild bees that the males appear earlier than the females, hence males occur in higher numbers at the commencement of the flight, and females at its termination. This is especially conspicuous for the *Eucera* species where the two sexes exhibit well-nigh separate swarming peaks in the diagrams (Figs 6 – 9).

1. Species with a short flight period

a) *Spring species.* This group of wild bees appears in early spring, at the end of March or in April. They are on the wing also at temperatures around 10 °C. The spring species disappear already in May, the adults of the next generation appearing from the brood comb next spring only. The characteristical representatives of this group are *Anthophora acervorum* LINNAEUS, *Osmia rufa* LINNAEUS, and *Osmia cornuta* LATREILLE. The species belonging here hibernate as adults in the pupal cocoon and have no role in the pollination of lucerne, having already disappeared at the end of spring.

b) *Early summer species.* Their flight begins at the end of spring and terminates already at high summer. They cannot be lucerne pollinators, since at blossoming time only some late specimens may yet be on the wing. They hibernate as pupae; a characteristical representant is *Osmia atrocoerulea* SCHILL.

c) *Summer species.* These insects appear in June, and fly until the middle or end of August; some of them are excellent lucerne pollinators. Characteristical representatives of the group are *Melitta leporina* PANZER. The members of the group hibernate as prepupae or fully developed larvae.

2. Species of medium flight period

These animals begin flying in the middle or at the end of spring and disappear at high or late summer. Their flight terminates during the flowering of lucerne, and some of them are important pollinators. The species hibernate as prepupae or pupae; the *Eucera* species are characteristic to the group.

3. Species with a long flight period

a) *Bivoltine species.* The first, spring generation of the bivoltine *Andrena* and *Halictus* species begin flying in early spring, terminating their activities at the beginning of summer. The second or summer generation appears in high summer and disappears at the end of summer or during the autumn. The flight of the second generation coincides with the period of lucerne blossoming. The species hibernate as adults within the pupal cocon; some of them are important pollinators of lucerne.

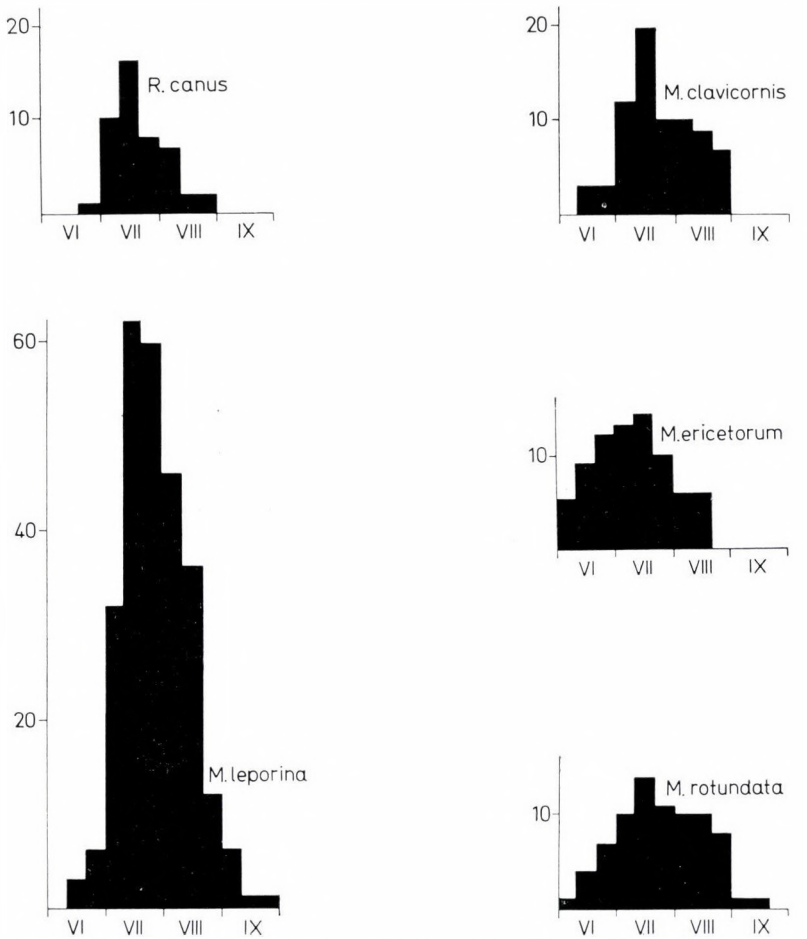
b) *Social, continuously breeding species.* Of our home wild bees, the *Bombus* species are social; the hibernated females begin to fly in early spring, and the first workers also appear soon. From this point on, the family continually raises fresh offsprings. The young females appear in summer, they hibernate, and the family gradually depopulates from the beginning of the autumn and dies out at its end.

Flight Period of Lucerne Pollinating Wild Bees

1. Species with a short flight period

Rhophitoides canus EVERSMANN (= *Rhophites canus)* appears only late, practically only at the beginning of July; it flies in July, and its numbers rapidly diminish in August (Fig. 1). This species has the shortest flight period in Hungary; its flight coincides with the main flowering time of lucerne. It exhibits a number of excellent qualities and seems to be bound to lucerne in all respects in Hungary, indeed, it might be regarded as entirely adapted to this plant.

The flight of *Melitturga clavicornis* LATREILLE (Fig. 2) resembles that of *R. canus*. It appears late, at the end of June, it is most numerous in July, its numbers diminishing in August, and usually vanishing at the end of the month. An excellent flower-opener (MÓCZÁR, 1959), the species abounds mainly in the warmer, sandy districts of the country, preponderantly in the south.



Figs 1—5: Flight diagrams of species with a short flight period: 1. *Rhophitoides canus* Ev., 2. *Melitturga clavicornis* Latr., 3. *Melitta leporina* Pz., 4. *Megachile ericetorum* Lep., 5. *Megachile rotundata* F.

The *Megachile* species are typically summer animals; *Megachile ericetorum* Lepeletier and *Megachile rotundata* Fabricius fly from June to August, some stragglers may, however, occur also in September. The main flight period coincides with the blossoming of lucerne. Of the two species studied, *Megachile ericetorum* Lepeletier (Fig. 4) is slightly earlier, whereas *Megachile rotundata* Fabricius (Fig. 5) is somewhat later in appearance, and the flight of the latter is more protracted. Their flight is rather short, having merely a single generation. They are rather rare in nature.

Melitta leporina Panzer flies from the end of June till the end of August. It is most abundant in July and the first days of August (Fig. 3). Some individuals

may occur also in mid-June and the beginning of September, but their amount is not significant. The peak of flight coincides with the period of lucerne flowering. Lucerne is its preferred food, the species nesting similarly to *R. canus*, in the soil of lucerne fields.

2. Species with a medium flight period

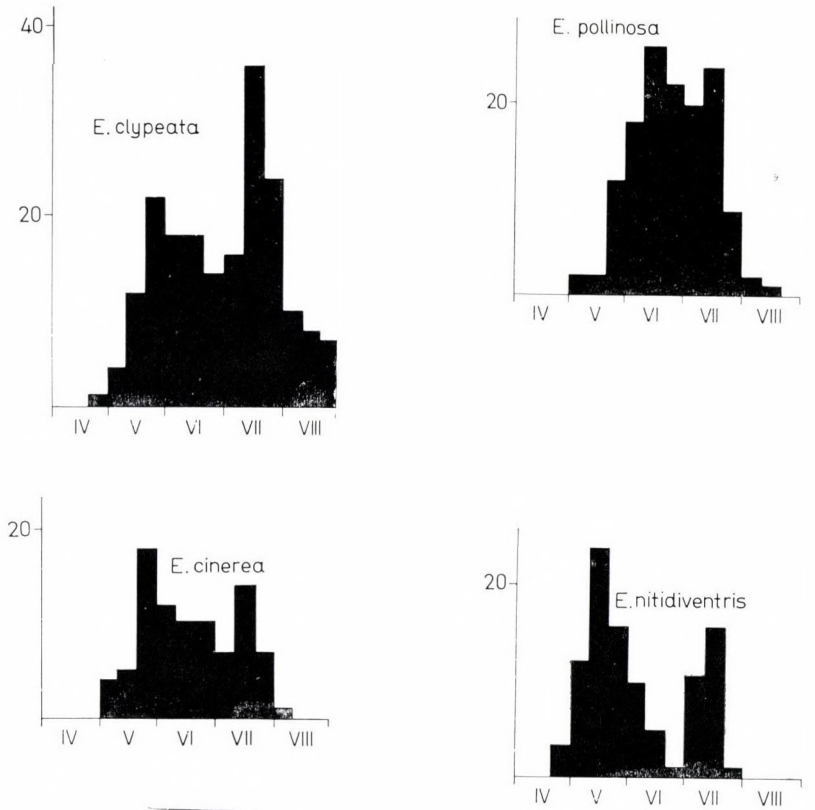
The *Eucera* species are to a certain extent transitional between the groups of short and long flying forms, because while the flight of *Eucera clypeata* ERICHS and *Eucera pollinosa* SMITH extends over four months, that of *Eucera cinerea* LEPELETIER and *Eucera nitidiventris* MOCSÁRY is no more than three. The *Eucera* species are animals of the late spring and early summer. *Eucera nitidiventris* (Fig. 9) is the earliest among them, possibly appearing already in April, exhibiting its peak in May and disappearing in July. Only the specimens of the final flight days partake in the pollination of lucerne. *Eucera nitidiventris* is followed by *Eucera pollinosa* (Fig. 7) and *Eucera cinerea* (Fig. 8). They appear in May, but become abundant in June and July, their number diminishing at the end of July and entirely disappearing in the first days of August. The two species are significant pollinators at the beginning of lucerne flowering. *Eucera clypeata* (Fig. 6), of the longest flight period, may appear at the end of April or the beginning of May, but becomes abundant only from the middle or the end of May. The flight peak appears in July, but the species is fairly abundant also in August, and vanishes at its end. Its flight coextends with the entire flowering period of the lucerne.

3. Species with a long flight period

The flight of the bivoltine *Andrena* species may commence at the end of March, extending to the very end of August, and some specimens may be found even in September. They are usually abundant already in April, and the females then on the wing already avidly collect pollen for their offsprings. The first (spring) generation consists of the hibernated animals, the second (summer) one of their brood; the flight is intensive during the entire blossoming period. The flight period of *Andrena ovatula* KIRBY (Fig. 10) is slightly longer than that of *Andrena flavipes* PANZER (Fig. 11); the two generations are more sharply delimited in the case of *Andrena flavipes*.

The flight period of the bivoltine *Halictus* species is longer than even that of the *Andrena* taxa, though it seems that, except for *Halictus calceatus* SCOPOLI, they appear only in April, but their numbers are significant even in September, indeed, in October. The flight period of *Halictus calceatus* (Fig. 15) is the longest of all; this species appears in masses from April till September, but it may be on the wing already at the end of March and some stragglers in October. *Halictus maculatus* SMITH (Fig. 13) and *Halictus patellatus* MORAWITZ[1] (Fig. 14) fly from

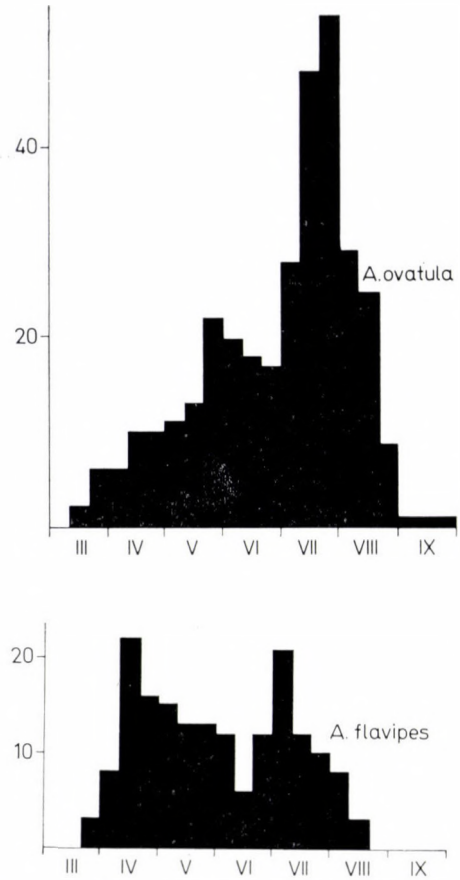
[1] L. MÓCZÁR discusses in his papers the species *Halictus rubicundus* CHRIST. In a revision, however, made by M. MÓCZÁR, the majority of the material originating from the lucerne survey proved to be *Halictus patellatus* MORAWITZ.



Figs 6—9: Flight diagrams of species with a medium flight period: 6. *Eucera clypeata* ER., 7. *Eucera pollinosa* SM., 8. *Eucera cinerea* LEP., 9. *Eucera nitidiventris* MOCS.

the end of April to the end of September. The flight peaks of the spring broods of these three species appear in May, those of the summer generation in July and August. *Halictus eurygnathus* BLÜTHGEN appears only late, in May (Fig. 12), flying until the end of September. It is abundant only in June; the two generations are rather overlapping. The *Halictus* species are the pollinators of early and late blossoming lucerne strains.

The Apoids discussed hitherto are solitary or live in nest colonies, whereas the *Bombus* species build communities numbering some dozens or hundreds of specimens. The composition of the community is not constant, since the place of the dead specimens is continuously taken over by new individuals. The bumblebees are therefore wild bees with the longest flight period, their breeding being uninterrupted. The hibernated, colony-founding females fly from the end of March or the first days of April, and the last individuals of the colonies can be found even in October. On their flight-diagrams, a gradual increase and then the dimin-

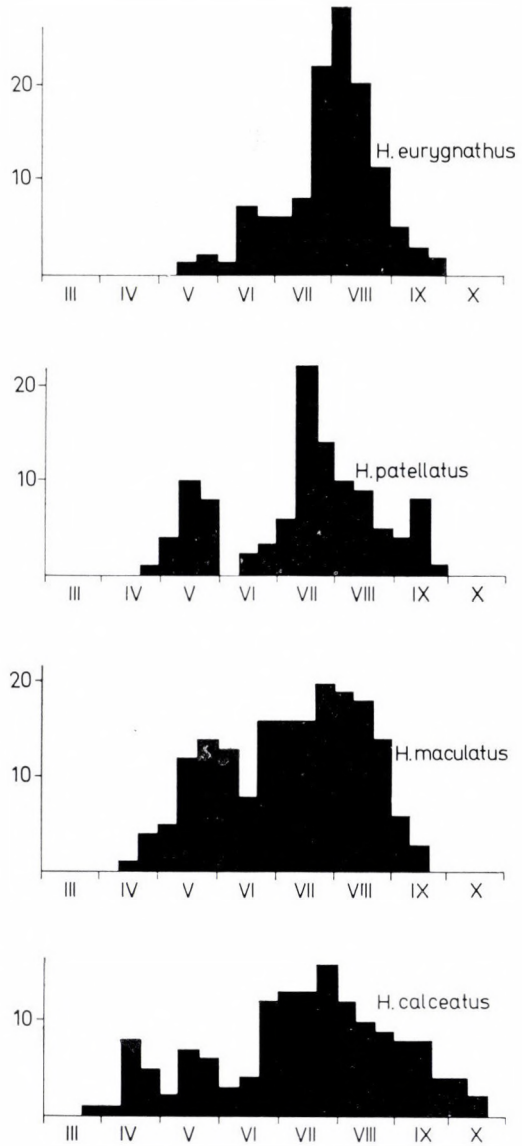


Figs 10—11: Flight diagrams of bivoltine species with a long flight period: 10. *Andrena ovatula* K., 11. *Andrena flavipes* Pz.

ishing of the colony is observable. The increase reaches the peak at the end of July and the first days of August, but the flight is still abundant in August and September. The flight of *Bombus lapidarius* LINNAEUS (Fig. 16) and *Bombus terrestris* LINNAEUS (Fig. 17) is rather uniform. Competing plants, especially *Trifolium pratense*, *Echium vulgare*, and *Carduus* species exert a very strong influence on them; the flight is abundant during the entire period of lucerne blossoming. In their habits, they resemble the honeybee to a certain extent.

Effect of Preventive Pest Control Measures on the Wild Bees

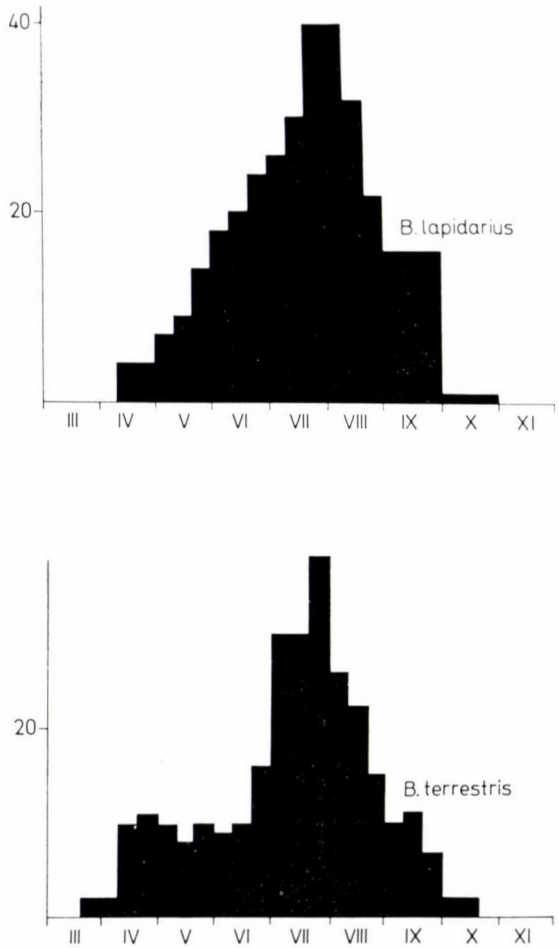
In plant cultivating circles there is a view recently prevailing that the decreas of lucerne seed crops is to be ascribed primarily to the action of insecticides sinc



Figs 12—15: Flight diagrams of bivoltine species with a long flight period: 12. *Halictus eurygnathus* BLÜTHG., 13. *Halictus patellatus* MOR., 14. *Halictus maculatus* SM., 15. *Halictus calceatus* SCOP.

they had considerably decreased the numbers of wild bees responsible for the pollination. Let us examine the question in details.

Among the wild bees participating in the pollination of lucerne, it is the species with a short flight period *(Rhophitoides canus, Melitta leporina, Melitturga*



Figs 16—17: Flight diagrams of social, continuously breeding species with a long flight period: 16. *Bombus lapidarius* L., 17. *Bombus terrestris* L.

clavicornis) which nest in the soil of lucerne fields. Those of a medium *(Eucera* spp.) and a long flight period *(Andrena* spp., *Halictus* spp., *Bombus* spp.) do not nest in the lucerne fields proper but along pathways and balks, in general in the undisturbed soils of sites with a sparse vegetation.

The species with a short flight period appear at the end of June and disappear already by the end of August. Short-effect insecticides applied until mid-June do not poison these species. Treatments made by substances with a residual effect up to the last days of May are also ineffective against these species owing to their later flight date. Flight terminates in August, hence chemical treatments conducted after the end of August in lucerne fields are also harmless to them.

The species with a long flight period do not nest in the lucerne fields, and visit them only at a time when flowering plants are (weeds or lucerne) present. The application of chemical agents therefore, whose effects pass by the commencement of the blossoming of lucerne, does not endanger them, provided, of course, that there are no flowering weeds suitable for feeding in the lucerne fields during the effective toxic period of the pesticide used. After the flowering of lucerne, the species with a long flight period do not visit the fields any more, hence treatments applied after flowering time do not affect them.

Accordingly, it is obvious that the period lasting from mid-June to the end of August is the dangerous one for the wild bees pollinating lucerne. In this period, the insecticides poison also the pollinating wild bees. During this dangerous period, the insecticides applied against insect pests in lucerne fields are usually Melipax and Thiodan. Literature considers these agents as harmless against bees. In the course of our field studies we have found, however, that these substances are in fact considerably toxic for them. Our results are summarized in Table 1.

Table 1

Effect of Melipax and Thiodan on the specimen number per hectare of the wild bees pollinating lucerne (1967)

Locality	First survey		Treatment at flowering time		Second survey		Third survey	
	date	wild bee ind/ha	date	treatment	date	wild bee ind/ha	data	wild bee ind/ha
Fegyvernek[2]	13 July	2227	17 July	Melipax 17 kg/ha	20 July	214	3 Aug.	115
Törökszent-miklós[2]	13 July	2192	17 July	Melipax 17 kg/ha	20 July	187	—	—
Szapárfalu[2]	17 July	2084	18 July	Melipax 17 kg/ha	20 July	95	3 Aug.	273
Halászi[3]	—	—	15 July	Melipax 17 kg/ha	19 July	64	—	—
Öcsöd[2]	19 July	2543	19 July	Thiodan 2.5 kg/ha	31 July	571	—	—
Berettyó-újfalu[4]	20 July	754	12 Aug.	Thiodan 1.7 kg/ha	15 Aug.	0	—	—
Mezőhék[2]	—	—	16 July	Thiodan 1.7 kg/ha	19 July	0	—	—

[2] In Com. Szolnok. Surveys made in 1967 in 30 sites of the area found 2182 (mean value) of wild bee specimens per hectare in untreated fields.

[3] In Com. Győr. Surveys made in 1967 in 20 sites of the area found 1553 (mean value) of wild bee specimens per hectare in untreated fields.

[4] In Com. Hajdu. Surveys made in 1967 in 25 sites of the area found 1657 (mean value) of wild bee specimens per hectare in untreated fields.

Prior to the chemical treatments, we have established, as is to be seen also from the Table, the presence of more than 2000 wild bee specimens per hectare in every field (with a single exception in Berettyóújfalu). In the days subsequent to the treatment, as well as during later and occasional surveys, the number of wild bees was at most 500 per hectare. A similarly low number of wild bees was found after treatments in fields where we had no occasion to investigate the situation prior to the treatment. During our surveys, identical weather conditions prevailed, hence the decrease in numbers could have been due only to the death of the animals. This conclusion is further corroborated by the fact that we have hardly found any opened lucerne flower after the treatment. In the course of our surveys prior to the use of insecticides, the species living in lucerne (mainly *Melitta leporina)* were present in masses; nor could they have left the fields owing to the implied repellent effects of the toxic agents. In any case, the presumably repellent action of the substances in question, with respect to the wild bees, is highly doubtful. The application of the insecticides coincides in several localities with the commencement of the flight of the second brood of the bivoltine species. Animals already active at the time of treatment fell victim to poisoning, but those hatching some days after treatment have escaped it. And these insects were fully active, despite the fact that the disagreeable odour of the agents could still strongly be felt. They have therefore escaped poisoning and no repellent effect could be observed.

Accordingly, the insecticides applied in lucerne fields up to the end of May and from the end of August do not poison the pollinating wild bees. Species which nest in the lucerne fields proper have a short flight period, they are active from the end of June till mid-August. Those which do not nest there, even if they show a long flight period, visit only fields in full blossom or those containing flowering weeds, hence treatments made before the above period, or after it, do not poison these animals. Measures taken on the stubble-fields and treatments after flowering time have therefore no toxic effects on the wild bees. However, administrations at the time of the green-bud stage of lucerne should be carefully made, and it is advisable to begin it at an earlier date than usual. During the time of flowering, all application of insecticides should possibly be avoided. If a treatment is absolutely necessary at the time of flowering, it should be postponed until the end of blossoming. Thus a smaller number of wild bees will be killed on the one hand (since at the time the number of visiting animals is less), and even if a part of the pollinators die we have still not obstructed the possibly greatest rate of pollination during the period of abundant visiting or flowering on the other. Thus, though the destruction of the wild bees cannot be avoided, only the smallest possible number of animals is killed and since the major part of the flowers is already pollinated the crop is also ensured. However, for the sake of higher lucerne seed yields, the maximum protection of the wild bees is also justified. If the lucerne field, in which pest control measures (under the conditions described above) are taken in the period of blossoming, remains for several years a seed-producing field, the decrease of wild bees will be demonstrable in the year following treatments.

The administration of toxic agents applied outside of the limits of the period designated above as perilous is advisable to be made in the form of spraying or wet dusting. In the course of dusting it is namely unavoidable that the flowering weeds, offering food around the lucerne fields, and the nesting sites remain free of poisons. By the careful application of spraying and wet dusting the treatment can be better delimited to the fields proper, thus the posioning of the above areas and consequently that of the wild bees can be obviated.

Effect of Weed Control

We have found, in the course of our investigations, that the deleterious effects of weed control are equal to, if not more pronounced than, those of insecticides. During the entire period of their flight the wild bees require a suitable quality of food, nectar and pollen. If feeding conditions are not guaranteed, the population will perish, and only the few animals will survive whose food is assured. Should the continuous supply of food be blocked for one or two weeks, destruction will set in. If the continuous supply of food is guaranteed, but the number of plants offering food is small, the diminishing of the population may also commence. An interruption of the course of steady food conditions will result in a certain shortening of the flight, incurring also the decrease of the numbers of the brood. The females live namely a shorter time than they might, and they construct fewer brood-cells than during a normal life-span. For the species whose flight commences before the flowering of lucerne, the suspension of the continuous supply of food results in a decrease of the population by the time of complete blossoming.

Mechanical weed control, the mewing of ruderal areas, but chiefly chemical weed control have essentially decreased the amount of weeds, that is, of food also substanding wild bees. This resulted in a decrease of their numbers. This holds especially for cultivated areas, thus also for the environs of lucerne fields. Weed control incurred not only a decrease of the total amount of available food, but also caused the repeated interruption, lasting occasionally for several weeks, of continuous feeding conditions.

Weed control and the decrease of food, respectively, had variously affected the given wild bee groups. The feeding possibilities of the spring species have hardly altered, but the decrease of food coinciding with the end of the flight of the early summer animals and with the middle of the flight of the species with a medium flight period was plainly observable. For the species flying in the summer and feeding on cultivated papilionaceous plants, feeding conditions have not suffered, but for those feeding on weeds they decreased to a considerable rate. Food decrease is substantial at the end of the flight of the first generation, and the beginning of that of the second brood, of the bivoltine wild bee species with a long flight period. The continually breeding species with a long flight period are able to adapt themselves rather plastically to adverse conditions; the radius of their activity is the multiple of that of the other species discussed here, as well as their foodplant spectra being also considerably wider.

The unfavourable situation, resulting from the causes discussed above, should be remedied without question. A direct result of the decrease of numbers of the wild bees is the diminishing yields of the lucerne seed crops. The giving up of weed control and of herbicides, respectively, would of course be an irreal demand, hence the continuous food supply of the wild bees pollinating lucerne must be assured by artificial means. To obtain this object, we have conducted wide-range investigations as to possible foodplants to be used in this respect, but a discussion of our proposals following our studies is beyond the scope of the present paper.

Acknowledgements

It is our agreeable duty to express our sincere thanks to Dr. L. MÓCZÁR, for making available for study the Apoid material, identified by Dr. M. MÓCZÁR, of the Hungarian Natural. History Museum. Thanks are also due to Dr. G. SZELÉNYI and Dr. G. A. MANNINGER for their critical remarks.

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Review of the Mycoflora of Hungary

Part IV*

By

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Research Institute for Plant Protection, Budapest

Class. Basidiomycetes
Subclass. Eubasidii
Ordo: Uredinales
Familia. Pucciniaceae

Genus. *Gymnosporangium* HEDWIG

Gymnosporangium amelanchieris ED. FISCHER
Syn.: G. aurantiacum CHEV.
SYDOW 2. III. p. 26.; PICBAUER 1. p. 14.; PODHRADSZKY in UBRIZSY 5. p. 425.
Gymnosporangium ariae-tremelloides KLEBAHN
Syn.: G. tremelloides HARTIG
KALCHBRENNER 2. I. p. 302.; HAZSLINSZKY 1. p. 142.; HAZSLINSZKY 3. p. 78.; MOESZ 12. col. 806.; HOLLÓS 7. p. 147.; BÄUMLER, HAZSLINSZKY, HOLUBY, KALCHBRENNER in RABENH. Fung. europ. exs. No. 897., MÁGOCSY-DIETZ, VARGA ap. MOESZ 17/a. p. 143.; PODHRADSZKY in UBRIZSY 5. p. 425.
Gymnosporangium aucupariae-juniperinum KLEBAHN
Syn.: Aecidium cornutum PERS.; Roestelia cornuta FRIES; G. juniperinum auct.; G. juniperi LINK
ENDLICHER 1. No. 83.; FUSS 1. IV. p. 125.; VIII. p. 232.; KALCHBRENNER 1. I. p. 302.; HAZSLINSZKY 3. p. 143.; HAZSLINSZKY 7. p. 15.; BORBÁS 3. p. 145.; RICHTER 1. No. 33.; BÄUMLER 1. II. No. 643.; SYDOW 2. III. p. 29.; BUBÁK 1. No. 60.; MOESZ 12. col. 806.; MOESZ 30. II. p. 373.; MOESZ 31. p. 95.; BÄUMLER, BÁNYAI, BOGSCH, BUDAI, FILARSZKY, GÁYER, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, LENGYEL, MÁGÓCSY-DIETZ, MÁRTON, MOESZ, RICHTER, SCHNELLER, SZEPESFALVY, SZÉPLIGETI ap. MOESZ 17/a. p. 144.; HAZSLINSZKY ap. MOESZ 13. p. 114.; PODHRADSZKY in UBRIZSY 5. p. 425.
Gymnosporangium aurantiacum CHEVALLIER
Syn.: G. amelanchieris ED. FISCHER
SAVULESCU 7. p. 537.; TÓTH 2. p. 57.

* Parts I—III appeared in Acta Phytopathologica 1967. Vol. 2. No. 2. pp. 153—178 Vol. 2. No. 3. pp. 267—285; Vol. 2. No. 4. pp. 388—411.
Data till 1940 are covering the entire territory of the Carpathian basin.

Gymnosporangium clavariaeforme (JACQU.) DE CANDOLLE

Syn.: Aecidium laceratum SOW.; Roestelia lacerata MÉRAT; R. oxyacanthae LINK HAZSLINSZKY 1. p. 143.; HAZSLINSZKY 2. p. 15.; HAZSLINSZKY 3. p. 79.; RICHTER 1. No. 32.; BORBÁS 3. p. 145.; BRESADOLA 1. No. 131.; BÄUMLER 1. II. No. 642.; SACCARDO 1. No. 116.; SYDOW 2. III. p. 62.; BUBÁK 1. No. 57.; MOESZ 2. No 53.; HOLLÓS 1. p. 141.; MOESZ 4. p. 145.; MOESZ 12. col. 806.; HOLLÓS 7. p. 147.; BÄUMLER, BÁNHEGYI, BOLLA, BORBÁS, BUDAI, CHYZER, FILARSZKY, GRESCHIK, HAZSLINSZKY, HOLUBY in LINHART Fung. hung. exs. No. 332., KMET, KOLOSVÁRY, KŐFARAGÓ-GYELNIK, KUPCSOK, MÁGÓCSY-DIETZ, MOESZ, RICHTER, SZARTÓRISZ, SZÉPLIGETI, SZURÁK, TUZSON, WAGNER ap. MOESZ 17/a p. 144.; BÁNHEGYI, FILARSZKY, MOESZ, SADLER, SZARTORISZ, SZÉPLIGETI ap. MOESZ 13. p. 115.; SAVULESCU 7. p. 541.; PODHRADSZKY in UBRIZSY 5. p. 425.; UBRIZSY 8. p. 365.

Gymnosporangium confusum PLOWRIGHT

BÄUMLER 1. V. No. 1870.; BUBÁK 1. No. 58.; SYDOW 2. III. p. 57.; GRESCHIK ap. MOESZ 17/a. p. 145.; SAVULESCU 7. p. 544.; PODHRADSZKY in UBRIZSY 5. p. 424.; UBRIZSY 8. p. 365.

Gymnosporangium juniperinum (L.) MARTIN

PICBAUER 1. p. 14.; SAVULESCU 7. p. 539.; TÓTH 2. p. 57.

Gymnosporangium mali-tremelloides KLEBAHN

Syn.: G. juniperinum (L.) MART.

KALCHBRENNER 1. I. p. 302.; HAZSLINSZKY 1. p. 143.; RICHTER 1. No. 49.; KMET 1. IV. p. 42.; SYDOW 2. III. p. 47.; BUBÁK 1. No. 60.; MOESZ 12. col. 806.; MOESZ 30. II. p. 373.; BÄUMLER, GRESCHIK, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KMET, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 245., RICHTER ap. MOESZ 17/a. p. 146.; SAVULESCU 7. p. 539.; PODHRADSZKY in UBRIZSY 5. p. 425

Gymnosporangium sabinae (DICKS.) WINTER

ENDLICHER 1. No. 84.; FUSS 1. IV. p. 125.; KALCHBRENNER 1. I. p. 302.; HAZSLINSZKY 1. p. 142.; HAZSLINSZKY 3. p. 78.; BÄUMLER 1. II. No. 644.; SYDOW 2. III. p. 53.; HOLLÓS 7. p. 148.; MOESZ 31. p. 95.; BÄUMLER, BOLLA, LINHART in Fung. hung. exs. No. 237., MÁGOCSY-DIETZ, MÁRTON, MOESZ, SCHNELLER, TUZSON, ZAHLBRUCKNER ap. MOESZ 17/a. p. 146.; UBRIZSY 1. p. 12.; UBRIZSY 2. p. 153.; KRENNER 1. p. 63.; SAVULESCU 7. p. 546.; PODHRADSZKY in UBRIZSY 5. p. 421.

Gymnosporangium torminali-juniperium ED. FISCHER

HAZSLINSZKY 1. p. 143.; HAZSLINSZKY 3 p. 79.; BÄUMLER 1. II. No. 643.; SYDOW 2. III. p. 31.; MOESZ 11. p. 100.; HOLLÓS 7. p. 148.; BÄUMLER, HAZSLINSZKY, KMET, MÁGOCSY-DIETZ, ap. MOESZ 17/a. p. 146.; SZÉPLIGETI ap. MOESZ 13. p. 115.; SAVULESCU 7. p. 537.; PODHRADSZKY in UBRIZSY 5. p. 425.

Gymnosporangium tremelloides A. BRAUN

HAZSLINSZKY 1. p. 144., 145.; HAZSLINSZKY 7. p. 10.; KMET 2. V. No. 380.; SACCARDO 1. No. 117.; HAZSLINSZKY, HOLUBY, SZÉPLIGETI in LINHART

Fung. hung. exs. No. 132., TUZSON ap. MOESZ 17/a. p. 147.; SZÉPLIGETI ap. MOESZ 13. p. 115.; PODHRADSZKY in UBRIZSY 5. p. 424.

Genus. *Kuehneola* MAGNUS

Kuehneola albida (KÜHN) MAGNUS

Syn.: Phragmidium albidum (KÜHN) DIETEL; K. uredinis (LINK) ARTHUR BUBÁK 1. No. 53.; SYDOW 2. III. p. 316.; BÄUMLER 1. V. No. 1871.; LINHART in Fung. hung. exs. No. 235. ap. MOESZ 17/a. p. 147.; SAVULESCU 7. p. 491.

Genus. *Gymnoconia* LAGERHEIM

Gymnoconia peckiana (HOWE) TROTTER

Syn.: G. interstitialis LGH.
PODHRADSZKY in UBRIZSY 5. p. 426.

Genus. *Phragmidium* LINK

Phragmidium disciflorum (TODE) JAMES

Syn.: Ph. subcorticium (SCHRANK) WINTER, Ph. mucronatum (PERS.) SCHLECHT. ENDLICHER 1. No. 65., 90.; SCHULZER 6. VII. p. 129.; KALCHBRENNER 1. I. p. 312.; FUSS 1. IV. p. 111., 126., VIII. p. 232.; SCHULZER 1. XVI. No. 39., 62., 63.; SCHULZER 2. p. 21., 24., 31., 33.; SCHULZER 7. XVIII. p. 709.; HAZSLINSZKY 1. p. 107., 172.; FUSS 2. p. 443.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 88.; BORBÁS 3. p. 145.; CSERNI 1. p. 105.; RICHTER 1. No. 27.; BRESADOLA 1. No. 135.; BÄUMLER 1. II. No. 650.; BUBÁK 1. No. 56.; MOESZ 2. No. 83.; RAPAICS 1. p. 218.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 119.; RAPAICS 4. p. 800.; MOESZ 11. p. 100.; MOESZ 12. col. 807.; HOLLÓS 7. p. 149.; MOESZ 30. p. 7., II. p. 373.; BÄUMLER, BÁNYAI, BORBÁS, BUDAI, FILARSZKY in Krypt. exs. No. 708/b., FUSS, HAJÓS in LINHART Fung. hung. exs. No. 130., HAYNALD, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 431., MÁRTON, MOESZ, NAGY, PÁTER, PÓSCH, RICHTER, SZÉPLIGETI, TAUSCHER, TUZSON, ZAHLBRUCKNER ap. MOESZ 17/a.p. 147.; AUGUSTIN, BORBÁS, MOESZ ap. MOESZ 13. p. 116. UBRIZSY 1. p. 12.; SAVULESCU 7. p. 505.; TÓTH 2. p. 57.; VASS-TÓTH 1. p. 158.; VASS 1. p. 50.; VASS—TÓTH 2. p. 48.; PODHRADSZKY in UBRIZSY 5. p. 418.; UBRIZSY 8. p. 365.

Phragmidium fragariastri (DC.) SCHROETER

FUSS 2. p. 442.; HAZSLINSZKY 3. p. 28.; HAZSLINSZKY 7. p. 14.; BRESADOLA 1. No. 132.; BÄUMLER 1. II. No. 645.; SIMONKAI 1. No. 1718.; BUBÁK 1. No. 54.; SYDOW 2. III. p. 102.; MOESZ 12. col. 807.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, BUDAI, CHYZER, DEGEN, GRESCHIK, JÁVORKA, KLEIN in LINHART Fung. hung. exs. No. 331., KMET, KŐFARAGÓ-GYELNIK, KÜMMERLE, LAUDON, MOESZ, SABRANSKY, SCHNELLER, SZEPESFALVY ap. MOESZ 17/a. p. 149.; MOESZ, SZEPESFALVY ap. MOESZ 13. p. 116.; UBRIZSY 1. p. 13.;

SAVULESCU 7. p. 501.; TÓTH 2. p. 57.; VASS 1. p. 50.; VASS–TÓTH 2. p. 48.; PODHRADSZKY in UBRIZSY 5. p. 417.; UBRIZSY 8. p. 366.

Phragmidium fusiforme SCHROETER

HAZSLINSZKY 2. p. 20.; HAZSLINSZKY 3. p. 88.; RICHTER 1. No. 26.; BÄUMLER 1. II. No. 646.; SACCARDO 1. No. 118.; MAGNUS 1. p. 46.; SYDOW 2. III. p. 122.; MOESZ 12. col. 807.; GREISIGER, GYŐRFFY, HAZSLINSZKY, KÁLOVICS, LENGYEL, MÁGOCSY-DIETZ, MÁRTON, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 330. ap. MOESZ 17/a. p. 149.; SAVULESCU 7. p. 511.; PODHRADSZKY in UBRIZSY 5. p. 417.

Phragmidium potentillae (PERS.) KARSTEN

ENDLICHER 1. No. 66.; BOLLA 1. p. 51.; KALCHBRENNER 1. I. p. 312.; FUSS 1. IV. p. 111., 126.; VIII. p. 2321.; FUSS 2. p. 442.; 445.; HAZSLINSZKY 1. p. 170., 172.; HAZSLINSZKY 2. p. 20.; HAZSLINSZKY 3. p. 88.; HAZSLINSZKY 5. p. 43.; HAZSLINSZKY 7. p. 13.; BORBÁS 2. p. 33.; RICHTER 1. No. 29.; BRESADOLA 1. No. 133.; BÄUMLER 1. II. No. 647.; SIMONKAI 1. No. 1716.; BUBÁK 1. No. 56.; MOESZ 2. No. 82.; HOLLÓS 1. pp. 141–142.; SYDOW 2. III. p. 99.; MOESZ 11. p. 100.; MOESZ 12. col. 807.; HOLLÓS 7. p. 150.; MOESZ 30. I. p. 7.; BARTH, BÄUMLER, BOLLA, FILARSZKY, FUSS, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KMET, KÜMMERLE, LAUDON, LINHART in Fung. hung. exs. No. 234., MÁGOCSY-DIETZ, MOESZ, POSCH, RICHTER, RÖMER in LINHART Fung. hung. exs. No. 432., SADLER, SCHNELLER, SZÉPLIGETI in LINHART Fung. hung. exs. No. 233., ZAHLBRUCKNER, ZSÁK ap. MOESZ 17/a. p. 150.; HAZSLINSZKY, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 116.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 496.; TÓTH 2. p. 57.; PODHRADSZKY in UBRIZSY 5. p. 419.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 366.

Phragmidium rosae-pimpinellifoliae (RABENH.) DIETEL

SYDOW 2. III. p. 113.; SZÉPLIGETI ap. MOESZ 17/a. p. 151.; SZÉPLIGETI ap. MOESZ 15. p. 116.; SAVULESCU 7. p. 503.; PODHRADSZKY in UBRIZSY 5. p. 419.; UBRIZSY 8. p. 366.

Phragmidium rubi (PERS.) WINTER

BOLLA 1. p. 51.; FUSS 1. IV. p. 126.; SCHULZER 1. XVI. No. 62.; SCHULZER 2. p. 32.; SCHULZER 7. p. 709.; HAZSLINSZKY 1. p. 172., 173.; FUSS 2. p. 442.; HAZSLINSZKY 3. p. 88.; BORBÁS 3. p. 145.; RICHTER 1. No. 30.; BÄUMLER 1. II. No. 648.; BRESADOLA 1. No. 134.; SIMONKAI 1. No. 1717.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 143.; MOESZ 4. II. p. 145.; MOESZ 11. p. 100.; HOLLÓS 7. p. 150.; MOESZ 30. I. p. 7.; MOESZ 31. p. 95.; BARTH, BÄUMLER, BERNÁTSKY, BUDAI, FUSS, GREINICH, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, KÜMMERLE, MÁGOCSY-DIETZ, MÁRTON, RICHTER, SADLER, SZEPESFALVY, SZÉPLIGETI, TIMKÓ, VESELSKY, WAGNER, ZAHLBRUCKNER ap. MOESZ 17/a. p. 151.; HAZSLINSZKY, MOESZ, KÜMMERLE, SADLER, SZEPESFALVY ap. MOESZ 13. p. 116.; SAVULESCU 7. p. 521.; TÓTH 2. p. 57.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 419.; UBRIZSY 8. p. 366.

Phragmidium rubi var. *candicantium* VLEUGEL

MOESZ ap. MOESZ 17/a. p. 152.

Phragmidium rubi-idaei (PERS.) KARSTEN

BOLLA 1. p. 48.; KALCHBRENNER 1. I. p. 312.; FUSS 1. IV. p. 126.; XVI. p. 24.; SCHULZER 2. p. 16., 33.; HAZSLINSZKY 1. p. 172., 173.; FUSS 2. p. 442., 446.; BORBÁS 2. p. 33.; HAZSLINSZKY 7. p. 13.; BÄUMLER 1. II. No. 649.; SYDOW 2. III. p. 147.; HOLLÓS 7. p. 150.; BÄUMLER, FUSS, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, LINHART in Fung. hung. exs. No. 236., MÁGOCSY-DIETZ in Krypt. exs. No. 106., MOESZ, PÓSCH, SCHULZER in LINHART. Fung. hung. exs. No. 131., TUZSON ap. MOESZ 17/a. p. 152.; SADLER ap. MOESZ 13. p. 116.; SAVULESCU 7. p. 516.; TÓTH 2. p. 57.; VÖRÖS 9. p. 102.; PODHRADSZKY in UBRIZSY 5. p. 419.; UBRIZSY 8. p. 366.

Phragmidium rubi-saxatilis LIRO

KMET ap. MOESZ 17/a. p. 152.; SAVULESCU 7. p. 524.

Phragmidium sanguisorbae (DC) SCHROETER

HAZSLINSZKY 1. p. 170.; HAZSLINSZKY 2. p. 20.; HAZSLINSZKY 7. p. 13.; RICHTER 1. No. 28.; BÄUMLER 1. IV. No. 1501.; V. No. 1873.; HOLLÓS 1. p. 142.; HOLLÓS 7. p. 150.; BÄUMLER, BUDAI, HAZSLINSZKY, HOLUBY, LINHART in Fung. hung. exs. No. 232., MÁGOCSY-DIETZ in Krypt. exs. No. 102., MOESZ, SCHNELLER, STAUB, SZÉPLIGETI ap. MOESZ 17/a. p. 152.; MÁGOCSY-DIETZ, MOESZ, STAUB, SZÉPLIGETI ap. MOESZ 13. p. 116.; SAVULESCU 7. p. 514.; TÓTH 2. p. 57.; PODHRADSZKY in UBRIZSY 5. p. 419.; UBRIZSY 8. p. 367.

Phragmidium tuberculatum MÜLLER

SYDOW 2. III. p. 115.; BÄUMLER 1. V. No. 1875.; GRESCHIK, HOLUBY, MOESZ ap. MOESZ 17/a. p. 153.; SAVULESCU 7. p. 509.; TÓTH 2. p. 57.; VASS – TÓTH 1. p. 158.; PODHRADSZKY in UBRIZSY 5. p. 419.

Phragmidium violaceum (SCHULTZ) WINTER

BOLLA 1. p. 51.; HAZSLINSZKY 3. p. 88.; HAZSLINSZKY 5. p. 43.; RICHTER 1. No. 31.; BÄUMLER 1. II. No. 651.; RAPAICS 1. p. 216.; SYDOW 2. III. p. 141.; MOESZ 11. p. 100.; HOLLÓS 7. p. 150.; BÄUMLER, BOLLA, BUDAI, GÁYER, HAZSLINSZKY, HOLLÓS, HOLUBY, KMET, LINHART in Fung. hung. exs. No. 235., MÁGOCSY-DIETZ, MOESZ, PÓSCH, SZÉPLIGETI, TUZSON ap. MOESZ 17/a. p. 153.; MOESZ 13. p. 116.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 518.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 419.

Genus. *Puccinia* PERSOON

Puccinia absinthii DE CANDOLLE

Syn.: P. artemisiella SYDOW

KALCHBRENNER 1. I. p. 309.; HAZSLINSZKY 1. p. 154.; HAZSLINSZKY 2. p. 17.; FUSS 2. p. 441., 445.; HAZSLINSZKY 3. p. 84.; RICHTER 1. No. 19.; BÄUMLER 1. II. No. 635., V. No. 1827.; BRESADOLA 1. No. 117.; SACCARDO 1. No. 109.; SYDOW 2. I. p. 12.; MOESZ 2. No. 62.; HOLLÓS 1. p. 134.; HOLLÓS 6/a. p. 104.; MOESZ 11. p. 100.; MOESZ 21. p. 85.; HOLLÓS, 7. p. 150.; ANGYAL, BÄUMLER in Krypt. exs. No. 2203/a., GREINICH, GRE-

SCHIK, HAZSLINSZKY, HOLLÓS in Krypt. exs. No. 2203/b., HOLUBY, KMET, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 216, 226., MOESZ, SZÉPLIGETI ap. MOESZ 17/a. p. 154.; ANGYAL, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 116.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 990.; TÓTH 2. p. 57.; TÓTH 10. p. 9.; DOHY—KIRÁLY in UBRIZSY 5. p. 463.; UBRIZSY 8. p. 367.

Puccinia acetosae (SCHUM.) KÖRNICKE

BOLLA 1. p. 48.; SYDOW 2. I. p. 581.; MOESZ 11. p. 100.; HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/a. p. 155.; MOESZ ap. MOESZ 13. p. 117.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 841.; TÓTH 2. p. 57.; VASS 1. p. 51.; DOHY—KIRÁLY in UBRIZSY 5. p. 461.

Puccinia aconiti-rubrae LÜDI

Syn.: Aecidium aconiti-napelli (DC.) WINTER

FUSS 1. IV. p. 125.; KALCHBRENNER 1. p. 304.; HAZSLINSZKY 1. p. 137.; FUSS 2. p. 437.; MOESZ 12. col. 804.; SYDOW 2. IV. p. 348.; HAZSLINSZKY 3. p. 77.; GYŐRFFY, LINHART in Fung. hung. exs. No. 246., SIMKOVITS, TUZSON ap. MOESZ 17/a. p. 155.; PICBAUER 1. p. 14.; PICBAUER 2. p. 193.; SAVULESCU 7. p. 691.

Puccinia actaeae-agropyri ED. FISCHER

SYDOW 2. I. p. 828.; BOROS, BUDAI, GREINICH, GRESCHIK, KMET, MOESZ, VAJDA ap. MOESZ 17/a. pp. 155—156.; PICBAUER 1. p 11.; PICBAUER 2. p. 189.

Puccinia actaeae-elymi MAYOR

SYDOW 2. IV. p. 352.; HOLLÓS 2. p. 13.; MOESZ, PÉNZES ap. MOESZ 17/a. p. 156.; PICBAUER 1. p. 11.; SAVULESCU 7. p. 692; TÓTH 2. p. 57.; UBRIZSY 8. p. 367.

Puccinia adoxae HEDWIG

ENDLICHER 1. No. 87.; HAZSLINSZKY 1. p. 166.; BÄUMLER 1. II. No. 591.; SYDOW 2. I. p. 204.; BÁNHEGYI, BENEDEK, MOESZ, PÉNZES, SABRANSKY ap. MOESZ 17/a. p. 156.; BÁNHEGYI, BENEDEK, GREGUSS, MOESZ, PÉNZES ap. MOESZ 13. p. 117.; SAVULESCU 7. p. 949.; TÓTH 2. p. 57.; SZIGETI—TÓTH 1. p. 20.

Puccinia aecidii-leucanthemi FISCHER

Syn.: Aecidium leucanthemi DC.

BOLLA 1. p. 49.; HAZSLINSZKY 1. p. 140.; HAZSLINSZKY 3. p. 84.; BÄUMLER 1. II. No. 663.; SACCARDO 1. No. 128. SYDOW 2. I. p. 664.; BÄUMLER, HOLUBY, KMET in Flora exs. Austro-Hung. No. 3154., LINHART in Fung. hung. exs. No. 46., MOESZ, TRAUTMANN ap. MOESZ 17/a. p. 156.; MOESZ, TRAUTMANN ap. MOESZ 13. p. 117.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 799.

Puccinia aegopodii (SCHUM.) MARTIN

BOLLA 1. p. 51.; KALCHBRENNER 1. I. p. 308.; HAZSLINSZKY 1. p. 166.; HAZSLINSZKY 2. p. 19.; FUSS 2. p. 440.; BORBÁS 3. p. 145.; BÄUMLER 1. II. No. 592.; SACCARDO 1. No. 111.; LIRO 3. p. 114.; SYDOW 2. I. p. 354.; MOESZ 12. col. 807.; BÄUMLER, FILARSZKY, GRESCHIK, HAZSLINSZKY, HOLUBY

in LINHART Fung. hung. exs. No. 118., KMET, MÁRTON, MOESZ, PÉNZES, SCHILBERSZKY in Krypt. exs. No. 1304., SZÉPLIGETI ap. MOESZ 17/a. p. 157.; MOESZ, PÉNZES, SCHILBERSZKY, SZÉPLIGETI ap. MOESZ 13. p. 117.; SAVULESCU 7. p. 899.; VASS—TÓTH 3. p. 85.; UBRIZSY 8. p. 367.

Puccinia agropyri ELLIS et EVERHART

Syn.: P. (clematidis) agropyri KLEB.; Aecidium clematidis DC.

FUSS 1. IV. p. 125.; KALCHBRENNER 1. I. p. 304.; HAZSLINSZKY 1. p. 137.; FUSS 2. p. 438., 440.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 77.; HAZSLINSZKY 7. p. 11.; BÄUMLER 1. II. No. 659., V. No. 1828.; RICHTER 1. No. 52.; BRESADOLA 1. No. 144.; SACCARDO 1. No. 126.; SYDOW 2. I. p. 824.; BUBÁK 1. No. 28.; MOESZ 2. No. 63.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 109.; HOLLÓS 7. p. 151.; BÄUMLER, BERNÁTSKY, BORBÁS, BUDAI, FILARSZKY, GRESCHIK, HAMBALKÓ, HAZSLINSZKY, HOLLÓS, HOLUBY, JÁVORKA, KMET, LINHART in Fung. hung. exs. No. 144., 437., MÁGOCSY-DIETZ in Krypt. exs. No. 1705/6., MÁRTON, MOESZ, RICHTER, SCHNELLER, STAUB, SZÉPLIGETI, TAUSCHER, TUZSON, ZSÁK ap. MOESZ 17/a. p. 157.; BERNÁTSKY, BORBÁS, HAMBALKÓ, HAZSLINSZKY, JÁVORKA, MÁGOCSY-DIETZ, MOESZ, SADLER, STAUB, SZÉPLIGETI, TAUSCHER, ZSÁK ap. MOESZ 13. p. 117.; PICBAUER 1. p. 11.; PICBAUER 2. p. 189.; UBRIZSY 6. in SZÉKESSY p. 23.; SAVULESCU 7. p. 695; TÓTH 2. p. 57.; UBRIZSY 8. p. 367.

Puccinia agropyrina ERIKSSON

SYDOW 2. I. p. 712.; HOLLÓS 7. p. 151.; SAVULESCU 7. p. 697.; UBRIZSY 8. p. 368.

Puccinia agrostidis PLOWRIGHT

Syn.: P. agrostis PLOWR.; Aecidium aquilaegiae PERS

FUSS 1. IV. p. 125.; KALCHBRENNER 1. I. p. 304.; HAZSLINSZKY 1. p. 137.; FUSS 2. p. 437.; BORBÁS 3. p. 145.; RICHTER 1. No. 50.; SYDOW 2. I. p. 718.; MOESZ 12. col. 807.; FILARSZKY, GRESCHIK, HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ, RICHTER, SZÉPLIGETI ap. MOESZ 17/a. p. 158.; SAVULESCU 7. p. 705.; DOHY—KIRÁLY in UBRIZSY 5. p. 470.

Puccinia albescens (GREV.) PLOWRIGHT

HAZSLINSZKY 1. p. 132.; BÄUMLER, HAZSLINSZKY, MOESZ ap. MOESZ 17/a. p. 158.; SAVULESCU 7. p. 951.

Puccinia alpina FUCKEL

HAZSLINSZKY 1. p. 148. ap. MOESZ 17/a. p. 158.

Puccinia ambigua (ALB. et SCHW.) LAGERHEIM

Syn.: P. difformis KTZE et SCHUM.

HAZSLINSZKY 1. p. 156.; HOLLÓS 7. p. 151.; BUDAI, HAZSLINSZKY, KMET, SZÉPLIGETI ap. MOESZ 17/a. p. 159.; SZÉPLIGETI ap. MOESZ 13. p. 117.; SAVULESCU 7. p. 954.; TÓTH 2. p. 59.

Puccinia angelicae (SCHUM.) FUCKEL

PICBAUER 1. p. 13.; SAVULESCU 7. p. 905.

Puccinia annularis (STRAUSS) SCHLECHTENDAL

HAZSLINSZKY 1. p. 159.; HAZSLINSZKY 2. p. 18.; HAZSLINSZKY 3. p. 85.;

BÄUMLER 1. II. No. 594.; SYDOW 2. I. p. 300.; BUBÁK 1. No. 29.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 100.; HOLLÓS 7. p. 151.; BÄUMLER, HAZSLINSZKY, HOLLÓS in Krypt. exs. No. 2712., HOLUBY, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/a. p. 159.; MOESZ ap. MOESZ 13. p. 118. PICBAUER 1. p. 13. PICBAUER 2. p. 192.; SAVULESCU 7. p. 921.; LEHOCZKY 1. p. 236.

Puccinia anthoxanthi DIETEL

SAVULESCU 7. p. 778.; DOHY – KIRÁLY in UBRIZSY 5. p. 469.

Puccinia antirrhini DIETEL et HOLWAY

HUSZ 3/c. p. 240.; HUSZ 4/a. p. 292.; HUSZ 4/b. p. 39.; HUSZ, KRENNER, MOESZ ap. MOESZ 17/a. p. 159.; MOESZ 42.; HUSZ, KRENNER, MOESZ ap. MOESZ 13. p. 118.; KRENNER 1. p. 63.; SAVULESCU 7. p. 971.; TÓTH 2. p. 57.; SZIGETI – TÓTH 1. p. 20.; DOHY – KIRÁLY in UBRIZSY 5. p. 471.; UBRIZSY 8. p. 368.

Puccinia apii DESMAZIÈRES

LIRO 3. p. 101.; SYDOW 2. I. p. 360.; LINHART in Fung. hung. exs. No. 121. ap. MOESZ 17/a. p. 159.; SAVULESCU 7. p. 893.; DOHY – KIRÁLY in UBRIZSY 5. p. 461.

Puccinia arenariae (SCHUM.) WINTER

Syn.: P. lychnidearum LINK.

BOLLA 1. p. 51.; KALCHBRENNER 1. I.306., 308.; HAZSLINSZKY 1. p. 164., 165.; HAZSLINSZKY 2. p. 18., 19.; FUSS 2. p. 440.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 87.; HAZSLINSZKY 7. p. 11.; RICHTER 1. No. 8.; BÄUMLER 1. II. No. 595., IV. No. (595.); SYDOW 2. I. p. 553.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 107.; MOESZ 4. II. p. 145.; HUSZ 1. p. 101.; MOESZ 11. p. 100.; MOESZ 12. col. 807.; HOLLÓS 7. p. 152.; MOESZ 30., II. p. 373.; MOESZ 31. p. 95.; BÄUMLER, BÁNYAI, BOLLA, DEGEN, FILARSZKY, FUSS, GREINICH, GRESCHIK, HAZSLINSZKY, HOLUBY, JÁVORKA, KALCHBRENNER, KÁRPÁTI, KMET, KÜMMERLE, LAUDON, LINHART in Fung. hung. exs. No. 116., 217., MÁGOCSY-DIETZ in Krypt. exs. No. 40/a., MICHAELIS, MOESZ, PÉNZES, SABRANSKY, SZADLER, SZÉPLIGETI ap. MOESZ 17/a. p. 159.; DEGEN, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 118.; SAVULESCU 7. p. 847.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 58.; VASS – TÓTH 1. p. 158.; VASS – TÓTH 2. p. 48.; TÓTH 10. p. 9.; DOHY – KIRÁLY in UBRIZSY 5. p. 467.; UBRIZSY 8. p. 368.

Puccinia argentata (SCHULTZ) WINTER

HAZSLINSZKY 1. p. 149.; BRESADOLA 1. No. 119.; SYDOW 2. I. p. 451.; BÄUMLER 1. IV. No. 1949.; MOESZ 12. col. 807.; BÄUMLER in Krypt. exs. No. 33/b, BUDAI, GRESCHIK, HAZSLINSZKY ap. MOESZ 17/a.p. 161.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 868.; DOHY – KIRÁLY in UBRIZSY 5. p. 463.

Puccinia ari-phalaridis (PLOWR.) KLEBAHN

Syn.: P. phalaridis PLOWR.; Aecidium ari DESM.

LINHART in Fung. hung. exs. No. 140. ap. MOESZ 17/a. p. 161.; SAVULESCU 7. p. 727.

Puccinia aristolochiae (DC.) WINTER

FUSS 1. IV. p. 112.; HAZSLINSZKY 1. p. 115.; FUSS 2. p. 444.; MOESZ 30. II. p./373.; BÁNYAI ap. MOESZ 17/a. p. 161.; SAVULESCU 7. p. 834.

Puccinia arnicae-scorpioides (DC.) MAGNUS

HUSZ 1. p. 101. ap. MOESZ 17/a. p. 161.

Puccinia aromatica BUBÁK

LIRO 3. p. 23.; SYDOW 2. I. p. 370.; BÄUMLER 1. V. No. 1829.; KMET ap. MOESZ 17/a. p. 161.; PICBAUER 2. p. 189.; SAVULESCU 7. p. 884.; TÓTH 2. p. 58.

Puccinia arrhenatheri (KLEB.) ERIKSSON

Syn.: Aecidium magelhaenicum auctorum, non BERK.

KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 134.; BORBÁS 2. p. 33.; BÄUMLER 1. II. No. 664., V. No. 1830.; SYDOW 2. I. p. 729.; MOESZ 2. No. 64.; HOLLÓS 7. p. 152.; BÄUMLER in RABENH.—WINTER Fung. eur. exs. No. 3221., BERNÁTSKY, FILARSZKY, HAZSLINSZKY, KÜMMERLE, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 143.; MOESZ ap. MOESZ 17/a. p. 162.; BERNÁTSKY, FILARSZKY, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 118.; SAVULESCU 7. p. 701.; VASS 1. p. 51.; UBRIZSY 8. p. 368.

Puccinia artemisiella SYDOW

SAVULESCU 7. p. 994.; VASS—TÓTH 2. p. 48.

Puccinia artemisiicola SYDOW

SYDOW 2. I. p. 14. ap. MOESZ 17/a. p. 162.; SAVULESCU 7. p. 980.

Puccinia asarina KUNTZE

ENDLICHER 1. No. 86.; KALCHBRENNER 1. I. p. 310.; HAZSLINSZKY 1. p. 161.; HAZSLINSZKY 2. p. 18.; HOLUBY 2. p. 18.; RICHTER 1. No. 10.; BÄUMLER 1. II. No. 596.; SIMONKAI 1. No. 1730.; SYDOW 2. I. p. 584.; BUBÁK 1. No. 30.; MAGNUS 1. p. 46.; MOESZ 12. col. 807.; MOESZ 30. II. p. 373.; MOESZ 31. p. 95.; BÄUMLER, BÁNYAI, BOLLA, BUDAI, ÉHIK, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KMET, KÜMMERLE, LÁNYI, LINHART in Fung. hung. exs. No. 25., MOESZ, RICHTER, SZÉPLIGETI, VISNYA ap. MOESZ 17/a. p. 162.; KÜMMERLE, MOESZ ap. MOESZ 13. p. 118.; PICBAUER 1. p. 13.; SAVULESCU 7. p. 835.; TÓTH 2. p. 58.; VÖRÖS 9. p. 102.; UBRIZSY 8. p. 368.

Puccinia asparagi DE CANDOLLE

KALCHBRENNER 1. I. p. 311.; HAZSLINSZKY 1. p. 161.; FUSS 2. p. 441.; HAZSLINSZKY 3. p. 86.; HAZSLINSZKY 7. p. 12.; HAZSLINSZKY 12. p. 371.; BÄUMLER 1. II. No. 597.; SYDOW 2. I. p. 616.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 107.; RAPAICS 1. p. 216.; HOLLÓS 7. p. 151.; BÄUMLER, DEGEN, FUSS, HAZSLINSZKY, LÁNYI, LINHART in RABENH. Fung. eur. exs. No. 3507. et in Fung. hung. exs. No. 223., MÁGOCSY-DIETZ in Krypt. exs. No. 24., MOESZ, VAJDA ap. MOESZ 17/a. p. 163.; DEGEN, MÁGOCSY-DIETZ, MOESZ, VAJDA ap. MOESZ 13. p. 119.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 816.; TÓTH 2. p. 58.; DOHY—KIRÁLY in UBRIZSY 5. p. 465.; UBRIZSY 8. p. 368.

Puccinia asperulae-cynanchicae WURTH

BÄUMLER 1. II. No. 610.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 100.; MOESZ

30. p. 7.; BÄUMLER, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 324. ap. MOESZ 17/a. p. 163.; SZÉPLIGETI ap. MOESZ 13. p. 119.; SAVULESCU 7. p. 961.

Puccinia asperulae-odoratae WURTH

LINHART in Fung. hung. exs. No. 429. et in RABENH.—WINTER Fung. eur. exs. No. 3512/b., MOESZ ap. MOESZ 17/a. p. 163.; SAVULESCU 7. p. 963.

Puccinia asperulina (JUEL) LAGERHEIM

BUBÁK 1. No. 31.; TUZSON—BUBÁK ap. MOESZ 17/a. p. 163.; SAVULESCU 7. p. 966.

Puccinia asteris DUBY

SYDOW 2. I. 16.; KMET in Flora Austro-Hung. exs. No. 3164., MOESZ in Flora hung. exs. No. 306. ap. MOESZ 17/a. p. 163.; SAVULESCU 7. p. 978.; DOHY—KIRÁLY in UBRIZSY 5. p. 468.

Puccinia astrantiae KALCHBRENNER

FUSS 1. IV., VIII.; KALCHBRENNER 1. I. p. 309.; HAZSLINSZKY 1. p. 165., 166.; LIRO 3. p. 122.; SYDOW 2. I. p. 362.; GRESCHIK, HOLUBY, KALCHBRENNER ap. MOESZ 17/a. p. 163.; SAVULESCU 7. p. 900.

Puccinia athamantae (DC.) LINDROTH

BOLLA 1. p. 51.; HAZSLINSZKY 1. p. 150.; HAZSLINSZKY 7. p. 12.; LIRO 3. p. 103.; SYDOW 2. I. p. 405.; HOLLÓS 7. p. 152.; BÄUMLER, BOLLA, HAZSLINSZKY, HOLUBY, LAUDON ap. MOESZ 17/a. p. 164.; SAVULESCU 7. p. 913.

Puccinia atragenes HAUSMANN

BRESADOLA 1. No. 128.; SYDOW 2. I. p. 539.; SAVULESCU 7. p. 856.; TÓTH 2. p. 58.

Puccinia australis KÖRNICKE

BUDAI, MOESZ, SZÉPLIGETI ap. MOESZ 17/a. p. 164.; SZÉPLIGETI ap. MOESZ 13. p. 119.; SAVULESCU 7. p. 761.

Puccinia balsamitae (STRAUSS) RABENHORST

SCHULZER 2. p. 28.; HAZSLINSZKY 1. p. 154.; HAZSLINSZKY 3. p. 84.; BÄUMLER 1. II. No. 599.; BRESADOLA 1. No. 120.; SYDOW 2. I. p. 163.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; AUGUSTIN, BÄUMLER, GRESCHIK, HAZSLINSZKY, HOLLÓS in Krypt. exs. No. 2309., KMET, MÁGOCSY-DIETZ, MOESZ, NAGY, SZÉPLIGETI, ZAHLBRUCKNER ap. MOESZ 17/a. p. 164.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 119.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 988.; TÓTH 2. p. 58.

Puccinia bardanae CORDA

HAZSLINSZKY 1. p. 153.; HAZSLINSZKY 2. p. 16.; HAZSLINSZKY 3. p. 83.; HAZSLINSZKY 7. p. 12.; RICHTER 1. No. 18.; BÄUMLER 1. II. No. 613., V. No. 1831.; SACCARDO 1. No. 108.; SYDOW 2. I. p. 114.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 105.; MOESZ 11. p. 100.; MOESZ 21. p. 85.; HOLLÓS 7. p. 153.; BÄUMLER, BUDAI, HAZSLINSZKY, KMET in Flora exs. Austro-Hung. No. 3160., LINHART in Fung. hung. exs. No. 31., MÁGOCSY-DIETZ, MÁRTON, MOESZ, RICHTER, SZÉPLIGETI ap. MOESZ 17/a. p. 165.; MOESZ, SZÉPLIGETI

ap. MOESZ 13. p. 119.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 1060.; TÓTH 2. p. 58.; TÓTH 10. p. 9.; UBRIZSY 8. p. 368.

Puccinia barkhausiae-rhoeadifoliae BUBÁK

BUBÁK 1. No. 32.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; HOLLÓS 7. p. 153.; MOESZ ap. MOESZ 17/a. p. 165.; BUBÁK, MOESZ ap. MOESZ 13. p. 119.; SAVULESCU 7. p. 1019.

Puccinia baryana THÜMEN

Syn.: P. pulsatillae KALCHBRENNER; P. compacta DUBY.; P. anemones-virginianae SCHROETER

KALCHBRENNER 1. I. p. 307., 308.; HAZSLINSZKY 1. p. 163.; HAZSLINSZKY 3. p. 83.; RICHTER 1. No. 9.; BRESADOLA 1. No. 129.; SYDOW 2. I. p. 536.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 107.; MAGNUS 1. p. 46.; BÄUMLER 1. V. No. 1832.; HOLLÓS 7. p. 161.; FILARSZKY, GRESCHIK, HAZSLINSZKY, KALCHBRENNER, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ in Flora Hung. exs. No. 808., RICHTER ap. MOESZ, 17/a. p. 165.; MOESZ ap. MOESZ 13. p. 119.; SAVULESCU 7. p. 853.

Puccinia baryi (BERK. et BR.) WINTER

HAZSLINSZKY 7. p. 13.; BÄUMLER 1. IV. No. 1495.; SYDOW 2. I. p. 737.; MOESZ 11. p. 100.; MOESZ 12. col. 807.; BÄUMLER in Krypt. exs. No. 1124., BOROS, BUDAI, HAZSLINSZKY, HOLUBY, MOESZ ap. MOESZ 17/a. p. 166.; BOROS, MOESZ ap. MOESZ 13. p. 119.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 736.; VASS—TÓTH 2. p. 48.

Puccinia bäumleriana BUBÁK

BUBÁK 2/a. p. 22.; BÄUMLER 1. V. No. 1833.; BÄUMLER in Krypt. exs. No. 1706. ap. MOESZ 17/a. p. 166.

Puccinia behenis (DC.) OTTH

Syn.: Aecidium behenis DC.; P. lychnidearum LINK; P. silenes SCHROET.

KALCHBRENNER 1. I. p. 304., 308.; HAZSLINSZKY 1. p. 149., 164.; BORBÁS 2. p. 33.; RICHTER 1. No. 21.; SYDOW 2. I. p. 560.; MOESZ 2. No. 79.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 107.; HOLLÓS 7. p. 162.; BÄUMLER 1. V. No. 1834; KMET, MÁRTON, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 326. ap. MOESZ 17/a. p. 166.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 119.; SAVULESCU 7. p. 851.; TÓTH 2. p. 58.

Puccinia betonicae (ALB. et SCHW.) DE CANDOLLE

FUSS 1. IV. p. 126., VIII. p. 232., XVI. p. 24.; SCHULZER 1. No. 56.; KALCHBRENNER 1. I. p. 310.; SCHULZER 2. p. 27.; HAZSLINSZKY 1. p. 156.; FUSS in Herb. Norm. Florae Transsilv. No. 3., MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 312. ap. MOESZ 17/a. p. 167.; SAVULESCU 7. p. 936.; DOHY—KIRÁLY in UBRIZSY 5. p. 463.

Puccinia bistortae (STR.) DE CANDOLLE

Syn.: P. cari-bistortae KLEB.; P. angelicae-mamillata KLEB.; P. mei-mamillata SEMADENI

FUSS 2. p. 441.; SYDOW 2. I. p. 572.; MAGNUS 1. p. 46.; MOESZ 12. col. 808.; FILARSZKY, KÜMMERLE ap. MOESZ 17/a. p. 167.; SAVULESCU 7. p. 845.

Puccinia bromina ERIKSSON

Syn.: Aecidium lithospermi THUEM.

PICBAUER 1. p. 11.; SAVULESCU 7. p. 713.; TÓTH 2. p. 58.; VASS 1. p. 51.; VASS–TÓTH 2. p. 48.; VASS–TÓTH 3. p. 85.; UBRIZSY 8. p. 369.

Puccinia bullata (PERS.) WINTER

FUSS 1. IV. p. 111.; HAZSLINSZKY 1. p. 150.; BÄUMLER 1. II. No. 601., SACCARDO 1. No. 106.; LIRO 3. p. 106.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 106.; MAGNUS 1. p. 46.; PÉTERFI ap. MOESZ 1. p.; MOESZ 11. p. 100.; BÄUMLER, HAZSLINSZKY, KMET, MÁGOCSY-DIETZ, MOESZ in Flora hung. exs. No. 807., TUZSON ap. MOESZ 17/a. p. 168.; HAZSLINSZKY, MÁGOCSY-DIETZ ap. MOESZ 13. p. 120.; SAVULESCU 7. p. 909.

Puccinia bupleuri-falcati (DC.) WINTER

HAZSLINSZKY 1. p. 132.; LIRO 3. p. 133., 134.; SYDOW 2. I. p. 365.; MOESZ 2. No. 65.; BÄUMLER 1. V. No. 1835.; BÄUMLER in Krypt. exs. No. 2713., BEER, BOLLA, BORBÁS, BUDAI, HAZSLINSZKY, HOLUBY, JÁVORKA, MOESZ, PÉNZES ap. MOESZ 17/a. p. 168.; BORBÁS, JÁVORKA, MOESZ, PÉNZES ap. MOESZ 13. p. 120.; SAVULESCU 7. p. 894.; UBRIZSY 8. p. 369.

Puccinia calthae LINK

KALCHBRENNER 1. I. p. 304., 308.; HAZSLINSZKY 1. p. 136., 163.; RICHTER 1. No. 23.; SACCARDO 1. No. 97.; SYDOW 2. I. p. 541.; ROUPPERT 1. p. 311.; BÄUMLER 1. V. No. 1836.; MOESZ 12. col. 807.; GRESCHIK, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 430., LOJKA, MOESZ, RICHTER ap. MOESZ 17/a. p. 168.; SAVULESCU 7. p. 854.

Puccinia calthaecola SCHROETER

Syn.: P. zopfii WINTER ppe.

SAVULESCU 7. p. 858.; TÓTH 2. p. 58.

Puccinia campanulae CARMICHAEL

BRESADOLA 1. No. 126., GRESCHIK ap. MOESZ 17/a. p. 169.

Puccinia carduorum JACKY

HAZSLINSZKY 1. p. 151.; HAZSLINSZKY 2. p. 15.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 83.; HAZSLINSZKY 7. p. 12.; PLOWRIGHT 1. No. 18.; BRESADOLA 1. p. 116.; SYDOW 2. I. p. 33.; HOLLÓS 1. p. 134.; HOLLÓS 6/a. p. 104.; HUSZ 1. p. 101.; MOESZ 12. col. 807.; HOLLÓS 7. p. 153.; BÄUMLER, BUDAI, GREINICH, GRESCHIK, HAZSLINSZKY, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ, RICHTER, SZÉPLIGETI ap. MOESZ 17/a. p. 169.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 120.; PICBAUER 1. p. 11.; PICBAUER 2. p. 192.; SAVULESCU 7. p. 1031.; TÓTH 2. p. 58.; VASS 1. p. 51.

Puccinia caricis (SCHUM.) REBENTISCH

Syn.: P. ribesii-caricis KLEB. ppe.; P. urticae-caricis (SCHUM.) REBENT. ppe.; P. pringsheimiana Kleb. ppe.

SYDOW 2. I. p. 648.; BÄUMLER, BÁNHEGYI, BOROS, BUDAI, DEGEN, FILARSZKY, GREINICH, HAZSLINSZKY, HOLUBY, KMET, MOESZ ap. MOESZ 17/a. p. 170.; BÁNHEGYI, FILARSZKY ap. MOESZ 13. p. 120.; UBRIZSY 2. p. 153.; TÓTH 2.

p. 58.; VASS—TÓTH 1. p. 158.; VASS 1. p. 51.; VASS—TÓTH 2. p. 48.; VASS—TÓTH 3. p. 85.; DOHY—KIRÁLY in UBRIZSY 5. p. 466.

Puccinia (urticae) caricis (SCHUM.) REBENTISCH f. sp. *urticae-pilosae* HASLER
VASS—TÓTH 2. p. 48.; see p. 108.

Puccinia caricis-montanae FISCHER
HAZSLINSZKY 7. p. 11.; SYDOW 2. I. p. 662.; MOESZ 30. p. 7; BÄUMLER, GÁYER, HAZSLINSZKY, HOLUBY, LINHART Fung. hung. exs. No. 425., MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 426. ap. MOESZ 17/a. p. 170.; SZÉPLIGETI ap. MOESZ 13. p. 20.; SAVULESCU 7. p. 800.

Puccinia carlinae JACKY
BÄUMLER 1. II. No. 613.; SYDOW 2. I. p. 35.; SAVULESCU 7. p. 1033.

Puccinia carniolica VOSS
SYDOW 2. I. p. 406. ap. MOESZ 17/a. p. 171.

Puccinia carthami (HUTZELM.) CORDA
HAZSLINSZKY 1. p. 153.; HAZSLINSZKY 2. p. 16.; HOLLÓS 1. p. 134.; HOLLÓS 6/a. p. 104.; HAZSLINSZKY, MÁGOCSY-DIETZ ap. MOESZ 17/a. p. 171.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 1035.

Puccinia caulincola SCHNEIDER
Syn.: P. schneideri SCHROET.
FUSS 1. IV. p. 111.; SACCARDO 1. No. 110. SYDOW 2. I. p. 302.; MOESZ 30. II. p. 373.; BÁNYAI, KMET ap. MOESZ 17/a. p. 171.; PICBAUER 2. p. 192.; SAVULESCU 7. p. 941.; DOHY—KIRÁLY in UBRIZSY 5. p. 463.

Puccinia celakowskyana BUBÁK
Syn.: P. punctata LINK ppe.
BÄUMLER 1. IV. No. 1496.; SYDOW 2. I. p. 216.; BÄUMLER, MOESZ ap. MOESZ 17/a. p. 171.; SAVULESCU 7. p. 953.; VASS—TÓTH 2. p. 48.; TÓTH 10. p. 9.; UBRIZSY 8. p. 369.

Puccinia centaureae DE CANDOLLE
BÄUMLER, HAYNALD, HAZSLINSZKY, HOLLÓS in Krypt. exs. No. 2306/a., b., KOSZILKOV, LAUDON, MOESZ, PÉNZES, RÖMER, SZÉPLIGETI, WAGNER ap. MOESZ 17/a. p. 172.; HAZSLINSZKY, KOSZILKOV, MOESZ ap. MOESZ 13. p. 120.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 1041.; TÓTH 2. p. 58.; VASS 1. p. 51.; VASS—TÓTH 2. p. 49.; TÓTH 10. p. 9.; UBRIZSY 8. p. 369.

Puccinia centaureae DE CANDOLLE f. sp. *nigrae* HASLER
LINHART in Fung. hung. exs. No. 427., MÁRTON ap. MOESZ 17/a. p. 172.; SAVULESCU 7. p. 1042.

Puccinia centaureae DE CANDOLLE f. sp. *scabiosae* HASLER
KALCHBRENNER 1. I. p. 309.; HAZSLINSZKY 1. p. 152.; HAZSLINSZKY 2. p. 16.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 83.; BÄUMLER 1. II. No. 613., V. No. 1838.; SYDOW 2. I. p. 40.; BUBÁK 1. No. 33.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 104.; MOESZ 4. II. p. 145.; MOESZ 11. p. 100.; HOLLÓS 7. p. 154.; BÄUMLER, GRESCHIK, HAZSLINSZKY, HOLUBY ap. MOESZ 17/a. p. 172.; SAVULESCU 7. p. 1042.

Puccinia cerasi (BÉRENG.) CASTAGNE

RAPAICS 1. p. 217.; SCHILBERSZKY 6. p. 163.; HOLLÓS 7. p. 154.; GREINICH in Flora Hung. exs. No. 201. ap. MOESZ 17/a. p. 173.; UBRIZSY 1. p. 13.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 484.; DOHY—KIRÁLY in UBRIZSY 5. p. 462.

Puccinia cesatii SCHROETER

KALCHBRENNER 1. I. p. 311.; HAZSLINSZKY 1. p. 162.; BÄUMLER 1. II. No. 603., IV. No. (603.); SYDOW 2. I. p. 723.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; RAPAICS 1. p. 213.; BÄUMLER, BUDAI, GREINICH, HOLUBY, KALCHBRENNER in RABENH. Fung. eur. exs. No. 595.; MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 314. ap. MOESZ 17/a. p. 173.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 120.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 782.; TÓTH 2. p. 58.; SZIGETI—TÓTH 1. p. 20.; UBRIZSY 8. p. 369.

Puccinia chaerophylli PURTON

Syn.: P. pimpinellae WINTER

KALCHBRENNER 1. I. p. 308.; HAZSLINSZKY 1. p. 150.; HAZSLINSZKY 7. p. 11.; RICHTER 1. No. 12.; BRESADOLA 1. No. 109.; BÄUMLER 1. II. No. 621. V. No. 1839.; LIRO 3. p. 14.; SYDOW 2. I. p. 367.; BÄUMLER, BORBÁS, GRE; SCHIK, HAZSLINSZKY, HOLUBY, MÁGÓCSY-DIETZ in Krypt. exs. No. 27.-No. 1121., MOESZ, RICHTER, SCHNELLER, SZÉPLIGETI ap. MOESZ 17/a. p, 173.; MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 120.; SAVULESCU 7. p. 883.; TÓTH 2. p. 58.; UBRIZSY 8. p. 369.

Puccinia chondrillina BUBÁK et SYDOW

HAZSLINSZKY 2. p. 17.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; HOLLÓS 7. p. 154.; GREINICH, HOLUBY, MOESZ in Flora hung. exs. No. 1., TAUSCHER ap. MOESZ 17/a. p. 174.; MOESZ ap. MOESZ 13. p. 120.; SAVULESCU 7. p. 1044.; LEHOCZKY 1. p. 237.; UBRIZSY 8. p. 369.

Puccinia chrysosplenii GREVILLE

BÄUMLER 1. II. No. 604; SYDOW 2. I. p. 493; HUSZ 1. p. 102.; BÄUMLER, KMET ap. MOESZ 17/a. p. 174.; SAVULESCU 7. p. 865.

Puccinia cichorii (DC.) BELLYNCK

HAZSLINSZKY 1. p. 154.; BÄUMLER 1. II. No. 613.; V. No. 1841.; SYDOW 2. I. p. 49.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; HOLLÓS 7. p. 154.; BÄUMLER, HAZSLINSZKY, LAUDON, MOESZ ap. MOESZ 17/a. p. 174.; MOESZ ap MOESZ 13 p 120 ; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 1047.; TÓTH 2. p. 58.; VASS 1. p. 51.; VÖRÖS 9. p. 102.; SZIGETI—TÓTH 1. p. 20.; TÓTH 10. p. 9.; DOHY—KIRÁLY in UBRIZSY 5. p. 462.; UBRIZSY 8. p. 369.

Puccinia circaeae PERSOON

FUSS 1. IV. p. 126.; BOLLA 1. p. 50.; HAZSLINSZKY 1. p. 167.; FUSS 2. p. 441.; HAZSLINSZKY 2. p. 19.; HAZSLINSZKY 3. p. 85.; BÄUMLER 1. II. No. 605., IV. No. (605.); SACCARDO 1. No. 114.; SYDOW 2. I. p. 423.; HOLLÓS 7. p. 154.; BÄUMLER, BOLLA, BUDAI, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KMET, LINHART in Fung. hung. exs. No. 21., MÁGOCSY-DIETZ, MOESZ, SABRANSKY, SZÉPLIGETI, WIERZBICKY ap. MOESZ 17/a. p. 174.; SAVULESCU

7. p. 877.; Tóth 2. p. 58.; Vass–Tóth 1. p. 158.; Vass 1. p. 51.; Ubrizsy 8. p. 369.

Puccinia cirsii Lasch

Kalchbrenner 1. I. p. 309.; Hazslinszky 1. p. 151.; Fuss 2. p. 440.; Hazslinszky 3. p. 83.; Hazslinszky 7. p. 12.; Richter 1. No. 18.; Bäumler 1. II. No. 613.; Sydow 2. I. p. 55.; Hollós 1. p. 135.; Moesz 11. p. 100.; Moesz 12. col. 807.; Hollós 7. p. 154.; Bäumler, Hazslinszky, Holuby, Kalchbrenner, Kmet, Mágocsy-Dietz, Moesz, Porcius in Linhart Fung. hung. exs. No. 225., Richter, Szépligeti in Linhart Fung. hung. exs. No. 321/a. ap. Moesz 17/a. p. 174.; Moesz, Szépligeti ap. Moesz 13. p. 120.; Picbauer 1. p. 12.; Savulęscu 7. p. 1004.; Tóth 2. p. 58.; Vass 1. p. 51.; Vass–Tóth 2. p. 49.; Tóth 10. p. 9.

Puccinia cirsii-eriophori Jacky

Syn.: P. cnici Martin ppe.

Moesz 12. col. 808.; Győrffy ap. Moesz 17/a. p. 175.; Picbauer 1. p. 12.; Savulescu 7. p. 1007.

Puccinia cirsii-lanceolati Schroeter

Syn.: P. cnici Martin ppe.

Hazslinszky 1. p. 151.; Hazslinszky 3. p. 83.; Hazslinszky 7. p. 12.; Bäumler 1. II. No. 613., V. No. 1843.; Sydow 2. I. p. 52.; Hollós 1. p. 135.; Hollós 6/a. p. 105.; Hollós 7. p. 154.; Bäumler, Hazslinszky, Holuby, Kmet, Laudon, Mágocsy-Dietz, Moesz ap. Moesz 17/a. p. 175.; Moesz ap. Moesz 13. p. 120.; Ubrizsy 8. p. 369.

Puccinia cnici Martin

Savulescu 7. p. 1006.; Tóth 2. p. 58.; Vass 1. p. 51.

Puccinia cnici-oleracei Persoon

Moesz ap. Moesz 17/a. p. 175.; Savulescu 7. p. 984.

Puccinia coaetanea Bubák

Fuss 2. p. 436.; Moesz 30. p. 7.; Moesz ap. Moesz 17/a. p. 176.; Picbauer 1. p. 10. Savulescu 7. p. 964.

Puccinia commutata Sydow

Linhart in Fung. hung. exs. No. 222. ap. Moesz 17/a. p. 176.; Savulescu 7. p. 948.

Puccinia conglomerata (Strauss) Kuntze et Schumacher

Magnus 1. p. 46.; Husz 1. p. 102.; Moesz 12. col. 808.; Greschik, Linhart in Fung. hung. exs. No. 122., Mágocsy-Dietz, Moesz in Flora hung. exs. No. 403. ap. Moesz 17/a. p. 176.; Picbauer 1. p. 14.; Picbauer 2. p. 192.; Savulescu 7. p. 1074.

Puccinia conii (Str.) Fuckel

Kalchbrenner 1. I. p. 308.; Hazslinszky 1. p. 150.; Richter 1. No. 12.; Sydow 2. I. p. 375.; Páter 2. p. 274.; Hollós 1. p. 137.; Hollós 6/a. p. 106.; Páter 3. p. 274.; Moesz 11. p. 100.; Hollós 7. p. 155.; Augustin, Hazslinszky, Kalchbrenner, Kálovits, Kmet, Kováts, Moesz, Páter ap. Moesz 17/a. p. 176.; Savulescu 7. p. 907.; Tóth 2. p. 58.

Puccinia convolvuli (PERS.) CASTAGNE

HAZSLINSZKY 1. p. 137., 160.; HAZSLINSZKY 2. p. 15., 18.; FUSS 2. p. 438.; HAZSLINSZKY 3. p. 85.; RICHTER 1. No. 17.; SYDOW 2. I. p. 320.; BUBÁK 1. No. 35.; MOESZ 21. p. 85.; BÄUMLER 1. V. No. 1844.; MOESZ 11. p. 101.; HOLLÓS 7. p. 155.; FUSS, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 422., MÁGOCSY-DIETZ in LINHART Fung hung exs. No. 421., MOESZ in Krypt. exs. No. 1119/b., RICHTER ap. MOESZ 17/a. p. 176.; MOESZ ap. MOESZ 13. p. 120.; PICBAUER 2. p. 188.; SAVULESCU 7. p. 919.; TÓTH 2. p. 58.; VASS – TÓTH 1. p. 158.; VASS 1. p. 51.; SZIGETI – TÓTH 1. p. 20.; UBRIZSY 8. p. 369.

Puccinia coronata CORDA

Syn.: Aecidium rhamni PERS.; Ae. frangulae SCHUM.; P. coronifera KLEB.; P. lolii NIELS

FUSS 1. VIII. p. 232., XVI. p. 24.; KALCHBRENNER 1. I. p. 303., 311.; HAZSLINSZKY 1. p. 136.; HAZSLINSZKY 2. p. 20.; FUSS 2. p. 437.; HAZSLINSZKY 3. p. 86.; HAZSLINSZKY 7. p. 13.; BORBÁS 3. p. 145.; BRESADOLA 1. No. 111.; SYDOW 2. I. p. 700.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 108.; MOESZ 12. col. 808.; HOLLÓS 7. p. 155.; BERNÁTSKY, FUSS, GRESCHIK, GYŐRFFY, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 329/a., LÜTKEMÜLLER in Krypt. exs. No. 29/b., MOESZ, SZÉPLIGETI ap. MOESZ 17/a. p. 177.; MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 120.; UBRIZSY 1. p. 13.; UBRIZSY 2. p. 153.; PICBAUER 2. p. 190.; UBRIZSY 6. in SZÉKESSY p. 23.; SAVULESCU 7. p. 740.; SZIGETI – TÓTH 1. p. 21.; HUSZ – KIRÁLY in UBRIZSY 5. p. 458.

Puccinia coronata CORDA f. *agrostis* ERIKSSON

MICHAELIS, MOESZ ap. MOESZ 17/a. p. 177.; SAVULESCU 7. p. 742.; UBRIZSY 8. p. 369.

Puccinia coronata CORDA f. *calamagrostis* ERIKSSON

BÄUMLER ap. MOESZ 17/a. p. 177.; SAVULESCU 7. p. 742.

Puccinia coronata CORDA f. *holci* KLEBAHN

BÄUMLER ap. MOESZ 17/a. p. 177.; SAVULESCU 7. p. 742.

Puccinia coronata CORDA f. *phalaridis* KLEBAHN

LINHART in Fung. hung. exs. No. 36. ap. MOESZ 17/a. p. 177.; SAVULESCU 7. p. 742.

Puccinia coronifera KLEBAHN

Syn.: P. coronata auct. ppe.; P. lolii SYD.

KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 136.; 169.; FUSS 1. VIII. p. 232., IV. p. 125.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 76.; BÄUMLER 1. II. No. 606., V. No. 1845.; SYDOW 2. I. p. 705.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 108.; RAPAICS 1. p. 213.; MOESZ 4. II. p. 145.; MOESZ 11. p. 101.; HOLLÓS 7. p. 157.; MOESZ 30. p. 7., II. p. 373.; BÄUMLER, BÁNHEGYI, BÁNYAI, BOLLA, DEGEN, GREINICH, GRESCHIK, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 230. I., MÁRTON, MOESZ, SCHNELLER, SZEPESFALVY, SZÉPLIGETI, WAGNER ap. MOESZ 17/a. p. 178.; DEGEN, HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ, SADLER, SZÉPLIGETI, TRAUTMANN ap. MOESZ 13. p. 121.;

UBRIZSY 1. p. 13.; SAVULESCU 7. p. 743.; TÓTH 2. p. 58.; VASS–TÓTH 1. p. 158.; VASS 1. p. 51.; HUSZ–KIRÁLY in UBRIZSY 5. p. 458.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 369.

Puccinia coronifera KLEBAHN f. *agropyri* ERIKSSON

GREINICH, HAZSLINSZKY, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 230. II. ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 747.

Puccinia coronifera KLEBAHN f. *alopecuri* ERIKSSON

MOESZ ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 746.; UBRIZSY 8. p. 369.

Puccinia coronifera KLEBAHN f. *arrhenatheri* KLEBAHN

BÄUMLER, BUDAI, MOESZ ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 747.

Puccinia coronifera KLEBAHN f. *avenae* ERIKSSON

BÁNHEGYI ap. MOESZ 1.; FUSS, HAZSLINSZKY, HOLLÓS, MÁGOCSY-DIETZ ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 745.

Puccinia coronifera KLEBAHN f. *bromi* MÜHLETHALER

BÄUMLER, BUDAI ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 747.

Puccinia coronifera KLEBAHN f. *epigaei* ERIKSSON

GREINICH ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 747.

Puccinia coronifera KLEBAHN f. *glyceriae* ERIKSSON

BÄUMLER, GREINICH, HUNYOR in Fung. hung. exs. No. 329/b.; MOESZ ap. MOESZ 17/a. p. 178.; SAVULESCU 7. p. 747.; TÓTH 2. p. 58.

Puccinia coronifera KLEBAHN f. *lolii* (NIELS) ERIKSSON

BÄUMLER, BUDAI, MOESZ ap. MOESZ 17/a. p. 179.; SAVULESCU 7. p. 746.; UBRIZSY 8. p. 369.

Puccinia crepidicola SYDOW

SYDOW 2. I. p. 71.; BÄUMLER, MOESZ , WIERZBICKY ap. MOESZ 17/a. p. 179.; MOESZ ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 1030.; UBRIZSY 8. p. 369.

Puccinia crepidis SCHROETER

HAZSLINSZKY 1. p. 152.; HAZSLINSZKY 5. p. 43.; BÄUMLER 1. II. No. 613.; SYDOW 2. I. p. 64.; HOLLÓS 7. p. 155.; BÄUMLER, MÁRKUS, MOESZ ap. MOESZ 17/a. p. 180.; MOESZ ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 1015.; TÓTH 2. p. 58.

Puccinia cruciferarum RUDOLPHI

FUSS 2. p. 441.; SYDOW 2. I. p. 510.; WAGNER ap. MOESZ 17/a. p. 180.; SAVULESCU 7. p. 863.

Puccinia cyani (SCHLEICH.) PASSERINI

HAZSLINSZKY 1. p. 152.; HOLLÓS 1. p. 134.; HOLLÓS 6/a. p. 104.; HAZSLINSZKY, MOESZ ap. MOESZ 17/a. p. 180.; MOESZ ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 1037.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 58.; UBRIZSY 8. p. 369.

Puccinia cynodontis DESMAZIÈRES

Syn.: Aecidium plantaginis CES.

HAZSLINSZKY 1. p. 131.; BORBÁS 2. p. 33.; BÄUMLER 1., II. No. 607.; IV. No. (607); BUBÁK 1. No. 36.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; SYDOW 2. IV. p. 310.; MOESZ 11. p. 101.; BÄUMLER, GREINICH, HAZSLINSZKY, LINHART in Fung. hung. exs. No. 141., No. 313., MOESZ, PÉNZES, TUZSON

ap. MOESZ 17/a. p. 180.; BUBÁK, HAZSLINSZKY, MOESZ, PÉNZES, SADLER, TUZSON ap. MOESZ 13. p. 121.; PICBAUER 1. p. 11.; PICBAUER 2. p. 190.; SAVULESCU 7. p. 764.; TÓTH 2. p. 58.; UBRIZSY 8. p. 370.

Puccinia dactylidina BUBÁK

BUBÁK 1/a. p. 219.; BÄUMLER ap. MOESZ 17/a. p. 181.; DOHY—KIRÁLY in UBRIZSY 5. p. 471.

Puccinia dentariae (ALB. et SCHW.) FUCKEL

HAZSLINSZKY 1. p. 164.; SYDOW 2. I. p. 512.; HAZSLINSZKY, PÉNZES ap. MOESZ 17/a. p. 181.; PÉNZES, VAJDA ap. MOESZ 13. p. 121.; PICBAUER 2. p. 192.; VASS—TÓTH 1. p. 158.; VASS—TÓTH 3. p. 85.

Puccinia depauperans (VIZE) SYDOW

HOLLÓS 7. p. 155. ap. MOESZ 17/a. p. 181.

Puccinia dictyoderma LINDROTH

BÄUMLER 1. II. No. 632.; BÄUMLER 5. p. 328.; LINDROTH 1. p. 8.; LIRO 3. p. 11.; BÄUMLER, BOROS, MOESZ in Flora hung. exs. No. 606., PÉNZES, SABRANSKY ap. MOESZ 17/a. p. 181.

Puccinia dioicae MAGNUS

KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 140.; SYDOW 2. I. p. 654. ap. MOESZ 17/a. p. 181.

Puccinia dispersa ERIKSSON

Syn.: P. rubigo-vera (DC.) SCHRÖTER ppe.

KALCHBRENNER 1. I. p. 304.; HAZSLINSZKY 1. p. 138.; FUSS 2. p. 436., 440.; BÄUMLER 1. II. No. 628.; SIMONKAI 1. No. 1658., 1728.; SYDOW 2. I. p. 710.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; RAPAICS 1. p. 212.; BÄUMLER 1. V. No. 1846.; HOLLÓS 7. p. 155.; BÄUMLER, BUDAI, FILARSZKY, FLEISCHMANN, HAZSLINSZKY, HOLUBY, KMET, KOSZILKOV, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ, TUZSON—BUBÁK in Krypt. exs. No. 2104. ap. MOESZ 17/a. p. 181.; MOESZ, KOSZILKOV, TUZSON ap. MOESZ 13. p. 121.; UBRIZSY 1. p. 13.; PICBAUER 1. p. 11.; SAVULESCU 7. p. 711.; HUSZ—KIRÁLY in UBRIZSY 5. p. 457.; UBRIZSY 8. p. 370.

Puccinia divergens BUBÁK

Syn.: P. carlinae JACKY ppe.

BÄUMLER 1. II. No. 613., V. No. 1837.; HOLLÓS 1. p. 134.; HOLLÓS 6/a. p. 104.; MOESZ 2. No. 67.; MOESZ 11. p. 100.; BÄUMLER, KMET, MOESZ ap. MOESZ 17/a. p. 182.; MOESZ, SZEPESFALVY ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 1034.

Puccinia doronicella SYDOW

HAZSLINSZKY 1. p. 164.; FUSS 2. p. 441.; BUBÁK 1. No. 37.; MOESZ ap. MOESZ 17/a. p. 182.; SAVULESCU 7. p. 1050.

Puccinia drabae RUDELPHI

Syn.: P. drabae HAZSL.; P. hazslinszkyi DE TONI

HAZSLINSZKY 1. p. 148.; SYDOW 2. I. p. 513.; BUBÁK 1. No. 38.; MOESZ 2. No. 70.; MOESZ 12. col. 808.; DEGEN in Flora Hung. exs. No. 404., FILARSZKY, HOLUBY, JÁVORKA, KÜMMERLE, MOESZ ap. MOESZ 17/a. p. 182.;

DEGEN, FILARSZKY, JÁVORKA, KÜMMERLE, MOESZ ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 864.

Puccinia dracunculina FAHRENDORFF

SAVULESCU 7. p. 995.; UBRIZSY 8. p. 370.

Puccinia echinopsis DE CANDOLLE

HAZSLINZSKY 1. p. 153.; HAZSLINSZKY 2. p. 16.; HAZSLINSZKY 3. p. 83.; HAZSLINSZKY 5. p. 43.; HAZSLINSZKY 11. p. 362.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; MOESZ 11. p. 101.; HOLLÓS 7. p. 155.; MOESZ 31. p. 96.; BERNÁTSKY, BUDAI, HAZSLINSZKY, JÁVORKA, KMET, LAUDON, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI, ZSÁK ap. MOESZ 17/a. p. 183.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 1052.; TÓTH 2. p. 59.; UBRIZSY 8. p. 370.

Puccinia epilobii DE CANDOLLE

SYDOW 2. I. p. 428.; LENGYEL in Krypt. exs. No. 1405. ap. MOESZ 17/a. p. 183.; SAVULESCU 7. p. 881.; VASS–TÓTH 2. p. 49.

Puccinia epilobii-tetragoni (DC.) WINTER

Syn.: Puccinia pulverulenta GREVILLE

HAZSLINSZKY 3. p. 75.; RICHTER 1. No. 20.; BÄUMLER 1. II. No. 608.; BUBÁK 1. No. 39.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 106.; HOLLÓS 7. p. 156.; GRESCHIK, HAYNALD, HOLUBY, MOESZ, RICHTER, SABRANSKY ap. MOESZ 17/a. p. 183.; SAVULESCU 7. p. 879.; DOHY–KIRÁLY in UBRIZSY 5. p. 464.; UBRIZSY 8. p. 370.; TÓTH 2. p. 60.; VASS 1. p. 52.

Puccinia eryngii DE CANDOLLE

HAZSLINSZKY 3. p. 83.; LIRO 3. p. 38.; SYDOW 2. I. p. 379.; HOLLÓS 7. p. 156.; HAZSLINSZKY ap. MOESZ 17/a. p. 184. SAVULESCU 7. p. 889.; TÓTH 2. p. 59.

Puccinia expansa LINK

SYDOW 2. I. p. 146. ap. MOESZ 17/a. p. 184.

Puccinia extensicola PLOWRIGHT

MOESZ ap. MOESZ 17/a. p. 184.; SAVULESCU 7. p. 797.

Puccinia falcariae (PERS.) FUCKEL

Syn.: Aecidium falcariae PERS.

SADLER 1. V. No. 25.; ENDLICHER 1. No. 80.; FUSS 1. IV. p. 125.; KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 132., 150.; FUSS 2. p. 438.; BORBÁS 3. p. 145.; RICHTER 1. No. 67.; BÄUMLER 1. II. No. 630.; V. No. 1848.; BRESADOLA 1. No. 123.; SIMONKAI 1. No. 1651.; SYDOW 2. I. p. 380.; MOESZ 2. No. 71.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 106.; BARTH, BÄUMLER, BERNÁTSKY, BUDAI, FILARSZKY, FUSS, GRESCHIK, GREINICH, HAZSLINSZKY, HOLUBY, JÁVORKA, KMET, KOVÁCS, LAUDON, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 123., MÁRKUS, MOESZ, MÜLLER, PÉNZES, SCHILBERSZKY in Krypt. exs. No. 916.; SIMKOVITS, SZEPESFALVY, SZÉPLIGETI ap. MOESZ 17/a. p. 184.; BERNÁTSKY, HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ, MÜLLER, SCHILBERSZKY, SIMKOVITS, SZÉPLIGETI ap. MOESZ 13. p. 121.; UBRIZSY 1. p. 13.; UBRIZSY 6. in SZÉKESSY p. 23.; SAVULESCU 7. p. 896.; TÓTH 2. p. 59.; VASS 1. p. 51.; UBRIZSY 8. p. 370.

Puccinia festucae PLOWRIGHT

Syn.: Aecidium periclymeni SCHUM.

KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 135.; RICHTER 1. No. 48.; SYDOW 2. I. p. 753.; MOESZ 12. col. 808.; GRESCHIK, LENGYEL, RICHTER, TUZSON ap. MOESZ 17/a. p. 185.; PICBAUER 2. p. 190.; SAVULESCU 7. p. 749.; DOHY–KIRÁLY in UBRIZSY 5. p. 471.

Puccinia festucina SYDOW

Syn.: Aecidium muscari LINHART

SAVULESCU 7. p. 724.; UBRIZSY 8. p. 370.

Puccinia fuckelii SYDOW

Syn.: Puccinia jurineae Tranzschel non COOKE

HOLLÓS 7. p. 156.; et ap. MOESZ 17/a. p. 185.; PICBAUER 1. p. 13.; PICBAUER 2. p. 192.; SAVULESCU 7. p. 1075.

Puccinia galanthi UNGER

HAZSLINSZKY 1. p. 155.; HAZSLINSZKY 3. p. 87.; SYDOW 2. I. p. 606.; BÄUMLER 1. V. No. 1850.; LINHART in Fung. hung. exs. No. 24. et in RABENH.–WINTER Fung. europ. exs. No. 2707., SCHNELLER ap. MOESZ 17/a. p. 186.; DOHY–KIRÁLY in UBRIZSY 5. p. 464.

Puccinia galii (PERS.) WINTER

Syn.: P. galiorum LINK; P. punctata LINK ppe.

BOLLA 1. II. No. 610.; KALCHBRENNER 1. I. 303., 308.; HAZSLINSZKY 1. p. 137., 156.; HAZSLINSZKY 2. p. 17.; FUSS 2. p. 440.; HAZSLINSZKY 3. p. 77.; BÄUMLER 1. II. No. 610.; BRESADOLA 1. No. 105.; SYDOW 2. I. p. 244.; MOESZ 2. No. 78.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 101.; HOLLÓS 7. p. 161.; MOESZ 31. p. 96.; BÄUMLER, BUDAI, GÁYER, GREINICH, HAZSLINSZKY, HOLUBY, KMET, LINHART in Fung. hung exs. No. 124., MÁGOCSY-DIETZ, MOESZ, PÓSCH ap. MOESZ 17/a. p. 186.; MOESZ ap. MOESZ 13. p. 121.; SAVULESCU 7. p. 956.; UBRIZSY 8. p. 370.

Puccinia galii-silvatici OTTH

FUSS 1. IV. No. 111.; HAZSLINSZKY 1. p. 137–156.; FUSS 2. p. 436.; BORBÁS 2. p. 33.; BRESADOLA 1. No. 105.; BUBÁK 1. No. 40.; HUSZ 1. p. 102.; BÄUMLER, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, KŐFARAGÓ-GYELNIK, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 17/a. p. 186.; KŐFARAGÓ-GYELNIK, MOESZ ap. MOESZ 13. p. 121.; PICBAUER 1. p. 11.; PICBAUER 2. p. 188.; SAVULESCU 7. p. 959.; TÓTH 2. p. 59.; VASS–TÓTH 2. p. 49.; UBRIZSY 8. p. 370.

Puccinia gentianae (STRAUSS) MARTIN

FUSS 1. IV. p. 111.; BOLLA 1. p. 50.; KALCHBRENNER 1. I. p. 310.; HAZSLINSZKY 1. p. 161.; FUSS 2. p. 441.; BÄUMLER 1. II. No. 611., III. No. 1396.; SYDOW 2. I. p. 341.; MOESZ 2. No. 72.; MAGNUS 1. p. 45.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, BOLLA, BUDAI, GRESCHIK, HAYNALD, HAZSLINSZKY, JÁVORKA, KALCHBRENNER in Fung. europ. exs. No. 887., MÁGOCSY-DIETZ, RÖHMER in LINHART Fung. hung. exs. No. 424., SZÉPLIGETI, WIERZBICKY, ZAHLBRUCKNER in Krypt. exs. No. 1118. ap. MOESZ 17/a. p. 187.;

JÁVORKA ap. MOESZ 13. p. 122.; SAVULESCU 7. p. 944.; TÓTH 2. p. 59.; UBRIZSY 8. p. 370.

Puccinia gigantea KARSTEN

TUZSON—BUBÁK in Krypt. exs. No. 2005. ap. MOESZ 17/a. p. 188.

Puccinia glechomatis DE CANDOLLE

KALCHBRENNER 1. I. p. 310.; HAZSLINSZKY 1. p. 158.; HAZSLINSZKY 2. p. 18.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 84.; MÄUMLER 1. II. No. 640., V. No. 1852.; SACCARDO 1. No. 115.; SYDOW 2. I. p. 277.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 101.; HOLLÓS 7. p. 156.; ACÉL, BÄUMLER, GREINICH, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI, VIDA—LINHART in LINHART Fung. hung. exs. No. 215. ap. MOESZ 17/a. p. 188.; HAZSLINSZKY, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 122.; SAVULESCU 7. p. 922.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 59.; VASS—TÓTH 1. p. 59.; VASS 1. p. 51.; UBRIZSY 8. p. 370.

Puccinia glumarum (SCHMIDT) ERIKSSON et HENNINGS

SYDOW 2. I. p. 707.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; RAPAICS 1. p. 212.; HUSZ 3/b. p. 81.; TAUSCHER ap. MOESZ 17/a. p. 188.; HUSZ ap. MOESZ 13. p. 122.; UBRIZSY 1. p. 13.; SAVULESCU 7. p. 734.; SZIGETI—TÓTH 1. p. 21.; HUSZ—KIRÁLY in UBRIZSY 5. p. 455.

Puccinia graminis PERSOON

Syn.: Aecidium berberidis GMEL.

ENDLICHER 1. No. 72., 73., 82., 89.; FUSS 1. IV. p. 125., VIII. p. 232.; KALCHBRENNER 1. I. p. 303., 311.; SCHULZER 1. No. 37., 58.; SCHULZER 2. p. 29.; SCHULZER 6. p. 129.; HAZSLINSZKY 1. p. 135., 169.; FUSS 2. p. 436.; HAZSLINSZKY 2. p. 20.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 76.; BORBÁS 3. p. 145.; RICHTER 1. No. 24.; BÄUMLER 1. II. No. 612., V. No. 1849.; BRESADOLA 1. No. 110.; SIMONKAI 1. No. 1650., 1727.; ERIKSSON 1.; LINHART 7.; SYDOW 2.; BUBÁK 1. No. 41.; MOESZ 2. No. 73.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 108.; MAGNUS 1. p. 45.; RAPAICS 1. p. 212.; VARGA 2.; MOESZ 11. p. 101.; MOESZ 12. col. 808.; MOESZ 20.; MOESZ 32.; SCHILBERSZKY 7.; SCHILBERSZKY 8.; HUSZ 3/a.; HUSZ 3/b; HUSZ 3/d; HOLLÓS 7. p. 156.; BÄUMLER, BOLLA, BUDAI, GRESCHIK, HAYNALD, HAZSLINSZKY, HOLUBY, KÜMMERLE, LINHART in Fung. hung. exs. No. 126., MÁGOCSY-DIETZ, MÁRTON, MOESZ, PÓSCH, RICHTER, SCHILBERSZKY, STAUB, SZÉPLIGETI ap. MOESZ 17/a. p. 189.; HAYNALD, KŐFARAGÓ-GYELNIK, MÁGOCSY-DIETZ, MOESZ, RICHTER, SCHILBERSZKY, STAUB, SZÉPLIGETI ap. MOESZ 13. p. 122.; UBRIZSY 1. p. 13.; PICBAUER 1. p. 11.; PICBAUER 2. p. 189.; SAVULESCU 7. p. 754.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 59.; VASS 1. p. 51.; VASS—TÓTH 2. p. 49.; TÍMÁR 1 p 237 ; SZIGETI-TÓTH 1. p. 21.; UBRIZSY 8. p. 370.

Puccinia graminis PERSOON f. sp. *agrostis* ERIKSSON et HENNINGS

MOESZ ap. MOESZ 17/a. p. 189.; SAVULESCU 7. p. 760.

Puccinia graminis PERSOON f. sp. *avenae* ERIKSSON et HENNINGS

BÄUMLER, BOLLA, BUDAI, HAZSLINSZKY, MÁGOCSY-DIETZ, MÁRTON,

MOESZ, SABRANSKY ap. MOESZ 17/a. p. 189.; MOESZ ap. MOESZ 13. p. 122.; SAVULESCU 7. p. 759.

Puccinia graminis PERSOON f. sp. *poae* ERIKSSON et HENNINGS

BÄUMLER, MOESZ, PÉNZES ap. MOESZ 17/a. p. 190.; MOESZ, PÉNZES ap. MOESZ 13. p. 122.; SAVULESCU 7. p. 760.

Puccinia graminis PERSOON f. sp. *secalis* ERIKSSON et HENNINGS

BARTH, BÄUMLER, BOLLA, BUDAI, FUSS, GREINICH, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, LÁNYI, LINHART in Fung. hung. exs. No. 34., MOESZ, PÉNZES, PÓSCH, SZÉPLIGETI, ZÓLYOMI ap. MOESZ 17/a. p. 190.; MÁGOCSY-DIETZ, MOESZ, PÉNZES, SZÉPLIGETI ap. MOESZ 13. p. 122.; SAVULESCU 7. p. 756.; UBRIZSY 8. p. 370.

Puccinia graminis PERSOON f. sp. *tritici* ERIKSSON et HENNINGS

BÄUMLER, BUDAI, DEGEN, GREINICH, HAZSLINSZKY, KMET, MÁGOCSY-DIETZ, MOESZ, VARGA ap. MOESZ 17/a. p. 190.; SAVULESCU 7. p. 759.

Puccinia grisea (STRAUSS) WINTER

Syn.: P. globulariae DC.

HAZSLINSZKY 1. p. 159.; BORBÁS 2. p. 53.; BÄUMLER 1. V. No. 1853.; DEGEN, MOESZ, SADLER ap. MOESZ 17/a. p. 191.; DEGEN, MOESZ, SADLER ap. MOESZ 13. p. 122.; PICBAUER 1. p. 13.; PICBAUER 2. p. 192.; VÖRÖS 9. p. 102.

Puccinia helianthi SCHWEINITZ

HAZSLINSZKY 1. p. 155.; HAZSLINSZKY 2. p. 17.; HAZSLINSZKY 3. p. 84.; FUSS 2. p. 441.; HAZSLINSZKY 7. p. 12.; RICHTER 1. No. 19.; SIMONKAI 1. No. 1729.; BÄUMLER 1. III. No. 1397.; SACCARDO 1. No. 98.; SYDOW 2. I. p. 92.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; RAPAICS 1. p. 214.; MOESZ 21. p. 85.; HOLLÓS 7. p. 157.; BARTH in THUEMEN Myc. Univ. No. 549/b; BÄUMLER, BUDAI, CHYZER, GRESCHIK, GYŐRFFY, HAZSLINSZKY, KMET, KRENNER, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 227.; MOESZ, RICHTER, TAUSCHER in RABENH. Fungi eur. exs. No. 1586–1857., in THUEMEN Fung. aust. exs. No. 369., in THUEMEN Herb. Mycol. oecon. No. 10. et in F. SCHULTZ Herb. Norm. Nov. Ser. Cent. 7. No. 695. ap. MOESZ 17/a. p. 191.; MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 122.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 1001.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 59.; VASS–TÓTH 1. p. 158.; VASS 1. p. 51.; DOHY–KIRÁLY in UBRIZSY 5. p. 460.; UBRIZSY 8. p. 371.

Puccinia helvetica SCHROETER

SYDOW 2. I. p. 209.; LINHART in Fung. hung. exs. No. 416. ap. MOESZ 17/a. p. 192.; SAVULESCU 7. p. 967.

Puccinia heraclei GREVILLE

PICBAUER 2. p. 189.; SAVULESCU 7. p. 890.

Puccinia herniariae UNGER

PICBAUER 1. p. 13.; PICBAUER 2. p. 192.; SAVULESCU 7. p. 851.

Puccinia hieracii (SCHUM.) MARTIUS

KALCHBRENNER 1. I. p. 309.; HAZSLINSZKY 1. p. 152.; HAZSLINSZKY 2. p. 15.; FUSS 2. p. 440.; BÄUMLER 1. II. No. 613.; BRESADOLA 1. No. 116.; SYDOW

2. I. p. 96.; BUBÁK 1. No. 42.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; HUSZ 1. p. 102.; HOLLÓS 7. p. 157.; MOESZ 12. col. 808.; BUDAI, BÄUMLER, GYŐRFFY, HAZSLINSZKY, HOLUBY, KALCHBRENNER, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI, WIERZBICKY, ZAHLBRUCKNER ap. MOESZ 17/a. p. 192.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 123.; PICBAUER 1. p. 12.; PICBAUER 2. p. 191;. SAVULESCU 7. p. 1053.; TÓTH 2. p. 59.; VASS—TÓTH 1. p. 159.; VASS 1. p. 51.; VASS—TÓTH 2. p. 49. UBRIZSY 8. p. 371.

Puccinia holcina ERIKSSON

SYDOW 2. I. p. 715. ap. MOESZ 17/a. p. 193; SAVULESCU 7. p. 731.; DOHY—KIRÁLY in UBRIZSY 5. p. 471.

Puccinia hordei-murini BUCHWALD

SAVULESCU 7. p. 732.; UBRIZSY 8. p. 371.

Puccinia hypochaeridis OUDEMANS

Syn.: P. hyoseridis (SCHUM.) LIRO

RICHTER 1. No. 18.; SYDOW 2. I. p. 100.; HUSZ 1. p. 102.; RICHTER—HAZSLINSZKY, SZÉPLIGETI—MOESZ ap. MOESZ 17/a. p. 193.; SZÉPLIGETI ap. MOESZ 13. p. 123.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 1058.

Puccinia iridis (DC.) WALLROTH

HAZSLINSZKY 1. p. 167.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 82.; BÄUMLER 1. II. No. 614.; SYDOW 2. I. p. 599.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 107.; HOLLÓS 7. p. 157.; BÄUMLER, GREINICH, HOLUBY in LINHART Fung. hung. exs. No. 315., KRENNER, MAGYAR in Krypt. exs. No. 915., MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 219., PÉTERFI ap. MOESZ 17/a. p. 193.; KRENNER, MAGYAR, MÁGOCSY-DIETZ, SADLER ap. MOESZ 13. p. 123.; SAVULESCU 7. p. 824.; TÓTH 2. p. 59.; DOHY—KIRÁLY in UBRIZSY 5. p. 468.

Puccinia jaceae OTTH

KMET, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 17/a. p. 193.; SZÉPLIGETI ap. MOESZ 13. p. 123.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 1039.; VASS 1. p. 51.

Puccinia jueliana DIETEL

GREISIGER, GYŐRFFY ap. MOESZ 17/a. p. 194.

Puccinia jurineae COOKE non TRANZSCHEL.

Syn.: P. fuckelii SYD. ppe.

HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105. ap. MOESZ 17/a. p. 194.

Puccinia komarovi TRANZSCHEL

MOESZ ap. MOESZ 17/a. p. 194.; MOESZ ap. MOESZ 13. p. 123.; MOESZ 43. p. 101.

Puccinia lactucarum SYDOW

HAZSLINSZKY 1. p. 153.; HAZSLINSZKY 2. p. 17.; FUSS 2. p. 440.; BÄUMLER 1. II. No. 625.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; MOESZ 12. col. 808.; BÄUMLER, BUDAI, DEGEN, FUSS, HAZSLINSZKY, MOESZ, SCHNELLER, VAJDA, VARGA ap. MOESZ 17/a. p. 194.; DEGEN, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 123.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 1010.; TÓTH 2. p. 59.; VASS 1. p. 51.

Puccinia lapsanae (SCHULTZ.) FUCKEL

HAZSLINSZKY 2. p. 16.; HAZSLINSZKY 3. p. 84.; BRESADOLA 1. No. 107.; BÄUMLER 1. IV. No. 1497.; SYDOW 2. I. p. 113.; MOESZ 2. No. 74.; BUBÁK 1. No. 43.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; MAGNUS 1. p. 45.; HOLLÓS 7. p. 157.; MOESZ 30. II. p. 373.; MOESZ 31. p. 96.; BÄUMLER, BÁNYAI, BUDAI, GÁYER, GRESCHIK, JÁVORKA, KMET, LAUDON, MÁGOCSY-DIETZ, MOESZ, PÉNZES, SZÉPLIGETI ap. MOESZ 17/a. p. 194.; JÁVORKA, MOESZ, PÉNZES, SZÉPLIGETI ap. MOESZ 13. p. 123.; SAVULESCU 7. p. 1020.; TÓTH 2. p. 59.; VASS—TÓRH 1. p. 159.; VASS 1. p. 51.; TÓTH 10. p. 10.; VÖRÖS 9. p. 102.; UBRIZSY 8. p. 371.

Puccinia laserpitii LINDER

PICBAUER 1. p. 10.; PICBAUER 2. p. 189.

Puccinia leontodontis JACKY

HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 105.; SYDOW 2. I. p. 115.; HOLLÓS 7. p. 157.; MOESZ ap. MOESZ 17/a. p. 195.; BORBÁS, MOESZ ap. MOESZ 13. p. 123.; SAVULESCU 7. p. 1062.; VASS 1. p. 51.

Puccinia libanotidis LINDROTH

FUSS 1. VIII. p. 23.; FUSS 2. p. 440.; BUBÁK 1. No. 44.; GRESCHIK ap. MOESZ 17/a. p. 195.; SAVULESCU 7. p. 914.

Puccinia liliacearum DUBY

BOLLA 1. p. 50.; BÄUMLER 1. II. No. 615.; SYDOW 2. I. p. 628.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 107.; HOLLÓS 7. p. 157.; MOESZ 31. p. 96.; BÄUMLER, BERNÁTSKY, GÁYER, HAZSLINSZKY, LINHART in Fung. hung. exs. No. 29., MÁGOCSY-DIETZ in Krypt. exs. No. 38.; SCHMIDT, SCHNELLER, TUZSON, ZAHLBRUCKNER ap. MOESZ 17/a. p. 195.; BERNÁTSKY, HAZSLINSZKY, MÁGOCSY-DIETZ, TUZSON ap. MOESZ 13. p. 123.; PICBAUER 1. p. 159.; SAVULESCU 7. p. 819.; VASS—TÓTH 3. p. 85.; DOHY—KIRÁLY in UBRIZSY 5. p. 464.

Puccinia limosae MAGNUS

HAZSLINSZKY 3. pp. 85—86. ap. MOESZ 17/a. p. 195.; SAVULESCU 7. p. 789.

Puccinia linosyridis-caricis FISCHER

PICBAUER 1. p. 11.

Puccinia lojkaiana THÜMEN

Syn.: P. ornithogali HAZSL.

THÜMEN 1. p. 183.; HAZSLINSZKY 7. pp. 12—13.; SYDOW 2. I. p. 630.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 100., 107.; BÄUMLER 1. V. No. 1855.; HOLLÓS 7. p. 157.; BÄUMLER in Krypt. exs. No. 1127., FILARSZKY, KÜMMERLE in Flora hung. exs. No. 707.; LINHART in Fung. hung. exs. No. 117.; MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 415., MOESZ, SZÉPLIGETI ap. MOESZ 17/a. p. 195.; FILARSZKY, KÜMMERLE, LINHART, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 123.; PICBAUER 1. p. 13.; SAVULESCU 7. p. 820.; SZIGETI—TÓTH 1. p. 21.

Puccinia longissima SCHROETER

Syn.: Uredo sedi DC.; Endophyllum sedi LÉV.

BÄUMLER 1. II. No. 658.; SYDOW 2. I. p. 758.; BÄUMLER in RABENH.—WINTER Fung. eur. exs. No. 3128/b. ap. MOESZ 17/a. p. 196.; DOHY—KIRÁLY in UBRIZSY 5. p. 468.

Puccinia lycoctoni FUCKEL

HAZSLINSZKY 3. p. 77.; SYDOW 2. I. 537.; MOESZ 17/a. p. 196.; MÁGOCSY-DIETZ ap. MOESZ 13. p. 124.

Puccinia magnusiana KÖRNICKE

HAZSLINSZKY 3. p. 86.; BÄUMLER 1. II. No. 616.; SYDOW 2. I. p. 786.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; MOESZ 11. p. 101.; HOLLÓS 7. p. 158.; BARTH, BÄUMLER in Krypt. exs. No. 1305., BÁNHEGYI, BUDAI, GREINICH, HAYNALD, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MÁRTON, MOESZ in Flora hung. exs. No. 2., PÉNZES, SZÉPLIGETI ap. MOESZ 17/a. p. 196.; MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 124.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 752.; TÓTH 2. p. 59.; DOHY—KIRÁLY in UBRIZSY 5. p. 469.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 371.

Puccinia major DIETEL

HUSZ 1. p. 102. ap. MOESZ 17/a. p. 197.; SAVULESCU 7. p. 1017.

Puccinia malvacearum MONTAGNE

HOLUBY 1. p. 164.; HOLUBY 2. p. 351., 389.; HAZSLINSZKY 2. p. 19.; MIKA 3. p. 138.; HAZSLINSZKY 3. p. 87.; BÄUMLER 1. II. No. 617.; SIMONKAI 1. No. 1726.; PÁTER 2. p. 200.; SYDOW 2. I. p. 477.; PÁTER 3. p. 158.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 106.; GRÓF 1. p. 271.; MAGNUS 1. p. 46.; RAPAICS 1. p. 218.; MOESZ 4. p. 145.; MOESZ 11. p. 101.; MOESZ 12. col. 808.; MOESZ 21 p. 85.; HOLLÓS 7. p. 158.; MOESZ 30. II. p. 373.; MOESZ 31. p. 96.; AUGUSTIN, BARTH, BÄUMLER, BÁNYAI, BERNÁTSKY, BORBÁS, BUDAI, FANTA, FILARSZKY, FUSS, GÁYER, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLUBY, HORVÁTH, KMET, KÜMMERLE, MARGITTAI, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 22., MÁRTON, MOESZ, PÓSCH, SZÉPLIGETI, WAGNER, ZAHLBRUCKNER ap. MOESZ 17/a. p. 197.; MÁGOCSY-DIETZ, TOMEK, SZÉPLIGETI ap. MOESZ 13. p. 124.; UBRIZSY 1. p. 14.; KRENNER 1. p. 64.; SAVULESCU 7. p. 870.; TÓTH 2. p. 59.; TIMÁR 1. p. 237.; SZIGETI—TÓTH 1. p. 21.; DOHY—KIRÁLY in UBRIZSY 5. p. 467.; UBRIZSY 8. p. 371.

Puccinia maydis BÉRENGER

Syn.: P. sorghi SCHWEINITZ; Aecidium oxalidis THÜM.

HAZSLINSZKY 1. p. 162.; FUSS 2. p. 441.; HAZSLINSZKY 3. p. 86.; RICHTER 1. No. 11.; BÄUMLER 1. II. No. 84., V. No. 1856.; SYDOW 2. I. p. 831.; HOLLÓS 1. p. 140.; HOLLÓS 6/a. p. 109.; RAPAICS 1. p. 213.; MOESZ 11. p. 101.; MOESZ 21. p. 85.; HOLLÓS 7. p. 158.; BÄUMLER in Krypt. exs. No. 1123.; BUDAI, HAZSLINSZKY, HOLUBY, KRENNER, LAUDON, LINHART in Fung. hung. exs. No. 27., MÁGOCSY-DIETZ, MÁRKUS, MÁRTON, MOESZ, PÓSCH, RICHTER, SCHILBERSZKY ap. MOESZ 17/a. p. 198.; MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 124.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 763.; HUSZ—KIRÁLY in UBRIZSY 5. p. 459.; UBRIZSY 8. p. 371.; TÓTH 2. p. 60.

Puccinia mamillata SCHRÖTER

PICBAUER 3. p. 178.

Puccinia melicae (ERIKSS.) SYDOW

BÄUMLER ap. MOESZ 17/a. p. 199.; SAVULESCU 7. p. 750.

Puccinia microsora KÖRNICKE

HAZSLINSZKY 3. p. 86. ap. MOESZ 17/a. p. 200.

Puccinia menthae PERSOON

Syn.: Uredo origani HAZSL.

ENDLICHER 1. No. 70.; BOLLA 1. p. 50.; KALCHBRENNER 1. I. p. 310.; HAZSLINSZKY 1. p. 157.; HAZSLINSZKY 2. p. 17., 18.; FUSS 2. p. 441.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 84.; BORBÁS 3. p. 145.; RICHTER 1. No. 16.; BÄUMLER 1. II. No. 618.; SACCARDO 1. No. 103.; SYDOW 2. I. p. 284.; IV. p. 566.; PÁTER 3. p. 158.; BUBÁK 1. No. 46.; MOESZ 2. No. 75.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; GRÓF 2. p. 657.; MOESZ 11. p. 101.; MOESZ 12. col. 808.; MOESZ 21. p. 85.; MOESZ 32. p. 14.; HOLLÓS 7. p. 158.; AUGUSTIN, BÄUMLER, BOLLA, BUDAI, GREINICH, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLUBY, HORVÁTH, HULJÁK, KMET, LÁNYI, LINHART in Fung. hung. exs. No. 30., 419., 420., MÁGOCSY-DIETZ, MÁRTON, MOESZ, PÁTER, PERLAKY, RICHTER, SABRANSKY, SZÉPLIGETI, SZURÁK, WIERZBICKY ap. MOESZ 17/a. p. 199.; AUGUSTIN, FILARSZKY, HAZSLINSZKY, KÜMMERLE, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 124.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 928.; TÓTH 2. p. 59.; VASS 1. p. 51.; DOHY—KIRÁLY in UBRIZSY 5. p. 462.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 371.

Puccinia millefolii FUCKEL

BÄUMLER 1. II. No. 598.; SYDOW 2. I. p. 2.; MOESZ 11. p. 101.; HOLLÓS 7. p. 159.; BÄUMLER, MOESZ ap. MOESZ 17/b. p. 72.; SAVULESCU 7. p. 983.

Puccinia moliniae TULASNE

SYDOW 2. I. p. 763.; SAVULESCU 7. p. 773.; UBRIZSY in litt.

Puccinia montana FUCKEL

SYDOW 2. I. p. 37.; MAGNUS 1. p. 46.; SAVULESCU 7. p. 1038.; VASS—TÓTH 2. p. 49.

Puccinia montivaga BUBÁK

PICBAUER 1. p. 12.; PICBAUER 2. p. 191.

Puccinia mulgedii SYDOW

HUSZ 1. p. 102.; LINHART in Fung. hung. exs. No. 322., HUSZ, LINHART, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 72.; PICBAUER 1. p. 10.; PICBAUER 2. p. 189.; SAVULESCU 7. p. 1012.

Puccinia nigrescens KIRCHNER

Syn.: P. salviae HAZSL. non UNGER

HAZSLINSZKY 1. No. 158.; HAZSLINSZKY 2. p. 17.; BORBÁS 2. p. 33.; RICHTER 1. No. 15.; SACCARDO 1. No. 100.; BÄUMLER 1. III. No. 1398.; SYDOW 2. I. p. 289.; MOESZ 12. col. 808.; MOESZ 18. p. 13.; HOLLÓS 7. p. 159.; BÄUMLER, GRESCHIK, HAYNALD, HAZSLINSZKY, MÁGOCSY-DIETZ in Krypt. exs. No. 25.; MOESZ, RICHTER, SADLER, SZÉPLIGETI in LINHART Fung. hung.

exs. No. 320., WIERZBICKY ap. MOESZ 17/b. p. 72.; MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 124.; SAVULESCU 7. p. 932.; TÓTH 2. p. 59.; VASS 1. p. 52.

Puccinia oblongata (LINK) WINTER

KALCHBRENNER 1. I. p. 311.; HAZSLINSZKY 1. p. 168.; BÄUMLER 1. II. No. 619.; BRESADOLA 1. No. 122.; SYDOW 2. I. p. 647.; HUSZ 1. p. 102.; MOESZ 12. col. 808.; BÄUMLER, HAZSLINSZKY, MARGITTAI, MOESZ ap. MOESZ 17/b. p. 73.; SAVULESCU 7. p. 813.

Puccinia obscura SCHROETER

Syn.: Aecidium bellidis THUEM.

SYDOW 2. I. p. 646.; HOLLÓS 7. p. 159.; DEGEN ap. MOESZ 17/b. p. 73.;, DEGEN ap. MOESZ 13. p. 125.; SAVULESCU 7. p. 810.; DOHY—KIRÁLY in. UBRIZSY 5. p. 468.

Puccinia opizii BUBÁK

Syn.: Aecidium lactucae-sativae SYDOW

BUBÁK 1. No. 47.; BÄUMLER 1. V. No. 1857.; HOLLÓS 7. p. 145., 159.; BÄUMLER, GREINICH, MOESZ ap. MOESZ 17/b. p. 73.; MOESZ ap. MOESZ 13. p. 125.; SAVULESCU 7. p. 791.; SZIGETI—TÓTH 1. p. 21.; DOHY—KIRÁLY in UBRIZSY 5. p. 468.

Puccinia oreoselini (STRAUSS) FUCKEL

SYDOW 2. I. p. 401.; BÄUMLER 1. V. No. 1858.; BUBÁK 1. p. 101. (26); BÄUMLER, HAMBALKÓ, MOESZ ap. MOESZ 17/b. p. 74.; BUBÁK, HAMBALKÓ MOESZ 13. p. 125.; PICBAUER 1. p. 13.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 915.; TÓTH 2. p. 59.

Puccinia paludosa PLOWRIGHT

FUSS 1. IV. p. 112.; HAZSLINSZKY 1. p. 140.; BÄUMLER 1. II. p. 665.; SYDOW 2. I. p. 672.; HOLUBY, LIBOSCH, SCHNELLER ap. MOESZ 17/b. p. 74.; PICBAUER 1. p. 11.; PICBAUER 2. p. 190.; SAVULESCU 7. p. 790.

Puccinia passerinii SCHROETER

MOESZ ap. MOESZ 17/b. p. 74.; MOESZ ap. MOESZ 13. p. 125.; SAVULESCU 7. p. 832.; TÓTH 2. p. 59.

Puccinia perplexans PLOWRIGHT

Syn.: Aecidium ranunculacearum DC. ppe.

HOLLÓS 7. p. 159.; BÄUMLER, MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 74.; SZÉPLIGETI ap. MOESZ 13. p. 125.; SAVULESCU 7. p. 703.; DOHY—KIRÁLY in UBRIZSY 5. p. 470.; UBRIZSY 8. p. 371.

Puccinia persistens PLOWRIGHT

Syn.: Aecidium thalictri GREV.

FUSS 1. VIII. p. 232.; FUSS 2. p. 437.; BRESADOLA 1. No. 127.; SYDOW 2. I. p. 826.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 109.; HOLLÓS 7. p. 159.; GRESCHIK ap. MOESZ 17/b. p. 75.; SAVULESCU 7. p. 699.

Puccinia petasiti-pulchellae LÜDI

Syn : Aecidium petasitidis SYDOW

KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 140.; RICHTER 1. No. 25.;

MAGNUS 1. p. 47.; SYDOW 2. IV. p. 304.; GRESCHIK, HOLUBY, KMET, KÜMMERLE, MALY ap. MOESZ 17/b. p. 725.; SAVULESCU 7. p. 720.; TÓTH 2. p.59.

Puccinia petroselini (DC.) LINDROTH

Syn.: P. aethusae MART.; P. bullata PERS. ppe.

BOLLA 1. p. 48.; KALCHBRENNER 1. I. p. 308.; FUSS 1. p. 188. p. 24.; HAZSLINSZKY 1. p. 150.; HAZSLINSZKY 3. p. 83.; HAZSLINSZKY 5. p. 43.; FUSS 2. p. 441.; HAZSLINSZKY 7. p. 11.; SIMONKAI 1. No. 1722.; LIRO 3. p. 86.; SYDOW 2. I. p. 400.; RAPAICS 1. p. 218.; HOLLÓS 7. p. 159.; MOESZ 30. II. p. 373.; MOESZ 31. p. 96.; BÄUMLER, BÁNYAI, BOLLA, FUSS, HAZSLINSZKY, HOLUBY, KMET, MÁRTON, MOESZ ap. MOESZ 17/b. p. 75.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 903.; TÓTH 2. p. 59.; DOHY–KIRÁLY in UBRIZSY 5. p. 461.

Puccinia peucedani-parisiensis (DC.) LINDROTH

FUSS 2. p. 440.; MOESZ ap. MOESZ 17/b. p. 75.

Puccinia phlei-pratensis ERIKSSON et HENNINGS

RAPAICS 1. p. 213.; BÄUMLER 1. V. No. 1859.; MÁGOCSY-DIETZ ap. MOESZ 17/b. p. 76.; MÁGOCSY-DIETZ ap. MOESZ 13. p. 125.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 777.; VASS–TÓTH 1. p. 159.; DOHY–KIRÁLY in UBRIZSY 5. p. 468.; UBRIZSY 8. p. 371.

Puccinia phlomidis THÜMEN

Syn.: Aecidium phlomidis SADLER; Ae. phlomidis THÜMEN; Aecidium salviae HAZSL.

SADLER 1. XIII. p. 8.; HAZSLINSZKY 1. p. 133.; BORBÁS 2. p. 33.; MOESZ 18. p. 12., 13.; SYDOW 2. IV. p. 315.; MOESZ, SADLER ap. MOESZ 17/b. p. 76.; MOESZ, SADLER ap. MOESZ 13. p. 125.; SAVULESCU 7. p. 934.; UBRIZSY 6. in SZÉKESSY p. 23.

Puccinia phragmitis (SCHUM.) KÖRNICKE

Syn.: Aecidium rumicis SCHLECHT.

BOLLA 1. p. 50.; KALCHBRENNER 1. I. p. 311.; FUSS 1. et 2. p. 437., 441.; SCHULZER 1. No. 59., 67.; SCHULZER 2. p. 8., 9.; HAZSLINSZKY 1. p. 140., 162.; HAZSLINSZKY 2. p. 19.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 78., 86.; BORBÁS 3. p. 145.; BÄUMLER 1. II. p. 620.; BRESADOLA 1. No. 110., 114.; SIMONKAI 1. No. 1657., 1723., 1724.; SACCARDO 1. No. 105.; SYDOW 2. I. p. 789.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; MOESZ 11. p. 101.; MOESZ 12. col. 808.; HOLLÓS 7. p. 159.; MOESZ 31. p. 96.; BÄUMLER, BOLLA, BUDAI, FILARSZKY, GREINICH, HAZSLINSZKY, LENGYEL, LINHART in Fung. hung. exs. No. 26., 37.; LOJKA, MÁGOCSY-DIETZ, MOESZ in Flora hung. exs. No. 3., SADLER, SCHILBERSZKY in Krypt. exs. No. 30/b.; SCHNELLER, SZÉPLIGETI ap. MOESZ 17/b. p. 76.; LENGYEL, MÁGOCSY-DIETZ, MÜLLER, SADLER, SZÉPLIGETI, DEGEN, MOESZ, FILARSZKY, SCHILBERSZKY, HAZSLINSZKY, LOJKA ap. MOESZ 13. p. 126.; SAVULESCU 7. p. 767.; TÓTH 2. p. 59.; VASS–TÓTH 1. p. 159.; SZIGETI–TÓTH 1. p. 21.; DOHY–KIRÁLY in UBRIZSY 5. p. 469.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 371.

Puccinia picridis HAZSLINSZKY

HAZSLINSZKY 1. p. 152.; FUSS 2. p. 440.; SYDOW 2. I. p. 131.; BUBÁK 1. No. 49.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 100., 105.; MOESZ 11. p. 101.; HOLLÓS 7. p. 160.; HAZSLINSZKY, KÜMMERLE, MOESZ, SZÉPLIGETI, SZURÁK ap. MOESZ 17/b. p. 77.; KÜMMERLE, MOESZ, SZEPESFALVY, SZÉPLIGETI ap. MOESZ 13. p. 126.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 1066.; TÓTH 2. p. 59.; VASS–TÓTH 2. p. 49.; UBRIZSY 8. p. 372.

Puccinia pimpinellae (STRAUSS) MARTIUS

Syn.: Uredo pimpinellae STR.

HAZSLINSZKY 1. p. 150.; BORBÁS 2. p. 33.; HAZSLINSZKY 2. p. 15.; BÄUMLER 1. II. No. 621.; BRESADOLA 1. No. 109.; SYDOW 2. I. p. 409.; RAPAICS 1. p. 218.; MOESZ 31. p. 96.; BÄUMLER, HAZSLINSZKY, HOLUBY, MOESZ, SADLER, SCHNELLER ap. MOESZ 17/b. p. 77.; HAZSLINSZKY, MOESZ, SADLER ap. MOESZ 13. p. 126.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 887.; DOHY–KIRÁLY in UBRIZSY 5. p. 464.

Puccinia poarum NIELSEN

FUSS 1. IV. 112.; FUSS 2. p. 438.; KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 139.; HAZSLINSZKY 2. p. 15.; HAZSLINSZKY 3. p. 75., 78.; HAZSLINSZKY 7. p. 11.; BORBÁS 3. p. 145.; RICHTER 1. No. 25.; BÄUMLER 1. II. No. 622., IV. No. (622.); SYDOW 2. I. p. 796.; MOESZ 2. No. 76.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; MAGNUS 1. p. 45.; MOESZ 11. p. 101.; MOESZ 12. col. 808.; HOLLÓS 7. p. 160.; BÄUMLER, HOLLÓS in Krypt. exs. No. 2910., KMET in Flora exs. Austro-Hungarica No. 3555./IV., LINHART in Fung. hung. exs. No. 128., MOESZ ap. MOESZ 17/b. p. 78.; BERNÁTSKY, LÁNYI, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 126.; SAVULESCU 7. p. 717.; TÓTH 2. p. 59.; VASS–TÓTH 1. p. 159.; VASS 1. p. 52.; SZIGETI–TÓTH 1. p. 22.; DOHY–KIRÁLY in UBRIZSY 5. p. 470.; UBRIZSY 8. p. 372.

Puccinia podospermi DE CANDOLLE

Syn.: Aecidium amphigenum HAZSL.

HAZSLINSZKY 1. p. 133.; HAZSLINSZKY 3. p. 83.; MOESZ 18. p. 11.; SYDOW 2. IV. p. 305.; HAZSLINSZKY, HOLUBY, PÉNZES ap. MOESZ 17/b. p. 78.; MOESZ 13. p. 127.; SAVULESCU 7. p. 1024.; TÓTH 2. p. 59.; UBRIZSY 8. p. 372.

Puccinia polygoni ALBERTINI et SCHWEINITZ

HAZSLINSZKY 1. p. 160.; HAZSLINSZKY 2. p. 18.; HAZSLINSZKY 3. p. 85.; HAZSLINSZKY 7. p. 12.; BÄUMLER 1. II. No. 623.; BUBÁK 1. No. 50.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 107.; MOESZ 11. p. 101.; BÄUMLER, BUDAI, FUSS, GREINICH, HAZSLINSZKY, KMET, MÁGOCSY-DIETZ, MÁRTON, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 316. ap. MOESZ 17/b. p. 79.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 127.; SAVULESCU 7. p. 839.; TÓTH 2. p. 59.; VASS 1. 52.; TÍMÁR 1. p. 237.; UBRIZSY 8. p. 372.

Puccinia polygoni-amphibii PERSOON

HAZSLINSZKY 1. p. 160.; HAZSLINSZKY 2. p. 18.; SYDOW 2. I. p. 570., IV. p. 336.; MOESZ 11. p. 101.; MOESZ 21. p. 85.; BÄEMLER 1. V. No. 1860.; HOLLÓS 7. p. 160.; BÄUMLER, BARTH, BUDAI, GRESCHIK, HAZSLINSZKY, HOLUBY, KALCHBRENNER, LÁNYI, LÜTKEMÜLLER in Krypt. exs. No. 32/b., MÁGOCSY-

DIETZ in LINHART Fung. hung. exs. No. 119., MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 79.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 127.; SAVULESCU 7. p. 837.; TÓTH 2. p. 60.; UBRIZSY 8. p. 372.

Puccinia polygoni-vivipari KARSTEN

BRESADOLA 1. No. 118 ; HUSZ 1. p. 101.; MOESZ 30. p. 7.; DEGEN, GRESCHIK, KALCHBRENNER in RABENH. – WINTER Fung. eur. exs. No. 1383., MÁGOCSY-DIETZ, LINHART in Fung. hung. exs. No. 220., SZÉPLIGETI ap. MOESZ 17/b. p. 80.; PICBAUER 2. p. 191.; SAVULESCU 7. p. 844.

Puccinia porri (SOW.) WINTER

HAZSLINSZKY 2. p. 14.; FUSS 2. p. 437.; HAZSLINSZKY 7. p. 11.; BÄUMLER 1. II. No. 624.; SACCARDO 1. No. 101.; SYDOW 2. I. p. 611.; HOLLÓS 7. p. 160.; BARTH in THUEMEN Fung. austr. exs. No. 1118.; BÄUMLER, FUSS, MÁGOCSY-DIETZ in Krypt. exs. No. 26., MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 418. ap. MOESZ 17/b. p. 80.; MÁGOCSY-DIETZ, SZÉPLIGETI ap. MOESZ 13. p. 127.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 814.; VASS – TÓTH 3. p. 85.; DOHY – KIRÁLY in UBRIZSY 5. p. 469.; UBRIZSY 8. p. 372.

Puccinia praecox BUBÁK

HAZSLINSZKY 7. p. 11.; BORBÁS 3. p. 145.; BÄUMLER 1. II. No. 613.; BRESADOLA 1. No. 116.; MOESZ 2. No. 77.; MOESZ 11. p. 101.; HOLLÓS 7. p. 160.; GRESCHIK, HOLUBY, MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 80.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 127.; SAVULESCU 7. p. 1016.; VASS 1. p. 52.; UBRIZSY 8. p. 373.

Puccinia prenanthis (PERS.) LINDROTH

Syn.: P. chondrillae CORDA

BOLLA 1. p. 48.; HAZSLINSZKY 2. p. 17.; HAZSLINSZKY 3. p. 78.; SYDOW 2. I. p. 108.; BUBÁK 1. No. 34.; HUSZ 1. p. 101.; BÄUMLER 1. II. No. 625., V. No. 1840.; BÄUMLER, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 80.; SAVULESCU 7. p. 1009.

Puccinia prenanthis-purpureae (DC.) LINDROTH

KALCHBRENNER 3. p. 159.; HAZSLINSZKY 2. p. 15., 17.; HAZSLINSZKY 12. p. 153.; BRESADOLA 1. No. 106.; SYDOW 2. I. p. 135.; MOESZ 31. p. 96.; BUDAI, GRESCHIK, HAZSLINSZKY, KALCHBRENNER, KMET, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 81.; PICBAUER 1. p. 10.; PICBAUER 2. p. 189.; SAVULESCU 7. p. 1013.; UBRIZSY 8. p. 373.

Puccinia primulae (DC.) DUBY

BÄUMLER 1. II. No. 626.; BÄUMLER ap. MOESZ 17/b. p. 81.; PICBAUER 3. p. 177.; VASS 1. p. 52.; DOHY – KIRÁLY in UBRIZSY 5. p. 177.

Puccinia pringsheimiana KLEBAHN

Syn.: P. caricis (SCHUM.) REB. ppe.; P. ribesii caricis KLEB. ppe.; Aecidium grossulariae (GMEL.) SCHUM. ppe.

FUSS 1. IV. p. 125.; BOLLA 1. p. 50.; KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 138.; HAZSLINSZKY 3. p. 77.; BÄUMLER 1. II. No. 662.; HOLLÓS 7. p. 160.; BÄUMLER, GREINICH, GRESCHIK, HAZSLINSZKY, KANABÉ,

MÁGOCSY-DIETZ, MOESZ, NAGY ap. MOESZ 17/b. p. 81.; KRENNER, MOESZ ap. MOESZ 13. p. 127.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 787.; DOHY—KIRÁLY in UBRIZSY 5. p. 466.

Puccinia pulsatillae (OPIZ) ROSTRUP

Syn.: P. fusca (PERS.) WINTER

FUSS 1. IV. p. 126.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 107.; BÄUMLER, MÁGOCSY-DIETZ, TAMÁSSY, ZSÁK ap. MOESZ 17/b. p. 83.; MÁGOCSY-DIETZ, ZSÁK ap. MOESZ 13. p. 127.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 853.; SZIGETI—TÓTH 1. p. 22.; DOHY—KIRÁLY in UBRIZSY 5. p. 464.

Puccinia pygmaea ERIKSSON

BÄUMLER 1. V. No. 1861.; HOLLÓS 7. p. 161. ap. MOESZ 17/b. p. 83.; SAVULESCU 7. p. 708.; UBRIZSY 8. p. 373.

Puccinia pyrethri RABENHORST

HAZSLINSZKY 7. p. 12.; SIMONKAI 1. No. 1725.; SYDOW 2. I. p. 45.; BUBÁK 1. No. 51.; BÄUMLER 1. II. No. 635., V. No. 1862.; HOLLÓS 7. p. 161.; AUGUSTIN, BÄUMLER, GRESCHIK, HOLUBY, LINHART in Fung. hung. exs. No. 228., 323., MOESZ ap. MOESZ 17/b. p. 83.; AUGUSTIN, MOESZ ap. MOESZ 13. p. 127.; PICBAUER 2. p. 191.; TÓTH 2. p. 60.; VASS—TÓTH 2. p. 49.; SAVULESCU 7. p. 992.

Puccinia punctata LINK

SAVULESCU 7. p. 956.; TÓTH 2. p. 60.; VASS 1. p. 52.; VASS—TÓTH 2. p. 49.

Puccinia retifera LINDROTH

Syn.: Cystopus verrucosus HAZSL.

LIRO 3. p. 20.; SYDOW 2. I. p. 368.; HOLLÓS 1. p. 136.; MAGNUS 1. p. 46.; BÄUMLER, BORNMÜLLER in Krypt. exs. Vindob. No. 2805.; GRESCHIK, HOLUBY in LINHART Fung. hung. exs. No. 125., 136., in RABENH.—WINTER Fung. eur. exs. No. 2926., MOESZ ap. MOESZ 17/b. p. 84.; SAVULESCU 7. p. 892.

Puccinia rhododendri FUCREL

FUSS 2. p. 440.; SYDOW 2. I. p. 351. ap. MOESZ 17/b. p. 84.; SAVULESCU 7. p. 918.

Puccinia ribesii-caricis KLEBAHN

Syn.: P. caricis (SCHUM.) REB. ppe.

KRENNER 1. p. 64.; VÖRÖS 9. p. 102.; DOHY—KIRÁLY in UBRIZSY 5. p. 466.

Puccinia ribis DE CANDOLLE

HAZSLINSZKY 1. p. 149.; MOESZ 12. col. 808.; GRESCHIK, GYŐRFFY, HAZSLINSZKY, LOJKA ap. MOESZ 17/b. p. 84.; SAVULESCU 7. p. 866.; DOHY—KIRÁLY in UBRIZSY 5. p. 461.

Puccinia rossiana (SACC.) LAGERHEIM

Syn.: P. scillae LINHART

SYDOW 2. I. p. 631.; LINHART in Fung. hung. exs. No. 417. et in RABENH.—WINTER Fung. eur. exs. No. 3516. ap. MOESZ 17/b. p. 84.; SAVULESCU 7. p. 821.; DOHY—KIRÁLY in UBRIZSY 5. p. 464.

Puccinia rubigo-vera (DC.) WINTER

MOESZ 2. No. 68.; MOESZ, PÉNZES ap. MOESZ 17/b. p. 85.; MOESZ, PÉNZES ap. MOESZ 13. p. 127.; SAVULESCU 7. p. 710.

Puccinia salviae UNGER

Syn.: P. cornuta HAZSL.

HAZSLINSZKY 1. p. 158.; HAZSLINSZKY 2. p. 18.; BÄUMLER 1. V. No. 1863.; MOESZ 12. col. 808.; MOESZ 18. p. 13.; HOLLÓS 7. p. 161.; BÄUMLER, GRESCHIK, HAZSLINSZKY, HOLLÓS, LINHART in Fung. hung. exs. No. 414., MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 85.; PICBAUER 1. p. 13.; SAVULESCU 7. p. 924.; VASS 1. p. 52.

Puccinia saniculae GREVILLE

HAZSLINSZKY 1. p. 166.; HAZSLINSZKY 3. p. 83.; SYDOW 2. I. p. 414.; BUDAI, LAUDON, MOESZ ap. MOESZ 17/b. p. 85.; SAVULESCU 7. p. 897.

Puccinia saxifragae SCHLECHTENDAL

BUBÁK 1. No. 52.; SYDOW 2. I. p. 501.; SAVULESCU 7. p. 867.

Puccinia schirajewskii TRANZSCHEL

SAVULESCU 7. p. 1070.; VASS 1. p. 52.; CONSTANTINESCU et DIHORU 1. p. 87.

Puccinia schmidtiana DIETEL

Syn.: Aecidium leucoji LINHART

HAZSLINSZKY 3. p. 78.; SYDOW 2. I. p. 748.; BÄUMLER in Krypt. exs.No. 2404., LINHART in Fung. hung. exs. No. 48. et in RABENH.—WINTER Fung. eur. exs. No. 2718., MOESZ ap. MOESZ 17/b. p. 85.; SAVULESCU 7. p. 729.

Puccinia schroeteri PASSERINI

DOHY—KIRÁLY in UBRIZSY 5. p. 464.

Puccinia scillae-rubrae CRUCHET

Syn.: Aecidium scillae FUCKEL

SYDOW 2. IV. p. 361.; BÄUMLER 1. V. No. 1883.; KMET in Flora exs. Austro-Hung. No. 1567., LINHART in Fung. hung. exs. No. 408. et in RABENH.—WINTER Fung. eur. exs. No. 3409. ap. MOESZ 17/b. p. 85.; SAVULESCU 7. p. 723.

Puccinia scirpi DE CANDOLLE

Syn.: P. typhae KALCHBR.; Aecidium nymphoides DC.

KALCHBRENNER 1. I. p. 310.; SCHULZER 1. No. 60.; SCHULZER 2. p. 29.; HAZSLINSZKY 1. p. 167.; HAZSLINSZKY 2. p. 19.; SYDOW 2. I. p. 689.; MOESZ 21. p. 85.; GREINICH, KALCHBRENNER, MÁGOCSY-DIETZ, MOESZ, SCHEITZ, TUZSON ap. MOESZ 17/b. p. 86.; SAVULESCU 7. p. 806.; TÓTH 2. p. 60.

Puccinia scorzonerae (SCHUM.) JACKY

SYDOW 2. I. p. 142.; HOLLÓS 7. p. 161.; MOESZ ap. MOESZ 17/b. p. 86.; MÜLLER ap. MOESZ 13. p. 127.; SAVULESCU 7. p. 1023.; TÍMÁR 1. p. 237.; DOHY—KIRÁLY in UBRIZSY 5. p. 463.

Puccinia sesleriae REICHARDT

BÄUMLER 1. V. No. 1865.; LENGYEL ap. MOESZ 17/b. p. 86.; SAVULESCU 7. p. 781.

Puccinia sessilis SCHNEIDER

SAVULESCU 7. p. 725.; LEHOCZKY 1. p. 237.

Puccinia sileris Voss

Liro 3. p. 43.; Sydow 2. I. p. 416.; Holuby in Linhart Fung. hung. exs. No. 325. ap. Moesz 17/b. p. 86.; Picbauer 1. p. 10.

Puccinia silvatica Schroeter

Syn.: Aecidium taraxaci Schm. et Kze.

Bolla 1. p. 49.; Fuss 2. p. 436.; Borbás 2. p. 33.; Hazslinszky 1. p. 140.; Hazslinszky 3. p. 85.; 86.; Bäumler 1. II. No. 631.; Saccardo 1. No. 125.; Sydow 2. I. p. 657.; Hollós 1. p. 138.; Hollós 6/a. p. 107.; Hollós 7. p. 162.; Moesz 30. p. 7.; Bäumler, Budai, Hollós in Krypt. exs. No. 2715., Holuby, Kmet, Linhart in Fung. hung. exs. No. 129., 221., Mágocsy-Dietz, Moesz, Schneller, Szépligeti ap. Moesz 17/b. p. 87.; Mágocsy-Dietz, Moesz, Sadler, Szépligeti ap. Moesz 13. p. 127.; Picbauer 1. p. 12.; Savulescu 7. p. 795.; Tóth 2. p. 60.; Vass–Tóth 1. p. 159.; Tímár 1. p. 237.; Ubrizsy 8. p. 373.

Puccinia simplex (Koern.) Eriksson et Hennings

Syn.: P. hordei Otth; Aecidium ornithogaleum Bub.

Simonkai 1. No. 1728.; Sydow 2. I. p. 757., IV. p. 287.; Rapaics 1. p. 212.; Bäumler 1. V. No. 1866.; Moesz 11. p. 101.; Bäumler, Hazslinszky, Lányi, Linhart in Fung. hung. exs. No. 127., Moesz, Pósch ap. Moesz 17/b. p. 87.; Moesz ap. Moesz 13. p. 127.; Ubrizsy 1. p. 14.; Ubrizsy 2. p. 153.; Savulescu 7. p. 721.; Husz–Király in Ubrizsy 5. p. 456.; Ubrizsy 8. p. 373.

Puccinia singularis Magnus

Syn.: P. bäumleri Lagh.

Bäumler 1. II. No. 600.; Bäumler in Rabenh.–Winter–Pazschke Fung. eur. exs. No. 3916., Sabransky ap. Moesz 17/b. p. 88.; Savulescu 7. p. 860.

Puccinia smilacearum-digraphidis Klebahn

Syn.: Aecidium convallariae Schum.

Fuss 1. IV. p. 112.; Bolla 1. p. 49.; Hazslinszky 1. p. 142.; Fuss 2. p. 438.; Bäumler 1. II. No. 660.; Simonkai 1. No. 1659.; Sydow 2. I. p. 782.; Hollós 1. p. 139.; Hollós 6/a. p. 108.; Hollós 7. p. 162.; Bäumler in Flora hung. exs. No. 202., Bolla–Sabransky, Budai, Hazslinszky, Kümmerle, Linhart in Fung. hung. exs. No. 139., Moesz, Szépligeti ap. Moesz 17/b. p. 88.; Kümmerle, Moesz, Szépligeti ap. Moesz 13. p. 127.; Ubrizsy 2. p. 153.; Savulescu 7. p. 725.; Lehoczky 1. p. 237.; Dohy–Király in Ubrizsy 5. p. 469.; Ubrizsy 6. in Székessy p. 23.

Puccinia soldanellae (DC.) Fuckel

Sydow 2. I. p. 350.; Magnus 1. p. 45.; Husz 1. p. 102.; Moesz 12. col. 808.; Moesz 30. p. 7. II. p. 373.; Bányai, Győrffy, Linhart in Fung. hung. exs. No. 224. ap. Moesz 17/b. p. 88.; Picbauer 1. p. 10.; Picbauer 2. p. 189.; Savulescu 7. p. 946.

Puccinia sonchi Roberge

Haynald, Moesz ap. Moesz 17/b. p. 89.; Moesz ap. Moesz 13. p. 127.; Savulescu 7. p. 976.

Puccinia spergulae DE CANDOLLE

SACCARDO 1. No. 112.; SYDOW 2. I. p. 560.; KMET ap. MOESZ 17/b. p. 89.; SAVULESCU 7. p. 851.; DOHY—KIRÁLY in UBRIZSY 5. p. 467.

Puccinia stachydis DE CANDOLLE

HAZSLINSZKY 1. p. 158.; HAZSLINSZKY ap. MOESZ 17/b. p. 89.; SAVULESCU 7. p. 926.; TÓTH 2. p. 60.

Puccinia stipina TRANZSCHEL

Syn.: P. stipae (OPIZ) HORA; Aecidium thymi FUCKEL

HAZSLINSZKY 1. p. 140.; FUSS 2. p. 438.; BÄUMLER 1. III. No. 1399.; SYDOW 2. I. p. 816.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 109.; MOESZ 11. p. 102.; BÄUMLER in Krypt. exs. Vindob. No. 919., MOESZ ap. MOESZ 17/b. p. 89.; SAVULESCU 7. p. 775.

Puccinia suaveolens (PERS.) ROSTRUP

Syn.: P. obtegens (LINK) TUL.

BOLLA 1. p. 48.; KALCHBRENNER 1. I. p. 310.; HAZSLINSZKY 1. p. 151.; HAZSLINSZKY 2. p. 15.; HAZSLINSZKY 3. p. 83.; HAZSLINSZKY 7. p. 12.; BORBÁS 2. p. 33.; BÄUMLER 1. II. No. 634., V. No. 1842.; BRESADOLA 1. No. 115.; SACCARDO 1. No. 107.; SYDOW 2. I. p. 54.; MOESZ 2. No. 80.; HOLLÓS 1. p. 135.; HOLLÓS 6/a. p. 105.; MOESZ 11. p. 102.; MOESZ 12. col. 808.; HOLLÓS 7. p. 159.; MOESZ 30. II. p. 373.; LENGYEL in Krypt. exs. Vindob. No. 1130., LINHART in Fung. hung. exs. No. 120., SADLER ap. MOESZ 17/b. p. 90.; FILARSZKY, HAZSLINSZKY, KŐFARAGÓ-GYELNIK, LENGYEL, MÁGOCSY-DIETZ, MOESZ, PÉNZES, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 127.; SAVULESCU 7. p. 1048.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 60.; VASS 1. p. 52.; VASS—TÓTH 2. p. 49.; TÍMÁR 1. p. 237.; SZIGETI—TÓTH 1. p. 22.; TÓTH 10. p. 10.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 373.

Puccinia symphyti-bromorum MÜLLER

Syn.: P. bromina ERIKSS. p. min. pe.; P. dispersa auct. ppe.; Aecidium symphyti THÜM. ppe.; Ae. pulmonariae THÜM. ppe.

KALCHBRENNER 1. I. p. 304.; HAZSLINSZKY 1. p. 137.; BORBÁS 2. p. 33.; HAZSLINSZKY 7. p. 13.; BRESADOLA 1. No. 112.; SYDOW 2. I. p. 713.; HOLLÓS 1. p. 139.; HOLLÓS 6/a. p. 108.; HOLLÓS 7. p. 153.; BÄUMLER, BUDAI, GREINICH, GRESCHIK, LINHART in Fung. hung. exs. No. 35., MOESZ, SCHNELLER, HAZSLINSZKY, SZÉPLIGETI ap. MOESZ 17/b. p. 90.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 128.; UBRIZSY 1. p. 14.; PICBAUER 2. p. 190.; SAVULESCU 7. p. 713.; DOHY—KIRÁLY in UBRIZSY 5. p. 471.; UBRIZSY 8. p. 374.

Puccinia sweertiae (OPIZ) WINTER

SYDOW 2. I. p. 342. ap. MOESZ 17/b. p. 91.; PICBAUER 2. p. 188.

Puccinia tanaceti DE CANDOLLE

SYDOW 2. I. p. 162.; MOESZ 11. p. 102.; HOLLÓS 7. p. 162.; AUGUSTIN, MOESZ ap. MOESZ 17/b. p. 91.; SAVULESCU 7. p. 998.; VASS—TÓTH 2. p. 49.; UBRIZSY 8. p. 374.

Puccinia taraxaci (REBENT.) PLOWRIGHT

HAZSLINSZKY 2. p. 17.; HAZSLINSZKY 7. p. 11.; HAZSLINSZKY 11. p. 83.;

BÄUMLER 1. II. No. 613.; SYDOW 2. I. p. 165.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 102.; MOESZ 12. col. 808.; HOLLÓS 7. p. 162.; AUGUSTIN, BAKOS, BÄUMLER, HAZSLINSZKY, HOLUBY, KÜMMERLE, LAUDON, LÁNYI, LENGYEL, MÁGOCSY-DIETZ, MOESZ, PÉNZES, SIMKOVITS, SZÉPLIGETI, SZURÁK ap. MOESZ 17/b. p. 91.; AUGUSTIN, BAKOS, HAZSLINSZKY, KÜMMERLE, MOESZ, PÉNZES, SZEPESFALVY, SZÉPLIGETI ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 1071.; TÓTH 2. p. 60.; VASS 1. p. 52.; VASS–TÓTH 2. p. 49.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 374.

Puccinia taraxaci-serotini PICBAUER

PICBAUER 1. I. p. 11.; DEGEN, SZÉPLIGETI ap. MOESZ 17/b. p. 92.; DEGEN, SZÉPLIGETI ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 1073.

Puccinia tenuistipes ROSTRUP

BÄUMLER 1. IV. No. 1500.; BÄUMLER ap. MOESZ 17/b. p. 92.; SAVULESCU 7. p. 803.

Puccinia thalictri CHEVALLIER

Syn.: Tranzschelia thalictri (CHEV.) DIETEL

MOESZ ap. MOESZ 17/b. p. 92.; MOESZ ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 488.; DOHY–KIRÁLY in UBRIZSY 5. p. 464.

Puccinia thesii (DESV.) CHAILLET

Syn.: Aecidium thesii DESV.

KALCHBRENNER 1. I. p. 304.; HAZSLINSZKY 1. p. 134.; HAZSLINSZKY 3. p. 75., 87.; FUSS 1. IV. p. 125., VIII. p. 232.; BÄUMLER 1. II. No. 636.; SACCARDO 1. No. 123.; SYDOW 2. I. p. 586.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 107.; HOLLÓS 7. p. 162.; MOESZ 11. p. 102.; BÄUMLER in LINHART Fung. hung. exs. No. 319., DEGEN, FUSS, GOMBOCZ, GREINICH, HOLUBY, JÁVORKA, KMET, MÁRKUS, MOESZ, SIMONKAI, WIERZBICKY ap. MOESZ 17/b. p. 92.; DEGEN, GOMBOCZ, JÁVORKA, MOESZ ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 830.; SZIGETI–TÓTH 1. p. 22.; UBRIZSY 8. p. 374.

Puccinia thlaspeos SCHUBERT

FUSS 2. p. 441.; BRESADOLA 1. No. 130.; BÄUMLER 1. III. No. 1400.; SYDOW 2. I. p. 516.; BÄUMLER in Krypt. exs. Vindob. No. 926., HOLUBY, MOESZ ap. MOESZ 17/b. p. 93.; MOESZ ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 861.; SZIGETI–TÓTH 1. p. 22.

Puccinia tinctoriicola MAGNUS

Syn.: Uredo serratulae SCHUM.

HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 102.; BOROS, BUDAI, CZAKÓ in LINHART Fung. hung. exs. No. 428., LINHART in Fung. hung. exs. No. 321/b., MOESZ ap. MOESZ 17/b. p. 93.; BOROS, CZAKÓ, MOESZ ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 1068.; VASS–TÓTH 2. p. 49.; UBRIZSY 8. p. 374.

Puccinia tragopogi (PERS.) CORDA

Syn.: Aecidium tragopogi PERS.

FUSS 1. IV. p. 112.; BOLLA 1. p. 50.; KALCHBRENNER 1. I. p. 303.; FUSS 2. p. 436., 440.; BORBÁS 2. p. 33.; HAZSLINSZKY 1. p. 133., 135., 151.; HAZSLINSZKY 3. p. 84.; BÄUMLER 1. II. No. 637.; BRESADOLA 1. No. 124.; SYDOW

2. I. p. 168., IV. 306.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 30. p. 7.; MOESZ 31. p. 96.; BÄUMLER, BOLLA, BUDAI, FUSS, GÁYER, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, MOESZ, SCHULZER, WAGNER ap. MOESZ 17/b. p. 93.; MOESZ, SADLER ap. MOESZ 13. p. 128.; SAVULESCU 7. p. 1026.

Puccinia trailii PLOWRIGHT

KRENNER ap. MOESZ 17/b. p. 94.; SAVULESCU 7. p. 770.; DOHY—KIRÁLY in UBRIZSY 5. p. 469.

Puccinia triseti ERIKSSON

SAVULESCU 7. p. 730.; DOHY—KIRÁLY in UBRIZSY 5. p. 471.

Puccinia triticina ERIKSSON

Syn.: P. recondita ROB.; P. dispersa ERIKSS. et HENN. ppe.; P. rubigo-vera ppe.; P. straminis ppe.

SYDOW 2. I. p. 716.; RAPAICS 1. p. 212.; MOESZ 12. col. 808.; HUSZ 3/b. p. 77.; HUSZ 7.; HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ, TUZSON—BUBÁK in Krypt. exs. Vindob. No. 2105., VARGA ap. MOESZ 17/b. p. 94.; HUSZ, MOESZ ap. MOESZ 13. p. 129.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 706.; HUSZ—KIRÁLY in UBRIZSY 5. p. 451.

Puccinia uliginosa JUEL

SYDOW 2. I. p. 674. ap. MOESZ 17/b. p. 94.

Puccinia uralensis TRANZSCHEL

SACCARDO 1. No. 99.; SYDOW 2. I. p. 145.; JÁVORKA, KMET in Flora exs. Austro-Hung. No. 1962., SIMONKAI (SIMKOVITS) ap. MOESZ 17/b. p. 95.; SAVULESCU 7. p. 986.

Puccinia urticae-caricis (SCHUM.) REBENTISCH

Syn.: P. caricis DC. ppe.; Aecidium urticae SCHUM., P. urticae-caricis KLEBAHN BOLLA 1. p. 49., 50.; KALCHBRENNER 1. I. p. 304.; FUSS 1. XVI. p. 24.; SCHULZER 1. No. 70.; SCHULZER 2. p. 10.; HAZSLINSZKY 1. p. 138., 168.; FUSS 2. p. 441.; HAZSLINSZKY 2. p. 19.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 77., 85.; BÄUMLER 1. II. No. 602.; BRESADOLA 1. No. 113.; SACCARDO 1. No. 104., 124.; MOESZ 2. No. 66.; HOLLÓS 1. p. 138.; HOLLÓS 6/a. p. 107.; MAGNUS 1. p. 45.; MOESZ 11. p. 100.; MOESZ 12. col. 807.; HOLLÓS 7. p. 153.; MOESZ 30. p. 7.; BÄUMLER, BOLLA, BUDAI, FILARSZKY, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLUBY, KMET in Flora exs. Austro-Hung. No. 1960. I., LINHART in Fung. hung. exs. No. 38., MÁGOCSY—DIETZ in Krypt. exs. Vindob. No. 1411., MOESZ, NAGY, PALIK, STAUB, SZÉPLIGETI, VAJDA ap. MOESZ 17/b. p. 95.; MOESZ, STAUB, SZÉPLIGETI ap. MOESZ 13. p. 129.; UBRIZSY 6. in SZÉKESSY p. 23.; SAVULESCU 7. p. 784.; SZIGETI—TÓTH 1. p. 22.; DOHY—KIRÁLY in UBRIZSY 5. p. 466.

Puccinia urticae-caricis (SCHUM.) REBENTISCH f. sp. *urticae-acutiformis* KLEBAHN

BUDAI, MOESZ ap. MOESZ 17/b. p. 95.

Puccinia urticae-caricis (SCHUM.) REBENTISCH f. sp. *urticae-hirtae* KLEBAHN

GREINICH, HOLUBY, KMET in Flora exs. Austro-Hung. No. 1960. II. MOESZ ap. MOESZ 17/b. p. 95.

Puccinia valantiae PERSOON

KALCHBRENNER 1. I. p. 308.; HAZSLINSZKY 1. p. 166.; HAZSLINSZKY 2. p. 19.; BÄUMLER 1. II. No. 638.; SACCARDO 1. No. 113.; SYDOW 2. I. p. 218.; MAGNUS 1. p. 46.; HUSZ 1. p. 102.; MOESZ 11. p. 102.; BÄUMLER, FILARSZKY, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 96.; FILARSZKY, MOESZ ap. MOESZ 13. p. 129.; PICBAUER 1. p. 14.; SAVULESCU 7. p. 951.; LEHOCZKY 1. p. 237.

Puccinia veratri NIESSL

KALCHBRENNER 1. I. p. 310.; HAZSLINSZKY 1. p. 155. ap. MOESZ 17/b. p. 96.; PICBAUER 3. p. 178.; SAVULESCU 7. p. 823.

Puccinia veronicarum DE CANDOLLE

Syn.: P. veronicae SCHROETER; P. veronicarum MOESZ

BÄUMLER 1. II. No. 639.; SYDOW 2. I. p. 258.; MAGNUS 1. p. 46.; HOLLÓS 7. p. 162.; BÄUMLER, KMET, RŐMER in LINHART Fung. hung. exs. No. 214. ap. MOESZ 17/b. p. 96.; SAVULESCU 7. p. 970.

Puccinia verruca THÜMEN

SYDOW 2. I. p. 43.; BUDAI, VAJDA ap. MOESZ 17/b. p. 96.; PICBAUER 1. p. 14.; PICBAUER 2. p. 192.; SAVULESCU 7. p. 987.; TÓTH 2. p. 60.

Puccinia vincae (DC.) BERKELEY

FUSS 1. IV. p. 111.; HAZSLINSZKY 1. p. 155.; FUSS 2. p. 441.; BORBÁS 2. p. 33.; BÄUMLER 1. IV. p. 1499.; SYDOW 2. I. p. 338.; MOESZ 2. No. 81.; HOLLÓS 1. p. 136.; HOLLÓS 6/a. p. 106.; MOESZ 4. VII. p. 127.; HOLLÓS 7. p. 163.; BARTH in LINHART Fung. hung. exs. No. 317.; BÁNHEGYI, BÄUMLER, BERNÁTSKY, DEGEN, HOLLÓS in Krypt. exs. Vindob. No. 2101/a, JÁVORKA, MÁGOCSY-DIETZ, MOESZ, SADLER, SZANDOVICS, SZÉPLIGETI, TRAUTMAN, VAJDA ap. MOESZ 17/b. p. 97.; BÁNHEGYI, BERNÁTSKY, DEGEN, JÁVORKA, MÁGOCSY-DIETZ, MOESZ, SADLER, SZÉPLIGETI, TRAUTMANN, VAJDA, SZANDOVICS ap. MOESZ 13. ap. 29.; PICBAUER 1. p. 13.; SAVULESCU 7. p. 942.; VASS 1. p. 52.; DOHY–KIRÁLY in UBRIZSY 5. p. 463.; UBRIZSY 8. p. 374.

Puccinia violae (SCHUM.) DE CANDOLLE

Syn.: Aecidium violae SCHUM.

FUSS 1. IV. p. 111., VIII. p. 232., XVI. p. 24.; BOLLA 1. p. 48., 50.; SCHULZER 1. No. 68.; SCHULZER 2. p. 9.; HAZSLINSZKY 1. p. 141., 148.; HAZSLINSZKY 2. p. 15.; HAZSLINSZKY 3. p. 83., 87.; HAZSLINSZKY 7. p. 11.; FUSS 2. p. 436., 440.; BORBÁS 2. p. 33.; BORBÁS 3. p. 145.; RICHTER 1. No. 22.; BÄUMLER 1. II. No. 641.; SYDOW 2. I. p. 441.; HOLLÓS 1. p. 137.; HOLLÓS 6/a. p. 106.; MOESZ 11. p. 102.; HOLLÓS 7. p. 163.; MOESZ 31. p. 96.; BÄUMLER, BÁNHEGYI, BOLLA, BUDAI, DEGEN, FILARSZKY, GÁYER, GREINICH, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KARDOS, KMET, LAUDON, LÁNYI, LINHART in Fung. hung. exs. No. 33., MÁGOCSY-DIETZ in Krypt. exs. No. 1409., MÁRTON, MOESZ, SADLER, SCHNELLER, SZÉPLIGETI, SZURÁK, ZAHLBRUCKNER ap. MOESZ 17/b. p. 97.; FILÁRSZKY, KARDOS, MÁGOCSY-DIETZ, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 129.; SAVULESCU 7. p. 874.; TÓTH 2. p. 60.; VASS–TÓTH 1. p. 159.; VASS 1. p. 52.; VASS–TÓTH

2. p. 49.; SZIGETI–TÓTH 1. p. 22.; DOHY–KIRÁLY in UBRIZSY 5. p. 464.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY 8. p. 374.

Puccinia virgaurea (DC.) LIBERT

FUSS 2. p. 441.; BÄUMLER 1. V. No. 1868.; SYDOW 2. I. p. 152.; HOLLÓS 7. p. 163.; KMET in Flora exs. Austro–Hung. No. 3163. ap. MOESZ 17/b. p. 98; SAVULESCU 7. p. 974.

Puccinia volkartiana FISCHER

PICBAUER 1. p. 13.; PICBAUER 2. p. 192.

Puccinia vossii KOERNICKE

SYDOW 2. I. p. 299.; DEGEN, SCHILBERSZKY in Krypt. exs. Vindob. No. 39., SZÉPLIGETI ap. MOESZ 17/b. p. 98.; SCHILBERSZKY, SZÉPLIGETI ap. MOESZ 13. p. 129.; SAVULESCU 7. p. 937.

Puccinia zopfii WINTER

Syn.: P. calthaecola SCHROET. ppe.

ROUPPERT 1. p. 313.; BÄUMLER 1. V. No. 1869.; LINHART in Fung. hung. exs. No. 229. ap. MOESZ 17/b. p. 99.

Genus. *Tranzschelia* ARTHUR

Tranzschelia discolor (FUCK.) TRANZSCHEL et LITVINOV

UBRIZSY 8. p. 374.; GLITS in litt.

Tranzschelia fusca (PERS.) DIETEL

Syn.: Puccinia fusca (RELHAN) WINTER

FUSS 1. IV. p. 126.; KALCHBRENNER 1. I. p. 307.; HAZSLINSZKY 1. p. 147.; FUSS 2. p. 440.; HAZSLINSZKY 7. p. 11.; BRESADOLA 1. No. 125.; SYDOW 2. I. p. 531.; FUSS, GRESCHIK, HAZSLINSZKY, JÁVORKA, MÁRTON, ZÓLYOMI ap. MOESZ 17/a. p. 185.; SAVULESCU 7. p. 484.; DOHY–KIRÁLY in UBRIZSY 5. p. 464.

Tranzschelia pruni-spinosae (PERS.) DIETEL

Syn.: Puccinia pruni-spinosae PERS.; P. prunorum LINK; Aecidium punctatum PERS.

ENDLICHER 1. No. 85.; FUSS 1. IV. p. 125., 126.; BOLLA 1. p. 50., 51.; KALCHBRENNER 1. I. p. 308.; SCHULZER 2. p. 28., 35., 36.; HAZSLINSZKY 1. p. 122., 131., 147.; HAZSLINSZKY 2. p. 15.; FUSS 2. p. 440.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 75.; 83.; HAZSLINSZKY 7. p. 9.; RICHTER 1. No. 13., 51.; BÄUMLER 1. II. No. 606., 609., 627.; BRESADOLA 1. No. 121.; SIMONKAI 1. No. 1656., SACCARDO 1. No. 122.; SYDOW 2. I. p. 486., IV. p. 350.; HOLLÓS 6/a. p. 107.; HOLLÓS 1. p. 137.; RAPAICS 1. p. 217.; MOESZ 11. p. 101.; MOESZ 21. p. 85.; HOLLÓS 7. p. 145.; HUSZ 6. p. 361.; BÄUMLER, BOLLA, BUDAI, ENDRESZ, FUSS, GREINICH, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, KOSZILKOV, KUNSZT, LINHART in Fung. hung. exs. No. 47., MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 28., MÁRTON, MOESZ, PÓSCH, RICHTER, SZÉPLIGETI ap. MOESZ 17/b. p. 82.; MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 127.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 480.; TÓTH 2. p.

60.; VASS 1. p. 50.; VASS–TÓTH 2. p. 49.; SZIGETI–TÓTH 1. p. 22.; VASS–TÓTH 3. p. 85.; PODHRADSZKY in UBRIZSY 5. p. 416.; UBRIZSY 8. p. 374.

Genus. *Triphragmium* LINK

Triphragmium echinatum LÉVEILLÉ

SYDOW 2. III. p. 178. ap. MOESZ 17/b. p. 99.

Triphragmium filipendulae (LASCH) PASSERINI

Syn.: Uredo filipendulae LASCH

SYDOW 2. III. p. 176.; BORBÁS, MÁGOCSY-DIETZ in Krypt. exs. No. 2206., MOESZ ap. MOESZ 17/b. p. 99.; BORBÁS, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 129.; SAVULESCU 7. p. 528.; PODHRADSZKY in UBRIZSY 5. p. 420.; UBRIZSY 8. p. 374.

Triphragmium isopyri MOUGEOT et NESTLER

HOLLÓS 7. p. 163.; HOLLÓS ap. MOESZ 17/b. p. 99.; PODHRADSZKY in UBRIZSY 5. p. 420.

Triphragmium ulmariae (SCHUM.) LINK

KALCHBRENNER 1. I. p. 311.; HAZSLINSZKY 1. p. 145.; BRESADOLA 1. No. 143.; SYDOW 2. III. p. 173.; BÄUMLER 1. V. No. 1876.; GRESCHIK, KMET, LINHART in Fung. hung. exs. No. 231., MOESZ, TUZSON p. MOESZ 17/b.p. 99.; SAVULESCU 7. p. 529.; LEHOCZKY 1. p. 237.; PODHRADSZKY in UBRIZSY 5. p. 420.; UBRIZSY 8. p. 374.

Genus. *Uromyces* LINK

Uromyces acetosae SCHROETER

FUSS 1. IV. p. 112.; HAZSLINSZKY 1. p. 126.; FUSS 2. p. 437.; BRESADOLA 1. No. 91.; HOLLÓS 1. p. 141.; HOLLÓS 7. p. 163.; HAZSLINSZKY, MÁGOCSY-DIETZ, TUZSON ap. MOESZ 17/b. p. 99.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 584.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 427.; UBRIZSY 8. p. 374.

Uromyces aconiti-lycoctoni (DC.) WINTER

Syn.: Uredo lycoctoni KALCHBR.

KALCHBRENNER 1. I. p. 306.; HAZSLINSZKY 1. p. 121.; SYDOW 2. II. p. 206.; GRESCHIK, MÁGOCSY-DIETZ, SZÉPLIGETI, ZSÁK ap. MOESZ 17/b. p. 100.; MÁGOCSY-DIETZ, ZSÁK ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 603.; PODHRADSZKY in UBRIZSY 5. p. 427.

Uromyces alchemillae (PERS.) LÉVEILLÉ

Syn.: Trachyspora alchemillae (PERS.) FUCKEL

KALCHBRENNER 1. I. p. 307.; HAZSLINSZKY 1. p. 128.; RICHTER 1. No. 3.; BÄUMLER 1. II. No. 554.; BRESADOLA 1. No. 95.; SYDOW 2. II. p. 197.; MAGNUS 1. p. 45.; HUSZ 1. p. 102.; MOESZ 12. col. 809.; HRUBY ap. MOESZ 1.; HRUBY 1. col. 1078–1085.; FILARSZKY in Krypt. exs. No. 1402., GYŐRFFY, HAZSLINSZKY, HOLUBY, KALCHBRENNER, LINHART in RABENH.–WINTER Fung. eur. exs. No. 3007. et in LINHART Fung. hung. exs. No. 208., MÁGO-

CSY-DIETZ, MOESZ, RICHTER, RŐMER ap. MOESZ 17/b. p. 100.; PICBAUER 2. p. 188.; SAVULESCU 7. p. 489.

Uromyces ambiguus (DC.) LÉVEILLÉ

BOLLA 1. p. 48.; HAZSLINSZKY 1. p. 127.; HAZSLINSZKY 3. p. 82.; HAZSLINSZKY 7. p. 91.; HOLLÓS 1. p. 141.; BÄUMLER 1. V. No. 1819.; HOLLÓS 7. p. 163.; BÄUMLER, DEGEN, GREINICH, HAZSLINSZKY, HOLUBY, KALCHBRENNER, MÁGOCSY-DIETZ, MOESZ in Krypt. exs. Vindob. No. 2305. ap. MOESZ 17/b. p. 101.; DEGEN, MOESZ ap. MOESZ 13. p. 130.; UBRIZSY 1. p. 14.; PICBAUER 1. p. 9.; PICBAUER 2. p. 188.; PICBAUER 3. p. 177.; SAVULESCU 7. p. 567.; LEHOCZKY 1. p. 236.; PODHRADSZKY in UBRIZSY 5. p. 427.; UBRIZSY 8. p. 374.

Uromyces anthyllidis (GREV.) SCHROETER

HAZSLINSZKY 3. p. 82.; BÄUMLER 1. II. No. 555.; SYDOW 2. II. p. 64.; MAGNUS 1. p. 45.; RAPAICS 1. p. 215.; HUSZ 1. p. 102.; HRUBY 1. col. 1078–1085.; BÄUMLER, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 17., MÁGOCSY-DIETZ, HAZSLINSZKY, MOESZ ap. MOESZ 17/b. p. 101.; HAZSLINSZKY, MOESZ ap. MOESZ 13. p. 130.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 622.; LEHOCZKY 1. p. 236.; TÓTH 2. p. 60.; PODHRADSZKY in UBRIZSY 5. p. 427.; UBRIZSY 8. p. 374.

Uromyces apiosporus HAZSLINSZKY

FUSS 1. VIII. p. 232.; HAZSLINSZKY 1. p. 120.; HAZSLINSZKY 5. p. 44.; HAZSLINSZKY 11. p. 262.; FUSS 2. p. 442.; SYDOW 2. II. p. 44.; MOESZ 12. col. 809.; HRUBY 1. col. 1078–1085.; GYŐRFFY in Flora hung. exs. No. 402., HAZSLINSZKY, SIMKOVICS ap. MOESZ 17/b. p. 101.; SAVULESCU 7. p. 662.

Uromyces armeriae (SCHLECHTD.) LÉVEILLÉ

SYDOW 2. II. p. 41. ap. MOESZ 17/b. p. 102.; SAVULESCU 7. p. 663.

Uromyces astragali (OPIZ) SACCARDO

Syn.: U. jordianus BUBÁK; V. euphorbiae-astragali JORDI.

LEHOCZKY 1. p. 236.

Uromyces bäumlerianus BUBÁK

HAZSLINSZKY 1. p. 124.; BUBÁK 2/a. p. 22.; BUBÁK–KABÁT 1. p. 363.; SYDOW 2. II. p. 117.; MOESZ 4. II. p. 145.; BÄUMLER 1. V. No. 1820.; HAZSLINSZKY, RICHTER ap. MOESZ 17/b. p. 102.; SAVULESCU 7. p. 630.; PODHRADSZKY in UBRIZSY 5. p. 430.; UBRIZSY 8. p. 375.

Uromyces behenis (DC.) UNGER

Syn.: Aecidium behenis DC.

HAZSLINSZKY 1. p. 125., 139.; HAZSLINSZKY 2. p. 14.; BÄUMLER 1. II. No. 557.; IV. No. (557.); SYDOW 2. II. p. 219.; MOESZ 12. col. 809.; BÄUMLER, HAYNALD, HAZSLINSZKY, LINHART in Fung. hung. exs. No. 114.; MÁGOCSY-DIETZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 308. ap. MOESZ 17/b. p. 102.; SZÉPLIGETI ap. MOESZ 13. p. 130.; PICBAUER 1. p. 9.; PICBAUER 2. p. 188.; SAVULESCU 7. p. 595.; TÓTH 2. p. 60.; PODHRADSZKY in UBRIZSY 5. p. 430.; UBRIZSY 8. p. 375.

Uromyces betae (PERS.) LÉVEILLÉ

HAZSLINSZKY 2. p. 14.; HAZSLINSZKY 3. p. 82.; BÄUMLER 1. II. No. 558.; SACCARDO 1. No. 86.; SYDOW 2. II. p. 225.; RAPAICS 1. p. 213.; BÄUMLER, HOLUBY, KMET, LINHART in Fung. hung. exs. No. 19. ap. MOESZ 17/b. p. 103.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 590.; PODHRADSZKY in UBRIZSY 5. p. 430.

Uromyces borealis LIRO

HRUBY 1. col. 1078–1085.

Uromyces brandzae SAVULESCU

SAVULESCU 7. p. 635.; VASS–TÓTH 2. p. 49.; PODHRADSZKY in UBRIZSY 5. p. 431.

Uromyces cacaliae (DC.) UNGER

FUSS 1. VIII. p. 232.; KALCHBRENNER 1. I. p. 306.; HOHENBÜHEL–HEUFLER 1. p. 66.; HAZSLINSZKY 1. p. 121.; SACCARDO 1. No. 89.; SYDOW 2. II. p. 2. HRUBY 1. col. 1078–1085.; LINHART in Fung. hung. exs. No. 307., MOESZ ap. MOESZ 17/b. p. 103.; PICBAUER 3. p. 177.; SAVULESCU 7. p. 673.

Uromyces caraganae (THÜM.) MAGNUS

SZIGETI–TÓTH 1. p. 22.

Uromyces caricis-sempervirentis FISCHER

MOESZ ap. MOESZ 17/b. p. 103.; SAVULESCU 7. p. 566.

Uromyces caryophyllinus (SCHRANK) WINTER

BÄUMLER 1. II. No. 559.; MOESZ 2. No. 55.; SYDOW 2. II. p. 211.; MOESZ 11. p. 102.; HOLLÓS 7. p. 164.; BÄUMLER, BUDAI, DEGEN, MOESZ ap. MOESZ 17/b. p. 103.; DEGEN, KARDOS, MOESZ ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 597.; PODHRADSZKY in UBRIZSY 5. p. 431.; UBRIZSY 8. p. 375.

Uromyces coronillae-variae VIENNOT–BOURGIN

SAVULESCU 7. p. 623.; TÓTH 2. p. 60.; PODHRADSZKY in UBRIZSY 5. p. 432.; UBRIZSY 8. p. 375.

Uromyces cristatus SCHROETER et NIESSL

VASS–TÓTH 2. p. 49.; PODHRADSZKY in UBRIZSY 5. p. 432.

Uromyces cristulatus TRANZSCHEL

TÓTH 2. p. 60.; PODHRADSZKY in UBRIZSY 5. p. 432.

Uromyces dactylidis OTTH

BÄUMLER 1. II. No. 560.(?), 667., IV. No. 1491.; SYDOW 2. II. p. 310.; MOESZ 11. p. 102.; BÄUMLER in Krypt. exs. Vindob. No. 909., LINHART in Fung. hung. exs. No. 213., MOESZ ap. MOESZ 17/b. p. 103.; MOESZ ap. MOESZ 13. p. 130.; PICBAUER 1. p. 9.; PICBAUER 2. p. 188.; SAVULESCU 7. p. 554.; LEHOCZKY 1. p. 236.; SZIGETI–TÓTH 1. p. 23.; PODHRADSZKY in UBRIZSY 5. p. 432.; UBRIZSY 8. p. 375.

Uromyces ervi (WALLR.) WESTENDORP

HAZSLINSZKY 1. p. 135.; BORBÁS 2. p. 33.; BÄUMLER 1. V. No. 1821.; HAZSLINSZKY, MOESZ, SADLER ap. MOESZ 17/b. p. 104.; SADLER ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 612.; PODHRADSZKY in UBRIZSY 5. p. 432.

Uromyces erythronii (DC.) PASSERINI

FUSS 1. IV. p. 112.; SCHULZER 2. p. 10.; HAZSLINSZKY 1. p. 141.; FUSS 2. p. 442.; SIMONKAI 1. No. 1653.; SYDOW 2. II. p. 270.; MOESZ 30. II. p. 374.; BARTH, ÉHIK, FUSS, LOJKA in RABENH. Fung. eur. exs. No. 1890. ap. MOESZ 17/b. p. 104.; SAVULESCU 7. p. 573.

Uromyces euphorbiae-astragali (OPIZ) JORDI

Syn.: U. astragali SACC., BUB., SYDOW; U. punctatus SCHROET. ppe.

KALCHBRENNER 1. I. p. 305.; HAZSLINSZKY 1. p. 124.; HAZSLINSZKY 2. p. 13.; FUSS 2.p. 442.; BORBÁS 2. p. 33.; RICHTER 1. No. 4.; BÄUMLER 1. IV. No. 1491.; MOESZ 2. No. 54.; SYDOW 2. II. p. 67.; HOLLÓS 1. p. 140.; MOESZ 11. p. 102.; HOLLÓS 7. p. 164.; MOESZ 30. II. p. 374.; HRUBY 1. col. 1078–1085.; BÄUMLER, BÁNYAI, BUDAI, DEGEN, HAZSLINSZKY, HOLUBY, KMET, KÜMMERLE, LAUDON, LÁNYI, MÁGOCSY-DIETZ in Krypt. exs. No. 2304., MÁRTON MOESZ, PALLA, RICHTER, SZÉPLIGETI ap. MOESZ 17/b. p. 104.; DEGEN, LÁNYI, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 614.; TÓTH 2. p. 60.; VASS 1. p. 50.; TÓTH 10. p. 10.; PODHRADSZKY in UBRIZSY 5. p. 432.; UBRIZSY 8. p. 375.

Uromyces fabae (PERS.) DE BARY

Syn.: U. viciae-fabae (PERS.) JÖRSTAD; U. viciae-craccae CONST.

FUSS 1. IV. p. 11., VIII. p. 232.; BOLLA 1. p. 48., 50.; KALCHBRENNER 1. I. p. 303., 306.; SCHULZER 1. No. 40.; SCHULZER 2. p. 17.; HAZSLINSZKY 1. p. 123., 124.; FUSS 2. p. 442.; HAZSLINSZKY 2. p. 13.; HAZSLINSZKY 7. p. 9.; BÄUMLER 1. II. No. 562.; BRESADOLA 1. No. 97.; SACCARDO 1. No. 84.; BUBÁK 1. No. 23.; MOESZ 2. No. 56.; SYDOW 2. II. p. 103., 107.; HOLLÓS 1. p. 140.; RAPAICS 1. p. 213.; HUSZ 1. p. 102.; MOESZ 11. p. 102.; MOESZ 12. col. 809. HOLLÓS 7. p. 164., 165.; BÄUMLER, BOLLA, FUSS, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, KRENNER, LINHART in Fung. hung. exs. No. 411., LAUDON, MÁGOCSY-DIETZ, MÁRTON, MOESZ, PÓSCH, SZÉPLIGETI in LINHART Fung. hung. exs. No. 309. ap. MOESZ 17/b. p. 105.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 130.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 607.; TÍMÁR 1. p. 237.; TÓTH 2. p. 61.; VASS–TÓTH 2. p. 49.; PODHRADSZKY in UBRIZSY 5. p. 440.; UBRIZSY 8. p. 375.

Uromyces fabae (PERS.) DE BARY f. *viciae sepium* GÄUMANN

LEHOCZKY 1. p. 236.

Uromyces ficariae (SCHUM.) LÉVEILLÉ

BOLLA 1. p. 47.; HAZSLINSZKY 1. p. 121.; HAZSLINSZKY 3. p. 82.; BÄUMLER 1. No. 563.; SYDOW 2. II. p. 209.; HOLLÓS 1. p. 141.; HOLLÓS 7. p. 164.; BÄUMLER, BOLLA, BUDAI, DEGEN, HAZSLINSZKY, KMET in Flora exs. Austro–Hung. No. 1565., LINHART in Fung., hung. exs. No. 112., MOESZ ap. MOESZ 17/b. p. 106.; DEGEN, MOESZ, PÉNZES ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 604.; TÓTH 2. p. 60.; SZIGETI–TÓTH 1. p. 23.; VASS–TÓTH 3. p. 85.; PODHRADSZKY in UBRIZSY 5. p. 432.; UBRIZSY 8. p. 375.

Uromyces fischeri-eduardi MAGNUS

MOESZ 2. No. 57.; MOESZ ap. MOESZ 17/b. p. 106.; MOESZ ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 621.; UBRIZSY in litt.

Uromyces fulgens (HAZSL.) BUBÁK

Syn.: Aecidium fulgens HAZSL.

KALCHBRENNER 1. I. p. 305.; HAZSLINSZKY 1. p. 133.; BORBÁS 2. p. 33.; BÄUMLER 1. II. No. 564., V. No. 1822.; SYDOW 2. II. p. 90.; HOLLÓS 1. p. 140.; HOLLÓS 7. p. 164.; DEGEN, HAZSLINSZKY, MOESZ ap. MOESZ 17/b. p. 106.; DEGEN, HAZSLINSZKY, MOESZ ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 640.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces gageae BECK

SYDOW 2. II. p. 273.; BÄUMLER 1. V. No. 1823.; BÄUMLER, HAZSLINSZKY, LINHART in Fung. hung. exs. No. 405., MOESZ, PÉNZES, SZÉPLIGETI ap. MOESZ 17/b. p. 107.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 130.; SAVULESCU 7. p. 572.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces galegae (OPIZ) SACCARDO

Syn.: U. galegicola WORON

MOESZ 11. p. 102.; MOESZ 12. col. 809.; MOESZ 21. p. 85.; MOESZ ap. MOESZ 17/b. p. 107.; SAVULESCU 7. p. 625.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces genistae-tinctoriae (PERS.) WINTER

Syn.: U. genistae FUCKEL; Coleosporium fuscum HAZSL.; Uredo pteleae HAZSL. BOLLA 1. p. 48.; KALCHBRENNER 1. I. p. 305., 307.; HAZSLINSZKY 1. p. 106., 124.; HAZSLINSZKY 2. p. 13., 14.; HAZSLINSZKY 3. p. 82.; HAZSLINSZKY 5. p. 45.; HAZSLINSZKY 7. p. 9.; BORBÁS 2. p. 33.; BÄUMLER 1. II. No. 564.; BRESADOLA 1. No. 94.; SYDOW 2. II. p. 91., III. p. 656.; HOLLÓS 1. p. 140.; MOESZ 11. p. 102.; HOLLÓS 7. p. 164.; BÄUMLER, HAZSLINSZKY, HOLLÓS, LINHART in Fung. hung. exs. No. 209., MÁGOCSY-DIETZ, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ, 17/b. p. 107. HAZSLINSZKY, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 130.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 626.; TÓTH 2. p. 60.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 433.; UBRIZSY 8. p. 375.

Uromyces geranii (DC.) FRIES

BOLLA 1. p. 47.; KALCHBRENNER 1. I. p. 308.; HAZSLINSZKY 1. p. 165.; BRESADOLA 1. No. 90,; MAGNUS 1. p. 45.; HOLLÓS 1. p. 141.; MOESZ 12. col. 809.; HOLLÓS 7. p. 165.; BÄUMLER, GREINICH, HOLUBY, KMET, MÁGOCSY-DIETZ, MOESZ, PERLAKY ap. MOESZ 17/b. p. 108.; PERLAKY ap. MOESZ p. 130.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 660.; PODHRADSZKY in UBRIZSY 5. p. 433.; UBRIZSY 8. p. 375.

Uromyces graminis (NIESSL) DIETEL

Syn.: Aecidium seseli NIESSL

HAZSLINSZKY, STAUB ap. MOESZ 17/b. p. 109.; HAZSLINSZKY, STAUB ap. MOESZ 13. p. 130.; PICBAUER 1. p. 9.; SAVULESCU 7. p. 560.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces hedysari-obscuri (DC.) CARESTIA et PICCONE

Syn.: U. hedysari HAZSL.

KALCHBRENNER 1. I. p. 303., 306.; HAZSLINSZKY 1. p. 123.; FUSS 2. p. 442.; MOESZ 12. col. 809.; SYDOW 2. II. p. 99.; GYŐRFFY in Flora hung. exs. No.

706., KALCHBRENNER, LINHART in Fung. hung. exs. No. 210. ap. MOESZ 17/b. p. 109.; SAVULESCU 7. p. 643.

Uromyces heimerlianus MAGNUS

RICHTER 1. No. 5.; SYDOW 2. II. p. 361.; FILARSZKY, KÜMMERLE, RICHTER ap. MOESZ 17/b. p. 109.; FILARSZKY, KÜMMERLE ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 633.; TÓTH 2. p. 60.; TÍMÁR 1. p. 237.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces inaequialtus LASCH

Syn.: U. silenes (SCHLECHT.) FUCKEL; Aecidium sparsum HAZSL.

HAZSLINSZKY 1. p. 136., 138.; BÄUMLER 1. II. No. 576., IV. (No. 576.); BUBÁK 1. No. 26.; SYDOW 2. II. p. 218.; MOESZ 31. p. 96.; BARTH in Fung. hung. exs. No. 311., et in THUEMEN Fung. austr. exs. No. 1119., BÄUMLER, BUDAI, GÁYER, HAZSLINSZKY, KALCHBRENNER, KUNSZT, MICHAELIS ap. MOESZ 17/b. p. 109.; HAZSLINSZKY, SADLER ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 591.; VASS 1. p. 50.; VASS—TÓTH 2. p. 49.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces jordianus BUBÁK

Syn.: U. astragali JORDI

BUBÁK 1. No. 24.; SYDOW 2. II. p. 69.; HOLLÓS 1. p. 140.; BOROS, KÜMMERLE, ZSÁK ap. MOESZ 17/b. p. 110.; BUBÁK, KÜMMERLE, ZSÁK ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 624.; LEHOCZKY 1. p. 236.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces junci (DESM.) TULASNE

BÄUMLER 1. II. No. 566.; SYDOW 2. II. p. 288.; MOESZ 11. p. 102.; HOLLÓS 7. p. 165.; BÄUMLER, FILARSZKY—SCHILBERSZKY in Krypt. exs. Vindob. No. 25., LINHART in Fung. hung. exs. No. 413., MOESZ, PÉNZES, ZSÁK ap. MOESZ 17/b. p. 110.; FILARSZKY, MÁGOCSY-DIETZ, PÉNZES, SCHILBERSZKY, ZSÁK ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 577.; SZIGETI—TÓTH 1. p. 23.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces kabatianus BUBÁK

BÄUMLER 1. II. No. 565.; KÁLOVITS in Flora hung. exs. No. 504.; MICHAELIS, SCHNELLER ap. MOESZ 17/b. p. 110.; PICBAUER 1. p. 9.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 659.; PODHRADSZKY in UBRIZSY 5. p. 433.

Uromyces kalmusii SACCARDO

BÄUMLER ap. MOESZ 17/b. p. 110.

Uromyces laevis KÖRNICKE

Syn.: U. scutellatus FISCHER ppe.

MOESZ 17/b. p. 111.; MOESZ ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 657.; PODHRADSZKY in UBRIZSY 5. p. 433.; UBRIZSY 8. p. 375.

Uromyces lathyri-latifolii GUYOT

SAVULESCU 7. p. 639.; UBRIZSY 8. p. 375.

Uromyces lilii (LK.) FUCKEL

Syn.: U. erythronii (DC.) PASS. ppe.

BÄUMLER 1. II. No. 561., V. No. 1824.; BRESADOLA 1. No. 96.; SYDOW 2. II. p. 278.; HOLLÓS 7. p. 165.; MOESZ 31. p. 96.; BÄUMLER, GÁYER, SCHMIDT

ap. MOESZ 17/b. p. 111.; SAVULESCU 7. p. 574.; PODHRADSZKY in UBRIZSY 5. p. 434.; UBRIZSY 8. p. 375.

Uromyces limonii (DC.) LÉVEILLÉ

Syn.: Aecidium statices RUD.

HAZSLINSZKY 1. p. 121., 142.; FUSS 2. p. 438.; BORBÁS 2. p. 33.; SYDOW 2. II. p. 42.; HOLLÓS 1. p. 140.; BUDAI, HAZSLINSZKY, KŐFARAGÓ—GYELNIK, LINHART in Fung. hung. exs. No. 211., MOESZ, PÉNZES, RIGLER, TUZSON, ZSÁK ap. MOESZ 17/b. p. 111.; HAZSLINSZKY, MOESZ, PÉNZES, TUZSON ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 664.; TÓTH 2. p. 61.; TÍMÁR 1. p. 237.; PODHRADSZKY in UBRIZSY 5. p. 434.

Uromyces lineolatus (DESM.) SCHROETER

Syn.: U. scirpi (CAST.) BURRIL

HOLLÓS 1. p. 141.; MOESZ 11. p. 102.; HOLLÓS 7. p. 166.; SYDOW 2. II. p. 304.; BARTH in THUEMEN Fung. austr. exs. No. 1120., GREINICH, LINHART in Fung. hung. exs. No. 207., MOESZ, RECHINGER in Krypt. exs. No. 910, ap. MOESZ 17/b. p. 112.; MOESZ ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 563.; TÓTH 2. p. 61.; PODHRADSZKY in UBRIZSY 5. p. 437.; UBRIZSY 8. p. 376.

Uromyces loti BLYTT

KALCHBRENNER 1. I. p. 305.; HAZSLINSZKY 1. p. 124.; SYDOW 2. II. p. 110., HOLLÓS 1. p. 140.; HOLLÓS 7. p. 165.; HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 112.; MOESZ ap. MOESZ 13. p. 131.; SAVULESCU 7. p. 617.; PODHRADSZKY in UBRIZSY 5. p. 434.

Uromyces lupinicolus BUBÁK

SAVULESCU 7. p. 629.; PODHRADSZKY in UBRIZSY 5. p. 434.; UBRIZSY 8. p. 376.

Uromyces minor SCHROETER

Syn.: Aecidium trifolii HAZSL.

HAZSLINSZKY 1. p. 139.; HAZSLINSZKY 7. p. 9.; HOLUBY 2. p. 18.; HAZSLINSZKY 3. p. 77., 82.; BÄUMLER 1. II. No. 567.; SACCARDO 1. No. 90.; SYDOW 2. II. p. 135.; HUSZ 1. p. 102.; BÄUMLER in RABENH.—WINTER Fung. eur. exs. No. 3013. Suppl. a, b, DEGEN, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, LINHART in Fung. hung. exs. No. 310., TUZSON, SABRANSKY, SZÉPLIGETI ap. MOESZ 17/b. p. 112.; DEGEN, MÁGOCSY-DIETZ ap. MOESZ 13. p. 132.; SAVULESCU 7. p. 651.; PODHRADSZKY in UBRIZSY 5. p. 434.

Uromyces onobrychidis (DESM.) LÉVEILLÉ

BRESADOLA 1. No. 94.; SYDOW 2. II. p. 118.; HOLLÓS 1. p. 140.; HOLLÓS 7. p. 165.; GRESCHIK, MOESZ ap. MOESZ 17/b. p. 113.; MOESZ ap. MOESZ 13. p. 132.; SAVULESCU 7. p. 631.; TÓTH 2. p. 61.; PODHRADSZKY in UBRIZSY 5. p. 434.

Uromyces ononidis PASSERINI

HAZSLINSZKY 2. p. 14.; RICHTER 1. No. 4.; BÄUMLER 1. II. No. 568.; SYDOW 2. II. p. 119.; HOLLÓS 1. p. 140.; MOESZ 11. p. 102.; HOLLÓS 7. p. 165.; BÄUMLER in Krypt. exs. No. 1302., BUDAI, GYŐRFFY—KOL, MOESZ, MOESZ—

TIMKÓ in Flora hung. exs. No. 605. ap. MOESZ 17/b. p. 113.; PICBAUER 3. p. 177.; SAVULESCU 7. p. 631.; UBRIZSY 8. p. 376.

Uromyces orobi (PERS.) LÉVEILLÉ

Syn.: Aecidium orobi PERS.; Ae. orobi-tuberosi PERS.

SYDOW 2. II. p. 107. et ap. MOESZ 17/b. 113.; SAVULESCU 7. p. 610.; VASS 1. p. 50.; VASS—TÓTH 2. p. 49.; PODHRADSZKY in UBRIZSY 5. p. 434.

Uromyces ornithogali LÉVEILLÉ

SAVULESCU 7. p. 576.; VASS—TÓTH 3. p. 85.

Uromyces pallidus NIESSL

KALCHBRENNER 1. I. p. 305.; HOLLÓS 7. p. 165. ap. MOESZ 17/b. p. 113.; SAVULESCU 7. p. 613.; PODHRADSZKY in UBRIZSY 5. p. 434.

Uromyces phaseoli (PERS.) WINTER

Syn.: U. appendiculatus (PERS.) LINK

FUSS 1. IV. p. 111.; BOLLA 1. p. 48.; KALCHBRENNER 1. I. p. 306.; SCHULZER 1. No. 45.; SCHULZER 2. p. 26.; HAZSLINSZKY 1. p. 122.; FUSS 2. p. 442.; SIMONKAI 1. No. 1788.; HOLLÓS 1. p. 141.; SYDOW 2. II. p. 121.; RAPAICS 1. p. 215.; MOESZ 11. p. 102.; MOESZ 21. p. 85.; MOESZ 12. col. 809.; HOLLÓS 7. p. 164.; MOESZ 30. II. p. 374.; BARTH, BÄUMLER, BÁNYAI, FUSS, GREINICH, HAZSLINSZKY, LAUDON, LINHART in Fung. hung. exs. No. 20., MÁGOCSY-DIETZ in Krypt. exs. Vindob. No. 13/b., in Flora hung. exs. No. 705., MÁRTON, MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 114.; MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 132.; UBRIZSY 1. p. 14.; SAVULESCU 7. p. 644.; TÓTH 2. p. 60.; PODHRADSZKY in UBRIZSY 5. p. 427.; UBRIZSY 8. p. 376.

Uromyces phyteumatum (DC.) UNGER

HAZSLINSZKY 1. p. 125.; SYDOW 2. II. p. 17.; HOLUBY, KMET, ap. MOESZ 17/b. 114.; SAVULESCU 7. p. 673.

Uromyces pisi (PERS.) SCHROETER

Syn.: Aecidium euphorbiae GMEL. ppe.

BOLLA 1. p. 47.; KALCHBRENNER 1. I. p. 306.; SCHULZER 1. No. 44.; SCHULZER 2. p. 26.; HAZSLINSZKY 1. p. 123., 124., 134.; HAZSLINSZKY 2. p. 14.; HAZSLINSZKY 3. p. 82.; HAZSLINSZKY 7. p. 9.; SACCARDO 1. No. 87.; BÄUMLER 1. II. No. 570.; SYDOW 2. II. p. 125.; HOLLÓS 1. p. 141.; RAPAICS 1. p. 215.; HOLLÓS 7. p. 165.; MOESZ 30. II. p. 374.; MOESZ 38. p. 172.; BÄUMLER, BERNÁTSKY, BUDAI, CHYZER, GRESCHIK, HAZSLINSZKY, HAYNALD, HOLUBY, KALCHBRENNER, KMET in Flora exs. Austro—Hung. No. 3166., KÜMMERLE, LAUDON, LÁNYI, MÁGOCSY-DIETZ, MOESZ, PÉNZES, PÓSCH, RIGLER, SZÉPLIGETI, ZAHLBRUCKNER ap. MOESZ 17/b. p. 114.; BERNÁTSKY, MOESZ, PÉNZES, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 132.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 619.; LEHOCZKY 1. p. 236.; TÓTH 2. p. 61.; VASS—TÓTH 1. p. 159.; VASS 1. p. 50.; TÍMÁR 1. p. 237.; VÖRÖS 9. p. 102.; PODHRADSZKY in UBRIZSY 5. p. 434.; UBRIZSY 8. p. 376.

Uromyces poae RABENHORST

Syn.: Aecidium ficariae PERS.; Ae. ranunculacearum DC. ppe.

FUSS 1. IV. p. 125.; HAZSLINSZKY 1. p. 136.; FUSS 2. p. 437.; BÄUMLER 1.

II. No. 560.; Bresadola 1. No. 92.; Simonkai 1. No. 1652.; Sydow 2. II. p. 312.; Moesz 11. p. 102.; Hollós 7. p. 166.; Bäumler, Bolla, Greschik, Hazslinszky, Kmet, Linhart in Fung. hung. exs. No. 115., Mágocsy-Dietz, Moesz, Zahlbruckner ap. Moesz 17/b. p. 116.; Moesz—Mágocsy-Dietz ap. Moesz 13. p. 132.; Savulescu 7. p. 557.; Podhradszky in Ubrizsy 5. p. 436.; Ubrizsy 6. in Székessy p. 23.; Ubrizsy 8. p. 376.

Uromyces polygoni (Pers.) Fuckel

Syn.: Uredo polygonorum DC.

Fuss 1. IV. p. 111.; Bolla 1. p. 48.; Hazslinszky 1. p. 126.; Hazslinszky 2. p. 14.; Fuss 2. p. 442.; Hazslinszky 7. p. 9.; Bäumler 1. II. No. 571.; Bresadola 1. No. 89.; Sydow 2. II. p. 238.; Hollós 1. p. 141.; Hollós 7. p. 166.; Bäumler, Hazslinszky, Holuby, Jávorka, Kőfaragó-Gyelnik, Márton, Moesz, Szépligeti ap. Moesz 17/b. p. 116.; Jávorka, Kőfaragó-Gyelnik, Moesz, Szépligeti ap. Moesz 13. p. 132.; Savulescu 7. p. 579.; Tóth 2. p. 61.; Tímár 1. p. 237.; Podhradszky in Ubrizsy 5. p. 436.; Ubrizsy 8. p. 376.

Uromyces proëminens (DC.) Léveillé

Vajda ap. Moesz 17/b. p. 117.; Savulescu 7. p. 654.

Uromyces renovatus Sydow

Syn.: U. lupini Sacc.

Moesz ap. Moesz 17/b. p. 117.; Ubrizsy 2. p. 154.; Podhradszky in Ubrizsy 5. p. 436.

Uromyces reticulatus (Thüm.) Bubák

Kalchbrenner 1. I. p. 304., 306.; Hazslinszky 1. p. 127., 141.; Bresadola 1. No. 96.; Sydow 2. II. p. 261.; Greschik, Vajda ap. Moesz 17/b. p. 117.; Podhradszky in Ubrizsy 5. p. 436.

Uromyces rumicis (Schum.) Winter

Syn.: Uredo rumicum DC.

Kalchbrenner 1. I. p. 306., 310.; Hazslinszky 1. p. 126.; Borbás 2. p. 33.; Hazslinszky 3. p. 82.; Bäumler 1. II. No. 572.; Bresadola 1. No. 93.; Sydow 2. II. p. 240.; Hollós 1. p. 141.; Moesz 11. p. 102.; Hollós 7. p. 166.; Bäumler, Budai, Greinich, Greschik, Hazslinszky, Mágocsy-Dietz in Krypt. exs. No. 15., Moesz, Szépligeti in Linhart Fung. hung. exs. ap. Moesz 17/b. p. 117.; Moesz, Szépligeti ap. Moesz 13. p. 132.; Ubrizsy 2. p. 154.; Savulescu 7. p. 582.; tóth 2. p. 61.; Vass 1. p. 50.; Vass—Tóth 3. p. 85.; Podhradszky in Ubrizsy 5. p. 436.; Ubrizsy 8. p. 376.

Uromyces salsolae Reichardt

Reichardt 1. p. 842.; Bäumler 1. IV. No. 1492.; Sydow 2. II. p. 232.; Bäumler ap. Moesz 17/b. p. 118.; Savulescu 7. p. 587.; Podhradszky in Ubrizsy 5. p. 436.

Uromyces scillarum (Grev.) Winter

Hazslinszky 1. p. 126.; Hazslinszky 3. p. 82.; Bäumler 1. II. No. 569., 573.; Saccardo 1. No. 91.; Moesz 2. No. 59.; Sydow 2. II. p. 279.; Hollós

1. p. 141.; HOLLÓS 7. p. 166.; BÄUMLER, BERNÁTSKY, BOLLA, BUDAI, DEGEN, FILARSZKY, GRENICH, GYŐRFFY, HOLUBY, KMET, KÜMMERLE, LINHART in Fung. hung. exs. No. 15., MÁGOCSY-DIETZ in Krypt. exs. No. 17., MOESZ, PÉNZES, PÓSCH, SZÉPLIGETI, ZSÁK ap. MOESZ 17/b. p. 118.; BERNÁTSKY, DEGEN, GYŐRFFY, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ, PÉNZES, ZSÁK ap. MOESZ 13. p. 132.; SAVULESCU 7. p. 569.; VASS–TÓTH 3. p. 85.; PODHRADSZKY in UBRIZSY 5. p. 436.; UBRIZSY 8. p. 376.

Uromyces scleranthi ROSTRUP

SYDOW 2. II. p. 217.; HAYNALD–MOESZ ap. MOESZ 17/b. p. 118.; SAVULESCU 7. p. 601.

Uromyces scrophulariae (DC.) FUCKEL

Syn.: Aecidium scrophulariae DC.

HAZSLINSZKY 3. p. 75.; BÄUMLER 1. II. No. 574.; BUBÁK 1. No. 25.; SYDOW 2. II. p. 28.; HOLLÓS 1. p. 140.; MOESZ 12. col. 809.; HOLLÓS 7. p. 166.; BÄUMLER, BUDAI, FILARSZKY, GRESCHIK, KMET, LINHART in Fung. hung. exs. No. 113., MÁGOCSY–DIETZ, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 410., WIERZBICKY ap. MOESZ 17/b. p. 118.; SZÉPLIGETI ap. MOESZ 13. p. 132.; UBRIZSY 1. p. 14., 15.; PICBAUER 1. p. 10.; PICBAUER 2. p. 188., SAVULESCU 7. p. 669.; TÓTH 2. p. 61.; PODHRADSZKY in UBRIZSY 5. p. 438.

Uromyces scutellatus (SCHRANK) LÉVEILLÉ

SADLER 1. VII. No. 24.; ENDLICHER 1. No. 78.; FUSS 1. IV., III., VIII. p. 232.; HAZSLINSZKY 1. p. 127.; FUSS 2. p. 442.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 82.; BÄUMLER 1. II. No. 575.; SYDOW 2. II. p. 178.; HOLLÓS 1. p. 141.; HOLLÓS 7. p. 167.; MOESZ 30. p. 7., II. p. 374.; BÄUMLER, BÁNYAI, BUDAI, GREINICH, GYŐRFFY, HAZSLINSZKY, JÁVORKA, KMET, LINHART in Fung. hung. exs. No. 406., MOESZ, PÓSCH, SADLER, SZÉPLIGETI, WIERZBICKY ap. MOESZ 17/b. p. 119.; JÁVORKA, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 132.; TÓTH 2. p. 61.; VASS–TÓTH 1. p. 159.; TÍMÁR 1. p. 237.; SZIGETI–TÓTH 1. p. 23.; PODHRADSZKY in UBRIZSY 5. p. 438.

Uromyces striatus SCHROETER

Syn.: Aecidium cyparissiae DC. ppe.

SCHULZER 2. p. 18.; HAZSLINSZKY 1. p. 124.; HAZSLINSZKY 2. p. 14.; HAZSLINSZKY 7. p. 9.; RICHTER 1. No. 6.; BÄUMLER 1. II. No. 577.; SYDOW 2. II. p. 116.; HOLLÓS 1. p. 140.; MOESZ 11. p. 102.; HOLLÓS 7. p. 167.; MOESZ 30. p. 7.; BÄUMLER in Krypt. exs. Vindob. No. 14., FLEISCHMANN, GREINICH, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MÁRTON, MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 119.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 132.; UBRIZSY 2. p. 154.; SAVULESCU 7. p. 635.; TÓTH 2. p. 61.; VASS 1. p. 50.; TÍMÁR 1. p. 237.; SZIGETI–TÓTH 1. p. 23.; PODHRADSZKY in UBRIZSY 5. p. 438.; UBRIZSY 8. p. 376.

Uromyces sublaevis TRANZSCHEL

MOESZ 31. p. 96.; DEGEN, FILARSZKY, GÁYER, JÁVORKA, KÜMMERLE. MÁGOCSY-DIETZ, MOESZ, MÜLLER, SZÉPLIGETI, ZORKÓCZY ap. MOESZ, 17/b.

p. 120.; SAVULESCU 7. p. 656.; PODHRADSZKY in UBRIZSY 5. p. 438.; UBRIZSY 8. p. 377.

Uromyces terebinthi (DC.) WINTER

SYDOW 2. II. p. 143.; KÜMMERLE, LINHART in Fung. hung. exs. No. 305. ap. MOESZ 17/b. p. 120.

Uromyces thapsi (OPIZ) BUBÁK

SCHULZER 2. p. 10.; HAZSLINSZKY 1. p. 140.; HAZSLINSZKY 2. p. 15.; BORBÁS 2. p. 33.; HAZSLINSZKY 3. p. 82.; BÄUMLER 1. II. No. 574., V. No. 1825.; SYDOW 2. II. p. 29.; HOLLÓS 1. p. 140.; MOESZ 11. p. 102.; HOLLÓS 7. p. 167.; BÄUMLER, BOROS, BUDAI, GREINICH, HAZSLINSZKY, HOLUBY, KMET, LINHART in Fung. hung. exs. No. 306., MÁRTON, MOESZ, SCHULZER in LINHART Fung. hung. exs. No. 18., SIMKOVITS ap. MOESZ 17/b. p. 120.; SADLER ap. MOESZ 13. p. 133.; UBRIZSY 1. p. 15.; PICBAUER 1. p. 10.; SAVULESCU 7. p. 667.; PODHRADSZKY in UBRIZSY 5. p. 438.

Uromyces tinctoriicola MAGNUS

MOESZ 11. p. 102.; BOLLA, MÁGOCSY-DIETZ, MOESZ, NAGY, SABRANSKY, SZÉPLIGETI ap. MOESZ 17/b. p. 121.; MOESZ, SSÉPLIGETI ap. MOESZ 13. p. 133.; SAVULESCU 7. p. 655.; TÓTH 2. p. 61.; SZIGETI—TÓTH 1. p. 23.; PODHRADSZKY in UBRIZSY 5. p. 438.

Uromyces trifolii (HEDW. f.) LÉVEILLÉ

HAZSLINSZKY 1. p. 124.; FUSS 2. p. 442., 445.; BÄUMLER 1. II. No. 578.; SYDOW 2. II. p. 133.; HOLLÓS 1. p. 141.; HOLLÓS 7. p. 167.; BÄUMLER, HAVASS, HAZSLINSZKY, HODOSSY, HOLUBY, LÁNYI, MÁGOCSY-DIETZ ap. MOESZ 17/b. p. 121.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 647.; TÓTH 2. p. 61.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 438.; UBRIZSY 8. p. 377.

Uromyces trifolii-hybridi PAUL.

GREINICH, MOESZ ap. MOESZ 17/b. p. 122.; PODHRADSZKY in UBRIZSY 5. p. 439.

Uromyces trifolii-repentis (CAST.) LIRO

SACCARDO 1. No. 85.; SYDOW 2. II. p. 132.; RAPAICS 1. p. 215.; MOESZ 21. p. 85.; BÄUMLER, GREINICH, KMET, MOESZ ap. MOESZ 17/b. p. 122.; MOESZ ap. MOESZ 13. p. 133.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 649.; TÓTH 2. p. 61.; VASS 1. p. 51.; PODHRADSZKY in UBRIZSY 5. p. 439.; UBRIZSY 8. p. 377.

Uromyces trigonellae PATOUILLARD

SAVULESCU 7. p. 632.; TÓTH 2. p. 61.; PODHRADSZKY in UBRIZSY 5. p. 439.

Uromyces valerianae (SCHUM.) FUCKEL

SYDOW 2. II. p. 20.; MOESZ 2. No. 61.; MOESZ 11. p. 102.; et FILARSZKY, LINHART in Fung. hung. exs. no. 212. ap. MOESZ 17/b. p. 122.; MOESZ ap. MOESZ 13. p. 133.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 671.; PODHRADSZKY in UBRIZSY 5. p. 439.

Uromyces veratri (DC.) SCHROETER

HAZSLINSZKY 3. p. 78., 82.; SYDOW 2. II. p. 285.; HUSZ 1. p. 102.; KALCHBRENNER in RABENH. Fung. eur. exs. No. 2476., LINHART in Fung. hung. exs. No. 16. ap. MOESZ 17/b. p. 122.; SAVULESCU 7. p. 570.; UBRIZSY 8. p. 377.

Uromyces verruculosus SCHROETER

BÄUMLER 1. III. No. 1393., V. No. 1826., SYDOW 2. II. p. 216.; BÄUMLER, HAZSLINSZKY, KMET ap. MOESZ 17/b. p. 122.; SAVULESCU 7. p. 600.

Uromyces viciae-craccae CONSTANTINEANU

SACCARDO 1. No. 88.; BUBÁK 1. No. 27.; SYDOW 2. II. p. 138.; BÄUMLER, MOESZ ap. MOESZ 17/b. p. 122.; BUBÁK, MOESZ ap. MOESZ 13. p. 133.; SAVULESCU 7. p. 638.; PODHRADSZKY in UBRIZSY 5. p. 439.; UBRIZSY 8. p. 377.

Genus. *Cumminsiella* ARTHUR

Cumminsiella sanguinea (PECK) ARTHUR

Syn.: Uropyxis sanguinea (PECK) ARTHUR

HUSZ 3/c. p. 140.; HUSZ 5. p. 120.; MOESZ 1..; HUSZ, MOESZ, NAGY ap. MOESZ 17/b. p. 123.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 531.; TÓTH 2. p. 57.; PODHRADSZKY in UBRIZSY 5. p. 420.

Genus. *Xenodochus* SCHLECHTENDAL

Xenodochus carbonarius SCHLECHTENDAL

Syn.: Phragmidium carbonarius (SCHL.) WINTER

BRESADOLA 1. No. 136.; SYDOW 2. III. p. 158.; GRESCHIK, MOESZ ap. MOESZ 17/b. p. 123.; MOESZ ap. MOESZ 13. p. 133.; SAVULESCU 7. p. 525.; PODHRADSZKY in UBRIZSY 5. p. 420.

Xenodochus tormentillae (FUCK.) MAGNUS

Syn.: Phragmidium tormentillae FUCKEL; Ph. obtusum (STR.) WINTER

BORBÁS 2. p. 33.; SYDOW 2. III. p. 106.; BÄUMLER 1. V. No. 1874.; GRESCHIK ap. MOESZ 17/b. p. 123.; BORBÁS ap. MOESZ 13. p. 133.; SAVULESCU 7. p. 493.; PODHRADSZKY in UBRIZSY 5. p. 420.

Genus. *Endophyllum* LÉVEILLÉ

Endophyllum euphorbiae-silvaticae (DC.) WINTER

KALCHBRENNER 1. I. p. 313.; BRESADOLA 1. No. 98.; HOLLÓS 7. p. 147. ap. MOESZ 17/b. p. 127.; SAVULESCU 7. p. 1078.; VASS—TÓTH 1. p. 158.; SZIGETI—TÓTH 1. p. 20.; DOHY—KIRÁLY in UBRIZSY 5. p. 472.; UBRIZSY 8. p. 378.

Endophyllum sempervivi (ALB. et SCHW.) DE BARY

KALCHBRENNER 1. I. p. 302.; HAZSLINSZKY 1. p. 130.; SYDOW 2. III. p. 530.; MAGNUS 1. p. 46.; MOESZ 12. col. 806.; MOESZ 28. p. 174.; FILARSZKY, HAZSLINSZKY, KALCHBRENNER, KARDOSS, MÁGOCSY-DIETZ, MOESZ, SZU-

RÁK ap. MOESZ 17/b. p. 127.; MOESZ, SZEPESFALVY ap. MOESZ 13. p. 113.; PICBAUER 1. p. 9.; SAVULESCU 7. p. 1079.; DOHY–KIRÁLY in UBRIZSY 5. p. 472.; UBRIZSY 8. p. 378.

Familia. Melampsoraceae

Genus. *Calyptospora* KÜHN

Calyptospora goeppertiana KÜHN

Syn.: Melampsora goeppertiana (KÜHN) WINTER; Aecidium columnare ALB. et SCHW.

KALCHBRENNER 1. I. p. 303.; HAZSLINSZKY 1. p. 130.; HAZSLINSZKY 3. p. 81.; SYDOW 2. III. p. 472.; MOESZ 12. col. 805.; MOESZ 30. p. 372.; BÁNYAI, BOROS, FILARSZKY, GRESCHIK, GYŐRFFY, KALCHBRENNER, KMET, LINHART in Fung. hung. exs. No. 240., MÁGOCSY-DIETZ in Krypt. exs. No. 816/b., SCHILBERSZKY in Krypt. exs. No. 816/a, TUZSON ap. MOESZ 17/b. p. 124.; SAVULESCU 7. p. 362.

Genus. *Chrysomyxa* UNGER

Chrysomyxa abietis (WALLR.) UNGER

SYDOW 2. III. p. 519.; MOESZ 12. col. 805.; PICBAUER 1. p. 9.; PICBAUER 2. p. 187.; FILARSZKY, GRESCHIK, HAZSLINSZKY, MOESZ, TUZSON ap. MOESZ 17/b. p. 124.; SAVULESCU 7. p. 399.

Chrysomyxa empetri (PERS.) ROSTRUP

PICBAUER 1. p. 9.; PICBAUER 2. p. 187.; LINHART in Fung. hung. exs. No. 342. ap. MOESZ 17/b. p. 124.

Chrysomyxa ledi (ALB. et SCHW.) DE BARY

SYDOW 2. III. p. 505. ap. MOESZ 17/b. p. 125.

Chrysomyxa pyrolae (DC.) ROSTRUP

HAZSLINSZKY 1. p. 115.; RICHTER 1. No. 43.; SYDOW 2. III. p. 518.; MOESZ 12. col. 805.; GRESCHIK ap. MOESZ 17/b. p. 125.; PICBAUER 1. p. 9.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 396.; PODHRADZSKY in UBRIZSY 5. p. 406.

Crysomyxa ramischiae LAGERHEIM

KALCHBRENNER 1. p. 307.; HAZSLINSZKY 1. p. 115.; BRESADOLA 1. No. 142.; MOESZ 12. col. 805.; GYŐRFFY, LINHART in Fung. hung. exs. No. 340, SZÉPLIGETI ap. MOESZ 17/b. p. 125.; SAVULESCU 7. p. 398.

Chrysomyxa rhododendri (DC.) DE BARY

SYDOW 2. III. p. 510.; FILARSZKY, LINHART in Fung. hung. exs. No. 243., RÖMER in Fung. hung. exs. No. 436., MOESZ, SCHULER ap. MOESZ 17/b. p. 125.; SAVULESCU 7. p. 394.; PODHRADSZKY in UBRIZSY 5. p. 406.

Genus. *Cronartium* FRIES

Cronartium flaccidum (A. et S.) WINTER

Syn.: Cr. asclepiadeum (WILLD.) FR.; Peridermium pini auct. ppe.

FUSS 1. IV. p. 125.; BOLLA 1. p. 50.; KALCHBRENNER 1. I. p. 302.; HAZS-

LINSZKY 1. p. 129., 144.; HAZSLINSZKY 3. p. 79.; BÄUMLER 1. II. p. 589., 590.; BRESADOLA 1. p. 104.; SIMONKAI 1. No. 1675.; SACCARDO 1. No. 96.; SYDOW 2. III. p. 562.; HOLLÓS 1. p. 142.; MOESZ 11. p. 99.; MOESZ 12. col. 806.; HOLLÓS 7. p. 147.; MOESZ 31. p. 95.; BÄUMLER, GREINICH, GRESCHIK, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 433., MÁGOCSY-DIETZ in Krypt. exs. No. 110., MOESZ, PALICSEK, TUZSON ap. MOESZ 17/b. p. 125.; MOESZ, PAVLICSEK ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 390.; TÓTH 2. p. 57.; VASS – TÓTH 1. p. 158.; SZIGETI – TÓTH 1. p. 20.; PODHRADSZKY in UBRIZSY 5. p. 403.; UBRIZSY 8. p. 378.

Cronartium gentianeum THÜMEN

FILARSZKY, PAX ap. MOESZ 17/b. p. 126.; PICBAUER 1. p. 8.; SAVULESCU 7. p. 392.

Cronartium quercuum MIYABE

Syn.: Epitea quercina SCHULZER

SCHULZER 2. p. 20. ap. MOESZ 17/b. 126.

Cronartium ribicolum DIETR.

Syn.: Cr. ribicola (LASCH.) WALDH.; Peridermium strobi KLEB.

SYDOW 2. III. p. 569.; MOESZ 11. p. 99.; HOLLÓS 7. p. 147.; GRESCHIK ap. MOESZ 17/b. p. 127.; SAVULESCU 7. p. 388.; PODHRADSZKY in UBRIZSY 5. p. 404.

Genus. *Hyalopsora* MAGNUS

Hyalopsora polypodii (PERS.) MAGNUS

BOLLA 1. p. 48.; HAZSLINSZKY 1. p. 115.; HAZSLINSZKY 5. p. 43.; FUSS 2. p. 444.; RICHTER 1. No. 45.; BÄUMLER 1. II. No. 668.; BRESADOLA 1. No. 149.; SIMONKAI 1. No. 1662.; SYDOW 2. III. p. 497.; MOESZ 12. col. 806.; MOESZ 31. p. 95.; BÄUMLER, BOROS, FILARSZKY, KMET, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ, PÉNZES, PÓSCH, TUZSON, ZAHLBRUCKNER ap. MOESZ 17/b. p. 127.; KÜMMERLE, MOESZ ap. MOESZ 13. p. 111.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 353.; ZELLER – TÓTH 1. p. 231.; PODHRADSZKY in UBRIZSY 5. p. 399.

Hyalopsora polypodii-dryopteridis (MOUG. et NIESTL) MAGNUS

Syn.: Hyalopsora aspidiotus (PECK) MAGNUS

SYDOW 2. III. p. 496.; MOESZ 12. col. 806.; MOESZ ap. MOESZ 17/b. p. 128.; SAVULESCU 7. p. 352.; TÓTH 2. p. 57.

Genus. *Melampsora* CASTAGNE

Melampsora allii-fragilis KLEBAHN

Syn.: Caeoma alliorum auct. ppe.

HAZSLINSZKY 1. p. 127.; MOESZ 2. No. 47.; SYDOW 2. III. p. 373.; BÄUMLER, MOESZ ap. MOESZ 17/b. p. 128.; MOESZ ap. MOESZ 13. p. 111.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 431.; PODHRADSZKY in UBRIZSY 5. p. 409.

Melampsora allii-populina KLEBAHN

Syn.: M. populina auct. ppe.; Caeoma alliorum auct. ppe.
MOESZ 2. No. 48.; KÜMMERLE, MOESZ ap. MOESZ 17/b. p. 128.; KÜMMERLE, MOESZ, SZEPESFALVY ap. MOESZ 13. p. 111.; PODHRADSZKY in UBRIZSY 5. p. 409.

Melampsora allii-salicis albae KLEBAHN

Syn.: M. salicis-albae KLEB. ppe.; Caeoma alliorum auct. ppe.
SYDOW 2. III. p. 372.; BÄUMLER, LINHART in Fung. hung. exs. No. 341. ap. MOESZ 17/b. p. 129.; SAVULESCU 7. p. 430.; PODHRADSZKY in UBRIZSY 5. p. 409.

Melampsora amygdalinae KLEBAHN
SYDOW 2. III. p. 370. ap. MOESZ 17/b. p. 129.; SAVULESCU 7. p. 448.

Melampsora cyparissiae MÜLLER

Syn.: M. euphorbiae (SCHUB.) CAST.; M. helioscopiae (PERS.) CAST.

f. sp. *cyparissiae* MÜLLER
SCHULZER 2. p. 21.; HAZSLINSZKY 1. p. 120.; HOLLÓS 1. p. 142.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, BUDAI, GREINICH, HAZSLINSZKY, KMET, MÁRKUS, MOESZ ap. MOESZ 17/b. p. 129.; MOESZ ap. MOESZ 13. p. 111.; SAVULESCU 7. p. 468.; PODHRADSZKY in UBRIZSY 5. p. 410.; UBRIZSY 6. in SZÉKESSY p. 23.

f. sp. *euphorbiae exiguae* MÜLLER
BOLLA 1. p. 48.; HAZSLINSZKY 1. p. 120.; HAZSLINSZKY 2. p. 13.; HAZSLINSZKY 7. p. 9.; SACCARDO 1. No. 95.; BÄUMLER 1. IV. No. (583.); MOESZ 2. No. 49.; HOLLÓS 1. p. 142.; MOESZ 31. p. 95.; BÄUMLER, FILARSZKY, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 17/b. p. 130.; FILARSZKY, MOESZ ap. MOESZ 13. p. 111.; SAVULESCU 7. p. 469.; PODHRADSZKY in UBRIZSY 5. p. 410.; UBRIZSY 8. p. 378.

f. sp. *euphorbiae-pepli* MÜLLER
HAZSLINSZKY 1. p. 120.; HAZSLINSZKY ap. MOESZ 17/b. p. 130.

Melampsora euphorbiae-amygdaloides MÜLLER
HAZSLINSZKY 1. p. 120.; KALCHBRENNER, MOESZ ap. MOESZ 17/b. p. 130.; SAVULESCU 7. p. 471.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora euphorbiae-dulcis OTTH
FUSS 2. p. 439.; SYDOW 2. III. p. 381.; MOESZ 11. p. 100.; BARTH, BÄUMLER, MOESZ ap. MOESZ 17/b. p. 130.; MOESZ ap. MOESZ 13. p. 111.; PICBAUER 1. p. 8.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 465.; PODHRADSZKY in UBRIZSY 5. p. 410.; UBRIZSY 8. p. 378.

Melampsora euphorbiae-gerardianae MÜLLER
HAZSLINSZKY 1. p. 120.; FUSS 2. p. 439.; RICHTER 1. No. 38.; SIMONKAI 1. No. 1710.; HOLLÓS 1. p. 142.; HOLLÓS 7. p. 149.; MOESZ 31. p. 95.; BÄUMLER, GÁYER, HAZSLINSZKY, KOVÁCS, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 334. ap. MOESZ 17/b. p. 130.; KOVÁCS, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 111.; PICBAUER 3. p. 177.; SAVULESCU 7. p. 464.; TÓTH 2. p. 57.; TÍMÁR 1. p. 237.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora evonymi-capraearum KLEBAHN

HAZSLINSZKY 3. p. 79.; SYDOW 2. III. p. 360.; HOLLÓS 7. p. 145.; BUDAI, GRESCHIK, LINHART in Fung. hung. exs. No. 44., MOESZ ap. MOESZ 17/b. p. 131.; SAVULESCU 7. p. 442.; TÓTH 2. p. 57.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora galanthi-fragilis KLEBAHN

Syn.: Caeoma galanthi (UNG.) SCHROET.; Uredo galanthi UNG.

BOLLA 1. p. 49.; HAZSLINSZKY 1. p. 127.; BÄUMLER 1. II. No. 672., V. No. 1879.; SYDOW 2. III. p. 374.; BÄUMLER in RABENH.—WINTER Fung. eur. exs. No. 3730., LINHART in Fung. hung. exs. No. 135., SCHNELLER ap. MOESZ 17/b. p. 131.; PICBAUER 3. p. 176.; SAVULESCU 7. p. 433.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora helioscopiae (PERS.) CASTELLANI s. str. MÜLLER

FUSS 1. IV. p. 111.; KALCHBRENNER 1. I. p. 313.; SCHULZER 2. p. 21.; HAZSLINSZKY 1. p. 120.; FUSS 2. p. 439.; BÄUMLER 1. II. No. 583.; BRESADOLA 1. No. 98.; SIMONKAI 1. No. 1710.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 378.; HOLLÓS 7. p. 149.; BÄUMLER, FUSS, HAZSLINSZKY, HOLLÓS, HOLUBY, KMET, MOESZ, NOÉ, SZÉPLIGETI ap. MOESZ 17/b. p. 131.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 111.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 470.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora hypericorum (DC.) SCHROETER

Syn.: Uredo hypericorum DC.

ENDLICHER 1. No. 71.; FUSS 1. IV. p. 111.; KALCHBRENNER 1. I. p. 307.; HAZSLINSZKY 1. p. 115.; FUSS 2. p. 444.; HAZSLINSZKY 3. p. 79.; BÄUMLER 1. II. No. 584.; SYDOW 2. III. p. 385.; MOESZ 11. p. 100.; HOLLÓS 7. p. 149.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, HAZSLINSZKY, MOESZ, SZÉPLIGETI, ZAHLBRUCKNER ap. MOESZ 17/b. p. 132.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 111.; SAVULESCU 7. p. 460.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora larici-capraearum KLEBAHN

Syn.: M. larici-populina KLEB. ppe.; Caeoma laricis (WEST.) HARTIG ppe.

MOESZ 2. No. 50.; SYDOW 2. III. p. 354.; GYŐRFFY, MOESZ, PÉNZES, ap. MOESZ 17/b. p. 132.; MOESZ ap. MOESZ 13. p. 111.; SAVULESCU 7. p. 435.; PODHRADSZKY in UBRIZSY 5. p. 410.

Melampsora larici-epitea KLEBAHN

Syn.: Caeoma laricis (WEST.) HARTIG ppe.

SYDOW 2. III. p. 10.; MOESZ 12. col. 806.; FILARSZKY, GRESCHIK, HAZSLINSZKY ap. MOESZ 17/b. p. 132.; SAVULESCU 7. p. 439.

Melampsora larici-pentandrae KLEBAHN

Syn.: M. larici-populina KLEB. ppe.; Caeoma laricis (WEST.) HARTIG ppe.

GYŐRFFY ap. MOESZ 17/b. p. 133.; SAVULESCU 7. p. 429.

Melampsora larici-populina KLEBAHN

Syn.: M. populina KLEB. ppe.; Caeoma laricis (WEST.) HARTIG ppe.

SYDOW 2. III. p. 347.; HAZSLINSZKY 3. p. 81.; GYŐRFFY ap. MOESZ 17/b. p. 133.; SZÉPLIGETI ap. MOESZ 13. p. 113.; UBRIZSY 1. p. 15.; SAVULESCU 7.

p. 451.; TÍMÁR 1. p. 237.; PODHRADSZKY in UBRIZSY 5. p. 411.; UBRIZSY 8. p. 378.

Melampsora larici-tremulae KLEBAHN

Syn.: M. laricis HARTIG; M. larici-populina KLEB. ppe.; Caeoma laricis (WEST.) HARTIG ppe.

HAZSLINSZKY 3. p. 79.; SYDOW 2. III. p. 339.; MOESZ 11. p. 100.; BÄUMLER, KALCHBRENNER, LAUDON, LÁNYI, LINHART in Fung. hung. exs. No. 43., et in RABENH.—WINTER Fung. eur. exs. No. 3731., MÁGOCSY-DIETZ ap. MOESZ 17/b. p. 133.; LÁNYI, MOESZ ap. MOESZ 13. p. 112.; SAVULESCU 7. p. 452.; VASS—TÓTH 2. p. 48.; PODHRADSZKY in UBRIZSY 5. p. 411.; UBRIZSY 8. p. 378.

Melampsora lini (PERS.) DESMAZIÈRES

BOLLA 1. p. 48.; FUSS 1. VIII. p. 232.; KALCHBRENNER 1. I. p. 313.; HAZSLINSZKY 1. p. 119.; FUSS 2. p. 439.; BORBÁS 2. p. 145.; HAZSLINSZKY 3. p. 81.; HAZSLINSZKY 7. p. 9.; RICHTER 1. No. 39.; BÄUMLER 1. II. No. 585.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 383.; HUSZ 1. p. 101.; MOESZ 11. p. 100.; MOESZ 12. col. 806.; HOLLÓS 7. p. 149.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, DEGEN, FUSS, GREINICH, GYŐRFFY, HAZSLINSZKY, HOLUBY, KMET in Flora exs. Austro—Hung. No. 1562., LINHART in Fung. hung. exs. No. 134., MÁRTON, MOESZ, SZÉPLIGETI, WIERZBICKY ap. MOESZ 17/b. p. 133.; DEGEN, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 112.; UBRIZSY 1. p. 15.; KRENNER 1. p. 63.; SAVULESCU 7. p. 462.; TÓTH 10. p. 9.; PODHRADSZKY in UBRIZSY 5. p. 412.; UBRIZSY 6. in SZÉKESSY p. 24.; UBRIZSY 8. p. 378.

Melampsora magnusiana WAGNER

Syn.: Caeoma chelidonii MAGNUS; C. fumariae LINK.

BÄUMLER 1. II. No. 671., V. No. 1880.; SACCARDO 1. No. 130.; SYDOW 2. III. p. 343.; BÄUMLER, KMET, LINHART in Fung. hung. exs. No. 137., PÓSCH ap. MOESZ 17/b. p. 134.; SAVULESCU 7. p. 458.; PODHRADSZKY in UBRIZSY 5. p. 413.

Melampsora orchidi-repentis (PLOWR.) KLEBAHN

Syn.: M. repentis PLOWR.; Caeoma orchidis (MART.) WINTER

FUSS 1. IV. p. 111.; FUSS 2. p. 444.; HAZSLINSZKY 1. p. 115.; SYDOW 2. III. p. 359.; KISS ap. MOESZ 17/b. p. 134.; SAVULESCU 7. p. 444.; PODHRADSZKY in UBRIZSY 5. p. 413.

Melampsora pinitorqua ROSTRUP

Syn.: M. tremulae TUL.

SAVULESCU 7. p. 454.; VÖRÖS 9. p. 102.; PODHRADSZKY in UBRIZSY 5. p. 413.

Melampsora pulcherrima MAIRE

Syn.: Caeoma pulcherrimum BUBÁK; Uredo mercurialis MART.; Caeoma mercurialis LINK ppe.

BOLLA 1. p. 49.; BÄUMLER 1. II. No. 673.; SACCARDO 1. No. 131. ap. MOESZ 17/b. p. 134.

Melampsora reticulata BLYTT

SYDOW 2. III. p. 362.; HUSZ 1. col. 806.; FILARSZKY, GYŐRFFY, HANASIEWIC,

HUSZ, MOESZ, TRAUTMANN ap. MOESZ 17/b. p. 135.; MOESZ ap. MOESZ 13. p. 113.; PICBAUER 3. p. 176.; SAVULESCU 7. p. 447.; PODHRADSZKY in UBRIZSY 5. p. 414.

Melampsora ribesii-purpureae KLEBAHN

Syn.: Melampsora ribesii-salicum BUBÁK

SYDOW 2. III. p. 364.; MOESZ 12. col. 807.; GRESCHIK, SZEPESFALVY ap. MOESZ 17/b. p. 135.; PICBAUER 1. p. 8.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 441.

Melampsora ribesii-viminalis KLEBAHN

SAVULESCU 7. p. 437.; UBRIZSY 8. p. 378.

Melampsora rostrupii WAGNER

Syn.: Caeoma mercurialis LINK

HAZSLINSZKY 1. p. 115.; SIMONKAI 1. No. 1663.; MOESZ 2. No. 51.; SYDOW 2. III. p. 344.; BÄUMLER, BUDAI, FILARSZKY in Krypt. exs. No. 1116., GRESCHIK, KMET in Flora exs. Austro–Hung. No. 1569., MOESZ, SCHNELLER ap. MOESZ 17/b. p. 135.; MOESZ ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 456.; TÓTH 2. p. 57.; PODHRADSZKY in UBRIZSY 5. p. 415.

Melampsora salicina LÉVEILLÉ

HAZSLINSZKY 1. p. 117.; SADLER, SZÉPLIGETI ap. MOESZ 13. p. 113.; VÖRÖS 9. p. 102.

Genus. *Melampsorella* SCHROETER

Melampsorella caryophyllacearum SCHROETER

Syn.: Aecidium elatinum ALB. et SCHW.; Melampsora cerastii (PERS.) WINTER; Melampsorella cerastii (PERS.) WINTER

FUSS 1. VIII. p. 232.; KALCHBRENNER 1. I. p. 302.; HAZSLINSZKY 1. p. 129.; HAZSLINSZKY 3. p. 78., 81.; RICHTER 1. No. 46.; BUBÁK 1. No. 63.; BRESADOLA 1. No. 145.; MAGNUS 1. p. 46.; SYDOW 2. III. p. 436.; BÄUMLER 1. II. No. 661., IV. No. 1493., V. No. 1881.; MOESZ 12. col. 807.; BÄUMLER, BOTHÁR, FILARSZKY, GRESCHIK, HAZSLINSZKY, JÁVORKA, KALCHBRENNER, KLEIN, LINHART in Fung. hung. exs. No. 434., MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 142., MOESZ, PÁPAI, TUZSON ap. MOESZ 17/b. p. 136.; MOESZ ap. MOESZ 13. p. 113.; PICBAUER 1. p. 9.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 369.; PODHRADSZKY in UBRIZSY 5. p. 400.

Melampsorella symphyti (DC.) BUBÁK

Syn.: Uredo symphyti DC.

ENDLICHER 1. p. 68.; KALCHBRENNER 1. I. p. 307.; HAZSLINSZKY 1. p. 108.; HAZSLINSZKY 7. p. 7.; BÄUMLER 1. II. No. 669.; BRESADOLA 1. No. 148.; MOESZ 2. No. 52.; HOLLÓS 1. p. 143.; SYDOW 2. III. p. 439.; MOESZ 12. col. 807.; HOLLÓS 7. p. 149.; BÄUMLER, BUDAI, CSAPODY, FANTA, GOMBOCZ, GREINICH, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, LINHART in Fung. hung. exs. No. 42., MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 136.; GOMBOCZ, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 372.; PODHRADSZKY in UBRIZSY 5. p. 401.

Genus. *Melampsoridium* KLEBAHN

Melampsoridium betulinum (PERS.) KLEBAHN

Syn.: Melampsora betulina (P.) DESM.; Uredo betulina PERS.
FUSS 1. IV. p. 111.; KALCHBRENNER 1. I. p. 313.; HAZSLINSZKY 1. p. 118.; HAZSLINSZKY 2. p. 13.; FUSS 2. p. 439.; HAZSLINSZKY 3. p. 81.; HAZSLINSZKY 7. p. 9.; RICHTER 1. No. 34.; BÄUMLER 1. II. No. 580.; BRESADOLA 1. No. 103.; SYDOW 2. III. p. 427.; MOESZ 12. col. 807.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, FUSS, GRESCHIK, HAZSLINSZKY, HOLUBY, KMET, MÁGOCSY-DIETZ, MÁRTON, MOESZ, RICHTER, SZÉPLIGETI in Fung. hung. exs. No. 333., TUZSON ap. MOESZ 17/b. p. 137.; SAVULESCU 7. p. 383.; PODHRADSZKY in UBRIZSY 5. p. 402.; UBRIZSY 6. in SZÉKESSY p. 24.; UBRIZSY 8. p. 378.

Melampsoridium carpini (NEES) DIETEL

Syn.: Melampsora carpini FUCKEL; Caeoma carpini NEES
KALCHBRENNER 1. p. 313.; HAZSLINSZKY 1. p. 117.; RICHTER 1. No. 37.; BÄUMLER 1. II. No. 581.; SYDOW 2. III. p. 430.; HOLLÓS 7. p. 148.; BÄUMLER, HAZSLINSZKY, KMET, MÁGOCSY-DIETZ, MOESZ, VIDA in LINHART Fung. hung. exs. No. 239. ap. MOESZ 17/b. p. 137.; MOESZ ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 386.; PODHRADSZKY in UBRIZSY 5. p. 402.

Genus. *Milesina* MAGNUS
Syn.: Milesia WHITE

Milesina feurichii MAGNUS

Syn.: Milesia feurichii (MAGNUS) FAULL
GYŐRFFY ap. MOESZ 17/b. p. 138.; SAVULESCU 7. p. 358.

Milesina murariae SYDOW

Syn.: Milesia murariae (MAGNUS) FAULL; Uredo murariae MAGN.
BUBÁK 1. No. 64.; BÄUMLER 1. IV. No. 1505.; SZÉP ap. MOESZ 17/b. p. 138.; SAVULESCU 7. p. 359.

Milesina scolopendrii JAAP

Syn.: Milesia scolopendrii (FUCKEL) ARTHUR et FAULL
MOESZ ap. MOESZ 17/b. 138.; p. SAVULESCU 7. p. 357.

Genus. *Pucciniastrum* OTTH

Pucciniastrum abieti-chamaenerii KLEBAHN

Syn.: Melampsora epilobii FUCK. ppe.
HAZSLINSZKY 1. p. 118.; BRESADOLA 1. No. 140.; SYDOW 2. III. p. 443.; FILARSZKY, TUZSON ap. MOESZ 17/b. p. 138.; SAVULESCU 7. p. 374.; TÓTH 2. p. 60.; PODHRADSZKY in UBRIZSY 5. p. 401.

Pucciniastrum agrimoniae (DC.) TRANZSCHEL

Syn.: Melampsora agrimoniae DC.
KALCHBRENNER 1. I. p. 312.; HAZSLINSZKY 1. p. 108.; HAZSLINSZKY 2. p. 12.; RICHTER 1. No. 44.; BRESADOLA 1. No. 147.; HOLLÓS 1. p. 143.; SYDOW 2.

III. p. 448.; MOESZ 11. p. 102.; HOLLÓS 7. p. 148.; HAZSLINSZKY, KMET, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 244., MOESZ ap. MOESZ 17/b. p. 138.; MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 113.; PICBAUER 1. p. 8.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 379.; PODHRADSZKY in UBRIZSY 5. p. 401.

Pucciniastrum circaeae (SCHUM.) SPEGAZZINI

Syn.: Melampsora circaeae THÜMEN

KALCHBRENNER 1. p. 308.; HAZSLINSZKY 1. p. 167.; No. 52.; BÄUMLER 1. IV. No. 1502.; SYDOW 2. III. p. 446.; HOLLÓS 7. p. 149.; MOESZ, SABRANSKY ap. MOESZ 17/b. p. 139.; PICBAUER 1. p. 8.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 377.; TÓTH 2. p. 60.; VASS–TÓTH 1. p. 158.; PODHRADSZKY in UBRIZSY 5. p. 402.; UBRIZSY 8. p. 378.

Pucciniastrum epilobii (PERS.) OTTH

Syn.: Melampsora epilobii FUCKEL

HAZSLINSZKY 2. p. 13.; BÄUMLER 1. IV. No. 1503.; SYDOW 2. III. p. 445.; HOLUBY, MOESZ, SZÉPLIGETI in LINHART Fung. hung. exs. No. 335. ap. MOESZ 17/b. p. 139.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 376.; PODHRADSZKY in UBRIZSY 5. p. 402.

Pucciniastrum pyrolae (KARST.) SCHROETER

Syn.: Thecopsora pyrolae KARST.

FUSS 1. IV., III.; KALCHBRENNER 1. I. p. 307.; MAGNUS 1. p. 46.; SYDOW 2. III. p. 456.; HUSZ 1. p. 102.; MOESZ 31. p. 96.; MÁGOCSY-DIETZ, SZÉPLIGETI, VAJDA ap. MOESZ 17/b. p. 139.; SAVULESCU 7. p. 381.; PODHRADSZKY in UBRIZSY 5. p. 402.

Genus. *Schroeteriaster* MAGNUS

Schroeteriaster alpinus (SCHROET.) MAGNUS

Syn.: Uromyces alpinus SCHROETER

MAGNUS 1. p. 45.; SYDOW 2. III. p. 401.; MOESZ ap. MOESZ 17/b. p. 139.; PICBAUER 2. p. 188.; SAVULESCU 7. p. 674.

Genus. *Thecopsora* MAGNUS

Thecopsora areolata (FR.) MAGNUS

Syn.: Th. strobilina (ALB. et SCHW.) SAVULESCU; Uredo padi KZE et SCHMIDT; Melampsora padi WINTER; Pucciniastrum padi DIETEL; Aecidium strobilinum (ALB. et SCHM.) REESS.

KALCHBRENNER 1. I. p. 313.; HAZSLINSZKY 1. p. 118.; HAZSLINSZKY 7. p. 8.; BÄUMLER 1. II. No. 657., V. No. 1877.; BRESADOLA 1. No. 141.; SYDOW 2. III. p. 461.; MOESZ 12. col. 809.; MOESZ 31. p. 96.; BÄUMLER in Krypt. exs. No. 934., FILARSZKY, GRESCHIK, HAZSLINSZKY, LINHART in Fung. hung. exs. No. 45., MÁRTON, MOESZ, TUZSON ap. MOESZ 17/b. p. 140.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 364.

Thecopsora galii (LINK) DE TONI

Syn.: Th. guttata (SCHROET.) SYD.; Melampsora galii (LK.) WINTER

BÄUMLER 1. IV. No. 1504.; SYDOW 2. III. p. 468.; SZÉPLIGETI in Fung. hung. exs. No. 337. ap. MOESZ 17/b. p. 140.; SZÉPLIGETI ap. MOESZ 13. p. 113.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 366.; PODHRADSZKY in UBRIZSY 5. p. 400.

Thecopsora vacciniorum (LINK) KARSTEN

Syn.: Th. myrtilli (SCHUM.) TRANZSCHEL; Melampsora vacciniorum SCHROETER; Pucciniastrum vacciniorum DIETEL

FUSS 1. IV. p. 111.; HAZSLINSZKY 1. p. 115.; SACCARDO 1. No. 94.; SYDOW 2. III. p. 464.; HUSZ 1. p. 102.; MOESZ 12. col. 809.; KMET, PAEDAGIUM, SZEPESFALVY, SZÉPLIGETI in LINHART Fung. hung. exs. No. 336. ap. MOESZ 17/b. p. 140.; PICBAUER 2. p. 186.; PICBAUER 3. p. 177.; SAVULESCU 7. p. 367.

Genus. *Uredinopsis* MAGNUS

Uredinopsis filicina (NIESSL) MAGNUS

Syn.: U. phegopteridis (PASS.) ARTHUR

SYDOW 2. III. p. 485. ap. MOESZ 17/b. p. 141.; PICBAUER 1. p. 8.; PICBAUER 2. p. 186.; SAVULESCU 7. p. 349.

Familia. Coleosporiaceae

Genus. *Coleosporium* LÉVEILLÉ

Coleosporium cacaliae (DC.) WAGNER

FUSS 1. VIII. p. 232.; KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 107.; FUSS 2. p. 443.; SYDOW 2. III. p. 601.; HUSZ 1. p. 101.; MOESZ 12. col. 805.; MOESZ 30. p. 6.; GYŐRFFY, HAZSLINSZKY, KALCHBRENNER in RABENH. Fung. eur. exs. No. 484., VESELSKY ap. MOESZ 17/b. p. 141.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 412.

Coleosporium campanulae (PERS.) LÉVEILLÉ

FUSS 1. IV., III., VIII. p. 232.; BOLLA 1. p. 49.; KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 109.; HAZSLINSZKY 2. p. 13.; HAZSLINSZKY 5. p. 43.; FUSS 2. p. 443.; RICHTER 1. No. 41.; BÄUMLER 1. II. No. 652.; SIMONKAI 1. p. 1665.; BUBÁK 1. No. 61.; MOESZ 2. No. 45.; MAGNUS 1. p. 47.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 630.; MOESZ 11. p. 99.; MOESZ 12. col. 805.; HOLLÓS 7. p. 146.; MOESZ 30. II. p. 372.; MOESZ 31. p. 95.; BARTH, BÄUMLER, BÁNYAI, BOROS, BUDAI, FILARSZKY, FUSS, GREINICH, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLLÓS, HOLUBY, JÁVORKA, KMET, KŐFARAGÓ-GYELNIK, LÁNYI, LENGYEL, LOJKA, MÁGOCSY-DIETZ, MÁRTON, MOESZ, NAGY, RICHTER, RŐMER in LINHART Fung. hung. exs. No. 338/a., SCHILBERSZKY in Krypt. exs. Vindob. No. 815., STANKOVICS, SZATALA, SZÉPLIGETI in LINHART Fung. hung. exs. No. 338/b., TUZSON ap. MOESZ 17/b. p. 141.; KŐFARAGÓ-GYELNIK, MÁGOCSY-DIETZ, MOESZ, NAGY, SCHILBERSZKY, SZÉPLIGETI ap. MOESZ 13. p. 110.; PICBAUER 2. p. 187.; SAVULESCU 7. p.

407.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 57.; VASS 1. p. 50.; TÓTH 10. p. 8.; PODHRADSZKY in UBRIZSY 5. p. 407.; UBRIZSY 8. p. 378.

Coleosporium doronici NAMYSLOWSKI

HUSZ 1. p. 101.; MOESZ 12. col. 805.; GYŐRFFY, HAZSLINSZKY, LENGYEL ap. MOESZ 17/b. p. 143.; SAVULESCU 7. p. 426.

Coleosporium euphrasiae (SCHUM.) WINTER

Syn.: Uredo euphrasiae SCHUM.; Uredo rhinanthacearum DC.

ENDLICHER 1. No. 69.; FUSS 1. IV. p. 111.; HAZSLINSZKY 1. p. 108.; FUSS 2. p. 443.; HAZSLINSZKY 7. p. 8.; RICHTER 1. No. 40.; BÄUMLER 1. II. No. 653.; BRESADOLA 1. No. 139.; MAGNUS 1. p. 46.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 638.; MOESZ 11. p. 99.; MOESZ 12. col. 805.; HOLLÓS 7. p. 146.; MOESZ 30. II. p. 373.; MOESZ 31. p. 95.; BÄUMLER, BUDAI, DEGEN, FILARSZKY, GYŐRFFY, HAZSLINSZKY, HOLUBY, KMET, MÁRTON, MOESZ, RICHTER, SZÉPLIGETI, ZAHLBRUCKNER ap. MOESZ 17/b. p. 143.; DEGEN, MOESZ ap. MOESZ 13. p. 110.; SAVULESCU 7. p. 403.; LEHOCZKY 1. p. 237.; PODHRADSZKY in UBRIZSY 5. p. 407.; UBRIZSY 8. p. 378.

Coleosporium inulae (KUNZE) FISCHER

Syn.: C. synantherarum FRIES; Uredo inulae KZE.

ENDLICHER 1. No. 67.; KALCHBRENNER 1. I. p. 307., 314.; HAZSLINSZKY 1. p. 108.; BÄUMLER 1. II. No. 656.; SACCARDO 1. No. 120.; SYDOW 2. III. p. 610.; HOLLÓS 7. p. 146.; BÄUMLER, HAZSLINSZKY, KALCHBRENNER, KMET, MÁGOCSY-DIETZ – SZABÓ in Krypt. exs. No. 2311/a. ap. MOESZ 17/b. p. 144.; MÁGOCSY-DIETZ, SZABÓ ap. MOESZ 13. p. 110.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 414.; PODHRADSZKY in UBRIZSY 5. p. 407.

Coleosporium melampyri (REBENT.) KLEBAHN

KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 108.; HAZSLINSZKY 2. p. 13.; BORBÁS 3. p. 145.; RICHTER 1. No. 40.; BÄUMLER 1. II. No. 653.; SACCARDO 1. No. 121.; MOESZ 2. No. 46.; HOLLÓS 1. p. 142.; MAGNUS 1. p. 47.; SYDOW 2. III. p. 640.; HUSZ 1. p. 101.; MOESZ 11. p. 99.; HOLLÓS 7. p. 146.; MOESZ 30. p. 7.; BÄUMLER, BUDAI, GRESCHIK, HAZSLINSZKY, HOLLÓS, KMET, LÁNYI, MÁRTON, MOESZ, PROCOPP – SZABÓ in Krypt. exs. No. 107/b., SZÉPLIGETI in LINHART Fung. hung. exs. No. 339., TUZSON in Krypt. exs. No. 107/c., ZAHLBRUCKNER, ZSÁK ap. MOESZ 17/b. p. 144.; HAZSLINSZKY, MOESZ, PROCOPP, SZABÓ, SZÉPLIGETI, ZSÁK ap. MOESZ 13. p. 140.; UBRIZSY 1. p. 15.; SAVULESCU 7. p. 405.; LEHOCZKY 1. p. 237.; TÓTH 2. p. 57.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 407.; UBRIZSY 8. p. 378.

Coleosporium petasitis (DC.) FISCHER

KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 107.; HAZSLINSZKY 2. p. 12.; BRESADOLA 1. No. 138.; SYDOW 2. III. p. 614.; MOESZ 12. col. 805.; HOLLÓS 7. p. 146.; BUDAI, GYŐRFFY, HOLUBY, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 145.; SAVULESCU 7. p. 416.; TÓTH 2. p. 57.; TÓTH 10. p. 9.; PODHRADSZKY in UBRIZSY 5. p. 407.; UBRIZSY 8. p. 379.

Coleosporium pulsatillae (STRAUSS) LÉVEILLÉ

BÄUMLER 1. II. No. 654.; SYDOW 2. III. p. 652.; MOESZ 11. p. 99.; MOESZ 30. p. 7.; BÄUMLER, GRESCHIK, MOESZ ap. MOESZ 17/b. p. 145.; SAVULESCU 7. p. 401.; PODHRADSZKY in UBRIZSY 5. p. 407.

Coleosporium senecionis (PERS.) FRIES

BOLLA 1. p. 49., 50.; KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 109.; HAZSLINSZKY 2. p. 13.; FUSS 2. p. 439.; HAZSLINSZKY 7. p. 8.; BORBÁS 3. p. 145.; BÄUMLER 1. II. No. 655.; BRESADOLA 1. No. 137.; SACCARDO 1. No. 119.; HOLLÓS 1. p. 143.; SYDOW 2. III. p. 617.; HUSZ 1. p. 101.; MOESZ 12. col. 805.; HOLLÓS 7. p. 146.; MOESZ 30. p. 7.; BARTH, BÄUMLER, FILARSZKY, GRESCHIK, GYŐRFFY, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KMET, KÖRNYEI, KÜMMERLE, LENGYEL, LINHART in Fung. hung. exs. No. 241., MÁGOCSY-DIETZ, MOESZ, SZÉPLIGETI ap. MOESZ 17/b. p. 145.; KÖRNYEI ap. MOESZ 13. p. 110.; PICBAUER 2. p. 187.; SAVULESCU 7. p. 417.; SZIGETI—TÓTH 1. p. 20.; PODHRADSZKY in UBRIZSY 5. p. 408.

Coleosporium sonchi (PERS.) LÉVEILLÉ

Syn.: C. sonchi-arvensis (PERS.) WINTER; Uredo sonchi arvensis PERS.

FUSS 1. IV. p. 111.; BOLLA 1. p. 49.; KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 108.; HAZSLINSZKY 2. p. 12.; FUSS 2. p. 443.; BORBÁS 2. p. 33.; RICHTER 1. No. 42.; BÄUMLER 1. II. No. 656.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 622.; MOESZ 11. p. 99.; HOLLÓS 7. p. 146.; BÄUMLER, HAZSLINSZKY, KMET, MÁGOCSY-DIETZ, MÁRTON, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 17/b. p. 146.; MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 110.; SAVULESCU 7. p. 420.; TÓTH 2. p. 57.; PODHRADSZKY in UBRIZSY 5. p. 408.

Coleosporium telekiae THÜMEN

Syn.: C. telekiae BUBÁK

THÜMEN 2. p. 21.; HAZSLINSZKY 1. p. 109.; FUSS 2. p. 443.; BUBÁK 1. No. 62.; SYDOW 2. III. p. 624.; AUGUSTIN, LOJKA in THUEMEN Mycoth. univ. No. 954., RŐMER in LINHART Fung. hung. exs. No. 435. ap. MOESZ 17/b. p. 147.; SAVULESCU 7. p. 422.

Coleosporium tussilaginis (PERS.) KLEBAHN

ENDLICHER 1. No. 67.; FUSS 1. IV. p. 111.; BOLLA 1. p. 49.; KALCHBRENNER 1. I. p. 314.; HAZSLINSZKY 1. p. 107.; HAZSLINSZKY 2. p. 12.; FUSS 2. p. 443.; HAZSLINSZKY 7. p. 7.; BORBÁS 2. p. 33.; BÄUMLER 1. II. No. 656., V. No. 1882.; HOLLÓS 1. p. 142.; SYDOW 2. III. p. 625.; HUSZ 1. p. 101.; MGESZ 11. p. 99.; MOESZ 12. col. 806.; HOLLÓS 7. p. 147.; MOESZ 30. II. p. 373.; BÄUMLER, BÁNYAI, BUDAI, FILARSZKY, GYŐRFFY, HAZSLINSZKY, HOLUBY, KALCHBRENNER, KMET, LÁNYI, LINHART in Fung. hung. exs. No. 41., MÁGOCSY-DIETZ, MÁRKUS, MÁRTON, MOESZ, NAGY, PÉTERFI, PÓSCH, SADLER, SCHNELLER, SZÉPLIGETI ap. MOESZ 17/b. p. 147.; KRENNER, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ 13. p. 110.; KRENNER 1. p. 63.; SAVULESCU 7. p. 424.; TÓTH 2. p. 57.; VASS 1. p. 50.; PODHRADSZKY in UBRIZSY 5. p. 409.

Coleosporium sp.

(Pinus silvestris, P. montana — Aecidii)

HAZSLINSZKY 1. p. 129.; MAGNUS 1. p. 47.; MOESZ 12. col. 806.; BÄUMLER, BOLLA, FILARSZKY, GYŐRFFY, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 242., MÁGOCSY-DIETZ, MOESZ, SCHNELLER, TUZSON ap. MOESZ 17/b. p. 147.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 110.

Genus. *Ochropsora* DIETEL

Ochropsora sorbi (OUD.) DIETEL

Syn.: O. ariae (FUCK.) SYD.; Aecidium leucospermum DC.

FUSS 1. IV. p. 125.; BOLLA 1. p. 50.; HAZSLINSZKY 1. p. 131.; HAZSLINSZKY 7. p. 10.; BORBÁS 2. p. 33.; SIMONKAI 1. No. 1655.; BÄUMLER 1. III. No. 1395.; SYDOW 2. III. p. 663.; MOESZ 31. p. 95.; GÁYER, GRESCHIK, HAZSLINSZKY, MOESZ ap. MOESZ 17/b. p. 148.; SAVULESCU 7. p. 477.; PODHRADSZKY in UBRIZSY 5. p. 416.

Uredineae imperfectae

Genus. *Aecidium* PERSOON

Aecidium aposeridis NAMYSLOWSKI

FUSS 1. VIII. p. 232.; FUSS 2. p. 437 ap. MOESZ 17/b. p. 149.; SAVULESCU 7. p. 1094.

Aecidium asperifolii PERSOON

Cfr. Puccinia rubigo-vera auct. ppe.

KALCHBRENNER 1. I. p. 304.; HAZSLINSZKY 1. p. 137.; HAZSLINSZKY 3. p. 77.; BÄUMLER 1. II. No. 628.; BRESADOLA 1. No. 112.; SIMONKAI 1. No. 1658.; MOESZ 12. col. 804.; HOLLÓS 7. p. 145.; MOESZ 30. p. 6.; BÄUMLER, FILARSZKY, GRESCHIK, HAZSLINSZKY, KALCHBRENNER in RABENH. Fung. eur. exs. No. 1086., MOESZ, NYÁRÁDY, RŐMER in LINHART Fung. hung. exs. No. 328., SZÉPLIGETI ap. MOESZ 17/b. p. 149.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 1089.

Aecidium circaeae CESATI et MONTAGNE

BUDAI ap. MOESZ 17/b. p. 149.

Aecidium crambes MOESZ

MOESZ 17/b. p. 149.; SAVULESCU 7. p. 1087.

Aecidium doronici RABENHORST

SYDOW 2. IV. p. 35.; MOESZ, TAUSCHER in RABENH. Fung. eur. exs. No. 2192. ap. MOESZ 17/b. p. 150.; MOESZ ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 1092.; UBRIZSY 8. p. 379.

Aecidium euphorbiae GMEL

SADLER 1. VII. No. 25.; ENDLICHER 1. No. 79.; FUSS 1. IV. p. 125., VIII. p. 232.; KALCHBRENNER 1. I. p. 304.; SCHULZER 1. No. 38.; BORBÁS 2. p. 33.; HAZSLINSZKY 1. p. 134.; HAZSLINSZKY 3. p. 75.; BORBÁS 3. p. 145.; RICHTER 1. No. 47.; SIMONKAI 1. No. 1654.; SACCARDO 1. No. 129.; MOESZ 2. No. 58.; MAGNUS 1. p. 47.; MOESZ 4. II. p. 145.; MOESZ 30. p. 6.; BARTH,

BOLLA, BUDAI, FILARSZKY, FUSS, GENERSICH, HAYNALD, HAZSLINSZKY, KALCHBRENNER, KMET in Flora exs. Austro–Hung. No. 3167., MÁGOCSY-DIETZ, MÁRTON, MOESZ, SADLER (ex herb. L. HEUFFEL), STAUB, WAGNER, WIERZBICKY ap. MOESZ 17/b. p. 150.; HAZSLINSZKY, MÁGOCSY-DIETZ, MOESZ, SADLER, STAUB ap. MOESZ 13. p. 113.; SAVULESCU 7. p. 1085.; TÓTH 2. p. 57.; TIMÁR 1. p. 237.

Aecidium isopyri SCHROETER

SACCARDO 1. No. 127.; SYDOW 2. IV. p. 258.; GRESCHIK, KMET ap. MOESZ 17/b. p. 151.; SAVULESCU 7. p. 1082.

Aecidium lithospermi THÜMEN

Cfr. Puccinia bromina ERIKSSON

HAZSLINSZKY 1. p. 138.; HAZSLINSZKY 7. p. 11.; BÄUMLER 1. II. No. 628.; MOESZ 2. No. 69.; HOLLÓS 1. p. 108.; SYDOW 2. IV. p. 123.; BÄUMLER, SZÉPLIGETI ap. MOESZ 17/b. p. 151.; BERNÁTSKY, STAUB, SZÉPLIGETI ap. MOESZ 13. p. 114.; SAVULESCU 7. p. 1090.; UBRIZSY 8. p. 369.

Aecidium muscari LINHART

Cfr. Puccinia festucina SYDOW

HAZSLINSZKY 3. p. 76.; HOLLÓS 1. p. 143.; SYDOW 2. IV. p. 286.; HOLLÓS 7. p. 145.; BUTUJÁS, GYŐRFFY, JÁVORKA, LINHART in Fung. hung. exs. No. 49., 138., 409. et in RABENH.–WINTER Fung. eur. exs. No. 2929., 3408., MOESZ, PÉTERFI, SZÉPLIGETI ap. MOESZ 17/b. p. 151.; MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 114.

Aecidium nonneae THÜMEN

SYDOW 2. IV. p. 126.; BARTH in LINHART Fung. hung. exs. No. 327., STAUB ap. MOESZ 17/b. p. 152.; STAUB ap. MOESZ 13. p. 114.; SAVULESCU 7. p. 1090.

Aecidium onosmatis THÜMEN

FUSS 1. IV. p. 125.; HAZSLINSZKY 1. p. 138.; FUSS 2. p. 436.; BAUMGARTEN ap. MOESZ 17/b. p. 152.; SAVULESCU 7. p. 1090.

Aecidium pulmonariae THÜMEN

BOLLA 1. p. 50.; HAZSLINSZKY 1. p. 138.; BORBÁS 2. p. 33.; HOLLÓS 1. p. 108.; SYDOW 2. IV. p. 127.; MOESZ 12. col. 805.; BOLLA, GRESCHIK, HAZSLINSZKY, MOESZ, MÜLLER, SADLER ap. MOESZ 17/b. p. 152.; HAZSLINSZKY, MÜLLER, SADLER ap. MOESZ 13. p. 114.; PICBAUER 1. p. 14.; SAVULESCU 7. p. 1090.

Aecidium raciborskii WROBL

MOESZ 12. col. 805.; GRESCHIK ap. MOESZ 17/b. p. 152.

Aecidium ranunculacearum DE CANDOLLE

BRESADOLA 1. No. 92.; MOESZ, RŐMER in LINHART Fung. hung. exs. No. 412. ap. MOESZ 17/b. p. 152.; MOESZ ap. MOESZ 13. p. 114.; SAVULESCU 7. p. 1082.

Aecidium scabiosae (DOZY et MOLK.) WINTER

HAZSLINSZKY 3. p. 78.; HOLUBY ap. MOESZ 17/b. p. 153.; SAVULESCU 7. p. 1092.

Aecidium shiraianum SYDOW
MOESZ 12. col. 805.; GYŐRFFY in Flora hung. exs. No. 806. ap. MOESZ 17/b. p. 153.; PICBAUER 3. p. 179.
Aecidium solms-laubachii BOY et JACZEWSKI
HOLLÓS 1. p. 143.; HOLLÓS 7. p. 145. ap. MOESZ 17/b. 154.
Aecidium thlaspineum SYDOW
HAZSLINSZKY 7. p. 10.; SZÉPLIGETI ap. MOESZ 17/b. p. 154.; MOESZ 13. p. 114.; SZÉPLIGETI ap. MOESZ 13. p. 114.

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(For further literature see: Acta Phytopathologica Hung. 1967. Vol. 2. Number 3. pp. 275–285.)

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Book Review

ROBERT N. GOODMAN, ZOLTÁN KIRÁLY and MILTON ZAITLIN: *The Biochemistry and Physiology of Infectious Plant Disease*. Pp. X + 354. Princeton, New Jersey. D. Van Nostrand Company, Inc., 1967., $ 12.95

This book is written primarily for the upper division or graduate students interested in the physiological and biochemical aspects of plant diseases. The emphasis in the title on "infectious" disease indicates that other pathological effects (air pollution, frost-damage, injury caused by insects and nematodes, malnutrition etc.) are not dealt with. The text is divided into the following chapters: The infection process. Photosynthesis. Respiration. Cell wall composition and metabolism. Nitrogen metabolism. Phenol metabolism. Growth regulator metabolism. Vascular transport. Toxins. Each chapter, except the first and the last, contains information in a concise form on the respective metabolic process in the healthy plant. These introductory chapters are followed, whenever possible, by a detailed discussion of the altered metabolism of the diseased plant. Viral, bacterial and fungal diseases are dealt with separately and each chapter is concluded by a section on the "Comparative Analysis of Disease Physiology" in which the common or different aspects of bacterial, viral and fungal diseases, from the point of view of the metabolic process in question, are shortly summarized. In the text there is an ample discussion of the experimental material and a number of critical experiments are described in detail. The book contains an impressive amount of figures and tables taken from original research papers. In view of the rapid development of this field, it was often impossible to give a final and unequivocal interpretation of experimental findings. In such cases the authors present alternative views. This attitude is certainly a correct one which greatly adds to the value of the book. For the research worker the very extensive coverage of the literature is of great importance. Each chapter is followed by a high number of literary references and so called "general references" not cited but annotated. The style is, with a few exceptions, very clear. The three authors succeeded to "melt" the material into a coherent picture. The book is greatly recommanded both for the university student and for the established research worker.

G. L. FARKAS

G. W. BRUEHL: *Barley Yellow Dwarf, a Virus Disease of Cereals and Grasses*. Monograph Number 1 Published by the American Phytopathological Society 1961. Pp. 1–52.

Barley yellow dwarf has been discovered for the first time by Oswald and Houston in California in 1851. Beyond doubt there were found even previously symptoms caused by viruses, and epidemics also occurred, but the real cause of the disease was not diagnosed in those days.

It causes significant damage in some countries and regions of the U. S. A. (California, Oregon, Washington, Idaho, Dakota, Kansas, Iowa, Wisconsin, Indiana, Missouri), but elsewhere it is less significant or has only local importance. Accordingly they will have it that there had even been a loss of 5—14 millions of bushels, valued to several millions of dollars.

The virus disease is to be found in Europe as well, almost in every country, among them in Germany, where Rademacher and Schwarz (1958) have reported about it and termed it *Hordeumvirus manescens.*

The main host plants of the virus are barley and oats but it is to be found also on wheat and besides on various grasses, which are partly susceptible and show symptoms, partly are only virus carriers without any symptoms.

The characteristic symptoms of yellow dwarf, shown by the two host plants are somewhat different as to the discolouration. The characteristic difference consists in the withering of the leaves on barley and on oat in the reddening of those. In other features the symptoms are in concordance with one another. The divergence of the single stocks of viruses interfere, too, with the evolution of the symptoms. With barley the withering starts from the apex of the leaf and advances towards the base leaf stalk and so goes the reddening with oat, too. The dwarf of the stocks ensues gradually. The sooner the infection occurs, the more intensive is the symptom of the dwarf. The flexibility of the leaves comes to an end, they become standing quite stiff on the leaf-stalk. In case of an early infection the stems are unfruitful-sterile-barren or the yield of seeds is deficient. The whitening and the sterility of the lower part of the ear is particularly distinct with the oat. The roots get considerably reduced.

Degenerative processes are taking place in the phloem of the plants. In the xilem there may be observed a gum-production. In the pathological process a reduction of the tissue comes into beeing in the meristem. The metabolism becomes abnormal resulting in a reduction of the sugar content and the accumulation of starch. Catalase and peroxidase activity is also enhanced.

The virus of the barley yellow dwarf is distributed by aphids, which — feeding in the phloem — infect the plants. Other forms of distribution are not known. According to Oswald and his co-workers five vectors are known which transmit the virus with more or less frequency. These are as follows: *Rhopalosiphum fitchii*, *Macrosiphum granarium*, *Rhopalosiphum maydis*, *Macrosiphum dirhodium* and *Toxoptera graminum.* The requirements of the vectors for host plants and temperature are quite different. These environmental factors influence overwintering and survival as well as forms of the metamorphosis. All these are important elements of epidemiology. *Rhopalosiphum fitchii* accommodates well to low temperature, while *Rh. maidis* requires a higher temperature for reproduction. In California *Rh. fitchii* is the most important vector. On the other hand, in Europe, *Rh. padi* has the most important role (Rademacher and Schwarz).

Comparative investigations have shown that different species of aphids transmit the virus with different efficiency. It occurred that the same vector which has been a host of the virus proved elsewhere to be indifferent.

Generally speaking 24 hr is necessary for the vector to feed on the contaminated plant and take up the virus, and another 24 hr are needed to infect the healthy plants. It is remarkable that the virus may be carried from aphid to aphid, as was reported by Orlov.

There are several strains of this virus which are differentiated on the basis of their activity in different aphids and the host range. Allen selected 16 strains and 7 types of the virus out of 43 isolates. However, these produced similar symptoms on the differential varieties.

The specialization of the aphids to carrying different strains of the virus is a usual phenomenon. For example *Rh. fitchii* is known as a much more efficient vector than *Macrosiphum granarium.*

The development of a virus epidemy is the consequence of several factors. The most significant factors are: the initial appearance, multiplication and mobility of the aphids, the possibility to transmit virus strains and the survival of the virus reservoirs etc.

Climate also has an effect on the migration of the aphids. The migration goes generally from South to North, and from West to East. Suffering used to induce migration of the population. In the states of the coast of the Pacific Ocean having a moderate climate (like California, Washington, Oregon), where the moist climate is scarcely interrupted by March dryness and grasses are growing well, there are many possibilities for the survival, even on non-primary hosts. From these hosts a great invasion of aphids can move to the early sown cereals.

In the valley of Columbia the climate is continental. Therefore, the alternate change of mild and severe winters cause the epidemies to be periodical.

The protection to barley yellow dwarf is problematic. Up to the present the most significant control measure is the system of the cultivation. The time of sowing is of decisive importance. Late sowing is disadvantageous. The quantity of seeds used for sowing is also influencing susceptibility. Danger is larger on scarcely sown areas, it is therefore, that the plant breeders complaints about the spreading of the virus are more frequent.

Crop-rotation has no direct influence on the spread of this virus, because the virus is not soil-borne. Grasses which are hosts of the virus or hosts of the vectors exert an indirect influence.

The effect of insecticides is not satisfactory. It has only a static influence on the primary invasion. Though most of the U. S. varieties are susceptible, the most resistant ones are used as sources of resistance. Barley varieties such as C. I. 1227, 1237, 2376, Abate, Rojo are considerably tolerant. Besides there are other important varieties of Abyssinian origin.

Among oat varieties Albion (C. I. 729) and Fulghum (C. I. 1915) are fairly tolerant. Kanota (C. I. 839) and Bond (C. I. 2733) are also tolerant in California.

The breeding for resistance by crossing is also an important way of controlling this disease. Very good intermediates have been obtained by crossing Rojo barley variety tolerant to the virus with the susceptible Club Mariout. *Avena strigosa* is also resistant.

J. SZIRMAI

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G. UBRIZSY and J. VÖRÖS

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Observations on Karyology, Hyphal Fusions, and Production of a New Isolate in *Colletotrichum falcatum*

By

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The conidia of both the light and dark type isolates of *Colletotrichum falcatum* are shown to be uninucleate prior to germination, becoming multinucleate as they germinate. Illustrations are given of hyphal fusions, 'side-to-side', 'end-to-side' and 'peg-to-peg', the latter sometimes at several different points between the same pair of hyphae. Besides, certain rare types of fusions, such as those between two conidia, and between a conidium and an appressorium have also been recorded. Nuclei in the process of migration are shown in the bridging hyphae. Intermixing of the two different isolates produced a new isolate, which differed in pathogenicity as well as cultural characters.

Introduction

The red rot fungus of sugarcane commonly occurs in its imperfect state, *Colletotrichum falcatum* Went, and is responsible for considerable loss to the crop in certain sugarcane-growing areas of the world. Considerable variation of cultural and pathogenic characters has been demonstrated in this state of the fungus. The perfect stage, *Glomerella tucumanensis* (Speg.) v. Arx and Müller (= *Physalospora tucumanensis* Speg.), does occur in nature but infrequently. In recent years, it has been shown that in certain fungi, genetic systems based on heterokaryosis and parasexuality operate in absence of the sexual (perfect) stage. This indicates that imperfect fungi have as efficient mechanisms for the production of new variations as those known in fungi reproducing sexually. DUTTA and GARBER (1960) have attempted to demonstrate the occurrence of heterokaryosis and parasexuality in *Colletotrichum lagenarium* (Pass.) Ell. and Halst., using certain biochemical characters as genetic markers.

The morphological and cytological aspects of heterokaryosis and parasexuality have not been elucidated in sufficient detail. The most obvious way of nuclear exchange is through hyphal or germ tube fusions. A detailed morphological account of hyphal fusions was presented by BULLER (1933). However, this suffers from lack of cytological details. In this paper an attempt has been made to

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provide as far as possible a full account of hyphal anastomoses as observed in *Colletotrichum falcatum*. BULLER's terminology has been adopted for describing the various types of hyphal fusions. The nuclear condition of germinated and ungerminated conidia has also been described. It has been pointed out elsewhere (SINGH and CHONA, 1964), that the ungerminated conidia in this fungus are ordinarily uninucleate. The production of a new isolate, presumably by hyphal fusion, and its cultural and pathogenic characteristics have also been discussed.

Materials and Methods

Conidia from 12–16-day-old cultures of five different isolates (Nos* 78, 244, 300, 382 and strain 'I') of *C. falcatum* were smeared on clean slides, fixed in Carnoy's fixative (JOHANSEN, 1940) containing formaldehyde (5%) and lactic acid (10%), and stained either in aceto- or propiono-carmine, or in Giemsa (BDH). Material to be stained in carmine was hydrolysed in 0.17 M solution of NaCl (20 minutes at room temperature). In case of staining with Giemsa, hydrolysis was made in HCl-perchloric acid. Iron-alum (4%) was used as a mordant for carmine (20–30 minutes) and ammonium molybdate (1.5%) for Giemsa (20 minutes).

Conidia of the two isolates (Nos 78, 244) were placed side by side on egg-albumen coated glass slides and incubated for 24 hours at 27° C** in moist chambers. Within this period conidia germinated and the germ tubes elongated considerably. The material was fixed in Carnoy's fixative and stained in carmine after it had been ascertained that a sufficient number of anastomoses between the germ tubes had occurred. In culture, hyphal anastomoses were effected by seeding oat-meal agar plates with conidial suspensions of the two isolates at two points, about 4 cm apart. These were incubated at 25° C*** for 7 days. After 6 days the margins of the colonies had begun to coalesce. Transfers were made from 3 different points along the line of mutual contact. In this way, 3120 pickings were made from 1040 culture plates. Apparently, 29 such picked cultures seemed quite different from either of the parents and 17 more also showed slight alterations. Single sporing and further subculturing of these cultures thrice showed that only 13 remained different from the parents, all the rest merged with one or the other parent in their morphology. Finally, those cultures appearing markedly different from the parents, but which looked more closer to each other in the details of morphology were again purified by single sporing, and 10 cultures, thus obtained after another seven subculturings, were established. These cultures exhibited good stability of their morphology and appearance during ten more generations or subculturings. Control studies were also conducted by mingling or

* Indian Type Culture Collection.
** Optimum temperature for the germination of conidia (SINGH, 1962).
*** Optimum temperature for the growth of the fungus.

mixing the two cultures of the same isolate, i. e. isolate No. 244 with itself, and isolate No. 78 with itself. A similar number of pickings were made from such "self-mixing" in each case of these isolates, as was done with "cross-mixing" of isolates Nos 78 and 244. On subculturing, the culture showing differences in case of each isolate, it was noticed that 11 slightly different cultures were obtained in the case of isolate No. 244, and 7 such different cultures were obtained in case of isolate No. 78. The minor differences shown by these cultures as compared to the parents were mycelial growth, little colour difference and amount of sporulation. After single sporing and further subculturing these cultures, two or three times, it was observed that all these merged with one or the other parents and not even one such culture was stable enough to carry on itself in the 3rd or 4th generation or in subculturing.

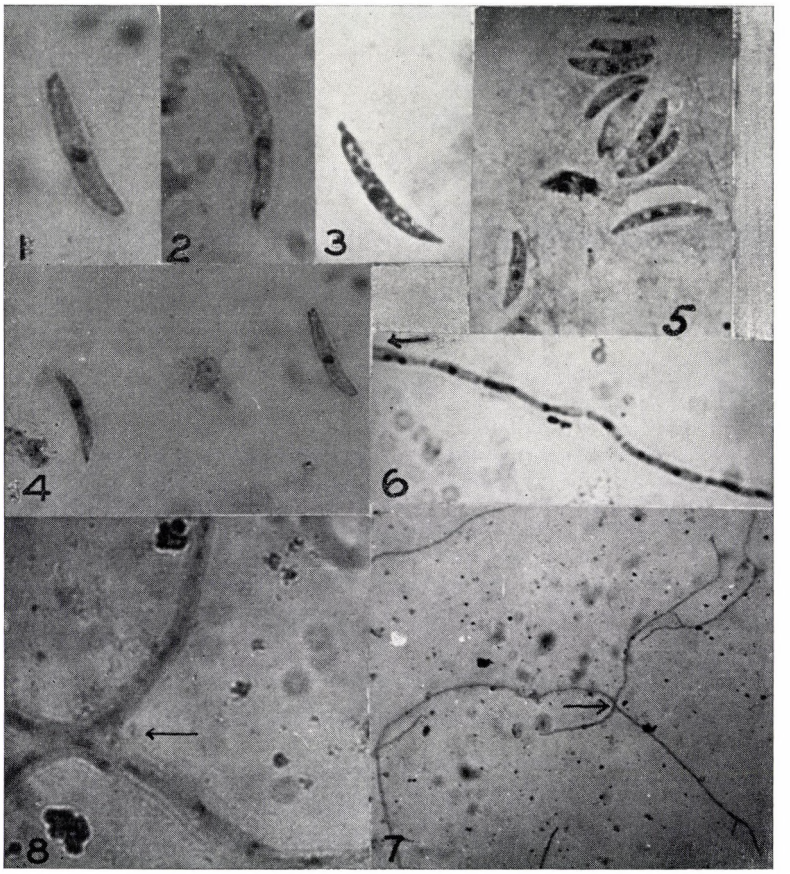
Observations

Nuclear condition in conidia: Young as well as nature conidia of all the five isolates were observed to be uniformly uninucleate (Figs 1, 2, 3, 4, 5). The nucleus is small and round with no difference in the size in different isolates, varying from $1.4-2.1\ \mu$ in all preparations. The nucleus is located ordinarily in the middle of the falcate conidium. Occasionally, it may lie nearer to either of the two ends, more often toward the concave side. The nuclei in Giemsa stained preparations were not found to be larger than those stained with carmine as was observed by LEAKEY (1960) in *Colletotrichum gossypii*. In carmine preparations, the nucleus appears as a deep red, spherical solidly stained mass (Fig. 26). However, with Giemsa staining, the stuctural details of the nucleus were better revealed, it consisting of a well developed nucleolus and elongated chromatin threads (Fig. 25). PINTO-LOPES (1949) referred to such an appearance of the nucleus as 'expanded or thread type'. PINTO-LOPES, however, considered this term for the nucleus at a particular stage of the development of a cell. We are using this term for describing the morphology of the nucleus, not in any way reflecting on the physiology or development of the cell or the nucleus.

Nuclear condition during germination: These studies were restricted to isolate No. 244. Just prior to or at the time of emergence of the germ tube, the nucleus of the conidium divides once, twice or three times with the result that the cell becomes 2, 4, or 8- nucleate (Figs. 27, 28, 29, 30). The nuclei are usually concentrated in the middle part of the conidium.

The formation of a septum in a conidium appears to be correlated to the type of the germination. Normally, a septum is observed in those conidia which germinate in a bipolar fashion. Each of the resulting cells is 2 or 3-nucleate (Figs. 31, 32). It has been observed that the two germ tubes do not arise simultaneously. Thus, it seems that a single conidium is capable of performing the functions of two conidia.

Spores with unipolar type of germination ordinarily remain aseptate (Fig.

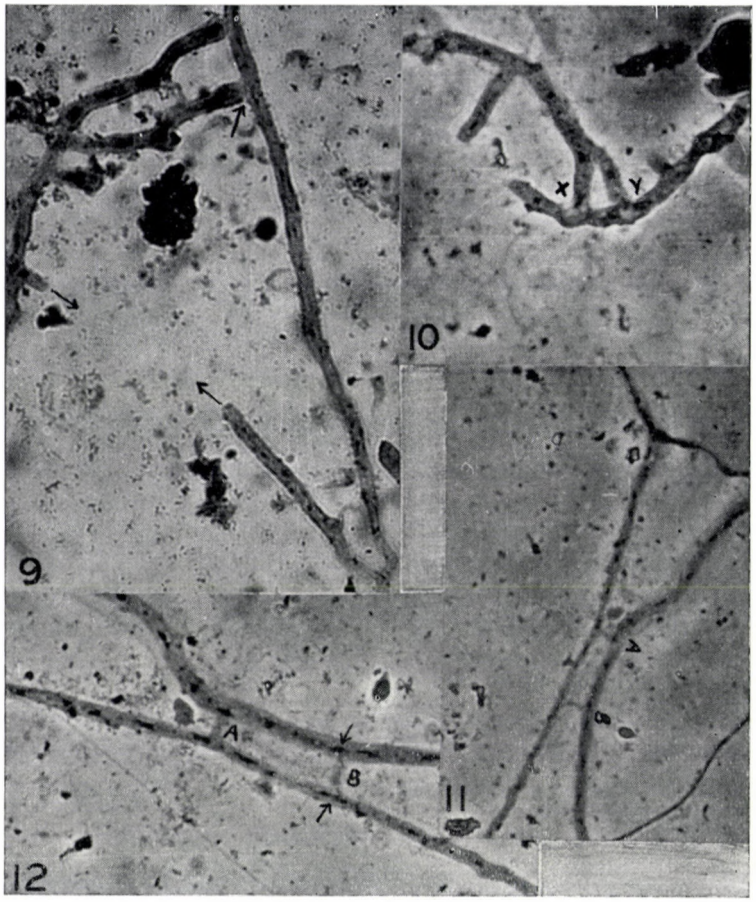


Figs 1, 2, 3, 4, 5. Uninucleate conidia. 1, 2 and 3: × 1360 (phase contrast); 4 and 5: × 840
Fig. 6. Portion of a germ tube showing distribution of nuclei in its cells, × 840. The spore has been removed in the picture due to space adjustment, but the position of the spore is being shown by the arrow
Figs 7 and 8. Direct fusion between germ tubes forming a fusion cell. 7: × 120; 8: The fusion cell, × 840

33). Occasionally, the conidia showing unipolar germination also develop a septum (Fig. 34). Conversely, some conidia, which had germinated from both ends, remain aseptate (Fig. 35). Sometimes, 3 or 4 germ tubes arise from a single conidium (Fig. 36).

The nuclei in the parent conidium persist even after the germ tubes have grown considerably.

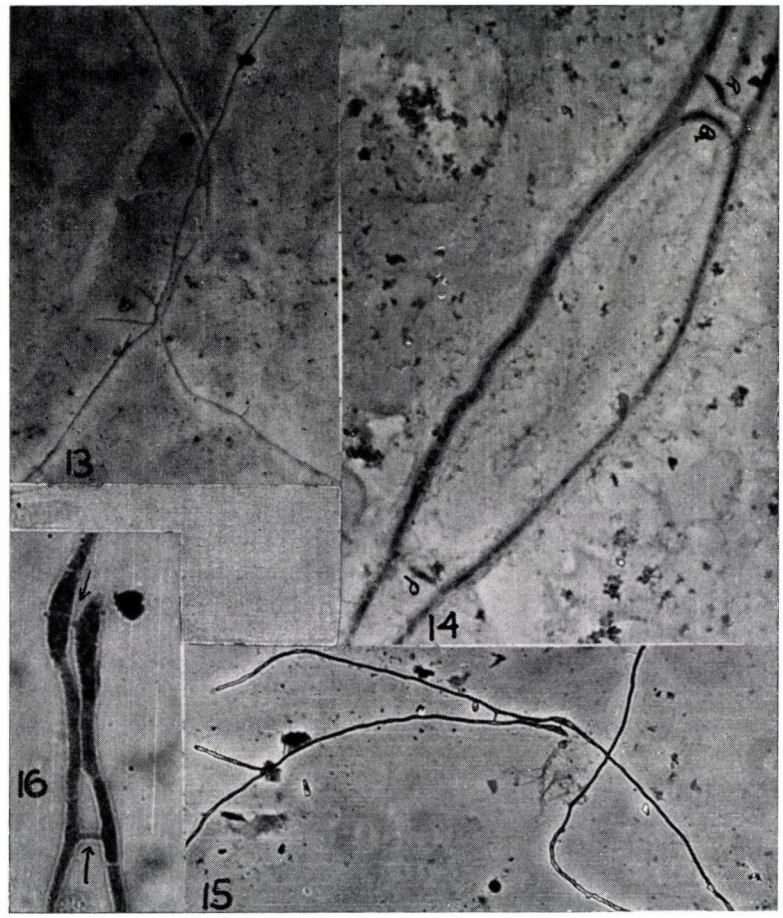
The cells of a germ tube (or better be called as germ hypha) may be one- to four-, occasionally five-nucleate. Cells nearer to the mother spore are ordinarily uni- or bi-nucleate (Figs 6, 32).



Figs 9 and 10. Direct hyphal fusions ('end-to-side'). 9: ×840; 10: ×840 (phase contrast). Figs 11 and 12. 'Peg-to-peg' fusion between two parallel germ tubes. 11: ×540 (phase contrast); 12: ×840 (phase contrast)

Hyphal fusions: Two types of hyphal or germ tube fusions have been chiefly observed, "direct" or "hyphal peg" fusion. The latter results in H-shaped configurations.

(a) *Direct hyphal fusions:* (i) Side-to-side: In Figs 37, 38, the intervening walls at the point of contact of two hyphae dissolved, resulting in a cross-like fusion cell which contains as many as 6 nuclei. In Figs 7, 8, the boundaries of the fusion cell cannot be discerned clearly because the septa are obscure. (ii) *End-to-side:* In Figs 9, 10, the two ends of a dichotomously branched hypha are fusing with the germ tube of another conidium. The intervening walls seem to have dissolved in one of the fusions (the lower one in Fig. 9). In Fig. 10, the intervening



Figs 13 and 14. Multiple fusion between two germ tubes of two conidia. 13: Three successive fusions, P, Q, R ×120 (phase contrast); 14: ×840 (phase contrast)
Figs 15 and 16. 'Peg-to-peg' fusion between two conidia and their germ tubes. 15: ×320 (phase contrast); 16: ×840 (phase contrast)

walls have completely dissolved at the points of contact, X and Y. A nucleus is seen at X, apparently in the process of migration.

In Figs 11 and 12, fusions occur between two parallel hyphae at two different places (A and B). There is no nucleus in the bridging hypha. At fusion B one of the two nuclei in the lower cell seems to be in telophase stage of division, and one of the two nuclei in the upper cell appears to be in the process of migration.

In Figs 13, 14 and 39, three successive fusions between two different germ tubes are observed. At fusion P (Fig. 39), the bridging hypha contains one nucleus. The upper cell shows a nucleus at its junction with bridging hypha and one of

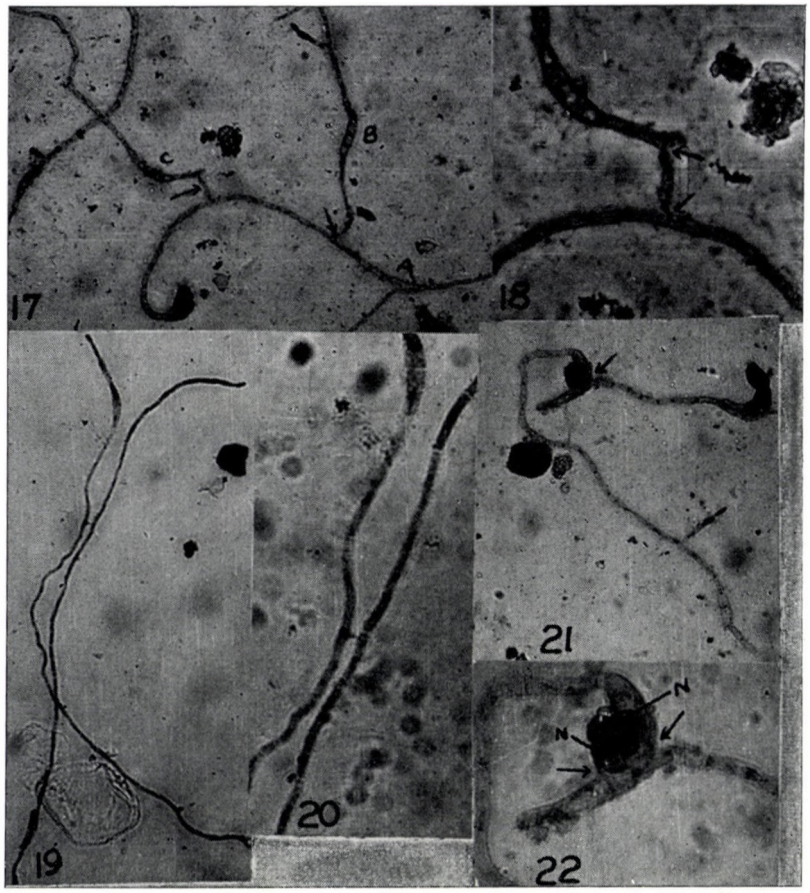


Fig. 17. Triple fusion between three germ tubes from three different conidia, A, B, C × 320
Fig. 18. One of the fusions in Fig. 17 showing nuclear migration. × 840 (phase contrast)
Figs 19 and 20. 'Peg-to-peg' fusion between two parallel running germ tubes. 19: × 320; 20: × 840
Figs 21 and 22. Fusion of a conidium with the appressorium of another conidium. 21: General view, × 320; 22: The point of contact with nuclear condition, showing nuclei in the appressorium (marked as N) and in the conidium, × 840

the nuclei on the right appears to be in telophase stage of division. At fusion Q, a nucleus is visible in the bridging hypha, while at R, two such nuclei are present.

In Figs 15 and 16, fusion through pegs between two conidia is noteworthy. The conidium on the right has put forth a peg which touches the other conidium. However, the intervening walls at the point of contact have not yet dissolved. It is an early stage of fusion. In another fusion between the germ tubes of the conidia, the bridging hypha provides protoplasmic continuity between the fusing cells.

In Figs 17 and 18, a case of multiple anastomoses is seen. One of the germ tubes of a spore (A) has first fused directly with the germ tube of a spore (B), and then by means of a peg with the germ tube of another spore (C). The nuclei in the latter case of fusion are seen in the process of migration (Fig. 18).

In Figs 19 and 20, a single fusion between the germ tubes of two spores is taking place by means of a thin connecting hypha.

An interesting type of fusion between an appressorium and a conidium was also noticed (Figs 21 and 22). The appressorium of a spore is fusing directly with the upper cell of a germinated spore. The outer walls of the appressorium as well as the conidial cell have dissolved away, but the inner walls seem to be intact. The appressorium is seen to have two nuclei.

Intermixing of isolates: As noted in 'Materials and Methods', ten isolates were finally picked up and established from the line of intermixing of the two isolates (Nos 78 and 244). Such isolates were established and maintained by propagating on oatmeal agar for ten generations or subculturings (each subculturing after 15 days). These established isolates were again single spored, subcultured and found to be quite stable in their morphology. Because of their similarity with each other and almost no morphological differences, all these isolates were grouped as one isolate.

Comparative summary description of the important characters of isolate Nos 78, 244 and the "new isolate" is as follows:

Isolate No. 78

Abundant mycelial growth; aerial mycelium mineral grey to olive-buff; colony thick, compact, even and felt-like; no obvious sign of sporulation; sporulation scanty; conidia $35 \times 7 \mu$, uni-nucleate, nuclei 1.4—2.1 μ in diameter, mostly straight or slightly curved; linear spread of infection is 26.7 and 23.1 cm on CO. 647 and CO. 312, respectively.

Isolate No. 244

Scanty mycelium growth; aerial mycelium white to light mineral grey; colony loose and cottony; sporulation obvious and very profuse, and in pink or salmon coloured spore masses; conidia $38 \times 7 \mu$, abundant, uni-nucleate, nuclei 1.4—2.1 μ in diameter, normally curved; linear spread of infection is 40.0 and 35.8 cm on Co. 647 and CO. 312, respectively.

"New Isolate"

Abundant mycelium growth; aerial mycelium light mineral grey to mineral grey; colony compact, loose at the margins; sporulation quite obvious and profuse; conidia $42.0 \times 9.2 \mu$, abundant, uninucleate, nuclei 2.4—2.6 μ in diameter, mostly normally curved; linear spread of infection is 69.0 and 61.5 cm on CO. 647 and CO. 312, respectively.

The new isolate resembled the isolate No. 78 in so far as the mycelial characters are concerned, i. e. there was more compact development of hyphae, and the mycelial growth was abundant and mineral grey in colour. On the other hand, it exhibited a markedly superior capacity for sporulation similar to the isolate No. 244. The spores were slightly bigger in size. However, no difference was observed in the shape and other morphological characters of the conidia. Conidia were uni-nucleate like the parent isolates (Fig. 23), but the nuclei seemed to be slightly bigger in the case of the "new isolate".

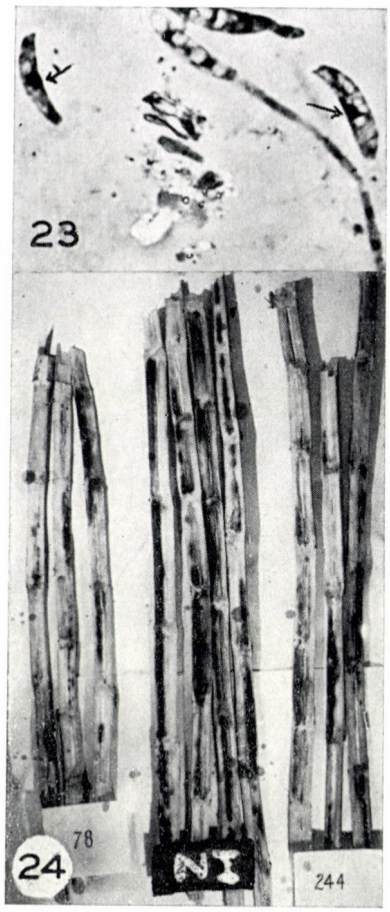


Fig. 23. Uninucleate conidia of the new isolate, two of the spores having germinated, the protoplasm seemed to be highly vacuolate, ×840 (phase contrast)
Fig. 24. Vertically split canes showing the linear spread of infection. The new isolate (N. I.) showing maximum infection spread as compared to the parent isolates, 78 and 244

The most significant difference was that concerning the pathogenicity. The data on pathogenicity of the parent isolates (Nos 78 and 244) and the "new isolate" (N. I.) have been presented in Table I and illustrated in Fig. 24. There was a marked increase in the linear spread of infection by the "new isolate" (N. I.) in both the cane varieties, CO. 647 and CO. 312. Reisolations were made and the stability of the isolate was maintained through another 18 generations or sub-culturings. Subsequently, one of these ten cultures of the "new isolate" reverted to the parental dark type (No. 78) by becoming predominantly mycelial, and loss in

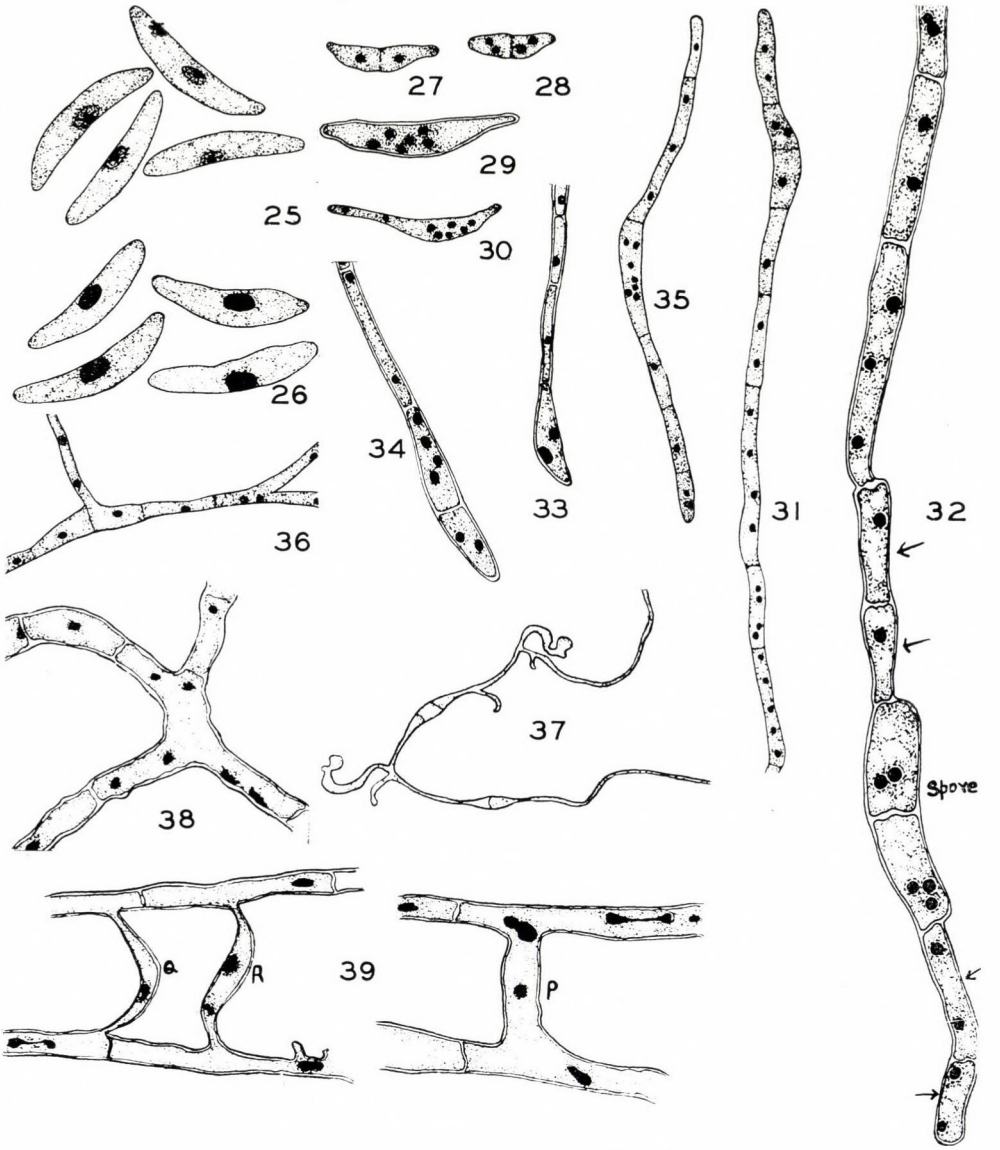


Fig. 25. Giemsa stained nuclei, ×1800
Fig. 26. Propiono-carmine stained nuclei, ×1800
Figs 27, 28, 29 and 30. Two, four, six and eight-nucleate stage in germinating conidia. 27, 28, 30: ×760; 29: ×1800
Figs 31 and 32. Bipolar germination in septate conidium, each cell with 2—3 nuclei. 31: ×750; 32: Also shows nuclear distribution in the cells of germ tube, uni- and bi-nucleate cells near the spore, ×1800
Fig. 33. Unipolar germination in aseptate conidium, ×1800
Fig. 34. Unipolar germination in septate conidium, ×1800
Fig. 35. Bipolar germination in aseptate conidium ×750
Fig. 36. Three germ tubes out of a conidium, ×750
Figs 37 and 38. Direct fusion between two germ tubes forming a cross-like fusion cell. 37: ×240; 38: The fusion cell with 6 nuclei, ×840
Fig. 39. Enlarged view of nuclear condition in the three fusions, shown in Figs. 13 and 14, ×1800

Table I

Comparative virulence of the light type, dark type and the "new isolate" on cane varieties CO. 647 and CO. 312

Isolate No.	CO. 647			CO. 312		
	Average L. S. (cm)	Range (cm)	No. of I affected	Average L. S. (cm)	Range (cm)	No. of I affected
New isolate	69.0	62.8—74.9	5—9	61.5	56.3—64.1	4—7
244	40.0	36.4—43.1	3—5	35.8	33.4—40.1	3—4
78	26.7	22.3—30.0	2—3	23.1	21.6—26.9	2—3

(L. S. = Linear spread of infection (total) on either side of the point of inoculation; I = Internodes involved)

the ability of sporulation and greater virulence. After another 10 generations, one more such culture became dark type.

Discussion

The conidia of all the isolates of *Colletotrichum falcatum* examined so far have been found to be uninucleate. As has been pointed out earlier (SINGH and CHONA 1964) this observation is in accord with that of MESSIAEN (1955) who has made cytological observations on a number of species of *Colletotrichum* and *Gloeosporium*. The nucleus of *G. falcatum* in well fixed preparations, stained in Giemsa, has been found to be of the 'expanded' or 'thread' type. However, the unexpanded type of nuclei have only been observed in carmine stained preparations. These differences in the appearance of the nuclei may possibly be due to different staining procedures.

The hyphal anastomoses observed in *C. falcatum* have been found to be of various types. Their classification is in accordance with that proposed by BULLER (1933). The direct fusion of germinating conidia, a conidium fusing with an appressorium, and 'end-to-side' fusion of two sister branches with another germ tube, are of rare occurrences.

CARVAJAL and EDGERTON (1943, 1944) had reported that in its perfect stage, *Glomerella tucumanensis* (Speg.) v. Arx and Müller (=*Physalospora tucumanensis* Speg.), the fungus is homothallic. Evidence has been produced here to demonstrate that genetic variability can be induced in the absence of the sexual stage. The isolates 244 and 78 are known to differ in their cultural characters and pathogenicity. Intermixing of these two strains resulted in the production of a new isolate (comprised of ten culture, grouped tentatively as one). This new isolate differed considerably in certain cultural characters from either of the parent

strains. It also exhibited a high degree of virulence, as determined by the linear spread of infection in canes. No exact basis for this sort of variation has been analysed. However, it is presumed to be due to either some sort of cytoplasmic inheritance or nuclear exchange and formation of new nuclear associations, since the conidia of the new isolate are uninucleate. The former can be presumed on the basis of the fact that cytoplasmic inheritance can also last through several generations. However, the latter possibility for nuclear exchange and formation of a new nuclear association seems to be more plausible in view of the observations of hyphal anastomoses, described in earlier pages. The greater virulence of the new isolate appears to be a result of heterosis (a result of somatic karyogamy). Surprisingly, the frequency of the phenomenon in this fungus, as seen from the figures, i.e. 10 out of 3120 pickings, seems to be quite high as compared to what has been recorded earlier in several fungi. The authors are not aware of any reason for that. However, they do suspect this to explain as to why this fungus has been showing such a tremendous amount of variation as reflected by its history in this country.

The loss of the characters, like high degree of sporulation and virulence, and reversal of the two new isolate cultures to the dark type parental isolate (No. 78) and none to the light type parent (No. 244) seems to follow the usual pattern of degeneration of fungi in culture, i.e. loss of certain physiological and pathogenic characters. This has been observed earlier by LUCAS (1943) and FOSTER (1949) in *C. falcatum* and several other fungi. They have termed it as "physiological degeneration" involving a detrimental change in the physiological potentialities of a culture, and regarded it as a continuous changeability of the fungus.

Though there has developed a considerable body of knowledge about heterokaryosis and parasexuality in fungi, the cytological aspects of these phenomena seem to have received little attention indeed. The phenomenon of nuclear fusion or the somatic karyogamy, mitotic haploidization and others have been deduced from genetic analysis only. It seems desirable to gain a cytological evidence for these events, thereby getting an integrated picture of these types of genetic variation in fungi.

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Increased Leaf Growth Regulated by Rust Infections, Cytokinins, and Removal of the Terminal Bud

By

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Infection of leaves of bean and broad bean with *Uromyces phaseoli* and *U. fabae* results in an increased leaf growth accompanied by increased leaf size and thickness. These symptoms can be simulated by treatment with cytokinins or removal of the terminal bud. Both of these treatments induce not only an increased leaf size and thickness but several other symptoms too, characteristic to rust infections, such as greening of the leaf tissues, higher level of protein and nucleic acid as well as increased incorporations of precursors of protein and nucleic acid synthesis. It is suggested that the removal of the terminal bud of Pinto beans induces these changes in primary leaves by a cytokinin-like effect. It is also assumed that the increased leaf growth caused by rust infections is a consequence of a cytokinin-like effect.

Introduction

As early as 1951 YARWOOD and COHEN reported on the increase in total growth of primary bean leaves infected by bean rust *(Uromyces phaseoli)*. Recently in our laboratory we were able to detect cytokinin-activity in rust infected leaves of beans and broad beans (KIRÁLY, POZSÁR and EL HAMMADY, 1966). Furthermore it was also shown that it is possible to simulate by treatments with cytokinins (kinetin and benzyladenine) some characteristic symptoms of the bean rust infection (POZSÁR and KIRÁLY, 1966). Of particular interest was the symptom which was caused by both rust infection and treatment with cytokinin in secondary growth. In this case a retardation in growth of secondary leaves (trifoliates) was experienced as a consequence of both rust infection and cytokinin effect on the primary leaves. The effect of kinetin and benzyladenine on leaf disc enlargement has been noted by SCOTT and LIVERMAN (1956), KURAISHI and OKUMURA (1956) and MILLER (1956). LEOPOLD and KAWASE (1964) reported on the stimulation of the growth of intact trifoliate bean leaves after treatment with benzyladenine. All these data pointed to the possible role of cytokinins in causing increased leaf growth in rust infected plants.

Materials and Methods

Plants were grown under ordinary greenhouse conditions. Twin leaves or half leaves were compared in the experiments as control or treated (infected), respectively. *Phaseolus vulgaris* var. *Pinto* and *Vicia faba* var. *Baranyai* infected with *Uromyces phaseoli* and *U. fabae*, were used in the experiments. Leaves were treated daily with cytokinins and leaf extracts exhibiting cytokinin-like activity for 6 successive days.

In the chlorophyll retention test, 1 cm sections of first leaves of barley were floated on test solutions and put into darkness at 27° C for 4–5 days. The optical density of leaf extracts was measured at 665 mμ. Leaf tissue of 100 mg fresh weight was extracted twice with 80 per cent methanol, then with a 1 : 1 v/v mixture of ethyl ether : methanol. This solution was assayed in a photometer.

To investigate the intensity of protein synthesis the incorporation of glycine-C^{14} and cysteine-S^{35} into the acid insoluble (protein) fraction was determined. Incorporation of the nucleic acid bases, adenine-C^{14} and guanine-C^{14}, into the nucleic acid fraction was also investigated indicating thereby the intensity of nucleic acid synthesis. In each case a 10 μC/50 ml solution was applied to leaf discs by floating them on the solution. Specific activity for glycine, cysteine, adenine and guanine was 26 mC/mM, 17 mC/mM, 11.4 mC/mM, and 1.0 mC/mM, respectively.

Protein and ribonucleic acid contents of tissues were assayed by methods described in another paper ((KLEMENT, KIRÁLY and POZSÁR, 1966). Similarly, the method of evaluation of radioactivity was outlined in this work.

The procedure for extraction of cytokinin-like substance(s) ("isolated cytokinins") from rust infected, decapitated as well as control primary leaves of Pinto bean was described in an earlier paper (KIRÁLY, EL HAMMADY and POZSÁR, 1967).

Results

Increase in leaf size

The increased size of primary bean leaves resulted from *(a)* rust infection, *(b)* treatment with cytokinins (kinetin and benzyladenine) and *(c)* treatment with extracts from healthy or rust infected leaves having cytokinin activity.

Similar to the observation of YARWOOD and COHEN (1951) we detected a considerable increase in leaf size after infection with rust (Fig. 1). If only half leaf of primary bean leaf was infected with rust or treated with benzyladenine leaf growth was asymmetric because of the stimulated growth of the affected half leaf (Figs 2 and 3).

The results seen in Tables 1 and 2 show the effect of rust infection on the increase of leaf size of beans and broad beans.

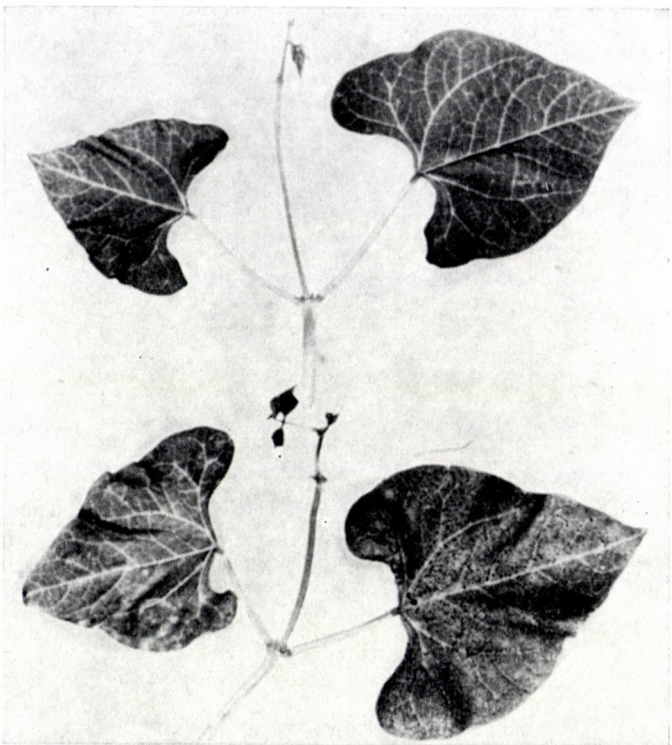


Fig. 1. Increase in leaf size of Pinto bean as a result of rust infection or treatment with a cytokinin. Upper twin leaves: left, control leaf; right, leaf treated with 30 ppm benzyladenine for 6 days. Lower twin leaves: left, control leaf; right, primary leaf infected with rust *(Uromyces phaseoli)* in the sporulation stage

Table 1

The effect of rust infection and treatments with cytokinins on the primary leaf size of Pinto bean

Treatment	Twin leaf size in cm^2		Increase of leaf size in per cent
	Before treatment	After 2 weeks	
Control (H_2O)	27.22	41.10	151
Rust infected: Sporulation stage	27.62	46.95	170
Control (H_2O)	20.01	36.18	181
Benzyladenine (30 ppm)	19.62	53.93	275
Control (H_2O)	19.32	34.78	180
Kinetin (50 ppm)	19.65	45.19	230

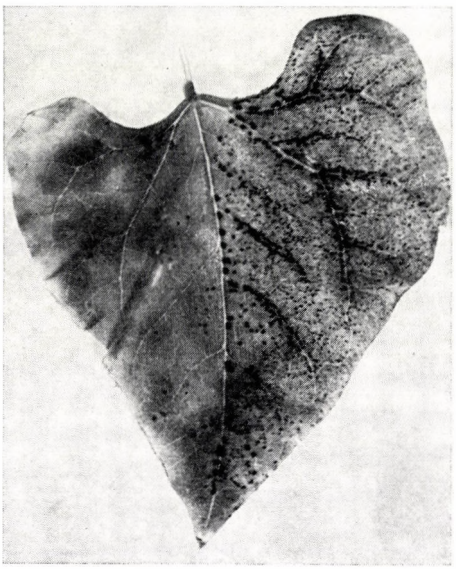


Fig. 2. The effect of rust *(Uromyces phaseoli)* infection on the growth of half leaf of Pinto bean as compared to the uninfected half leaf. Left, control; right, rust-infected

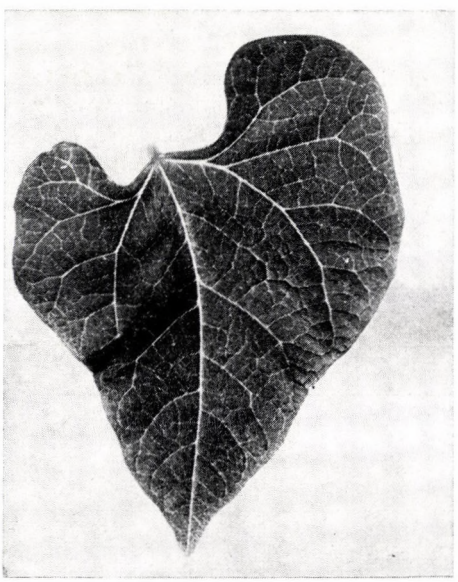


Fig. 3. The effect of treatment with benzyladenine (30 ppm) for 6 days on the growth of half leaf of Pinto bean as compared to the control (treated with water). Left, control; right, treated with benzyladenine

Table 2

The effect of rust infection and treatments with a cytokinin on the size of broad bean leaflets

Treatment	Size of the leaflet cm^2 after 2 weeks	Per cent
Control (H_2O)	13.38	100
Rust infected:		
Chlorotic stage	16.09*	120*
Sporulation stage	17.64	132
Benzyladenine (30 ppm)	26.63	199

* After 5 days.

It is of interest that rust infection affects leaf growth of broad bean as early as 4–5 days after inoculation, i.e. in the chlorotic stage. At this stage of disease one cannot experience a conspicuous effect of rust infection on the leaf size of bean; this is seen only later, namely, in the sporulation stage.

Similar to rust infections, treatments with extracts from rust infected leaves also increased the leaf size. Extracts from healthy leaves had some activity but lower than that of rusted leaves (Table 3).

Table 3

The effect of extracts from healthy and rust infected bean leaves on the primary leaf size of Pinto leaves, compared with benzyladenine

Treatment	Twin leaf size in cm^2		Increase of leaf size in per cent
	Before treatment	After 2 weeks	
Control (H_2O)	26.52	42.42	160
Extract from healthy plants	27.38	45.07	165
Control (H_2O)	27.5	44.64	162
Extract from rust infected plants	27.16	48.11	177
Control (H_2O)	20.01	36.18	181
Benzyladenine (30 ppm)	19.62	53.93	275

In another experiment the pathogen was killed in the chlorotic stage by heat, immersing bean leaves in hot water (50° C) for 20 seconds and placed into darkness at 27° C for 2–3 days. Leaf growth stimulation was not diminished by this treatment. After 2–3 days of the heat treatment characteristic *green islands*

appeared at the site of infection, showing a kinin-like activity at the infection centers. This phenomenon showed that cytokinins produced by the infection remained active after the killing of the fungus, or the stimulated cytokinin-activity of the infected host cells remained uninfluenced after the death of the pathogen.

In experiments where plants were treated with benzyladenine or "isolated cytokinin" the leaves remained dark green for a considerable long time. Control twin leaves became senescent in about 2–3 weeks and had a yellow or green-yellow color. On the other hand, cytokinin-treated leaves remained in a juvenile stage where the destruction of chlorophyll was retarded by the kinin-effect. These leaves contained more protein and RNA as well as more chlorophyll. About these results another paper will report which is under preparation.

The effect of decapitation

The influence of the removal of the terminal bud from bean plants is quite similar to that of the cytokinin treatments. The first characteristic of the decapitation is the dark green color of the primary leaves compared to the controls. In the latter case, primary leaves become senescent in about two weeks after the full development. This is attributed to the enhanced export of nutrients out of the old leaves toward the younger parts (POZSÁR and KIRÁLY, 1966). However, primary leaves of bean plants from which the terminal bud was removed did not become senescent and exhibited an increase in leaf growth. As is seen in Table 4 the leaf size

Table 4

The effect of the removal of the terminal bud on the size and dry matter content of primary leaves of Pinto bean, 10 days after decapitation

Treatment	Leaf size in cm^2		Increase in leaf size per cent	Dry weight	
	Before experiment	After experiment		mg/15 cm^2	Per cent
Intact plants	32.12	45.21	140	32.5	100
Decapitated plants	35.92	64.35	179	50.5	155

and the dry weight of the leaf discs (15 cm^2) are greater in decapitated plants than in the controls. Hence, this effect is similar to the action of benzyladenine or that of the extracts from plants having a cytokinin activity. Indeed, the removal of the terminal bud is able to induce higher levels of protein and nucleic acid and higher rate of synthesis of these substances, as well as higher cytokinin-activity in tissues of primary leaves (Tables 5, 6 and 7). All these data point to the possible relation between decapitation and increased leaf size as well as the juvenile character of the primary leaves of bean.

Table 5

Stimulatory effect of decapitation on the tissue level of protein and ribonucleic acid (RNA) in primary leaves of Pinto bean, two weeks after decapitation

Treatment	Protein mg/g fresh wt	Per cent	RNA μg ribose/g fresh wt.	Per cent
Control (intact)	15.3	100	7.1	100
Decapitated	23.0	150	10.1	142

Table 6

Stimulatory effect of decapitation on the incorporation of amino acids and nucleic acid bases into the acid insoluble fraction of Pinto bean leaves, two weeks after decapitation

Labelled compounds	C. p. m/100 mg fr. wt.		Increase in per cent
	Control (not treated) plant	Decapitated plant	
Glycine-C^{14}	1 071	1 435	134
Cysteine-S^{35}	47 517	62 532	132
Adenine-C^{14}	2 735	5 275	193
Guanine-C^{14}	1 763	2 733	155

Table 7

The effect of cytokinin-like substance(s) extracted from intact and decapitated plants on the dry matter content of primary leaves of Pinto bean and on the chlorophyll retention of detached barley leaves

Treatment	Dry weight		Chlorophyll content	
	mg/15 cm^2	Per cent	Extinction	Per cent
Control (H_2O)	39.0	100	0.47	100
Leaf extract from:				
Intact	42.5	108	0.51	108
Decapitated	47.9	123	0.54	114
Kinetin (50 ppm)	51.8	133	0.62	132

Discussion

As a consequence of increase in leaf size, the dry matter production is also increased in *(a)* rust infected, *(b)* in benzyladenine- or kinetin-treated bean leaves, *(c)* in primary leaves of bean attached to decapitated plants, and *(d)* in leaves which were treated with "isolated cytokinin" partially purified from extracts of healthy and rust-infected leaves (KIRÁLY, EL HAMMADY and POZSÁR, 1967), as well as leaves from decapitated plants.

However, dry matter production was affected not only by the increased leaf size but also by the increased thickness of the leaves. Discs cut from each treated plant had a higher fresh weight and dry weight than the untreated controls. The relative water content of the treated leaves was not changed, therefore, data are showing a real increase in dry matter production after the influence of cytokinins. This increase probably results from an activated cell division and enlargement which is a characteristic of the cytokinin effect (MILLER, 1961, LETHAM, 1967). The same result was gained in different experiments where twin primary leaves of the same bean plant or half leaves of the same leaf were compared for dry matter production as influenced by infection or cytokinin treatments.

Most of the experiments outlined above were carried out in fall or winter under relatively low light intensity. Increase in leaf size induced by cytokinins is more pronounced in winter experiments than in summer ones with a high light intensity. Furthermore, young (small) primary leaves respond to treatments with cytokinins more distinctly than the older and larger ones (Tables 1 and 3).

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The Change of Pigments and Sugar Content in the Chrome Mosaic Virus Infected Leaves of Grapevine

By

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The presence of chlorophyll a and b, xanthophylls and β-carotene was established in the healthy and chrome mosaic virus infected leaves of Szerémi Zöld and Red Veltliner grapevines. In both varieties the pigment level was lowest in the most strongly infected leaves. The presence of other pigments than those present in the healthy leaves was not observed in the infected leaves. The chlorophyll content, due to their mosaic like discoloration visible even in October, was higher in the infected leaves, than in the healthy leaves turned completely yellow during autumn. The dry matter and sugar contents of the infected leaves increased during the period between May and October, in spite of the infection, but was generally less than that of the healthy leaves. However, in October, the infected leaves of the Red Veltliner contained more sugar than the healthy ones.

Introduction

In a previous publication the authors (JÁKÓ, LEHOCZKY and SÁROSPATAKI, 1966) gave an account of the damage caused by the virus infection, at that time known as "Hungarian Yellow Mosaic", and of the nitrogen metabolism of the infected grapevine. According to the serological investigations of MARTELLI, QUACQUARELLI and LEHOCZKY (1967b) the virus seems related to the virus causing the black ring disease in tomatoes. The virus has been renamed "Chrome Mosaic Virus", hereafter CMV.

The CMV infection is characterized by the mosaic-like and later complete yellow discoloration of the leaves. The accumulation of the pigments, the decomposition of the green and yellow pigments of the leaves and the probable change in their proportion was investigated by means of chromatography. Since the reduction of the assimilating activity is a natural consequence of the chlorophyll deficiency, as caused by the CMV infection, it seemed advisable to complement the chromatographic tests by sugar determinations.

Materials and Methods

Leaves for the investigations were collected on the 9th and 22nd May and on the 23rd October, 1967, at the Badacsony vineyard of the Research Institute for Viticulture and Enology. At each date leaves were gathered from two healthy,

two moderately and two highly infected vinestocks of both varieties: Szerémi Zöld and Red Veltliner.

Leaf pigment chromatography: 2 g of fresh leaves were homogenized with the mixture of acetone: petrolether (1 : 2). The leaf residue was removed by filtration. The extract was washed with distilled water and dried over anhydrous Na_2SO_4 and made up to 40 ml with the mixture of acetone: petrolether. 4 ml of the stock solution was then transfered to a Whatmann 1 paper disc with the aid of a paperstrip of 2 cm width. The circular chromatogram was developed with a benzene-petrolether mixture (3 : 1) absorbed again by the paper strip cut out of the disc. The chromatograms were developed in a desiccator saturated with petrolether, in the dark, overnight. Each band containing one of the pigments was cut out and eluated in the mixture of ether : methylalcohol (100 : 5). The extracts were made up to 5 ml. The optical density of the green color was measured at 420 mμ and that of the yellow color at 450 mμ with an Unicam SP 500 type spectrophotometer. The values were plotted in diagram. The results obtained for the two parallel samples of both vine varieties were plotted separately.

The quantitative estimation of the chlorophyll content of the leaves was carried out by the method of COMAR–ZSCHEILE (1942). The chlorophyll was extracted from the leaves with acetone and was then transferred to ether. After washing with water and drying, the optical density of the ether extract was measured at 642.5 and 660 mμ wavelength. The concentration of chlorophyll a and b and the total chlorophyll content was determined using the equation given by the above authors. The mean value of the two parallel tests was tabulated.

The total sugar content of the leaves was established by Bertrand's method and expressed in invert sugar. The sugar percentage on dry basis was plotted in separate diagrams for the two parallels.

The total dry matter content of the leaves was determined by drying at 105° C to constant weight. The mean value of the two parallels was tabulated.

Results

1. The chlorophyll a and b, β-carotene and xanthophyll content of the vine leaves.

The following pigments of the vine leaves were separated: chlorophyll b (1), chlorophyll a (2), xanthophyll$_1$ (3), xanthophyll$_2$ (4) and β-carotene. (Figures 1, 2, 3.)

On comparing the chromatograms it appears that each of the five pigments is present in the leaves of the healthy, the moderately and the highly infected grapevines.

There was no evidence of the presence or accumulation of a yellow pigment in the infected leaves other than in the healthy ones. However, the reduction of the amount of the pigments present in the infected leaves is very marked.

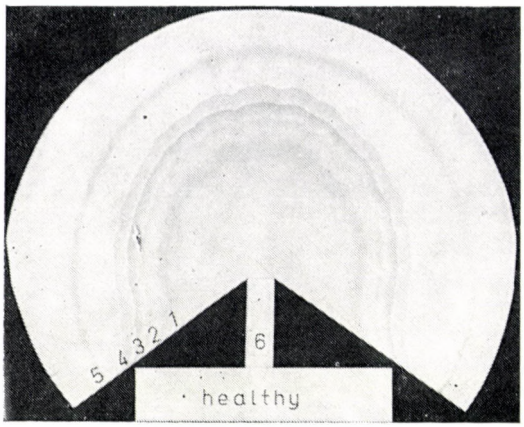


Fig. 1. The chromatogram of the pigments contained in the healthy leaves of the Red Veltliner vine variety. 1. Chlorophyll b, 2. chlorophyll a, 3. xanthophyll$_1$, 4. xanthophyll$_2$, 5. β-carotene

When the leaves gathered on the 22nd May from healthy and infected vinestocks were investigated it was found that the highly infected leaves contained much less chlorophyll a and b (Figures 4 and 5) as well as xanthophyll and β-carotene (Figures 6 and 7) than either the moderately infected or the healthy leaves. There was less pigment in the moderately infected leaves than in the healthy ones, but this difference was not so significant. Since the chromatograms belonging to the healthy leaves of the Red Veltliner variety could not be evaluated they were omitted from Figures 5 and 7.

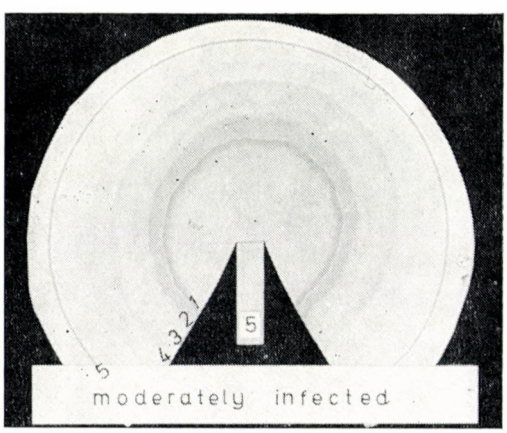


Fig. 2. The chromatogram of the pigments contained in the moderately infected leaves of the Red Veltliner vine variety. 1. Chlorophyll b, 2. chlorophyll a, 3. xanthophyll$_1$, 4. xanthophyll$_2$, 5. β-carotene

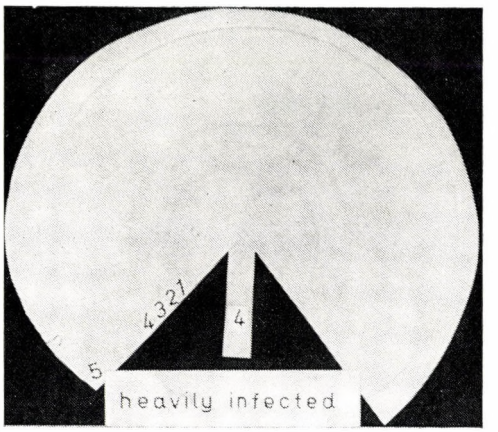


Fig. 3. The chromatogram of the pigments contained in the highly infected leaves of the Red Veltliner vine variety. 1. Chlorophyll a, 2. chlorophyll b, 3. xanthophyll$_1$, 4. xanthophyll$_2$, 5. β-carotene

Since control solutions of the leaf pigment are not available quantitative determinations with the chromatograms of the pigments could not be carried out. In order to obtain quantitative results the chlorophyll content of the leaves was determined also by the method of COMAR and ZSCHEILE (1942).

On the 23rd October, 1967, further samples were gathered from the healthy and infected vinestocks of both varieties: Szerémi Zöld and Red Veltliner. The date of the collection was so chosen that beside the healthy green leaves also yel-

Table 1

The chlorophyll content of the Szerémi Zöld and Red Veltliner vine varieties (Badacsony, 23. 10. 1967)

Sample	Chlorophyll			Chlorophyll			
	a	b	Total	a	b	Total	$C_a : C_b$
	g/100 g fresh leaf			g/100 g dry matter			g/100 g solids
Szerémi Zöld:							
healthy green leaves	0.714	0.353	1.067	2.210	1.100	3.310	2.009
CMV infected leaves	0.149	0.058	0.207	0.579	0.226	0.805	2.585
healthy yellow leaves	0.052	0.029	0.081	0.136	0.074	0.210	1.838
Red Veltliner:							
healthy green leaves	0.755	0.374	1.129	2.539	1.256	3.795	2.023
CMV infected leaves	0.157	0.078	0.235	0.633	0.316	0.949	1.687
healthy yellow leaves	0.065	0.025	0.090	0.175	0.070	0.245	2.436

low leaves could be gathered. Thus the effect of virus infection could be compared to the effect of the natural autumnal yellowing of the leaves in respect to both pigment and sugar content.

As is seen in Table 1 the chlorophyll content was lower in the CMV infected leaves and in the yellow leaves than in the healthy green leaves, not yet affected by the autumnal yellowing (Table 1). The total chlorophyll content on fresh lea.

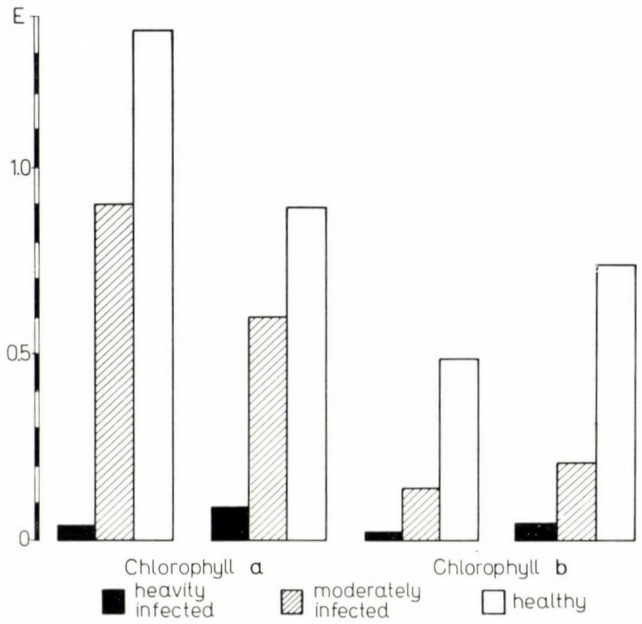


Fig. 4. The chlorophyll a and b content of the Szerémi Zöld vine variety. (Badacsony, 22. 5. 1967)

basis in the virus infected leaves amounted to about one fifth of that of the healthy green leaves. The chlorophyll content of the leaves turned yellow due to the autumnal change of colour was only one thirteenth of that of the unchanged green leaves. The total chlorophyll content on dry basis of the infected leaves was only one fourth of that of the green leaves and the chlorophyll content of the leaves turned naturally yellow was about one sixteenth of that of the green ones.

In the healthy leaves of both vine varieties the ratio (C_a : C_b) of chlorophyll a to b was found to be 2. In the infected leaves of the Szerémi Zöld variety this ratio surpassed 2, whereas in the Red Veltliner variety was below 2. In the healthy but yellow leaves just the opposite was true, the ratio was below 2 for the Szerémi Zöld variety and above 2 for the Red Veltliner variety. Thus the degree of decomposition of chlorophyll a and b depended not only on the vine variety but on the cause of yellowing as well.

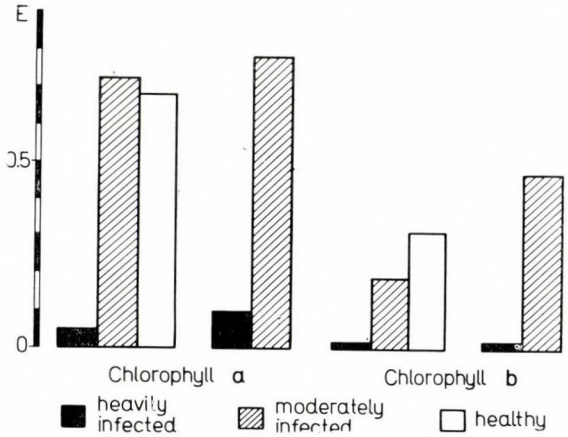


Fig. 5. The chlorophyll a and b content of the leaves of the Red Veltliner vine variety. (Badacsony, 22. 5. 1967)

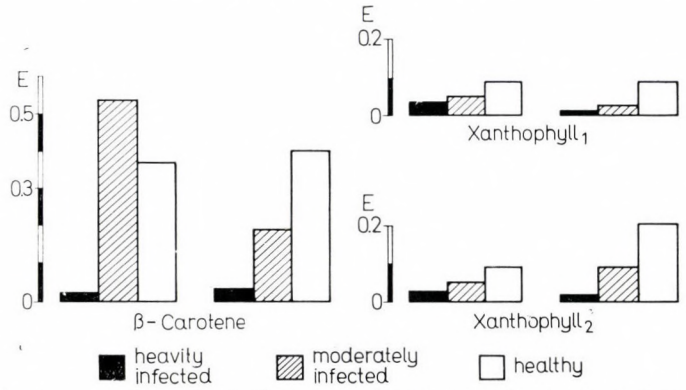


Fig. 6. The β-carotene and xanthophyll content of the Szerémi Zöld vine variety. (Badacsony, 22. 5. 1967)

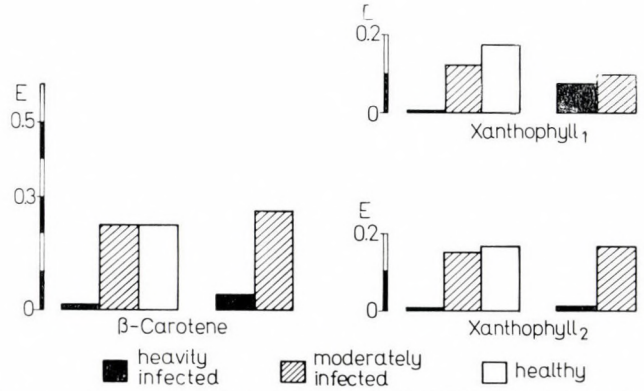


Fig. 7. The β-carotene and xanthophyll content of the Red Veltliner vine variety. (Badacsony, 22. 5. 1967)

The dry matter in the CMV infected leaves was lower, in the naturally yellowed leaves higher than in the healthy unchanged green leaves (Table 2). This explains why the extent of the reduction of the chlorophyll content differed according to the basis of calculation, i. e. whether it was calculated on the fresh or on the dry leaf basis. However, there is no doubt about it that the infected leaves contain more chlorophyll in the autumn than the naturally yellowed leaves, but less than the healthy unchanged leaves.

Table 2

The total solids content of the leaves of the Szerémi Zöld and Red Veltliner vine varieties

Sample	Total solids content g/100 g fresh leaf		
	9. 5. 1967	22. 5. 1967	23. 10. 1967
Szerémi Zöld:			
healthy green leaves	19.49	22.01	32.27
CMV infected leaves, moderately infected	17.25	21.44	—
CMV infected leaves, highly infected	17.09	19.62	25.79
healthy yellow leaves	—	—	38.00
Red Veltliner:			
healthy green leaves	16.08	24.60	29.75
CMV infected leaves, moderate infection	16.35	23.87	—
CMV infected leaves, highly infected	13.79	17.18	24.80
healthy yellow leaves	—	—	36.87

2. The total sugar content

On the 9th May, 1967, the total sugar content of both Szerémi Zöld and Red Veltliner varieties was below 1 per cent in the highly infected leaves, below 1.5 per cent in the moderately infected leaves and between 1 and 3.5 per cent in healthy leaves (Fig. 8). At this date the higher infected the leaves were, the less sugar they contained.

On the 23rd October, 1967, the sugar content in the infected leaves of the Szerémi Zöld variety was less than in the healthy leaves and thus was in good agreement with results previously obtained by the authors (Fig. 9). However in the Red Veltliner variety, in contrast to earlier results, the sugar content in the infected leaves was higher than in the healthy ones. That of the naturally yellowed leaves was generally between the two. It is interesting to note that all leaves whether healthy or infected, contained more sugar in October than in May.

On comparing the dry matter content of the leaves gathered at three different dates (Table 2) an enhancement of the dry matter content could be observed and at every date the dry matter content of the healthy leaves was higher than that of the infected ones.

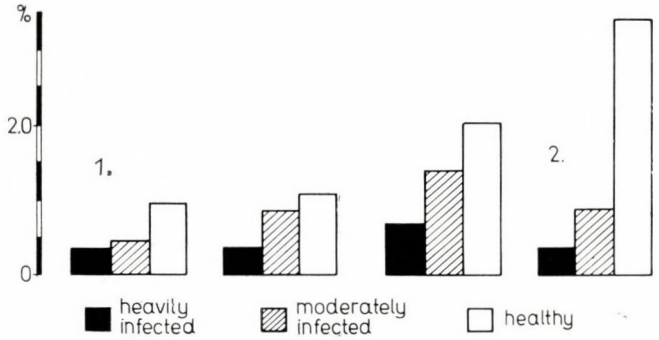


Fig. 8. The total sugar content of the Szerémi Zöld and Red Veltliner varieties. (Badacsony, 9. 5. 1967)

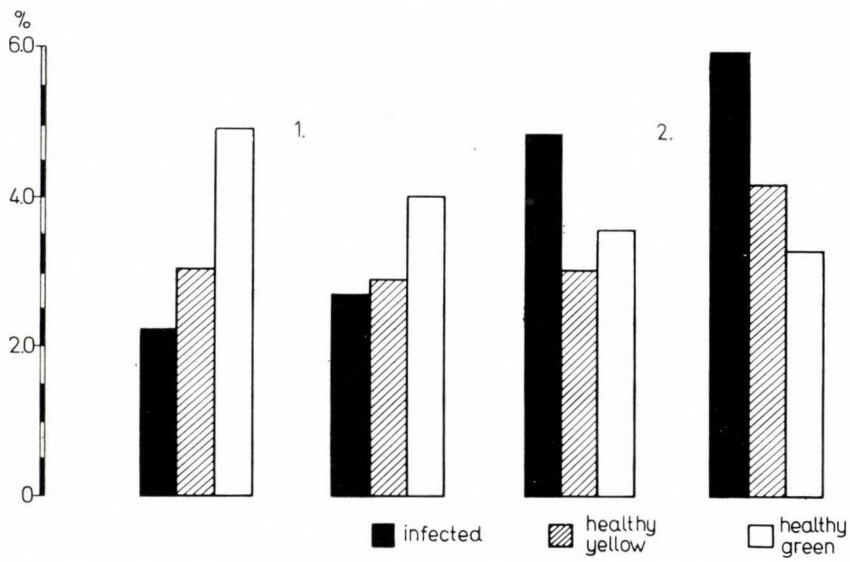


Fig. 9. The total sugar content of the Szerémi Zöld and Red Veltliner varieties. (Badacsony, 23. 10. 1967)

Discussion

Virus infection is generally characterized by the discoloration of the leaves and a slower development or decomposition of the chlorophyll content, which ultimately leads to a reduced photosynthetic activity (DIENER, 1963). Similar conclusion could be drawn of the above experiments. Since the chlorophyll content in the CMV infected leaves is substantially lower, photosynthesis is dis-

turbed, and thus, the total sugar and dry matter content is lower than in the healthy leaves. These observations are in good agreement with the fact that the CMV infected leaves contain more alcohol-soluble nitrogen and free amino acids than the healthy leaves (JÁKÓ, LEHOCZKY and SÁROSPATAKI, 1966). In consequence of the reduced photosynthetic activity and reduced assimilated sugar content the nitrogen metabolism increases and the alcohol-soluble nitrogen and free amino acids accumulate.

WYND (1943) found that generally a reduced carbohydrate content was characteristic of mosaic type virus infection. However, the observations on the accumulation of starch and reducing sugar in the virus infected leaves (MARTIN, 1954, BAWDEN, 1956, ORLOB and ARNY, 1961) and in the vineshoot (BOSC and CATAYÉE, 1961) deserve also some attention. These authors explain their observations by the inactivation of the enzymes transforming sugar into starch or by the disturbed sugar transfer. It may be presumed that the increased sugar content as observed in the infected leaves of the Red Veltliner variety in October is due to similar reasons. However, this phenomenon may be explained perhaps by the higher sensitivity of this variety to CMV infection.

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1,2,4-Triazoles

I. The Relationship of Structure to Phytotoxicity

By

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Seventeen derivatives of 3-amino-s-triazole have been prepared and their phytotoxic activities were evaluated under greenhouse conditions using pre- and post-emergence techniques. The results proved that 3-amino-1,2,4-triazole has the most ideal requirements for the herbicidal action among the compounds tested. The replacement of either of the two hydrogen atoms present on the triazole ring by different groups, as well as changing the position of the amino group, or its replacement either by chlorine atom or hydroxyl or mercapto group, reduce or eliminate the phytotoxic action.

Introduction

The reaction between aminoguanidine and organic acids has been used to yield 3-amino-1,2,4-triazole (MANCHOT and NOLL, 1905). After its introduction as a selective herbicide during the mid 1950's (ALVERSON, 1957), and the discovery of its defoliation properties (HALL et al. 1954) and intense phytocidal activity (HAUSER, 1954) the compound has received a considerable attention by researchers. Several of its derivatives have been prepared and some of them have been described to possess a considerable herbicidal activity. But none of these derivatives were, however, more active than the parent 3-amino-1,2,4-triazole.

SCHWEIZER and ROGERS (1964) studied the structural requirements of amitrole (3-amino-s-triazole) for physiological activity. The effect of eleven triazole and tetrazole compounds similar to amitrole on the growth of carrot explants, elongation of wheat roots, chlorophyll content in corn leaves and catalase activity were compared. Such a study does not, however, clarify the structural requirements of 3-amino-s-triazole molecule for the herbicidal action because the responses as observed by SCHWEIZER and ROGERS (loc. cit.) might be due more to the result than to the cause of the phytocidal action.

The aim of the present investigation is to relate the chemical constitution with the phytocidal activity of s-triazoles.

Materials and Methods

Chemical part

All the compounds investigated here are chemically known. Most of them have been prepared by known methods. The following compounds have been prepared according to the indicated references: 3-amino-1,2,4-triazole and its 5-methyl- (British Patent*), 5-ethyl- and 5-*iso*propyl- (REILLY and MADDEN, 1929), 5-phenyl- (GRINSTEIN and CHIPEN, 1961), 5-benzyl- (YATES and FARNUM, 1963), 5-phenoxy-methyl- and 5- (2,4-dichloro-phenoxy) methyl- (SHIRAKAWA et al., 1958) and 5-amino-derivatives (KAISER and ROEMER, 1953). Bis [3-(5-amino-s-triazolyl)], bis [3-(5-amino-s-triazolyl)] methane and bis[3-(5-amino-s-triazolyl)] ethane derivatives have been prepared according to SHREVE and CHARLESWORTH (1956). 4-Amino-s-triazole and its 3-hydrazino-derivative were prepared according to HERBST and GARRISON (1953), and KRÖGER et al. (1963), respectively. 3,4-Diamino-s-triazole has been prepared after carrying out simple modifications on the method described by GAITER (1915). 3-Chloro-1,2,4-triazole has been prepared by the method of MANCHOT and NOLL (1905), 3-mercapto-s-triazole according to BEYER et al. (1960). The methods described by MANCHOT and NOLL (1905) and GRUNDMANN and KREUTZBERGER (1957) for the preparation of 3-hydroxy-1,2,4-triazole were complicated and gave low yields. This compound has been prepared in an adequated yield according to a method suggested by the author. This method will be described in the experimental part.

Experiments

(A) *3,4-Diamino-1,2,4-triazole hydrobromide.* 3.4 g (0.02 mole) of diaminoguanidine hydrobromide was mixed thoroughly with 4 g of 98% formic acid, then boiled for 30 minutes under reflux. The mixture was evaporated and dried under vacuum at 100° C. The residual material was shaken with 5 ml methanol and distilled under vacuum. The shaking and distillation were repeated 3 or 4 times till the beginning of crystallization. The product was recrystallized from 70% ethanol after addition of 3 drops of 48% HBr to the solution. The yield was 2.9 g (80.7%), the m. p. was 215–216° C (decomp.). The analytical method applied determined one nitrogen atom only.

$C_2H_6N_5Br$	calculated (for one N)	% N 7.7	% Br 44.44
	found	% N 7.9	% Br 44.47

(B) *3-Hydroxy-1,2,4-triazole.* 3.35 g (0.03 mole) semicarbazide hydrochloride was thoroughly mixed with 2 g of 98% formic acid, then heated for one hour under reflux at 120° C. After evaporation to dryness, 2 *N* sodium carbonate solution was added till alkalinity and then boiled for a further one hour under reflux. The mixture was evaporated to dryness using vacuum distillation and

* British Patent 765, 728 Jan., 9. Chemical Abstr. 51: 13934. 1957.

extracted with hot ethanol. The yield was 2.2 g (86.3%), the m. p. 234–235° C (decomp.). The analytical method applied determined one nitrogen atom only (i. e. N_4 of the s-triazole ring).

$C_2H_3N_3O$	calculated (for one N)	% N 16.47
	found	% N 16.51

Biological part

The previous compounds have been formulated as 50% wettable powders. Their pre- and post-emergent toxicity have been tested under greenhouse conditions using wheat *(Triticum aestivum)* and white mustard *(Sinapis alba)* as standard testing plants. The used dosages based principally on the molecular weight of the tested compounds. Five dosage levels, 400, 200, 100, 50 and 25 g mole/ha/compound have been used. The treatments were replicated 5 times. The daily observations were collected and used in devising an arbitrary scale for measuring the phytotoxicity (MATOLCSY and GOMAA, 1968). The final results have been recorded 3 weeks after the treatment.

Results and Discussion

The final data for the pre- and post-emergence activity of the compounds tested are represented in Table 1 according to the devised scale of toxicity wherein 0 represents no toxicity and 10 indicates the death of plant.

The participation of the substituents can be studied only when comparing the phytotoxicities at molar dosages. From the represented data the following conclusions could be made:

1. All the compounds tested had a toxic action lower than 3-amino-s-triazole.
2. The presence of an alkyl group on the C-5 carbon atom reduced the activity as compared with the parent compound. The toxic action was in the order of 5-ethyl > 5-methyl > 5-isopropyl.
3. Replacement of the C-5 hydrogen with phenyl- or benzyl-radicals greatly diminished the phytotoxic action. The 5-phenyl derivative showed lower phytotoxic action than its 5-benzyl analogue. The phytotoxicity at the level of 400 mole/ha exhibited by both of these compounds was very weak.
4. Substitution of C-5 hydrogen with phenoxymethyl-, or 2,4 dichloro phenoxymethyl-groups greatly reduced the herbicidal activity of 3-amino-s-triazole.
5. On linking 3-amino-s-triazolyl radical with C-5 carbon atom of a second-s-triazole ring, either directly or through a hydrocarbon bridge the phytotoxicity was nullified.

All the bis (amino-s-triazoles) tested in this investigation were completely inactive. Both the wheat and mustard plants treated by these compounds with the

Table 1

The pre- and post-emergence activities of s-triazole derivatives

0 = no toxicity 10 = Plant death

Method of testing	Pre-emergence										Post-emergence									
Testing plant	mustard					wheat					mustard					wheat				
Dosage M/ha	400	200	100	50	25	400	200	100	50	25	400	200	100	50	25	400	200	100	50	25
Compound																				
3-Amino-1,2,4 triazole	10	10	10	10	9	10	10	10	9	7	8	8	6	5	4	7	7	6	3	2
3-Amino-5-methyl-s-triazole	4	4	3	2	1	3	3	2	1	1	5	5	4	2	1	4	4	3	1	0
3-Amino-5-ethyl-s-triazole	7	7	6	4	2	5	4	3	2	2	5	4	2	1	0	5	5	4	2	0
3-Amino-5-i. propyl-s-triazole	3	2	2	0	0	2	1	0	0	0	4	3	1	0	0	3	2	0	0	0
3-Amino-5-phenyl-s-triazole	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
3-Amino-5-benzyl-s-triazole	1	1	0	0	0	0	0	0	0	0	2	1	0	0	0	0	0	0	0	0
3-Amino-5-phenoxy methyl-s-triazole	4	2	1	0	0	0	0	0	0	0	5	3	2	1	1	4	2	2	0	0
3-Amino-5-(2,4-dichlorophenoxy methyl)-s-triazole	4	2	1	0	0	0	0	0	0	0	3	2	2	1	1	4	2	2	0	0
Bis-[3-(5-amino-s-triazolyl)]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Bis-[3-(5-amino-s-triazolyl)] methane	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1,2-Bis-[3-(5-amino-s-triazolyl)] ethane	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3,5-Diamino-s-triazole	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3,4-Diamino-s-triazole (HBr salt.)	2	1	0	0	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0
3-Hydrazino-4-amino-s-triazole (HCl salt)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
4-Amino-s-triazole	1	1	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
3-Chloro-s-triazole	4	2	0	0	0	4	3	2	2	0	2	2	0	0	0	2	1	0	0	0
3-Hydroxy-s-triazole	0	0	0	0	0	2	2	2	2	0	2	0	0	0	0	3	2	0	0	0
3-Mercapto-s-triazole	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

prescribed doses were healthy and no toxic symptoms appeared even at a dose as high as 400 mole/ha.

6. The phytotoxicity disappeared completely on replacing C-5 hydrogen with $-NH_2$ group. However, when a second amino group replaced the N-4 hydrogen (in 3,4-diamino-s-triazole), the phytotoxicity was greatly lowered.

7. 3-Hydrazino-4-amino-s-triazole was completely inactive even at the highest dose level (400 mole/ha). The tested plants did not suffer from toxicity.

8. Changing the position of the amino group from C-3 carbon to N-4 nitrogen resulted in an almost complete loss of activity. The phytotoxicity of 4-amino-s-triazole, the compound yielded as a result, became very weak.

9. Replacement of the amino group in 3-amino-s-triazole with a chlorine atom or hydroxy group resulted in a very weakly active compound. The phytotoxicity of 3-chloro-s-triazole was, however, greater than that of the hydroxy analogue. Both of the two compounds were more toxic to wheat than to mustard. The mercapto analogue was completely inactive.

The above mentioned conclusions indicated that: 1. 3-amino-1,2,4-triazole molecule has the most ideal requirements for the herbicidal activity among the tested compounts. 2. Replacement of the C-5 hydrogen by alkyl-, aryl-, aralkyl-, aryloxymethyl- or substituted aryloxymethyl-groups reduce the phytotoxicity but do not altogether eliminate it. 3. Replacement of the C-5 hydrogen by either $-NH_2$ group, or 3-amino-s-triazol-5-yl- or 3-amino-s-triazol-5-alkyl radicals eliminate the phytotoxicity from the parent 3-amino-s-triazole molecule. 4. Replacing the N-4 hydrogen by $-NH_2$ greatly reduce the phytotoxic action. 5. For highly herbicidal action the NH_2 must be present on carbon-3. 6. The highest herbicidal action is obtained when $-NH_2$ group and not -Cl atom or -OH or -SH groups are present on position-3 of the ring.

*

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Fungistatic Activity of Some Pyrimidine Antimetabolites. II.

Thio- and Aza-analogues of Uracil and Related Compounds

By

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Out of 25 potential pyrimidine antimetabolites 2,4-dithio-6-azauracil, 2-methylthio-3-methyl-4-thio-6-azauracil and 3-trichloromethylmercapto-6-azauracil revealed fungistatic activity against *Colletotrichum lindemuthianum* in the agar culture test. The character of their activity is divergent from that of 6-azauracil, indicating a different mechanism of action.

The activity of 3-trichloromethylmercapto-6-azauracil against 4 test fungi is about equal to that of N-trichloromethylmercapto-tetrahydro-phthalimide.

The biological action of 6-azauracil seems to be highly structure-specific; this might be connected with its influence on RNA biosynthesis.

In a previous report (Matolcsy and Doma, 1967) we have demonstrated that 6-azauracil known as an antagonist of uracil and as a systemic fungicide against powdery mildew (Dekker, 1962; Dekker and Oort, 1964; Oort and Dekker, 1964) reveals a direct fungistatic activity against *Colletotrichum lindemuthianum* and two other fungi.

The present study was aimed to test further potential pyrimidine antimetabolites for their fungistatic action in order to draw some conclusions concerning structure-activity relationship and mechanism of action.

Materials and Methods

In the chemical sense the compounds tested were derivatives of pyrimidine, 1,2,4-, 1,3,5-triazine and 1,2,4-triazole, as well as some open chain compounds yielding some of the above mentioned derivatives by cyclization. From the biochemical point of view the compounds tested were potential uracil-antimetabolites, mostly thio-, 5- and 6-aza-uracil-derivatives. Some of them can be regarded also as thio-, or 6-aza-analogues, respectively, of the intermediate products of uracil biosynthesis and as a consequence as potential precursors of 2-thiouracil and 6-azauracil, respectively.

The compounds tested were as follows: sodium salt and ethylester of β-ureidocrotonic acid, 5,6-dihydro-uracil, 2-thiouracil, β-thioureido-propionic acid, 5,6-dihydro-2-thiouracil, 2-thioorotic acid, semicarbazidoacetic acid and

its ethylester hydrochloride, α-ketoglutaric acid semicarbazone, dihydro-6-azauracil, 2-thio-6-azauracil, 4-thio-6-azauracil, 2,4-dithio-6-azauracil, 2-methylthio-6-azauracil, 5-bromo-6-azauracil, 6-azauracil-5-carboxylic acid, β-(6-azauracil-5-yl)-propionic acid, 2-methylthio-3-methyl-4-thio-6-azauracil, 3-trichloromethylmercapto-6-azauracil, 6-azathymine, 2-thio-6-azathymine, 5-azauracil-6-carboxylic acid and urazole (3,5-dioxy-1,2,4-triazole). 6-Azauracil, investigated in the previous study, was used as control substance.

Most of these compounds were synthetized by us by known methods, except 3-trichloromethylmercapto-6-azauracil which was not described in the literature. The mode of the synthesis of this compound will be published elsewhere. Some other derivatives were kindly submitted by Dr. J. GUT and. Dr. A. PÍSKALA from the Institute of Organic Chemistry and Biochemistry of the Czechoslovak Academy of Sciences, Prague and by Dr. V. ŠKARIĆ from the Institute Ruder Bošković, Zagreb.

The compounds were tested on *Colletotrichum lindemuthianum* by the same method as described in our earlier paper (MATOLCSY and DOMA, 1967).

3-Trichloromethylmercapto-6-azauracil was tested on 4 further fungal species with the "dried spray" and "wet spray" technique described by BÁNKI et al. (1966), comparatively with the known fungicide N-trichloromethylmercapto-tetrahydrophthalimide (captan) first synthetized by KITTLESON (1952).

Results and Discussion

Besides 6-azauracil used as a control substance three of these tested compounds revealed fungistatic activity in the culture tests: 2,4-dithio-6-azauracil, 2-methylthio-3-methyl-4-thio-6-azauracil and 3-trichloromethylmercapto-6-azauracil. The character of the biological effect of these three compounds, however, was different from that of 6-azauracil as none of them revealed the typical morphological deformations characteristic on the control substance.

In case of 2,4-dithio-6-azauracil the substrate-mycelia have not reached the inhibition disk within the 3–4 mm inhibition zone formed around the inhibition center. Within the completely transparent inhibition zone the conidia have not germinated (Fig. 1).

The primary zone border formed around the inhibition center of 2-methylthio-3-methyl-4-thio-6-azauracil is due to the initial inhibition of the substrate mycelial growth; within further 3–4 days this zone became completely dissolved and the normally growing mycelia reached the inhibition disk. Nevertheless, the shape of primary zone border is kept intact even after the overgrowth of the inhibition zone as it formes the border of the conidial acervulus colonies. This border zone, situated in a 1 cm ring around the inhibition center, is remained further on, indicating that activity is due to an inhibition of the sporulation rather than that of the vegetative growth (Fig. 2).

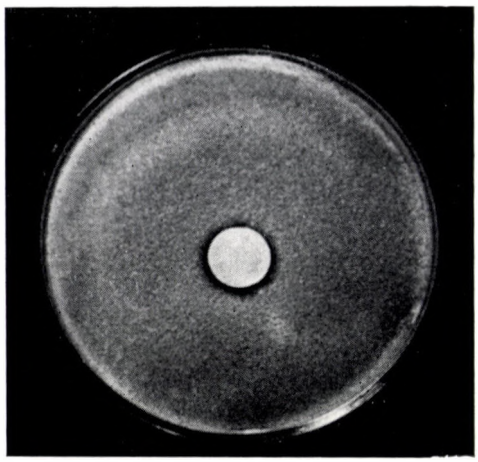


Fig. 1

The highest activity against *Colletotrichum lindemuthianum* was achieved by 3-trichloromethylmercapto-6-azauracil (Fig. 3). The effect manifested itself in the complete inhibition of the conidial germination. The subsequent dissolving of the inhibition zone was due to the successive spread of the substrate mycelia towards the inhibition center. Within the overgrown inhibition zone the conidia exposed to the original lethal concentration remained in an ungerminated state. This is in contrast to the 6-azauracil inhibition zone where the germinated conidia remained in a deformed state amongst the advancing hyphae as shown for comparison in Fig. 4.

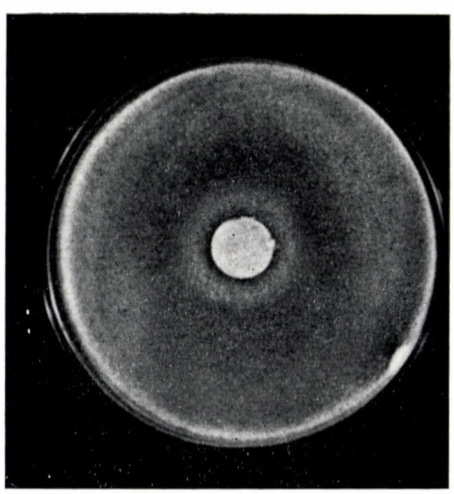


Fig. 2

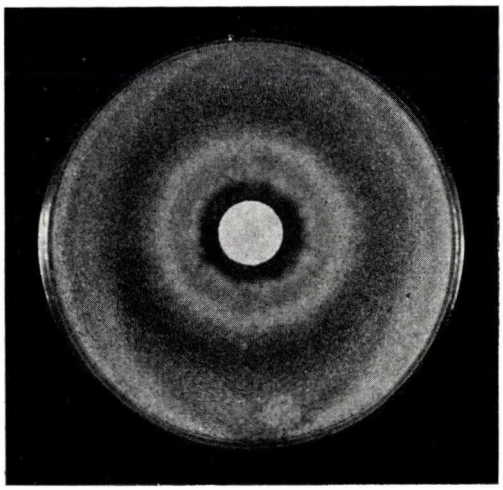


Fig. 3



Fig. 4. Arrow points at deformed conidium

The results of the dried spray and wet spray tests are shown in Table 1. 6-Azauracil gave a negligible activity in these tests, its values are, therefore, not presented. These data suggest that the fungistatic activity of 3-trichloromethylmercapto-6-azauracil is about equal to that of captan.

The fact that the three active compounds reveal symptoms divergent from those of 6-azauracil suggests that their action is based on a different mechanism. For the highly active 3-trichloromethylmercapto-6-azauracil this assumption obtained further support by the fact that 6-azauracil itself proved to be poorly active in the

Table 1

Fungistatic activity of 3-trichloromethylmercapto-6-azauracil as compared with that of captan (dr. sp. = "dry spray"; w. sp. = "wet spray")

	3-Trichloro-methyl-mercapto-6-azauracil	Captan
	Concentration of the test substance required for ED_{50} in %	
Alternaria tenuis		
inhibition of germination		
dr. sp.	$6 \cdot 10^{-3}$	$3 \cdot 10^{-3}$
w. sp.	10^{-4}	$3 \cdot 10^{-4}$
inhibition of growth		
dr. sp.	10^{-2}	$5 \cdot 10^{-3}$
w. sp.	10^{-5}	10^{-4}
Botrytis allii		
inhibition of germination		
dr. sp.	10^{-2}	10^{-2}
w. sp.	$4 \cdot 10^{-5}$	$2 \cdot 10^{-4}$
inhibition of growth		
dr. sp.	$7 \cdot 10^{-3}$	$7 \cdot 10^{-3}$
w. sp.	$5 \cdot 10^{-4}$	$2 \cdot 10^{-4}$
Aspergillus niger		
inhibition of germination		
dr. sp.	$2 \cdot 10^{-2}$	$5 \cdot 10^{-4}$
w. sp.	$6 \cdot 10^{-5}$	$2 \cdot 10^{-5}$
inhibition of growth		
dr. sp.	10^{-2}	$5 \cdot 10^{-4}$
w. sp.	$6 \cdot 10^{-5}$	$5 \cdot 10^{-5}$
Penicillium sativum		
inhibition of germination		
dr. sp.	$3 \cdot 10^{-2}$	$8 \cdot 10^{-4}$
w. sp.	$5 \cdot 10^{-5}$	$5 \cdot 10^{-5}$
inhibition of growth		
dr. sp.	$5 \cdot 10^{-3}$	$2 \cdot 10^{-4}$
w. sp.	$5 \cdot 10^{-5}$	10^{-5}

routine screening tests. The effectivity of this compound must be attributed, therefore, to the

$$CCl_3\text{-}S\text{-}N <$$

group characteristic on the trichloromercapto fungicides rather than to the 6-azauracil moiety which lost its original activity by substitution.

It should be mentioned that two of these compounds, 2,4-dithio-6-azauracil and 2-methylthio-3-methyl-4-thio-6-azauracil were found by ŠKODA et al. (1962) as highly effective against *Escheria coli*.

All three compounds found to be active in our tests are sulphur-derivatives of 6-azauracil containing the

$$-S-\underset{|}{C}=N-, \quad S=\underset{|}{C}-N= \quad \text{or} \quad \equiv C-S-N=$$

grouping, respectively, present in many fungicide molecules.

Our results have proved the high structure-specificity of 6-azauracil as regards its biological effect. Any alteration on the molecule, e.g. exchange of oxygen to sulphur, substitution in position 5 by bromine, methyl-, carboxy- or carboxyethyl-group, hydrogenation, transfer of the aza-nitrogen atom from position 6 to position 5, opening of the ring (e.g. glyoxylic acid semicarbazone investigated in our previous work) or its contraction to five membered ring (urazole) etc. results in a complete loss of activity or a change of its character.

Our results are in accordance with the findings of DEKKER (1962) demonstrating that analogues of the DNA-nucleobases (e.g. 6-azathymine) are inactive while activity is restricted to analogues of the RNA bases. This confirmes the assumption that the azauracil-type activity can be attributed to an interference with RNA biosynthesis. Considering the fact that the expected N-glycoside and hydrogen bondings can be formed only if definite electron-structural and spatial conditions are fulfilled, the strict structural requirements of activity seem to be justified.

*

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The Occurrence of Stripe Mosaic Disease of Maize in Hungary and Possibilities of Breeding for Virus Resistance

By

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Stripe mosaic virus of maize as it occurs in Hungary is related on the basis of biological and physical tests to maize mosaic virus from Italy and the Balcans, to red stripe mosaic virus of sorghum and, as judged from literary data, to maize dwarf mosaic virus from North America as well as to sugar cane mosaic virus. The disease occurs in two forms (mild and severe). The percentage distribution of any of these forms and accordingly the yield losses caused by the virus vary from year to year. The degree of resistance of 170 varieties, hybrids and lines was different. A useful hint for practical breeding for resistance is the finding that resistant individuals show mild symptoms with a long incubation period. Lines 333B, 333C, 129, 107A, WF9 and their hybrids were characterized by such "atypical" symptoms. Line 335 proved to be immune to the virus when infected artificially.

Correlations with related virus diseases

Virus disease of maize was found in Hungary for the first time in 1962 (SZIRMAI and PAIZS, 1963).

Highest infestations occurred on breeding stations where maize and sorghum cultures were close to each other. Sorghum plants showed red stripe symptoms similar to those which had been described from Italy (GOIDANICH, 1938), Yugoslavia (PANJAN, 1960), Bulgaria (KOVACHEVSKY, 1961), Roumania (POP, 1962) and Czechoslovakia (BENADA et al. 1964). It was suspected that both maize and sorghum plants were infected by the same virus. Similar cases have already been reported (LOVISOLO and ACIMOVIC, 1961; SAVULESCU and POP, 1962, and BENADA et al., 1964).

Comparative studies with some isolates obtained from the countries mentioned above have revealed that both red stripe disease of sorghum and stripe mosaic disease of maize originate from the same country, most probably Italy. As far as the geographical distribution of the disease is concerned, the epidemiological role of aphid vectors should be taken into consideration. SANDER (1961) has namely pointed out that the maize aphid is spreading very rapidly to the North.

There are several reports from the United States (WILLIAMS and ALEXANDER, 1964, 1965; WILLIAMS and LEONARD, 1965; JANSON et al., 1965; CALVERT et al.,

1965; BANCROFT et al., 1966; BUCHHOLTZ et al., 1966; MACKENZIE et al., 1966; SILL, 1966) on a virus disease of maize (maize dwarf mosaic) which causes serious losses in the Northern and Central States. A comparison of the descriptions and experimental results indicates that the European maize stripe mosaic and the American maize dwarf mosaic are identical or at least very closely related diseases.

A further analysis of the situation shows that maize dwarf mosaic, sorghum red stripe disease and maize stripe mosaic may all be regarded identical with sugarcane mosaic which is most likely a common source of all three virus diseases. As early as 1919 and 1920 BRANDES has considered maize mosaic to be identical with sugarcane mosaic because of similar symptoms and identical vectors. Results of electronmicroscopic and serological investigations carried out recently by DIJKSTRA and GRANCINI (1960) also support this view. In this respect European and American investigators are of the same opinion (GRANCINI, 1957; LOVISOLO, 1957; DIJKSTRA and GRANCINI, 1960; PANJAN, 1960; LOVISOLO and ACIMOVIC, 1961; WILLIAMS and ALEXANDER, 1964; BENADA et al., 1964; FRAZIER et al., 1965; SHEPHERD and HOLDEMAN, 1965; TOŠIĆ, 1965; HALL et al., 1966; WAGNER and DALE 1966).

Yield losses

Of the virus diseases of maize only stripe mosaic disease is known in Hungary. This disease is not very widespread yet and causes serious losses only in limited areas and in certain years. In 1962 on an experimental plot the average height of virus infected individuals (hybrid "Martonvásári 5") was 165 cm as compared to an average height of 205 cm for symptomless plants. The average grain yield was 123 g with infected and 296 g with symptomless plants. The rate of infestation amounted to 55 per cent. It can be calculated that the yield loss due to natural virus infection was 32 per cent.

In the same area in 1963 under conditions which did not favour virus infection the average heights were 203 and 211 cm, respectively, and the grain yields 178 g and 332 g, respectively. In this year the rate of infestation was 17 per cent. Correspondingly the yield loss due to virus infection amounted only to 7.9 per cent.

In extensive maize cultures the average infestation rate is still very low, it does not surpass 0.01 per cent. It seems therefore, that up to the present maize stripe mosaic disease has threatened only breeding stations and small confined areas. These, however, are to be regarded as critical infection sources. In these areas the yield losses show yearly fluctuations.

It is interesting to note that with the same rate of infestation the yield losses exhibit pronounced yearly variations. This may be due to differences in the severity of symptoms. In the last 3 to 4 years, for instance, in spite of fairly high infection rates yield losses were not so high probably due to milder symptom development.

Symptoms

The leaf symptoms of maize stripe mosaic disease consist of parallel short streaks along the veins (Fig. 2A). This type of symptom is characteristic of young sorghum plants infected with red stripe, as well as of mosaic infected sugarcane plants. Similar symptoms are encountered on the test plants *Sorghum* spp., *Panicum miliaceum*, *Digitaria sanguinolenta* and *Echinochloe crusgalli*. Whenever this basic type of symptom occurs neither corn-cobs nor plant growth are affected.

Maize plants (hybrid "Martonvásári 5") exhibiting another type of symptoms are characterized by longitudinal streaks on the stalk. In such plants the flowers are but partially pollinated, the corn-cobs are reduced and distorted and the grains bear reddish streaks. This syndrome is the expression of great yield losses and is closely related to the American dwarf mosaic symptoms.

Some physical properties of the virus

In our earlier experiments (SZIRMAI and PAIZS, 1963), which were carried out in August (average greenhouse temperature about 26° C) and in which crude sap of maize plants (hybrid "Martonvásári 5") showing mild symptoms was used, the thermal inactivation point was found to be $< 50°$ C, the dilution end point to be $< 10^{-3}$ and stability in vitro to be $< 4^{h}$. These values were lower than those reported by SEGHAL (1966) and TOŠIĆ (1965) but were in agreement with those described by LOVISOLO (1957).

In recent experiments, which were conducted in November (average greenhouse temperature about 18° C) higher values were obtained: 54° C for the thermal inactivation point, 2×10^{-4} for the dilution end point and 12 hours for the stability in vitro.

These experiments were repeated and it was found once more that at an average greenhouse temperature of 26° C lower values are obtained than at an average greenhouse temperature of 18° C.

The average length of the filamentous virus particles as measured in electronmicrographs was found to be 700 mμ (Fig. 1). The virus was prepared from maize leaves according to the dipping method described by BRANDES (1957).

The length of the virus particles varied from 300 mμ to 1100 mμ in the preparations. Several broken particles were found (marked by an arrow in Fig. 1) most probably due to the preparative work. One may suppose that the original length of the particles is somewhere above the average.

Varietal resistance

There is some evidence in the literature (BRANDES, 1920; TOŠIĆ, 1965; POP, 1966; HALL et al., 1966; STOREY and HOWLAND, 1966) suggesting that breeding for resistance is a promising way of controlling the mosaic disease of maize.

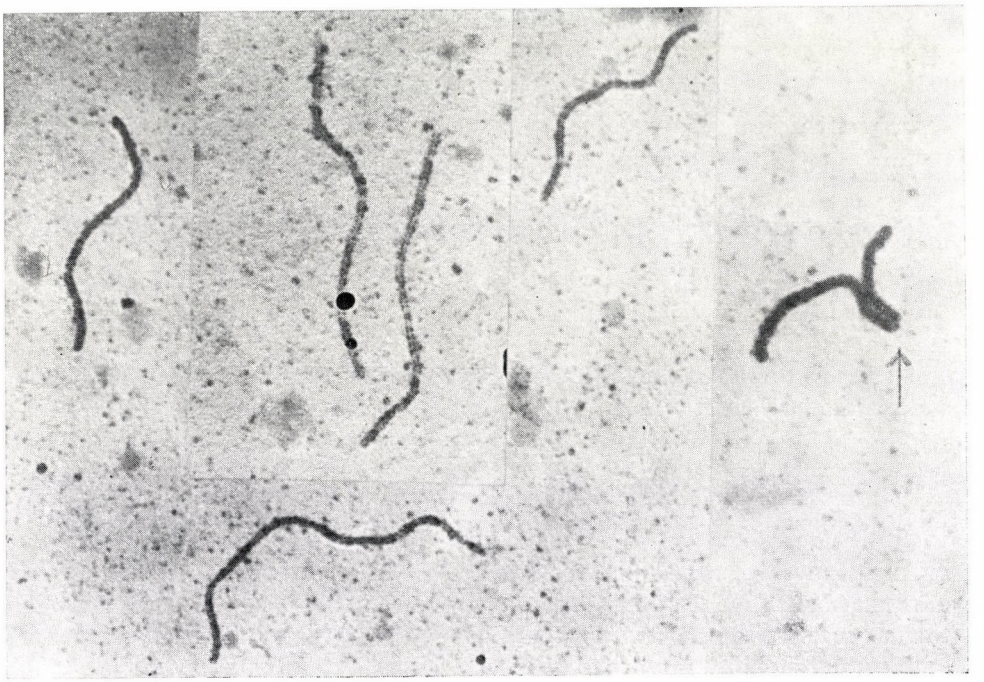


Fig. 1. Electron microscopic picture of maize streak mosaic virus

In our investigations we tested the resistance of some well known maize varieties and hybrids as well as of several lines kindly supplied by the Maize Breeding Station, School of Agriculture, Keszthely.

Materials and Methods

62 hybrids and lines from the Maize Breeding Station, School of Agriculture, Keszthely and 108 varieties and hybrids from other breeding stations were used. From each variety, hybrid and line, respectively, 24 plants were infected in three replications at different times. The infections were carried out by rubbing leaves, dusted with carborundum, of seedlings in the three-leaf stage between two fingers dipped into the virus containing sap. The sap was expressed from leaves of test plants infected with a standard virus inoculum. The symptoms were recorded 7, 14 and if necessary 30 days after infection. The average of three replications was regarded as the final result. The evaluations were based on the severity of symptoms, on the time of their appearance (early or late symptoms). The duration of the incubation period was also recorded.

Results

The results are shown in Table 1. In Table 1 out of 170 varieties, hybrids and lines, only those are listed which proved to be tolerant, resistant and immune, respectively. Tolerant varieties were grouped into two classes. Class A comprises those varieties which, although exhibiting typical symptoms, became infected only to a limited extent (less than 60 per cent of the individuals inoculated). Such varieties were designated as slightly tolerant. They made up 8.1 per cent of the total. Class B includes those varieties which showed typical symptoms only sporadically. With these varieties not more than 30 per cent of the individuals showed "atypical" symptoms after a considerable incubation period (about two weeks). Such varieties were designated as highly tolerant. They made up 4.1 per

Table 1

Classification of maize varieties, hybrids and lines according to their degree of resistance to maize stripe mosaic virus after artificial inoculation

Number of varieties, hybrids and lines tested	Tolerant A (slightly)	Tolerant B (highly)	Resistant	Immune
1	Keszthelyi hybrids	V 15 × 129 hybrid	15 × 335 hybrid	335 line
.	17, 22	V 15 × 107 hybrid	15 × 333C hybrid	
.		V 15 × 107A hybrid	333C line	
.	Martonvásári hybrid	V 15 × 333B hybrid	WF9	
.	26, 57	WF9 × Ml4 hybrid		
.	Szegedi hybrid	333B line		
.	70, 78	107A line		
.	Iregi hybrid			
.	12			
.	Szegedi sárga variety			
.	Aranyözön variety			
.	3, 4, 7			
.				
.				
.				
170	8.1%	4.1%	2.4%	0.6%

cent of the total. Those varieties which did not exhibit typical symptoms at all, only mild atypical ones after 2 to 4 weeks in 10 per cent of the total were regarded as resistant. The resistant varieties made up 2.4 per cent of the total. Only a single line proved to be immune. Individuals of this line showed no symptoms and did not contain virus either. With susceptible varieties about 90 to 100 per cent of the individuals became infected in one week.

The "atypical" symptoms mentioned above were characterized by the appearance on the leaves of wider and longer pale streaks instead of the typical short streaks (Fig. 2B). Plants bearing atypical symptoms showed no sign of

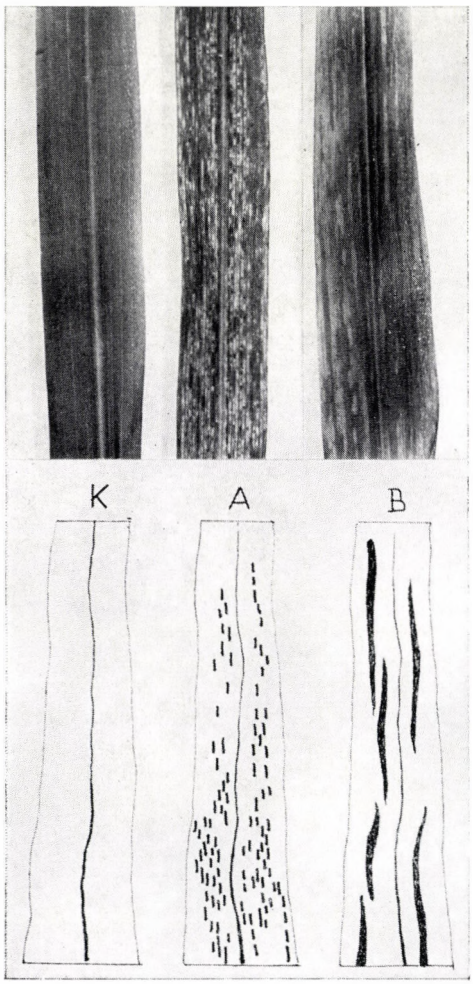


Fig. 2. Symptoms of maize stripe mosaic disease on leaves of susceptible and resistant maize plants. Control (K); typical symptoms consisting of short streaks on a susceptible plant (A); atypical symptoms consisting of pale long stripes on a resistant plant (B)

injury of any other kind. This type of symptom can be regarded as an indication of resistance.

To test the source of tolerance or resistance of certain hybrids some of the parents were also tested in the above manner. It was found that parents of tolerant

or resistant (B) hybrids also showed atypical symptoms upon infection with maize stripe mosaic virus. Consequently these parents were also tolerant or resistant. One of them was found to be immune.

Discussion

In the symptomatology of maize stripe mosaic disease as it occurs in Hungary, the most striking phenomenon is the existence of two types of symptoms. The milder type is the more common one whereas the severe type is more seldom and its occurrence varies from year to year. The reason for this fluctuation may be sought in the properties of either the host plant or the virus.

If we try to explain the appearance of severe symptoms by differences in the reaction of the host we may refer to the work of DEAN and COLEMAN (1959). They found that out of 72 sorghum varieties only 21 varieties reacted with severe necrotic symptoms, the rest exhibited simple mosaic pattern, a proof of the varietal difference in host reaction.

With hybrids or "unstabilized" varieties the reaction type of the parents may recur in a certain percentage of the offspring. It has been observed that some individuals of a certain maize variety reacted with mild red streaks upon infection just as infected sorghum plants did. This reaction type was characteristic of those varieties which due to their genetic machinery tended to form anthocyanins.

In field trials most work was done with two varieties: one was the hybrid "Martonvásári 5" and the other the sweet corn variety "Fekete mazsola". The latter showed an increased tendency towards severe necrotization. According to HALL et al. (1966) sweet corn varieties are more likely to get infected. These phenomena were not widespread even in those years in which severe symptoms could be observed; they occurred only in about 5 to 8 per cent of the whole stand.

Literary data indicate that early infections usually lead to severe symptoms and heavy damage to the plants. In May, 1964, 200 young maize plants (8 to 10 cm high, three-leaf stage) belonging to the above two varieties were mechanically infected in the field and compared throughout the vegetation period to plants growing nearby which became infected by aphids only in July. Although the yield of these maize plants infected in an early stage of development was less than of those infected in July, no severe symptoms were observed. From this it may be concluded that early infection does not always lead to the appearance of heavy symptoms.

As to the properties of the virus as a possible reason for the differences in the severity of symptoms, reference should be made to several authors (ABBOTT and TIPPETT, 1966; ZUMMO et al., 1967; WAGNER and DALE, 1966; MACKENZIE et al., 1966), who reported that different isolates from sugarcane, sorghum and maize caused symptoms of varying intensity.

It is not clear yet whether with certain strains of maize stripe mosaic virus there is such a high degree of specialization to insect vectors, as with strains of

barley yellow dwarf virus (OSWALD and HAUSTON, 1953; ROCHOW, 1958) or maize streak virus (HINZ, 1963). If there is, this could serve as a possible explanation for the yearly changes in the severity of symptoms. One could namely assume that certain strains which cause severe symptoms are specialized to some vector species which do not occur in every year with the same intensity.

Breeding for resistance seems to be a promising way of controlling the stripe mosaic disease of maize (BANCROFT et al. 1966; STOREY and HOWLAND, 1966; LOVISOLO and ACIMOVIC, 1961; POP, 1966). This is supported by the finding that among artificially infected varieties some exhibited extreme resistance. Tolerance or resistance in certain maize hybrids can be traced back to the parents. Tolerant or resistant maize varieties react to virus infection with the formation of atypical mild mosaic symptoms. This phenomenon facilitates the selection of tolerant or resistant lines in the breeding work. Reduced symptom development in resistant plants seems to be connected with retarded virus multiplication. In resistant plants the incubation period of atypical symptoms is two to three times longer than that of the typical symptoms in susceptible plants. This longer incubation period seems to be due to a decreased rate of virus multiplication as shown by experiments in which parallel infectivity tests were made with saps pressed from resistant and susceptible plants at a given time after infection. Rubbing with sap from diseased resistant plants resulted in 60 per cent less infections than rubbing with sap from diseased susceptible plants.

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Susceptibility, Hypersensitivity and Immunity to Potato Virus Y in Wild Species of Potatoes

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This investigation was initiated to study the reactions of wild potato species to potato virus Y (PVY). Three types of reaction — susceptibility, hypersensitivity and immunity — could be distinguished. Five selections of four *Solanum* species were found to be susceptible and 35 selections of 26 *Solanum* species to be hypersensitive to PVY. One selection of *Solanum chacoense* Bitt. (P. I. 275451), of *S. simplicifolium* Bitt. (P. I. 218223), of *S. stoloniferum* Schlechtd. (P. I. 275244) and of *S. vernei* Bitt. et Wittm. (P. I. 275155) each were immune to mechanical and graft inoculations with PVY.

Introduction

Solanum species, potato varieties and seedlings if inoculated with potato virus Y (PVY) may show one of the following reactions: *(a)* susceptibility to infection, *(b)* tolerance, *(c)* resistance to virus-increase and spread, *(d)* resistance connected with hypersensitivity and *(e)* extreme resistance or immunity. Immunity to PVY is heritable in potato [*Solanum tuberosum* L. (H. ROSS, 1958a; 1961a)]. Among *Solanum* species the following are the most important for breeding for virus resistance: *Solanum stoloniferum* Schlechtd. (syn. = *S. ajuscoense* Buk., *S. antipoviczii* Buk., *S. candelarianum* Buk., *S. longipedicellatum* Bitt., *S. malinchense* Hawkes, *S. neoantipoviczii* Buk., *S. tlaxcalense* Hawkes), *Solanum chacoense* Bitt. (syn. = *S. boergeri* Buk., *S. emmeae* Juz. et Buk., *S. garciae* Juz. et Buk., *S. gibberulosum* Juz. et Buk., *S. guaraniticum* Hassl., *S. horovitzii* Buk., *S. jujuyense* Hawkes, *S. knappei* Juz. et Buk., *S. muelleri* Bitt., *S. parodii* Juz. et Buk., *S. saltense* Hawkes, *S. schickii* Juz. et Buk., *S. subtilius* Bitt.) and *Solanum spectabile* (Corr.) Hawkes. Among the above wild *Solanum* species several individuals were found to bear resistance-conditioning genes (STELZNER, 1950; H. ROSS and BAERECKE, 1950; H. ROSS, 1958a; b; c; d; 1960; 1961b; 1962; EASTON et al., 1958; RUDORF, 1958; ANONYMOUS, 1958; ROTHACKER and WITT, 1959; WEBB and SCHULTZ 1961; R. W. ROSS and ROWE, 1965; 1966; HORVÁTH 1966a and others). Comprehensive works by ROTHACKER (1961), SCHICK and HOPFE (1962), WALKER (1965), RADCLIFFE and LAUER (1966) and BJÖRLING (1966) deal with the problems and the present status of virus resistance.

Materials and Methods

The selections of tuber-bearing *Solanum* species used in these studies were supplied by Prof. Dr. R. W. Ross (see P. I. numbers in the Table), Potato Introduction Station, Sturgeon Bay, Wisconsin, USA and by Prof. Dr. H. Ross (see R-numbers in the Table), Max-Planck-Institut, Köln-Vogelsang, German Federal Republic[1]. They included *S. acaule* Bitt., *S. acroscopicum* Ochoa, *S. ajanhuiri* Juz. et Buk., *S. berthaultii* Hawkes, *S. boliviense* Dun., *S. brachycarpum* Corr., *S. brevicaule* Bitt., *S. brevidens* Phil., *S. canasense* Hawkes, *S. cardiophyllum* Lindl., *S. chacoense* Bitt., *S. demissum* Lindl., *S. ehrenbergii* (Bitt.) Rydb., *S. famatinae* Bitt. et Wittm., *S. gourlayi* Hawkes, *S. gourreroense* Corr., *S. hjertingii* Hawkes, *S. hougashii* Corr., *S. infundibuliforme* Phil., *S. jamesii* Torr., *S. kurtzianum* Bitt. et Wittm., *S. leptophyes* Bitt., *S. megistacrolobum* Bitt., *S. polytrichon* Rydb., *S. simplicifolium* Bitt., *S. sparsipilium* (Bitt.) Juz. et Buk., *S. stoloniferum* Schlechtd., *S. tarijense* Hawkes, *S. vernei* Bitt. et Wittm. and *S. verrucosum* Schlechtd. In the tested plants of *Solanum* species grown from true seed all leaves have been inoculated when the plants reached 3 to 4 inches in height.

The inoculations with PVY were performed with an inoculum prepared from the mixture of four PVY strains (HORVÁTH, 1966b; c; 1967a; b). The PVY was carried in tobacco *(Nicotiana tabacum* L. cv. *Samsun)* plants. Mechanical inoculations were made with raw undiluted sap extracted from infected *Samsun* tobacco plants with a mortar and pestle and applied to carborundum (white, 500 mesh) dusted leaf surfaces with a glass spatula. In all mechanical inoculation every leaf of the plant to be tested was rubbed. After mechanical inoculation the plants were rinsed with water. Plant symptoms and/or subinoculations from inoculated plants to the appropriate indicator host were used as criteria for evaluating the susceptibility or immunity of selections to the PVY infection. *Nicotiana tabacum* L. cv. *Samsun* and *Solanum demissum A*6-hybrid were used as diagnostic hosts for PVY. *Solanum* species immune to mechanical inoculation were all tested by graft inoculation. Tomato (*Lycopersicon esculentum* Mill.) and/or tobacco *(Nicotiana tabacum* L. cv. *Samsun)* stocks of single top grafts were mechanically inoculated and both stock and scion tested for presence of PVY.

Results

Forty-four selections of 30 *Solanum* species with reported susceptibility, hypersensitivity and immunity to PVY were evaluated by mechanical and graft inoculation for immunity to this virus. Plant reactions of *Solanum* species following the mechanical inoculation are shown in the Table 1. In all tests when immune reactions resulted, all tests for virus recovery remained negative. *Solanum* species

[1] Thanks are due to Prof. Dr. R. W. Ross and to Prof. Dr. H. Ross for sending the seeds of these *Solanum* species.

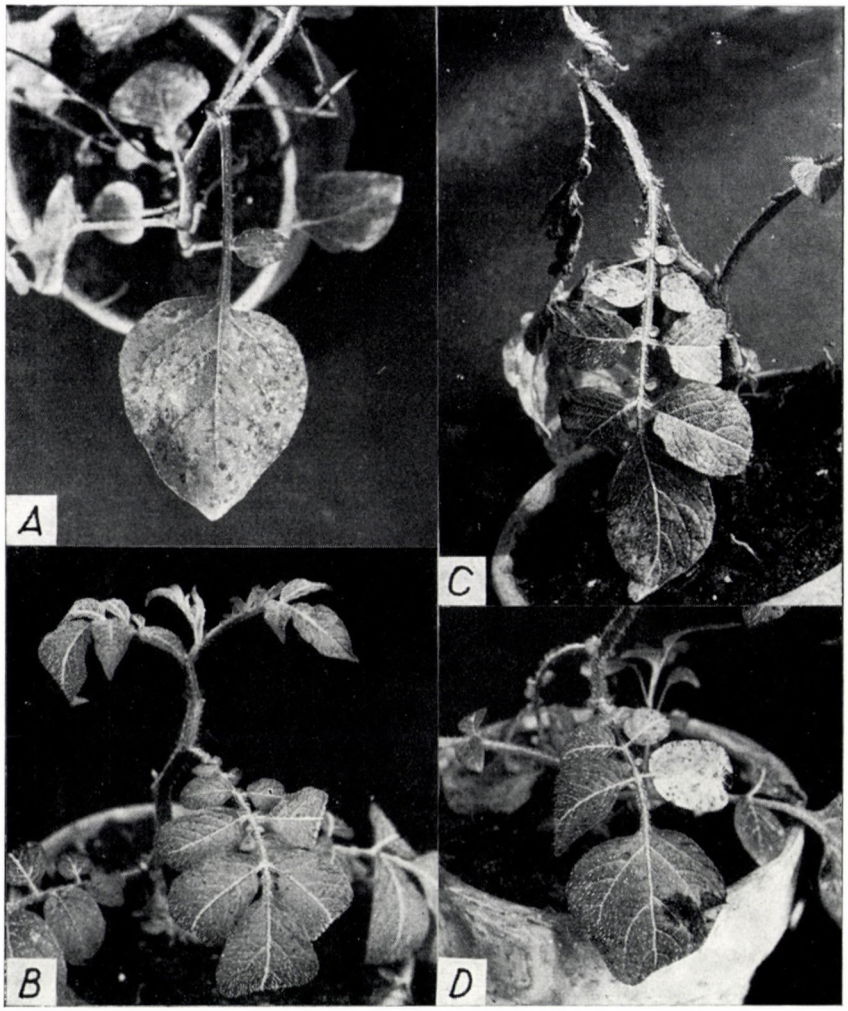


Fig. 1. Symptom reaction of potato virus Y on *Solanum guerreroense* Corr. A, B and C: P. I. 161730, D: P. I. 161727

immune to mechanical inoculation was further tested using grafts to make certain immunity would be expressed to different types of inoculation. *Solanum* species given in Table 1 immune to mechanical inoculation, were all tested by graft inoculation. The material previously found to be immune to mechanical inoculation was immune likewise to graft inoculations. One selection of *Solanum chacoense* Bitt. (P. I. 275451), one of *S. simplicifolium* Bitt. (P. I. 218223), one of *S. stoloniferum* Schlechtd. (P. I. 275244) and one of *S. vernei* Bitt. et Wittm. (P. I. 275155)

Table 1

Susceptibility, hypersensitivity and immunity of Solanum species to potato virus Y

Plant introduction or R number	Solanum species	Collector or donor number[1]	Method of inoculation[2]	
			Mechanical inoculation	Graft inoculation[3]
P. I. 208856	S. acaule Bitt.	Bru 33-Argentina	H	—
P. I. 230495	S. acroscopicum Ochoa	Och 2043-Peru	H	—
P. I. 255490	S. ajanhuiri Juz. et Buk.	CEEA 17-Bolivia	H	—
P. I. 265857	S. berthaultii Hawkes	EBS 1799	H	—
P. I. 265858	S. berthaultii Hawkes	EBS 1846-Bolivia	S	—
P. I. 265860	S. boliviense Dun.	EBS 1795-Bolivia	H	—
P. I. 275179	S. brachycarpum Corr.	Haw 1547-Mexico	H	—
P. I. 275180	S. brachycarpum Corr.	Haw 1577-Mexico	H	—
P. I. 265871	S. brevicaule Bitt.	EBS 1812-Bolivia	H	—
P. I. 245764	S. brevidens Phil.	Cor C. 15-Chile	H	—
P. I. 265864	S. canasense Hawkes	EBS 1831	S	—
P. I. 283063	S. cardiophyllum Lindl.	(218 × 298) × (218 × 297)	H	—
P. I. 275136	S. chacoense Bitt.	Hje 349-Argentina	H	—
P. I. 217451	S. chacoense Bitt.	Sleu 3566-Argentina	I	I
P. I. 186562	S. demissum Lindl.	Haw II. 18-Mexico	H	—
P. I. 275211	S. demissum Lindl.	Haw 1782-Guatemala	H	—
P. I. 275214	S. ehrenbergii (Bitt.)Rydb.	Haw 1429-Mexico	H	—
P. I. 275143	S. famatinae Bitt. et Wittm.	Hje 1893-Argentina	S	—
P. I. 265865	S. gourlayi Hawkes	EBS 1815-Argentina	H	—
P. I. 265883	S. gourlayi Hawkes	EBS 1850-Bolivia	H	—
P. I. 161727	S. guerreroense Corr.	Cor 14342-Mexico	H	—
P. I. 161730	S. guerreroense Corr.	Cor 14410-Mexico	H	—
P. I. 186560	S. hjertingii Hawkes	Haw 1114-Colombia	H	—
P. I. 275174	S. hjertingii Hawkes	Haw 1356-Mexico	H	—
P. I. 161174	S. hougasii Corr.	Cor 14253-Mexico	H	—
P. I. 161726	S. hougasii Corr.	Cor 14340-Mexico	H	—
P. I. 265867	S. infundibuliforme Phil.	EBS 1806	H	—
P. I. 275264	S. jamesii Torr.	CPC 386 × 388 — USA	H	—
P. I. 275265	S. jamesii Torr.	CCC 388 × 381	H	—
P. I. 175435	S. kurtzianum Bitt. et Wittm.	EBS 42	H	—
P. I. 205390	S. kurtzianum Bitt. et Wittm.	Bru — Argentina	H	—
P. I. 210056	S. leptophyes Bitt.	Hje 1592—Peru	H	—
P. I. 275147	S. megistacrolobum Bitt.	Hje 366—Argentina	H	—
P. I. 186545	S. polytrichon Rydb.	Haw 1090	H	—
P. I. 218223	S. simplicifolium Bitt.	EBS 190—Argentina	I	I
P. I. 218224	S. simplicifolium Bitt.	EBS 447—Argentina	S	—
R 58.452	S. simplicifolium Bitt.	EBS — 1082	S	—
R 63.44	S. sparsipilium (Bitt.) Juz. et Buk.	EBS 2189	H	—
P. I. 275244	S. stoloniferum Schlechtd.	Haw 1293—Mexico	I	I
P. I. 275248	S. stoloniferum Schlechtd.	Haw 1520—Mexico	H	—
P. I. 217457	S. tarijense Hawkes	Sleu 4002—Argentina	H	—
P. I. 230468	S. vernei Bitt. et Wittm.	CPC 2413—Argentina	H	—
P. I. 275155	S. vernei Bitt. et Wittm.	Hje 351—Argentina	I	I
P. I. 275257	S. verrucosum Schlechtd.	Haw 1532—Mexico	H	—

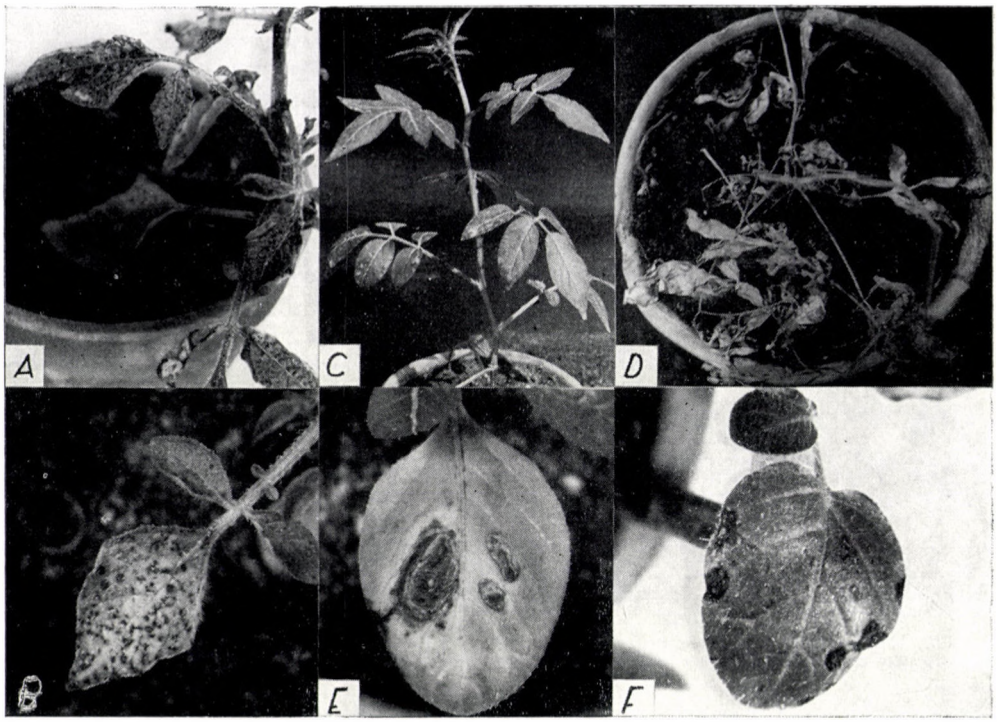


Fig. 2. Symptom reaction of potato virus Y on wild species of potatoes. A and B: *Solanum brachycarpum* Corr. P. I. 275180 and P. I. 275179., C and D: *S. chacoense* Bitt. P. I. 217451 (immune to potato virus Y) and P. I. 275136 (hypersensitive to potato virus Y), E: *S. boliviense* Dun. P. I. 265860, F: *S. leptophyes* Bitt. P. I. 210056

were immune to mechanical and graft inoculations with PVY (Fig. 2). Some selections developed hypersensitive black necrotic ring-like spots (*S. acaule* Bitt., P. I. 208856, *S. acroscopicum* Ochoa, P. I. 230495, *S. ajanhuiri* Juz. et Buk., P. I. 255490,

[1] A key to the prefixes is listed below: Bru—Brücher, E. H., Universidad Nacional de Cuyo, Mendoza, Argentina; CCC—Coleccion Central Colombiana, Centro Nacional de Investigaciones Agricolas Tibaitatá, Bogotá, Colombia; CEEA—Colleccion Estación Experimantal Altiplanó, Servició Agricola Interamericanó, La Paz, Bolivia; Cor—Correll, D. S., Texas Research Foundation, Renner, Texas; CPC—Commonwealth Potato Collection, John Innes Institute, Bayfordbury, Hertford, England; EBS—Erwin Bauer Sortiment, Max-Planck-Institut für Züchtungsforschung, Köln-Vogelsang, Germany; Haw—Hawkes, J. G., University of Birmingham, Edgbaston Birmingham, England; Hje—Hjerting, J. P., Copenhagen Botanical Gardens, Copenhagen, Denmark; Och—Ochoa, C. M., Universidad Nacional Agraria, Lima, Peru; Sleu—Sleumer, H., Universidad Nacional de Tucuman, Tucuman, Argentina.

[2] I = Immune plant reaction, H = Hypersensitive plant reaction, S = Susceptible plant reaction, — = No test.

[3] Graft inoculation of seedlings immune to mechanical inoculation.

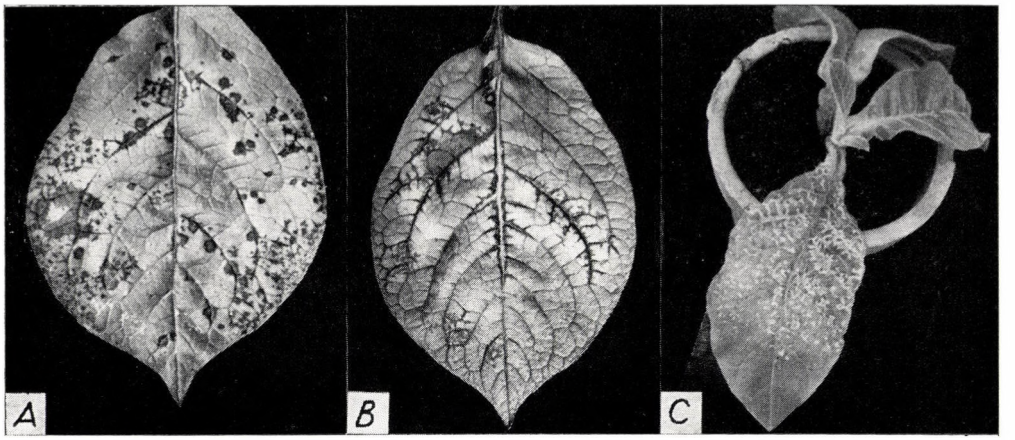


Fig. 3. Reaction of potato virus Y on test plants. A and B: *Solanum demissum A6*-hybrid, C: *Nicotiana tabacum* L. cv. *White Burley*

S. boliviense Dun., P. I. 265860 (Fig. 2), *S. cardiophyllum* Lindl., P. I. 283063, *S. demissum* Lindl. P. I. 186562, and P. I. 275211, *S. ehrenbergii* (Bitt.) Rydb., P. I. 275214, *S. gourlayi* Hawkes, P. I. 265883, *S. hjertingii* Hawkes, P. I. 186560, *S. kurtzianum* Bitt. et Wittm., P. I. 205390, *S. megistacrolobum* Bitt., P. I. 275147, *S. sparsipilium* (Bitt.) Juz. et Buk., R. 63.44, *S. stoloniferum* Schlechtd. P. I. 275248) and black necrotic ring-like spots and leaf abscission (*S. berthaultii* Hawkes, P. I. 265857,*S. brachycarpum* Corr., P. I. 275179 and P. I. 275180 (Fig. 2), *S. brevicaule* Bitt., P. I. 265871, *S. brevidens* Phil., P. I. 245764, *S. guerreroense* Corr., P. I. 161727 and P. I. 161730 (Fig. 1), *S. hougasii* Corr., P. I. 161726, *S. kurtzianum* Bitt. et Wittm,. P. I. 175435, *S. leptophyes* Bitt., P. I. 210056, (Fig. 2), *S. polytrichon* Rydb., P. I. 186545, *S. tarijense* Hawkes, P. I. 217457, *S. vernei* Bitt. et Wittm., P. I. 230468, *S. verrucosum* Schlechtd., P. I. 275257) and total necrosis (*S. chacoense* Bitt., P. I. 275136, *S. gourlayi* Hawkes, P. I. 265865, *S. hjertingii* Hawkes, P. I. 275174, *S. hougasii* Corr., P. I. 161174, *S. infundibuliforme* Phil., P. I. 265867, *S. jamesii* Torr., P. I. 275264 and P. I. 275265). All the remaining selections of the 44 listed in the Table (five selections of 4 *Solanum* species) were found to be susceptible (*S. berthaultii* Hawkes, P. I. 265858, *S. canasense* Hawkes, P. I. 265864, *S. famatinae* Bitt. et Wittm. P. I. 275143, *S. simplicifolium* Bitt., P. I. 218224 and R 58.452).

All plants of the *Solanum* species with visible PVY symptoms gave positive reactions on *Nicotiana tabacum* L. cv. *White Burley*, *Nicotiana tabacum* L. cv. *Samsun* and/or *Solanum demissum A6*-hybrid (Fig. 3). The fact that symptomless plants caused no reaction on *Nicotiana tabacum* L. cv. *White Burley*, *Nicotiana tabacum* L. cv. *Samsun* and/or *Solanum demissum A6*-hybrid indicated that they were not symptomless carriers of PVY.

Thanks are due to Mrs. K. MÖLLER, Miss É. BALOGH, Miss É. TARCSI and Miss I. BENKŐ for their helpful assistance and to Mrs. O. KASSAI and Mr. S. WECKERMANN for the preparation of photos.

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Studies on *Fusarium oxysporum* f. *lupini* Strains

Part I

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The pathogen of the infectious wilt of lupine, *Fusarium oxysporum* f. *lupini* Snyder et Hansen, is not a taxonomically homogeneous microorganism, but the aggregate of distinct strains or races out of which four were isolated in the Nyírség region of Hungary on the basis of cultural characteristics, morphological properties, the difference in the colour of the pigments and of biological specialization. With respect to the average dimensions of the macroconidia no difference was found between the strains and races which would exceed the limits of individual mutations. Thus the dimensions of the conidium remain the distinguishing characteristics of the *form*. Beyond its theoretical mycological implications the knowledge of the races has its practical importance. Specialization of the pathogen within the form hinders the successful development of resistance. If the resistant plant individuals are selected under conditions when no more than 1—2 races will cause the disease, only a race-specific resistance can be achieved. It is, therefore, expedient to select the resistant plant individuals from soil areas severely contaminated by the pathogen, namely, from areas in which all possible races characteristic of the region will be present in the soil. It should be further known which races are the most widely distributed ones in the area. The characteristics for the differentiation of the strains or races, as disclosed in this paper, should be included in the considerations.

Introduction

In recent years we have subjected to detailed study the pathogen of the infectious wilt of lupine, namely *Fusarium oxysporum* f. *lupini* Snyder et Hansen (1940). The comparison of a great number of isolates revealed certain *differences* with a conspicuous *similarity* between some groups. Both the difference and similarity manifested in the cultural characteristics, morphological properties, the pigment colours and pathogenity which promoted us to study the problem of biological specialization, and led finally to an investigation of the strains i. e. races.

Review of the Literature

Researchers both in Hungary and abroad isolated and identified several *Fusarium* species from the lupine. These species represent about six sections of the

Fusarium genus. In the present literature survey we wish to mention only those authors who studied the pathogens in the *Elegans* section or accept these as the phytopathogen organisms which may bring about the infectious wilt of the lupine.

RICHTER (1939, 1941, 1941/a) definitely maintains that the pathogen of the pernicious wilt of lupine is one of the forms of *Fusarium oxysporum*. RAJLLO (1950) claims that in Central Europe primarily *Fusarium lupini* Jacz. is responsible for the wilt diseases of *Lupinus luteus;* this pathogen was later identified as a fungus belonging to one of the forms of *Fusarium oxysporum*. According to WOLLENWEBER and REINKING (1935) the wilt of lupine is caused by forms of *Fusarium oxysporum* and *Fusarium vasinfectum*. In his experiments with *Fusarium oxysporum* f. *lupini* LAMBERTS (1951) was able to produce wilt disease on lupine. WUTTKE (1943), too, mentions *Fusarium oxysporum* in connection with the wilt of lupine. TROLL (1948) carried out resistance tests on lupine cultivars to *Fusarium oxysporum*. BARBACKI (1952) claims that *Fusarium oxysporum* Schlecht. is here of primary importance, BILAJ (1955) too, mentions *Fusarium oxysporum* Schlecht. as the pathogen responsible for the wilt of lupine.

Only a few Hungarian researchers have been engaged in the study of this pathogen. According to UBRIZSY (1942) the wilt of lupine in the Nyírség region was almost unexceptionally caused by *Fusarium oxysporum*. HUSZ (1935) pointed out that the suppression of white lupine cultivation was caused by *Fusarium oxysporum*. Mrs. CSUTI (1963) described the pathogen found in the Nyírség and South Somogy regions as *Fusarium oxysporum* f. *lupini* Snyder et Hansen. BÉRES (1965) used in his experiments *Fusarium oxysporum* inoculum which he had isolated from lupine.

Within the *Fusarium* genus *Fusarium oxysporum* f. *lupini* belongs to the *Elegans* section. For practical reasons SNYDER and HANSEN (1940) have converted the *Elegans* section into a single species, so that all members of the section form a natural group of species which may be called *Fusarium oxysporum*. This species is emended in such a way that it shall reflect all the fundamental morphological properties of the section. It will contain as many biological forms as the number of pathogens within it. The parasitic forms can be recognized primarily by their selective pathogenic character. This concept of the species does not refute the *phenomenon of variability* in spite of the simplification of the general concept of species.

Our investigations have indicated that *variability within the form produces such differential characteristics* that open the way to the disclosure of the existence of strains which are all different in many respects. So far we have been able to isolate four strains.

Material and Method

The material on which the comparison was based was collected from the Northern and Central Nyírség Region and from County Szabolcs-Szatmár in the years 1964 and 1965 (Fig. 1).

The pathogen was isolated by RAJLLO's (1950) method. First an initial culture and then a pure culture was prepared. Monoconidial cultures were used in the experiments. The cultures were incubated at 21 – 23° C. The standard medium for the measurement of the macroconidia was a 2% potato agar. Measurements were carried out on the 15th day. The colours of the pigments on rice were de-

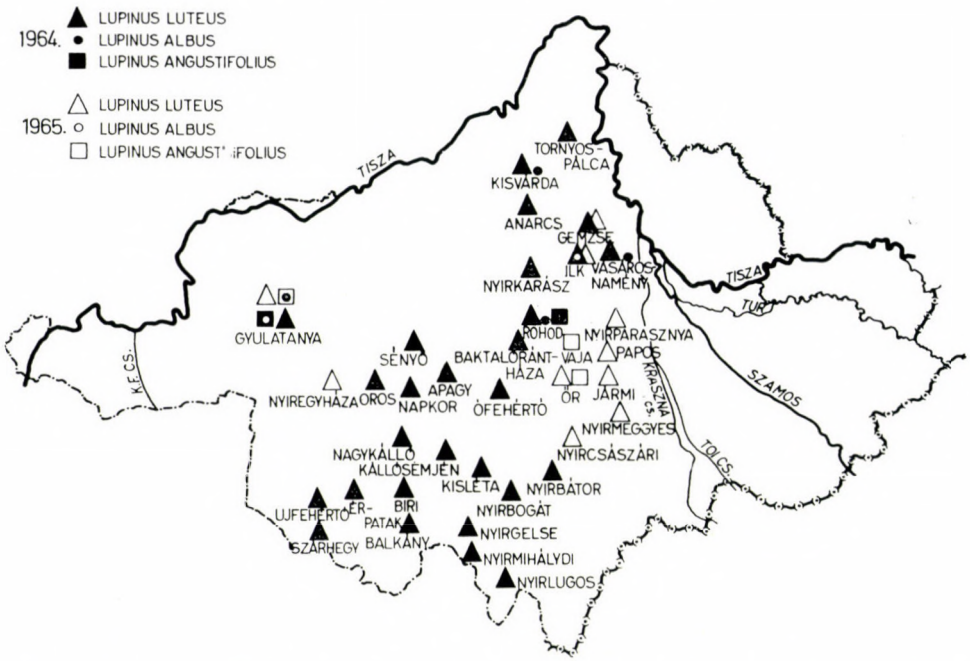


Fig. 1. The origin of *Fusarium oxysporum* f. *lupini* isolates from field crops (County Szabolcs-Szatmár)

scribed by means of the Pacht colour chart on the 15th and 30th day, rice being the generally accepted standard medium, as the colour differentiating powers of other media may vary. About 920 cultures were available for the comparison of the pigments. In the measurements the data of 22 cultures were compared and evaluated statistically. To find an answer to the problem of biological specialization the lupine species of the crops from which the various strains could be isolated had to enter the considerations. We had at our disposal the species and cultivar collection of the Gyulatanya Gardens belonging to the Agricultural Research Institute of the Nyírség Region.

Results

Pigmentation differences of the strains

Pigmentation as a taxonomical characteristics within the *Fusarium* genus is not equally useful for differentiation of taxonomical units. WOLLENWEBER used the pigments as diagnostic characters to describe the different species, varieties and forms. In certain cases, however, colour may be applied to the characterization of smaller units such as strains or races. In his monograph RAJLLO quotes a number of authors on the problems of physiological specialization according to whom the races differ also in respect to pigment formation, that is, the derivations can be considered as real differences between the races.

Our observation of 920 isolates indicated a sharp difference between the strains with respect to pigmentation, while there was a marked similarity between the isolates within the same strain or race. Thus it appeared justified to differentiate between the groups on the basis of colour differences.

On the 15th and 30th day the following differences in the colour of the pigments and the appearance of sclerotia on rice medium were observed for the different strains of *Fusarium oxysporum* f. *lupini:*

Strain No. I

15th day: The primary aerial mycelium is pale pink (purpureus 1–2) with a white shade. The edges around the rice seeds are sharply outlined and of purple colour (purpureus 6–7). At the bottom of the test tube the fungus spreads in a 1.5 cm zone with pale violet pigmentation (violaceus 1–2). In this zone the edges of the rice seeds are lavender blue (violaceus 3). No sclerotium is formed.

30th day: The primary aerial mycelium has a mauve colour (purpureus 3). The edges of the rice seeds are still purple (purpureus 6–7). The violet zone is completely lacking. No secondary mycelium or sclerotium is observed.

Strain No. II

15th day: The primary aerial mycelium has a mauve colour (purpureus 3). The edges of the rice seeds are sharply outlined and of purple colour (purpureus 6–7). No violet pigmentation appears at the bottom of the test tube. No sclerotium is formed.

30th day: The pale violet colour of the primary mycelium is completely masked by the secondary mycelium (violaceus 3–4). The edges of the rice seeds vary from medium violet to dark violet (violaceus 5–9). In an about 1 cm zone at the bottom of the test tube a mauve pigmentation appears (purpureus 3).

Strain No. III

15th day: The primary mycelium is pale violet (violaceus 2), with an occasional white shade. The edges of the rice seeds are sharply outlined and have a darker

violet colour (violaceus 5–6). Here and there purple spots of 4–8 mm in diameter are visible (purpureus 5–6). No sclerotium is formed.

30th day: The primary violet mycelium is masked by a secondary pink mycelium (purpureus 3). The edges of the rice seeds are bluish violet (lazulinus 6, violaceus 6–8). Here and there purple spots are visible (purpureus 6–7). No sclerotium was observed.

Strain No. IV

15th day: The primary aerial mycelium is pale pink (purpureus 1–2). The edges of the rice seeds are not sharply outlined, but have a deeper pink shade (purpureus



Fig. 2. Non-sclerotium forming strains (left). Sclerotium forming strains (right)

3–4). No discoloration appears at the contact zone of the rice seeds. The violet shades are completely missing. No secondary mycelium is formed. Sclerotia are formed but rarely single, more often in small groups. Their colour is greenish yellow (chlorinus 1). The sclerotia are covered by pinkish white mycelium.

30th day: The colours are as before and remain essentially unchanged even on the 45th day. (A shade deeper by one degree can be detected by means of the Pacht colour chart.) Sclerotia are present in considerable quantity, arranged in small groups (Fig. 2).

It was further found that the monoconidial cultures of the isolates show no essential colour change over three generations, that is, the pigment remains a

constant characteristic. This fact contributes to the applicability of pigment differences for the distinguishing of races.

The cultures obtained from the monoconidial cultures of isolates which had been collected from 38 areas of the Nyírség region showed within the strains or races only irrelevant colour differences. These minor deviations correspond to the individual variations of the single cultures.

As high variation in pigmentation is a widely distributed phenomenon among the fungi, not much importance was attributed to these differences *alone*. Therefore, other biological and morphological properties of the pathogens were also examined.

Biological specialization

These investigations have indicated that the host range of strains or races Nos I—IV are within the *Lupinus* genus, however, there are differences among the strains with respect to their biological specialization on the lupine species. All strains are pathogenic on *Lupinus luteus*. The strain which forms sclerotia is pathogenic on *Lupinus luteus* and *L. albus*. Strain No. I was isolated from all lupine species shown in Table 1.

Table 1

Classification according to pathogenity of strains (races*) isolated from various species of lupine

Lupine species	Fusarium oxysporum f. lupini			
	Strain I	Strain II	Strain III	Strain IV
Lupinus luteus	+	+	+	+
Lupinus albus	+	—	—	+
Lupinus angustifolius	+	—	—	—
Lupinus mutabilis	+	—	—	—
Lupinus Hartwegii	+	—	—	—
Lupinus hirsutus	+	—	—	—

* It should be noted that the "indirectly" identified races from the various *Lupinus* species are here in fact only strains, and direct inoculation experiments will prove whether they are distinct races or not.

In one of his publications RICHTER (1941a) mentions that he succeeded in isolating a "form" of different biological behaviour, namely a sclerotium producing "form" from *Lupinus albus*, *L. angustifolius* and *L. mutabilis*.

The problem whether this "form variety" may be classified into a highly variable subtype of *F. oxysporum* or should be treated as a separate species or variety will be, in his opinion, the task of further investigations.

It seems quite probable that RICHTER isolated our strain No. IV which we had isolated not only from *L. albus*, but in a number of cases also from *L. luteus*.

It should be mentioned further that strain No. IV which formed sclerotia differs from the non-sclerotia forming strains also in a lower requirement of temperature for growth and infection. This may explain the finding that the majority of isolates from plants which were infected by *Fusarium* at an early stage also formed sclerotia. Out of the 100 fungus cultures, isolated on May 11, 1966 from an early sown lupine, 96 developed sclerotia. The majority of the June and July isolates were strains which formed no sclerotia. Most of the isolates from infected plants of the August stubble crops showed also the characteristics of strains Nos I–III.

Morphological properties

On potato agar all strains form chlamydospores. The chlamydospores are apical or intercalary, and are situated singly, in pairs or occasionally in groups of three on the mycelium. Occasionally, more than three chlamydospores may appear in chain intervals. Regarding the formation of chlamydospores no morphological or dimensional difference was found between the strains which might have helped their differentiation. It was, however, observed that depending on the age of the culture apical chlamydospores are more frequent in younger cultures in which there are fewer multicellular intercalary chlamydospores, while in older cultures the latter predominate (Figs 3 and 4).

The *macroconidia* are formed in sporodochia. Strain No. IV (which forms sclerotia) is also outstanding because of its high sporulation ability; more than 90% of its macroconidia has three septa. On the other hand, strains Nos II and III show only a poor or fair sporulation tendency and form only few three septa macroconidia (Fig. 5).

The *microconidia* are formed on aerial mycelium and with respect to the morphology of the microconidia there is no difference between the strains. Strain No. I is quite remarkable, as it forms very many microconidia and only very few macroconidica. On the other hand, strain No. IV which forms sclerotium, forms hardly any microconidia, but a considerable quantity of macroconidia.

No difference was observed in the shape of the conidia. Analysis included the determination of the length and width of the macroconidia, of the number of septa and their percentage occurrence.

No essential differences were found in the average lengths and average widths of the macroconidia which might reflect a marked difference between the strains, thus the various strains cannot be characterized by dimensional differences (Table 2).

Those irrevelant dimensional differences which exist among the various strains do not exceed individual mutation which may be found between the single isolates within the same strain. This is shown in Table 3 which contains the data of ten isolates representing strain No. IV. It appears that the deviations in the average

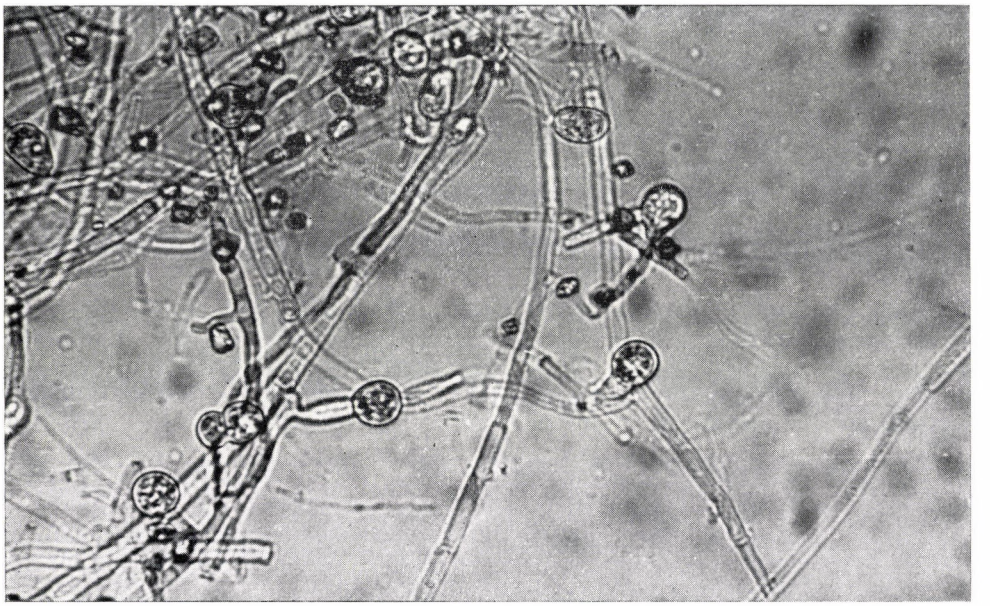


Fig. 3. The apical and intercalary chlamydospores of *Fusarium oxysporum* f. *lupini* from a 15-day old culture

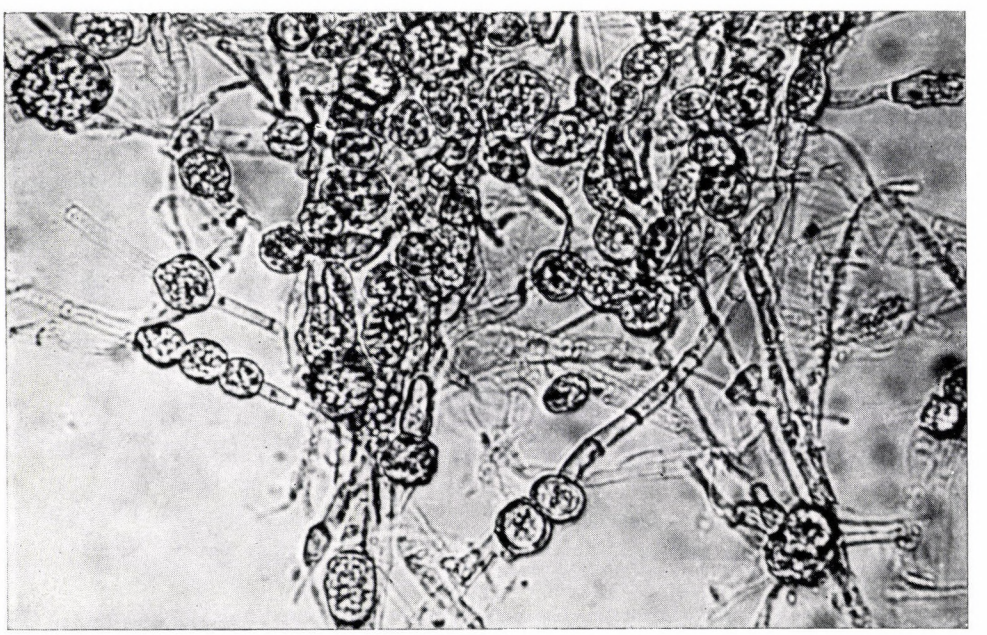


Fig. 4. Chlamydospores of *Fusarium oxysporum* f. *lupini* from a 30-day old culture

Table 2

Comparison of the morphological data of strains Nos I—IV

Strain	Number of septa	Percentage occurrence	Minimum and maximum lengths of conidia, in μ	Length			Minimum and maximum widths of conidia, in μ	Width		
				M $\pm$ m	δ	v		M $\pm$ m	δ	v
I.	1	33.3	19.4—34.3	—	—	—	2.98—4.47	—	—	—
	2	24.4	19.4—34.3	—	—	—	2.98—4.47	—	—	—
	3	42.3	19.4—46.2	31.20$\pm$0.30	5.0	16.20	2.98—5.96	4.55$\pm$0.02	0.33	7.52
	4	—	—	—	—	—	—	—	—	—
	5	—	—	—	—	—	—	—	—	—
II.	1	18.0	20.9—35.8	—	—	—	2.98—4.47	—	—	—
	2	15.0	22.4—40.2	—	—	—	4.47	—	—	—
	3	67.0	25.3—52.2	33.08$\pm$0.39	5.64	17.04	2.98—5.96	4.36$\pm$0.03	0.44	10.00
	4	—	—	—	—	—	—	—	—	—
	5	—	—	—	—	—	—	—	—	—
III.	1	12.0	19.4—32.8	—	—	—	3.72—4.47	—	—	—
	2	16.0	19.4—34.3	—	—	—	3.72—4.47	—	—	—
	3	72.0	20.9—49.2	30.58$\pm$0.68	6.84	22.36	2.98—5.96	4.46$\pm$0.01	0.05	1.12
	4	—	—	—	—	—	—	—	—	—
	5	—	—	—	—	—	—	—	—	—
IV.	1	1.5	19.4—34.3	—	—	—	2.98—4.47	—	—	—
	2	3.0	19.4—37.3	—	—	—	4.47	—	—	—
	3	94.3	19.4—49.2	33.08$\pm$0.50	5.04	15.23	2.98—5.96	4.46$\pm$0.01	0.17	3.81
	4	1.0	40.2—44.7	—	—	—	4.47—5.21	—	—	—
	5	0.2	47.7—49.2	—	—	—	4.47	—	—	—

Table 3

Individual mutations between the single cultures of strain IV (which forms sclerotia) from the aspect of the length and width of the macroconidia and of the percentage occurrence of septa

Strain	Number of septa	Percentage occurrence	Minimum and maximum widths of conidia, in μ	Length			Minimum and maximum widths of conidia, in μ	Width		
				M $\pm$ m	δ	v		M $\pm$ m	δ	v
135—1	1	—	—	—	—	—	—	—	—	—
	2	—	—	—	—	—	—	—	—	—
	3	99	23.9—49.2	35.12$\pm$0.53	5.24	14.92	2.98—4.47	4.42$\pm$0.02	0.23	5.20
	4	1	44.7	—	—	—	5.21	—	—	—
	5	—	—	—	—	—	—	—	—	—
135—2	1	7	19.4—25.3	—	—	—	3.72—4.47	—	—	—
	2	6	23.9—26.9	—	—	—	4.47	—	—	—
	3	85	20.9—43.2	31.25$\pm$0.56	5.16	16.51	4.47	4.47$\pm$0.00	0.00	0.00
	4	2	40.3	—	—	—	4.47—5.21	—	—	—
	5	—	—	—	—	—	—	—	—	—
148—1	1	4	19.4—22.4	—	—	—	2.98—3.70	—	—	—
	2	3	19.4—29.8	—	—	—	4.47	—	—	—
	3	92	19.4—44.7	33.52$\pm$0.67	6.52	19.45	2.98—5.96	4.47$\pm$0.02	0.20	4.47
	4	1	44.7	—	—	—	4.47	—	—	—
	5	—	—	—	—	—	—	—	—	—
155—1	1	—	—	—	—	—	—	—	—	—
	2	3	23.8—31.3	—	—	—	4.47	—	—	—
	3	95	22.4—44.7	32.51$\pm$0.37	3.64	11.19	2.98—4.47	4.43$\pm$0.01	0.15	3.38
	4	1	43.2	—	—	—	4.47	—	—	—
	5	1	49.1	—	—	—	4.47	—	—	—

	1	—	—	—	—	—	—	—	—	—
	2	8	19.4—29.8	—	—	—	4.47	—	—	—
156	3	90	20.8—44.7	32.28±0.62	5.92	18.33	4.47	4.47±0.00	0.00	0.00
	4	2	44.7	—	—	—	4.47	—	—	—
	5	—	—	—	—	—	—	—	—	—
	1	—	—	—	—	—	—	—	—	—
	2	—	—	—	—	—	—	—	—	—
159—1	3	99	23.8—44.7	34.88±0.43	4.12	11.81	2.98—4.47	4.43±0.03	0.27	5.98
	4	—	—	—	—	—	—	—	—	—
	5	1	47.7	—	—	—	4.47	—	—	—
	1	—	—	—	—	—	—	—	—	—
	2	—	—	—	—	—	—	—	—	—
341	3	97	25.3—44.7	33.12±0.47	4.60	13.89	4.47	4.47±0.00	0.00	0.00
	4	3	40.2—44.7	—	—	—	4.47	—	—	—
	5	—	—	—	—	—	—	—	—	—
	1	—	—	—	—	—	—	—	—	—
	2	1	22.4	—	—	—	4.47	—	—	—
424	3	99	22.4—41.7	33.12±0.50	5.00	15.09	4.47	4.47±0.00	0.00	0.00
	4	—	—	—	—	—	—	—	—	—
	5	—	—	—	—	—	—	—	—	—
	1	3	19.4—37.3	—	—	—	4.47	—	—	—
	2	7	25.3—28.3	—	—	—	4.47	—	—	—
433	3	90	26.8—41.7	32.32±0.32	3.04	9.41	3.72—5.96	4.52±0.02	0.20	4.42
	4	—	—	—	—	—	—	—	—	—
	5	—	—	—	—	—	—	—	—	—
	1	1	23.8	—	—	—	4.47	—	—	—
	2	2	29.8—34.3	—	—	—	4.47	—	—	—
441	3	97	20.9—44.7	34.60±0.51	5.08	14.68	4.47	4.47±0.00	0.00	0.00
	4	—	—	—	—	—	—	—	—	—
	5	—	—	—	—	—	—	—	—	—

lengths of the macroconidia are in general 1–3 μ, with a maximum difference of 3.8 μ between cultures Nos 135–1 and 135–2. These differences are not taxonomically significant in the case of the *Fusarium* genus which has an extremely high mutability. These investigations thus confirm that the conidium dimensions in the Table are "*form* characteristics", are applicable to the identification of the form, but not to the differentiation betwen strains or races (Table 3).

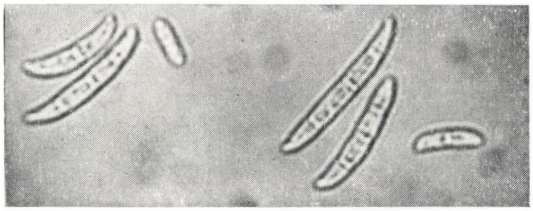


Fig. 5. Micro- and macroconidia from a 15-day old culture

There is, however, a difference in *the number and percentage occurrence of septa*. In the case of strain No. IV there are some, though few, macroconidia with 4–5 septa, while the macroconidia of strains Nos I–III have only 1–3 septa. Macroconidia with three septa dominate in all strains, nevertheless their percentage occurrence at the expense of macroconidia with 1–2 septa in strain No. IV is conspicuously high.

Discussion

It seems probable that further research will lead to the identification of more strains and races. The differences between the *strains* or *races* appear primarily in pigmentation, in the formation of sclerotia, in biological specialization, in the pathogenity and in a variation of the number of septa. For the identification of the form the conidium dimensions serve as distinguishing characteristics.

In addition to *theoretical mycological implications* the search and identification of races are of practical importance in breeding for resistance. Specialization within the form of the pathogen hinders successful breeding for resistance.

As we are dealing with soil born pathogens, it is useful to select the resistant plant individuals from those areas of the strongly infected soil in which all the possible races of that region will be present in the soil; should one or another of the races be missing, an artificial inoculation with the missing race is necessary. Thus it is important to know the most frequently occurring races in that area. This can be established by means of the characteristics which we have found to be suitable for the differentation of strains or races. In addition, the number of conidia per g. of soil for successful infection, with an approximately 100% destruction must also be determined.

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Die Porlinge Ungarns und ihre phytopathologische Bedeutung (Polypori Hungariae)

II. Teil

von

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Der I. Teil dieser Abhandlung erschien ungarisch im Heft 1–2 (p. 201–221) des Jahrganges 1965 der »Wissenschaftlichen Mitteilungen der Universität für Forstwirtschaft und Holzindustrie« (IGMÁNDY, 1965a) und umfasste die Gattungen *Gloeoporus* Mont., *Xanthochrous* Pat. und *Polystictus* Fr. em. Ames. In diesem Teil werden die Genus *Fomes* Fr. em. Gillet und *Ganoderma* Karst. em. Pat. der Familie Polyporaceae sowie deren Arten behandelt.

Zuerst werden die Gattungen beschrieben und der Bestimmungsschlüssel der Arten bekanntgemacht, darauf folgen die heimischen Abbildungen der Arten und die Literatur. Einige allbekannte Arten ausgenommen, wird auch das beweisende Herbariummaterial aufgezählt. Die beweisenden Exemplare sind im Herbarium der Botanischen Abteilung des Ungarischen Nationalmuseums (HMN) und des Lehrstuhls für Forstschutz der Universität für Forstwirtschaft und Holzindustrie (HE) untergebracht. Nach dieser Einleitung werden die Verbreitung der Arten in Ungarn, und deren phytopathologische Bedeutung geschildert.

Fomes (Fr.) Gillet

Der Fruchtkörper sitzt gewöhnlich seitlich auf, meistens ± halbkreisförmig, manchmal sich ausbreitend und auf die Unterlage aufliegend *(F. annosus)*; mit einer verschiedentlich dicken, vom Trama gut abgesonderten Rinde überzogen; Trama weisslich, blass holzfarbig, rosa oder rostbraun – braun; Substanz schwammig, korkartig oder fast holzig; die Röhrenschicht vom Trama in der Materie ganz verschieden, in der Farbe aber ± einheitlich; die Röhren sind geschichtet, ihre Öffnungen sind klein, regelmässig und rund; im Hymenium sind keine Cystiden und Stacheln; Sporen rundlich – walzig, hyalin; Sporenstaub weiss. Die Arten sind perennierend.

In Ungarn sind fünf Arten bekannt.

Schlüssel zur Bestimmung der Arten

1a Trama des Fruchtkörpers rostgelb – braun, weich schwammig; die Rinde dick, weisslich – haselnussfarbig – grauschwärzlich, die Lösung von KOH verfärbt es blutrot; Sporen länglich – ellipsoid, $14-18 \times 5-7\ \mu$:
1. F. fomentarius

1b Farbe vom Trama nicht wie bei 1a; die Rinde verfärbt die Lösung von KOH nicht, oder nur bräunlich 2

2a Farbe des Fleisches rosa; Rinde dünn, schwärzlich; Sporen 5,5–6,5 × ×2,0–2,5 μ. Auf Nadelbäumen:

2. F. roseus



Abb. 1. *Fomes fomentarius* (L.) Kickx. an *Salix*. Sitke

2b Trama weiss – weisslich – blass kremfarbig 3

3a Auf die Einwirkung von Azeton, Benzol, Chloroform löst sich die Rinde schleimig auf, und bekommt einen lackartigen glänzenden Überzug; Farbe der Rinde orange-rot – schwärzlich, die Krempe des Fruchtkörpers ist heller und bildet einen ausgeprägten Rand; Sporen ellipsoid, 5,0–8,0 × 2,5–4,0 μ:

3. F. marginatus

3b Von den vorherigen verschiedene Merkmale 4

4a Form des Fruchtkörpers sehr verschieden; konsolenförmig, flach-zurückgekrempelt oder auf die Unterlage wie eine Kruste aufliegend (auch in diesem Fall ist die Rinde auf der Seite gut erkennbar), dünn; die Rinde kastanien-, oder schwärzlichbraun; Sporen kugelig – el-



Abb. 2. Schädigung der *Fomes fomentarius* (L.) Kickx. im alten Buchenbestand. Szilvásvárad

lipsoid, $4{,}0-6{,}0 \times 3{,}5-4{,}0$ μ. Auf Nadelbäumen, nur ausnahmsweise auf Laubbäumen:

4. F. annosus

4b Der Fruchtkörper konsolenförmig, gross (15 – 50 cm), dick; Farbe der Rinde krem – graubraun; Sporen eiförmig, $7{,}0-9{,}0 \times 5{,}0-6{,}5$ μ. Auf Laubbäumen, vorwiegend auf der Robinie:

5. F. fraxineus

1. *Fomes fomentarius (L.) Kickx*

(Abb. 1, 2)

Die synonymen Namen: Boletus fomentarius L. – *Polyporus fomentarius* (L.) Fr. – *Ochroporus fomentarius* (L.) Schroet. – *Ungulina fomentaria* (L.) Pat. – *Fomes mirus* Kalchbrenner, Fungi e Sibiria., 1878., u. a.

Abbildung: Schulzer, 1869, p. 755. – Bánhegyi, Bohus, Kalmár, Ubrizsy, 1953, Tafel 55/2. – Igmándy, 1958, Abb. 9. – Győrfi, 1963, Abb. 108 und 110. – Igmándy, 1964, p. 101. – Ubrizsy, 1965, II. p. 149. – Haracsi, 1966, B. p. 144.

Literatur: Schulzer bezeichnet das Vorkommen in Ungarn nachstehend (1857): »Überall auf Eichen, Weiden, Eschen, Erlen, aber vorwiegend auf Buchen«. Hazslinszky (1895) schreibt auch in diesem Sinn. Das Vorkommen des Porlings in Ungarn wird von mehreren Autoren bekundet: Hollós (1913: Zwischen Donau und Theiss, 1933: Umgebung von Szekszárd), Fehér und Bessenyei (1933: Sopron), Bohus (1939: Vértes Gebirge), Ubrizsy (1941: Nyírség), Moesz (1942: Umgebung von Budapest), Igmándy (1958: Umgebung von Sopron).

Die phytopathologische Bedeutung des Porlings wird von Haracsi (1953, 1966), Győrfi (1963), Igmándy (1964), Ubrizsy (1965) und anderen gewürdigt.

Der aus dem Trama des Porlings erzeugte »Zunder« diente vornehmlich zum Anfechten des Feuers, und hatte in früheren Zeiten grosse Bedeutung. Die Zundererzeugung wurde in mehreren Gegenden als Hausindustrie betrieben (Küzdi, 1893, Radványi, 1912). Nach Istvánffy (1899) wurde der Zunderbedarf Europas grösstenteils aus Ungarn gedeckt. Die früheren mykologischen Abhandlungen (Schulzer 1869, Istvánffy, 1899, Szemere, 1930, Hollós, 1933, usw.) befassen sich ausführlich mit der Herstellung des Zunders und mit dessen Verbrauch.

Herbarium: Da diese Art nicht selten ist, wurde vom Aufzählen des Herbariummaterials Abstand genommen.

Vorkommen und phytopathologische Bedeutung: Diesen Porling kann man in der Tiefebene, aber auch hoch im Gebirge, also überall antreffen. Man findet ihn fast auf allen Laubbäumen: *Acer* (*pseudoplatanus* L., *platanoides* L.), *Aesculus hippocastanum* L., *Alnus glutinosa* (L.) Gaertn., *Betula*, *Carpinus*, *Fagus*, *Fraxinus*, *Juglans* (*nigra* L., *regia* L.), *Populus* (*alba* L., *tremula* L., *nigra* v. *italica* Müench., *euramericana* (Dode) Guin.), *Prunus avium* L., *Quercus* (*robur* L., *sessiflora* Salisb., *cerris* L., *borealis* Michx), *Salix*, *Tilia*, *Ulmus*, ausnahmsweise auch auf Nadelbäumen: *Abies*. Das häufige Vorkommen des Pilzes wird durch das Vorhandensein oder durch den gesundheitlichen Zustand der Wirtpflanze bestimmt. Am häufigsten ist er auf der Buche, wie das schon Schulzer (1857) feststellte, dann auf der Birke, aber manchmal tritt er epidemisch auf alten Bluteichen, und auf überhaltenen Zuchtpappeln u.a. m. auf. In alten überhaltenen Buchenbeständen ist er fast an allen Stämmen anzutreffen, und auf einem eingegangenen Stamm findet man dutzendweise die Fruchtkörper. *Fomes fomentarius* ist ein typischer Wundenparasit. Die Infizierung erfolgt durch abiotischen oder biotischen

Faktoren verursachten Wunden. Vornehmlich im weichen Laubholz und in der Buche geht die Zerstörung schnell vor sich und die angegriffenen Bäume werden sehr oft ein Opfer des Windbruchs. Auf den liegenden Stämmen sowie auf Stöcken lebt der Schwamm noch lange weiter. Dieser Porling verursacht die Weissfäule, kann aber auch zur Falschkernbildung führen und ist daher ein bedeutender Schädling der Forstwirtschaft.

2. Fomes roseus (A. et S.) Cooke

Die synonymen Namen: Polyporus roseus (A. et S.) Fr. – *Ungulina rosea* (A. et S.) Pat. – *Fomitopsis rosea* (A. et S.) Karst. – *Polyporus lilacino-roseus* Schulzer, Mpt., p. 763. 1869.

Abbildung: SCHULZER, 1869, p. 763 – ? BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, 55/3 Taf.

Literatur: IGMÁNDY (1958) erwähnt mehrere Fundorte in der Umgebung von Sopron.

Herbarium: Sopron (HE., von mehreren Fundorten; Haracsi, Igmándy, Juharos, Rumszauer, Stubnya).

Vorkommen und phytopathologische Bedeutung: Nach den bisherigen Beobachtungen ist es eine seltene Art im Western Ungarns, vornehmlich in den künstlich begründeten Fichtenbeständen. Im Bestand, im Holzschlag auf Fichten-*(Picea)* Stock, 250–450 m Meereshöhe. Ist Saprophyt, und verursacht Rotfäule. Hat keine forstwirtschaftliche Bedeutung, da er nur selten vorkommt.

3. Fomes marginatus (Fr.) Gillet

(Abb. 3.)

Die synonymen Namen: Polyporus marginatus Fr. – *Placodes marginatus* (Fr.) Quél. – *Ungulina marginata* (Fr.) Pat. – *Fomes ungulatus* (Schaef.) Sacc. – *Polyporus pinicola* (Sw.) Gill. – *Fomitopsis pinicola* (Sw.) Karst. u. a.

Abbildung: SCHULZER, 1869, p. 763. – BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Taf. 55/4.

Literatur: SCHULZER (1857) ohne die Fundorte anzugeben, erwähnt folgende Wirtpflanzen: Fichte u. Tanne, Buche, Birke, Rosskastanie. Auch am verbauten Holz. In seinem Manuskript erwähnt er (1869) Nyárád und Mohács als Fundorte. In diesem, aber auch in seinen späteren Veröffentlichungen betont er (1878b), dass die von den Mykologen seines Zeitalters für verschiedene Arten gehaltenen *marginatus* und *pinicola* zur selben Art gehören. HAZSLINSZKY (1895), sich auf SCHULZER berufend, veröffentlicht die heimische Fundorte dieser Art. HOLLÓS (1913) fand den Porling zwischen Donau und Theiss bei Pusztavacs, Nyárlőrinc (=Kohárysentlőrinc) und in der Gemarkung von Kecskemét; in Transdanubien bei Szekszárd (1933). PILÁT (1936/42) erwähnt ein Vorkommen im Bergwerk von Tatabánya auf gezimmertem Holz (gesammelt von TILES). Diesen Fundort erwähnt auch MOESZ (1941). Fundorte erwähnt noch MOESZ (1942) in der Umgebung von Budapest; IGMÁNDY (1958) gibt viele Orte bei Sopron an.

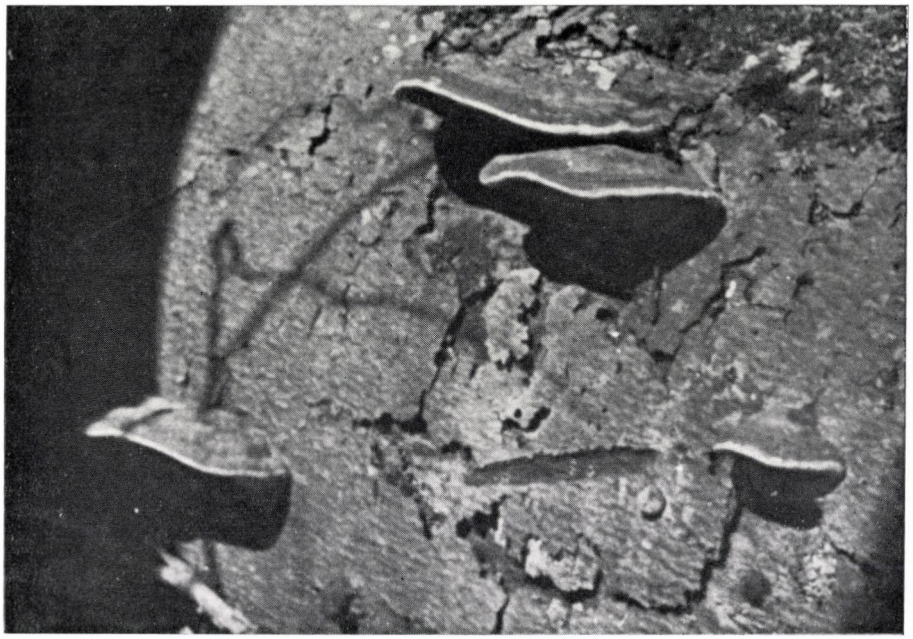


Abb. 3. *Fomes marginatus* (Fr.) Gillet an *Fagus*. Szilvásvárad

Herbarium: Alsógöd (HMN., Hambalkó) – Bakonyszombathely (HE., Stubnya) – Bikács (HE., Igmándy) – Budapest (HMN., Bohus, Szemere u. a.) – Diósjenő (HE., Rumszauer) – Fenyőfő (HMN., Filarszky) – Gödöllő (HE., Igmándy) – Kecskemét (HMN., Hollós) – Kunfehértó (HE., Igmándy) – Pári (HE., Igmándy) – Röjtökmuzsaj (HE., Igmándy) – Sitke (HE., Haracsi – Igmándy) – Sopron (HE., HMN., Bohus, Igmándy u. a.) – Szekszárd (HMN., Hollós) – Szilvásvárad (HE., Igmándy) – Visegrád (HE., Igmándy – Pagony).

Vorkommen und phytopathologische Bedeutung: Von der Tiefebene bis in die höchstgelegenen Teile des Landes. In den Nadelwaldungen der Höhenlagen, vornehmlich in den Fichtenbeständen, in alten Buchenwäldern ist er nicht selten, kommt aber sonst sporadisch vor. Im Bestand und im Schlage, in Parkanlagen und Obstkulturen, auf Nadel- *(Picea, Abies, Pinus)* und Laub- *(Fagus, Betula, Tilia, Alnus, Carpinus, Populus, Prunus)* stöcken, auf liegenden, eingegangenen Bäumen, hauptsächlich auf Fichten und Buchen. Seltener als Wundenparasit auf Obstbäumen, Pappelarten, und anderen Laubbäumen *(Prunus mahaleb* L., *P. armeniaca* L., *P. avium* L., *Pirus sativa* Lam. et DC., *Populus canescens* Sm., *P. nigra* L., *Ailanthus)*. Der Schwamm verursacht Rotfäule. Der durch den vor allem saprophytischen Schwamm verursachte Schaden ist unbedeutend. Eingeschlagenes Holz wird nur dann befallen, wenn es längere Zeit im Walde gelagert



Abb. 4. *Fomes annosus* (Fr.) Cooke an *Picea*-Wurzeln. Sopron

wird. Mit dem wirksamen Abbau des zurückgelassenen Holzes (hauptsächlich Fichten- u. Tannenstöcke) fördert dieser Porling den Rohstoffkreislauf.

4. Fomes annosus (Fr.) Cooke

(Abb. 4)

Die synonymen Namen: Polyporus annosus Fries. – *Fomitopsis annosa* (Fr.) Karst. – *Ungulina annosa* (Fr.) Pat. – *Polyporus cryptarum* (Bull.) Fr. – *Fomes cryptarum* (Bull.) Cooke – *Trametes radiciperda* Hartig. u. a.

Abbildung: Győrfi, 1943/44, Abb. 7, 8 u. 10. – Ubrizsy, 1952, Taf. XXXV. – Bánhegyi, Bohus, Kalmár, Ubrizsy, 1953, Taf. 56/1. – Győrfi, 1963, Abb. 75 u. 76. – Ubrizsy, 1965. II, p. 362. – Haracsi, 1966, B., p. 144.

Literatur: Pilát (1936/42) erwähnt Várpalota als Fundort (gesammelt durch Moesz). Moesz (1941) veröffentlicht ausser Várpalota auch Tokod, wo er den Schwamm auf Grubenholz fand. Győrfi (1943/44) fand ihn auf Fichte in Sopron. Haracsi (1944), Igmándy (1953) schreiben über den in den Soproner Fichtenbeständen verursachten Schaden. Igmándy (1958) zeichnet die Soproner Fundorte auf.

Herbarium: Alsóújlak (HE., Igmándy–Pagony) – Bakonyszentlászló (HE., Haracsi–Igmándy) – Bak (HE., Igmándy) – Baktalórántháza (HE., Igmándy)

– Bélapátfalva (HE., Igmándy) – Budapest (HE., Igmándy) – Csákánydoroszló (HE., Igmándy) – Cserénfa (HE., Igmándy) – Daraboshegy (HE., Igmándy) – Isaszeg (HMN., Visnya) – Miskolc: Jávorkút (HE., Igmándy) – Nádasd (HE., Igmándy–Pagony) – Pornóapáti (HE., Győrfi) – Réde (HE., Igmándy) – Röjtökmuzsaj (HE., Pagony) – Sopron (HE., Haracsi, Igmándy, Pagony, u. a.) – Szakonyfalu (HE., Igmándy) – Szalafő (HE., Igmándy–Varga) – Szentpéterfa (HE., Igmándy) – Tokod (HMN., Holl, Tiles) – Várpalota (HMN., Holl, Faller) – Zagyvaróna (HE., Igmándy).

Vorkommen und phytopathologische Bedeutung: Vom Tiefland bis in das Gebirge anzutreffen, wo Nadelholz stockt. In den Nadelholzkulturen ist er nicht selten. Befällt Fichten (*Picea excelsa* Link.), Weiss- u. Schwarzföhren (*Pinus silvestris* L., *P. nigra* Arn.), Tannen (*Abies alba* Mill.) usw., seltener Laubholz (*Betula, Tilia*), und Sträucher (*Ribes*). Der gefürchteste Schädling der ungarischen Nadelholzbestände – hauptsächlich der Fichten –, aber auch in den an Ungarn angrenzenden Ländern. Der Pilz ist ein kernzerstörender Wundparasit, infiziert durch an den Wurzeln, oder am Wurzelstock erlittenen Verletzungen. Die Fäule, die von den Wunden ausgeht und mehrere Meter weit emporsteigt, verursacht bei Fichten und Tannen in älteren Beständen einen grossen technischen Schaden. Noch grösser ist der Schaden, den der Pilz durch die Zersetzung der Wurzeln verursacht. Die wurzelkranken Bäume werden oft vom Wind geworfen, der Bestand wird dadurch gelichtet, es entstehen Lücken, und er verliert die Standfestigkeit. Den Anfall kann man mit Gewissheit feststellen, da sich der Wurzelkopf des Baumes kegelförmig verdickt, dann tritt starker Harzfluss auf. Im vorgeschrittenen Stadium ist die Schädigung an den Spechtlöchern, und an den Ameisenhaufen beim Stock zu erkennen. Das befallene Holz zeigt erst eine violette Verfärbung. Der Schwamm verursacht Weissfäule. Den grössten Schaden verursacht der Schwamm dort, wo die Wurzeln der Fichte, aber auch anderer Nadelbäume wegen ungünstiger Standortsverhältnisse absterben (Bodentrockenheit, oder Nässe) und dem Schwamm den Eintritt ermöglichen (Haracsi, 1966). Die Fruchtkörper des *Fomes annosus* erscheinen in der Regel im hohlen Stock, an den Wurzeln aber erst nach dem Tode des Baumes. Die Wurzeln des durch den Wind geworfenen Baumes werden oft durch die flachen krustenartigten Fruchtkörper ganz überzogen.

5. *Fomes fraxineus (Fr.) Cooke*

(Abb. 5)

Die synonymen Namen: Polyporus fraxineus Fr. – *Ungulina fraxinea* (Fr.) B. et G. – *Polyporus cystisinus* (Berk.) Bond. et Sing. u. a.

Abbildung: Moesz, 1942, p. 425. – Igmándy, 1961, Abb. 1, 6. – Igmándy, 1963, p. 386. – Igmándy, 1965b, p. 590. – Ubrizsy, 1965, II. p. 361.

Literatur: Nach Moesz (1942) fand Lohwag den Pilz in Budapest (Margareteninsel), auf dem Wurzelstock eines Aesculus-Stammes. – Igmándy (1958) erwähnt zwei Fundorte bei Sopron und Umgebung. In späteren Veröffentlichun-



Abb. 5. *Fomes fraxineus* (Fr.) Cooke am Wurzelkopf der *Robinie*. Hetes

gen desselben Verfassers (1961, 1963, 1965b) werden Schadenerregungen des Pilzes in ungarischen Robinienbeständen beschrieben.

Herbarium: Baja (HE., Igmándy) – Bakonyszentlászló (HE., Igmándy) – Baktalórántháza (HE., Igmándy) – Budapest (HMN., Mágócsy) – Bugac (HE., Vörös) – Daraboshegy (HE., Igmándy) – Hercegszántó (HE., Igmándy) – Hetes (HE., Varga) – Ikervár (HE., Igmándy) – Kajdacs (HMN., Hollós) – Kám (HE., Varga) – Kecskemét (HMN., Hollós) – Nagycenk (HE., Haracsi – Igmándy) – Nagykanizsa (HE., Turny) – Pamuk (HMN., Szemere) – Pörböly (HE., Pagony) – Pusztavacs (HE., Stubnya) – Réde (HE., Igmándy) – Röjtökmuzsaj (HE., Haracsi) – Sopron (HE., Igmándy) – Sümeg (HMN., Gammel).

Vorkommen und phytopathologische Bedeutung: Der Schwamm ist in Ungarn im Flachland – aber auch im Hügelland verbreitet angepflanzten Robinienwaldungen und Alleen überall anzutreffen. Selten auf anderen Laubbäumen (*Aesculus*, *Gleditschia*, *? Malus*, *Populus canescens* Sm.). Die Fruchtkörper bilden sich nur selten an den befallenen Stämmen. Hauptsächlich findet man den Fruchtkörper an Stubben, und am Wurzelkopf alter Bäume. Der Porling infiziert durch die am Wurzelkopf und an den Wurzeln entstandenen Wunden. Die Fäulnis breitet sich vom Wurzelkopf der alten und überhaltenen Bäume aus und verursacht einen grossen Schaden. In jungen und mittelalten Beständen dringt die

Fäulnis nicht hoch in den Stamm hinauf, sondern beschränkt sich nur auf einen Durchmesser von 2—5 cm um das Mark des Baumes.

Der Schwamm verursacht Weissfäule mit einem radialen und ringförmigen Verlauf. Radial, da die Markstrahlen der Fäule ± widerstehen, und ringförmig, da der Schwamm das Frühjahrsholz der Jahrringe leichter zermodert und da sich daher das Holz an den Jahrringen spaltet (IGMÁNDY, 1961, 1963, 1965b).

Anmerkung: Der unter dem Namen *Fomes ulmarius* (Sow.) Sacc. beschriebene Pilz (Synonym *Polyporus ulmarius* (Sow.) Fr. — *Ungulina ulmaria* (Sow.) Pat. — *Fomitopsis ulmaria* (Sow.) Bond. et Sing. u. a.) ist bei uns noch nicht vorgekommen.

Ganoderma Karst. em. Pat.

Der Fruchtkörper ist, mit oder ohne Stiel, 0,1—1,5 mm dick, im Querschnitt mit einer rostbraun — schwarzbraunen Rinde überzogen, oft lackartig glänzend; Trama zunderartig-korkig, von blasskrem bis rotbraun; Farbe der Röhrenschicht bräunlich, ein- oder mehrschichtig; Öffnung der Röhren winzig, ± kreisförmig; im Hymenium keine Stacheln; Sporen ovoid, charakteristisch ist die glatte, hyaline äussere (Epispor), und die warzige innere (Endospor) Haut; Sporenstaub gelbbräunlich. Einjährige oder perennierende Arten. In Ungarn kommen 5 Arten vor.

Schlüssel zur Bestimmung der Arten

1a Die lackartige, oder harzige Rinde des Fruchtkörpers zerfliesst auf die Einwirkung von Azeton, Benzol und Chloroform, und bekommt einen dauerhaften, lackartigen glänzenden Überzug. 2

1b Die Rinde des Fruchtkörpers ändert sich auf die Einwirkung von Azeton, Benzol und Chloroform nicht 4

2a Die Rinde des Fruchtkörpers ist sehr dünn (0,1—0,2 mm), besteht aus nebeneinander stehenden keulenförmig verdickten Hyphen (Abb. 6); Trama krem bis hellbraun; Röhren einschichtig; Fruchtkörper einjährig, mit oder ohne Stiel; Sporen $7-10 \times 5-7\ \mu$ 3

2b Die Rinde des Fruchtkörpers ist dicker (0,3—0,6 mm), besteht aus einem unregelmässigen Hyphengewebe; Trama rostbraun, Röhren geschichtet; Fruchtkörper ohne Stiel, perennierend; Sporen $10,0-11,5 \times 7,0-8,5\ \mu$:
3. G. cupreolaccatum

3a Auf dem Stiel, der mit einer lackartigen Haut überzogen ist, sitzt der Hut seitlich. Sporen $7,0-10,5 \times 5,0-7,0\ \mu$. Fruchtkörper am Boden oder auf Stubben von Laubbäumen, selten auf lebenden Bäumen:
1. G. lucidum

3b Der Fruchtkörper ist gross, hat einen Durchmesser von 10—40 cm, ohne Stiel, konsolenartig; Sporen $7,0-10,5 \times 5,0-7,0\ \mu$. Fruchtkörper am Wurzelkopf von Laubbäumen als Parasit, oder auf Stubben:.
2. G. resinaceum

4a Fruchtkörper meistens auffallend flach; ist mit einer 0,2 – 0,5 mm dicken Kruste überzogen, die sich mit dem Nagel eindrücken lässt; Trama meistens dünn, gelblichbraun-braun; Sporen 6,0 – 8,0 × 4,0 – 7,0 μ:

4. G. applanatum



Abb. 6. Hyphen in den Rinden von *Ganoderma lucidum* (Leyss.) Karst

4b Fruchtkörper regelmässig gewölbt, ist mit einer 0,5 – 2,0 mm dicken Kruste überzogen, die sich mit dem Nagel nicht, oder nur schwer eindrücken lässt; Trama dick, dunkel rostbraun – schokoladebraun; Sporen 9,0 – 12,0 × 7,0 – 8,5 μ:

5. G. linhartii

1. Ganoderma lucidum (Leyss.) Karst.

(Abb. 6, 7)

Die synonymen Namen: Boletus lucidus Leyss. – *Polyporus lucidus* (Leyss.) Fr. – *Boletus laccatus* Timm. – *Ganoderma laccatum* (Timm.) Pat. u. a.

Abbildung: SCHULZER, 1869, p. 789. — BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Tafel 54/5.

Literatur: SCHULZER (1857, 1869) erwähnt diesen als eine, im ganzen Lande überall vorkommende Art. HAZSLINSZKY (1895) schreibt folgendes: »Wächst in Nadel-, sowie in Laubwäldern, am abwechslungsreichsten auf der Tiefebene

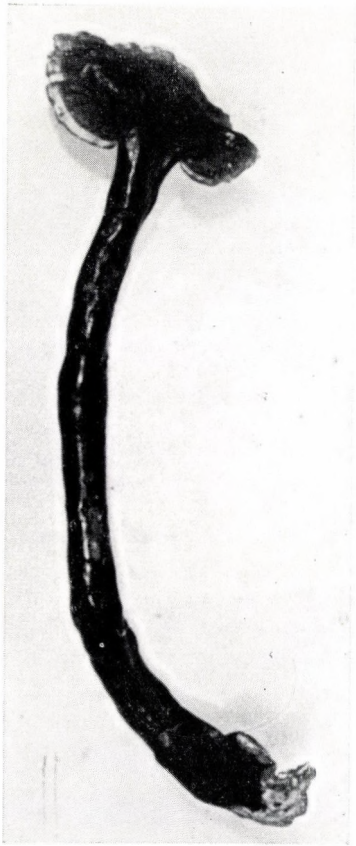


Abb. 7. *Ganoderma lucidum* (Leyss.) Karst. Sopron

östlich der Theiss, wo dieser immer aus der Erde (auf moderndem Holz) wächst . . « Er unterscheidet drei Abarten: 1. ohne Stiel, 2. ohne Hut, und 3. verzweigend. Diese letzte Form hat er in den Wäldern bei Debrecen und Nyíregyháza beobachtet. HOLLÓS (1913, 1933) hat zwischen der Donau und der Theiss folgende Fundorte aufgemerkt: Cegléd, Kecskemét, Kunszentmárton, Nagykőrös, Nyáregyháza, Nyárlőrinc (= Kohányszentlőrinc), Pusztavacs. An diesen Orten ist der Pilz in Eichenwäldern gemein. In Transdanubien wurde der Schwamm bei Szekszárd

gefunden. MOESZ (1942) erwähnt in der Gemarkung von Budapest viele Fundorte. Nach UBRIZSY (1942, 1953, 1965) kommt dieser Pilz in den Eichenbeständen der Nyírség massenhaft vor und schädigt auf den Wurzeln. IGMÁNDY (1958) erwähnt ihn als eine nicht seltene Art, da er bei Sopron oft massenhaft vorkommt.

Herbarium: Da dieser Schwamm sehr verbreitet und nicht selten ist, kann man vom Aufzählen der Fundorte Abstand nehmen.

Vorkommen und phytopathologische Bedeutung: Diese Art kommt in ganz Ungarn vor. Häufig in den Eichen- und Zerreichenwäldern der Tiefebene, des Hügellandes und der Vorgebirge. Manchmal in Kreisen (Hexenringen), mit grosser Stückzahl je m^2. Der Schwamm wächst meistens auf der Humusschicht der Bodenoberfläche, auf erdbedecktem Holz (Äste, Stöcke), aber auch auf Stöcken, seltener auf absterbenden Stämmen *(Salix)*. Verursacht Weissfäule. Diese Art ist saprophytisch; in Ungarn wurde dieser Schwamm vom Verfasser auf Holzteilen der Eiche, Zerreiche und der Weide, aber auch der Weissbuche, Platane, des eschenblättrigen Ahorns (*Acer negundo* L.) beobachtet. Obwohl dieser Schwamm kein seltener ist, ist seine Lebensweise noch wenig bekannt und in vielen Beziehungen sogar umstritten (Parasitismus usw.).

2. *Ganoderma resinaceum Boud.*

(Abb. 8)

Die synonymen Namen: Ganoderma lucidum (Leys.) Karst. *var. resinaceum* Boud. u. a.

Literatur: IGMÁNDY (1958) erwähnt drei Fundorte bei Sopron.

Herbarium: Bejcgyertyános (HE., Igmándy) – Budapest (HE., Szőke) – Karancsalja (HE., Igmándy) – Kazár (HE., Igmándy) – Keszthely (HE., Koronky) – Őriszentpéter (HE., Igmándy – Varga) – Pécs (HMN., Reuter) – Pörböly (HE., Pagony) – Sitke (HE., Igmándy) – Sopron (HE., Győry, Igmándy, Szappanos, Varga).

Vorkommen und phytopathologische Bedeutung: nach den bisherigen Feststellungen kommt der Schwamm hauptsächlich in Eichenwäldern vor. Man kann ihn in den Stieleichenbeständen des Tieflandes, ebenso in den Zerr- und Traubeneichenbeständen des Hügellandes, sowie der Gebirge auffinden. In alten überhaltenen Beständen ist er meistens auch beobachtet worden, wo er als Wurzelkopfvermoderer von Bedeutung ist. Man findet ihn auch im Kahlschlag auf Stöcken. Ausser der Stiel-, Trauben- und Zerreiche, ist sein Vorkommen bisher am Wurzelkopf und Stamm von *Robinie*, *Salix* und *Pirus sativa* Sam. et DC. mit Gewissheit festgestellt worden.

Früher hat man den Pilz für eine Unterart von *Ganoderma lucidum* Karst. gehalten. Morphologisch ist er diesem sehr ähnlich, besonders dann, wenn der Vergleich mit Exemplaren aus Herbarien vollzogen wird. Gross ist aber der Unterschied ihrer Lebensweise. Solange *lucidum* meistens als Saprophyt auf abgestorbener organischer Masse lebt, ist *resinaceum* ein typischer Wundenparasit, der nach



Abb. 8. *Ganoderma resinaceum* Boud. an *Salix*. Keszthely

dem Absterben des Holzes und seiner Nutzbarmachung auf dem zurückgelassenen Stubben als Saprophyt weiter lebt, usw. Erregt Weissfäule.

3. Ganoderma cupreolaccatum Kalchbr. n. c.

Die synonymen Namen: Polyporus laccatus Kalchbr. – *Ganoderma laccatum* (Kalchbr.) B. et G. – *Fomes laccatus* (Kalchbr.) Lloyd – *Ganoderma Pfeifferi* Bres. – *Polyporus cupreolaccatus* Kalchbrenner, in Wettstein, über einem neuen Polyporus., Öst. Bot. Zeitschrft., 1885. u. a.

Bemerkungen zu der Nomenklatur dieser Art: WETTSTEIN (1885) schreibt in einer Abhandlung über diesen Pilz: »*. . . . im Jahre* 1882 *. . . . . c. Kalchbrenner . . . . . den Pilz als neu erkannte und ihn in einem Briefe unter dem Namen Polyporus cupreolaccatus als neue Art aufstellte.*« WETTSTEIN hat später mit KALCHBRENNER's Einwilligung die Benennung auf *laccatus* vereinfacht. Die Priorität gebührt auf alle Fälle der Benennung *laccatus* oder *cupreolaccatus* gegenüber des *Pfeifferi*, mit welchem Namen *Patouillard* (nach BRESADOLA) erst 1889 den Schwamm beschrieb. In der neueren Literatur bedient man sich der Benennung *Pfeifferi* (DONK, 1933, PILÁT 1936–42, BONDARCEV, 1953, KREISEL 1961, JAHN, 1963. u. a.). Die Benennung *laccatus* wurde durch einigen Verfassern vermutlich deshalb abgelehnt, weil eine *Ganoderma*-Art – nämlich *lucidum* – schon früher

als *laccatum* bezeichnet wurde (*Polyporus laccatus* [Timm.], Persoon in Myc. Eur., 1825). Nach dem Grundsatz der Priorität, und da die Benennung auf ein charakteristisches Merkmal hinweist, hält Verfasser die Benennung *cupreolaccatum* als gerechtfertigt.

Literatur: Nach der Verlautbarung von MOESZ (1942) hat SCHILBERSZKY den Schwamm in Nagytétény auf einem Pfirsichbaum gesammelt (*Prunus persica* [L.] Stok.). Das Herbariumexemplar konnte vom Verfasser im Nationalmuseum nicht vorgefunden werden.

Herbarium: Erdősmecske (HE., Vaday) – Pusztavám (HE., Haracsi).

Vorkommen und phytopathologische Bedeutung: Wie in ganz Europa, so ist auch bei uns diese Art sehr selten. HARACSI sammelte den Pilz auf einem Ahornstock (*Acer*) in einem Buchenbestand.

4. Ganoderma applanatum (Pers. ex Wallr.) Pat.

(Abb. 9)

Die synonymen Namen: Polyporus applanatus Pers. ex Wallr. – *Fomes applanatus* (Pers. ex Wallr.) Gill. – *Elfvingia applanata* (Pers. ex Wallr.) Karst. – *Polyporus vegetus* Fr. u. a.

Abbildung: SCHULZER, 1869, p. 755. – BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Tafel 55/1. – IGMÁNDY, 1964. p. 102. – UBRIZSY, 1965, II. p. 379. – HARACSI, 1966, B. p. 146.

Literatur: SCHULZER (1857) charakterisiert das Vorkommen mit einem Wort »überall.« Als Wirtpflanze nennt er folgende Bäume: Buche, Erle, Linde, Rosskastanie, Föhre und Eiche. Auf der letzteren ist der Pilz aussergewöhnlich häufig. In seinem Manuscript (1869) gibt SCHULZER mehrere Fundorte im Komitat Baranya an (Székelyszabar, Bellye, usw.). Später (1880) beschreibt er die Bildung der Konidiensporen dieser Art. [Die geschlechtslose (asexuelle) Sporenbildung dieser Art bezweifelt OVERHOLTS (1953, p. 100–101.)]. HAZSLINSZKY (1895), sich auf SCHULZER berufend, beschreibt ebenfalls das Vorkommen dieser Art in Ungarn. Seine Fundortangabe in bezug auf das Komitat Szabolcs ist unrichtig! HOLLÓS (1913) fand den Schwamm zwischen der Donau und der Theiss auf Eichen und Weiden (Kecskemét, Nagykőrös, Pusztavacs), und in der Gegend von Szekszárd (1933) auch auf der Robinie. SZEMERE (1930) sammelte den Pilz in Sopron, BOHUS (1939) im Vértes-Gebirge, UBRIZSY (1941, 1942) in Debrecen auf Pappel und Eiche, in Bátorliget (1953) ebenfalls auf Stieleiche. MOESZ (1942) erwähnt bei Budapest zwei, IGMÁNDY (1958) bei Sopron viele Fundorte.

Herbarium: Bélapátfalva (HE., Igmándy) – Budapest (HMN., Pénzes) – Diósjenő (HE., Rumszauer) – Gödöllő (HE., Véssey) – Karancsalja (HE., Ig nándy) – Kecskemét (HMN., Hollós) – Körmend (HE., Igmándy–Pagony) – Marcaltő (HE., Csötönyi) – Mátraháza (HMN., Moesz) – Miskolc, Lillafüred (HE., Igmándy) – Neszmély (HE., Haracsi) – Nagyvisnyó (HE., Igmándy) – Pusztavacs (HE., Balázs) – Sopron (HE., Deák, Igmándy) – Szekszárd

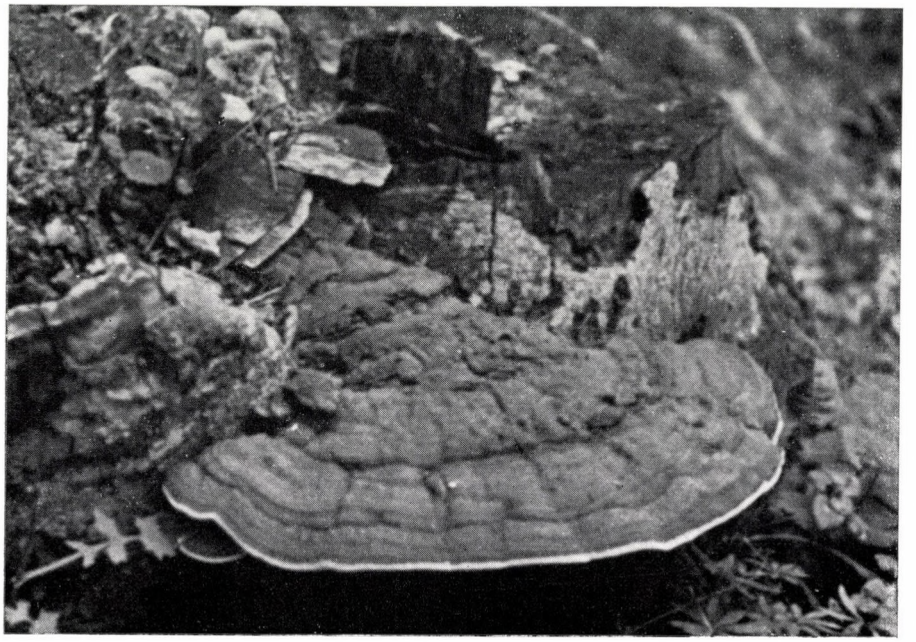


Abb. 9. *Ganoderma applanatum* (Pers. ex Wallr.) Pat. auf einem Buchenstock. Szilvásvárad

(HMN., Hollós) – Szilvásvárad (HE., Győry, Igmándy) – Vértes-hegység (HMN., Bohus) – Zagyvaróna (HE., Igmándy).

Vorkommen und phytopathologische Bedeutung: Man trifft diese Art in der Tiefebene, aber auch in den Buchenwäldern des Gebirges überall an. Im Walde, in Parkanlagen sowie in Alleen. Nach den bisherigen Beobachtungen lebt diese Art auf *Acer*, *Aesculus*, *Alnus*, *Carpinus*, *Fagus*, *Prunus*, *Quercus*, *Robinia*, *Salix* und *Tilia*. Meistens auf *Fagus* und *Salix*. Das Vorkommen ist sporadisch, in alten überhaltenen Buchenbeständen ist sie häufig. Den Fruchtkörper findet man meistens auf Stöcken und auf liegenden Bäumen, seltener auf verletzten, schon eingehenden Stämmen. Diese Art lebt hauptsächlich saprophytisch, und nur selten parasitisch auf einem kränkelnden Baum. In diesem Fall erfolgt die Infektion am Stock des Stammes und verursacht Stockfäule. Verursacht Weissfäule. Hat nur eine geringe wirtschaftliche Bedeutung.

Die früheren mykologischen und pflanzenpathologischen Werke stuften auch die folgende Art hierher ein, obwohl sich diese nicht nur morphologisch, sondern nach den bisherigen – zwar spärlichen – Beobachtungen auch in der Lebens- und Schädigungsweise von jener unterscheidet.



Abb. 10. *Ganoderma linhartii* (Kalchbr.) n. c. an *Quercus robur*. Sitke

5. Ganoderma linhartii (Kalchbr.) n. c.

(Abb. 10)

Die synonymen Namen: ? Polyporus australis Fr. – *Polyporus australis* Fr. in Kalchbrenner, Diagnosen etc. Verh. zool. -bot. Ges., 1868. – *Polyporus adspersus* Schulzer, Mycologisches, Flora, 1878a. – *Polyporus (fomentarius) adspersus* Schulz., in Linhart, Fungi hungarici. Nr. 55. 1884. – *Polyporus (Placoderm.) Linharti* Kalchbrenner, in Linhart, Fungi hungarici. Nr. 252., 1884. – *Ganoderma europaeum* Steyaert u. a.

Bemerkungen zu der Nomenklatur dieser Art: FRIES beschrieb unter dem Namen *Polyporus australis* eine aus dem südpazifischen Raum stammende Art. Dieselbe Benennung hatte er später auf eine ähnliche, aber aus Europa stammende *Ganoderma*-Art angewendet (siehe KALCHBRENNER, 1868; STEYAERT 1961). KALCHBRENNER beschrieb diese Art (1868) nach FRIES's Meinungsäusserung unter der Benennung *Polyporus australis*, später aber (1884, bei LINHART) als eine neue Art und nannte sie *Polyporus (Placoderm.) Linharti*. Schon im Jahre 1878 schrieb SCHULZER über diesen Pilz, und gab ihm den Namen »*adspersus*«, gab aber keine bestimmte Diagnose. Man kann zweifellos feststellen, dass der in der Reihe Fungi hungarici unter Nummer 55 angeführte *Polyporus (fomentarius) adspersus* Schulz, mit dieser Art identisch ist. Im Jahre 1961 beschrieb *Steyaert* den besprochenen Pilz als eine neue Art *(europaeum)*.

Literatur: KALCHBRENNER (1884 bei LINHART) beschrieb den Pilz nach einem von LINHART bei Mosonmagyaróvár auf *Populus nigra* gesammelten Exemplar.

Herbarium: Balatonendréd (HMN., Mágocsy) – Bősárkány (HE., Igmándy – Varga) – Gyöngyössolymos (HE., Nagy) – Hőgyész (HE., Igmándy) – Lövő (HE., Igmándy) – Mosonmagyaróvár (HMN., Linhart, *typus*) – Nagylózs (HE., Igmándy) – Pölöske (HE., Igmándy) – Pörböly (HE., Igmándy) – Sárvár (HE., Igmándy – Varga) – Sitke (HE., Igmándy – Varga) – Szekszárd (HMN., Hollós).

Vorkommen und phytopathologische Bedeutung: Nach den bisherigen Beobachtungen und Feststellungen kommt diese Art hauptsächlich im Tiefland und im Hügelland vor. Die Wirtpflanzen sind folgende: *Fagus*, *Gleditschia*, *Morus*, *Populus* (*nigra?; alba* L.), *Quercus* (*borealis* Mich,; *robur* L.; *sessiliflora* Salisb.). Lebt vorwiegend als Wundenparasit auf lebenden Bäumen im Walde, im Schlag, in Parkanlagen und Baumreihen, aber auch auf Stöcken und verbauten (Zaun-) Holz. Nach den bisherigen in- und ausländischen Feststellungen (JAHN, 1963) ist diese Art hauptsächlich ein Wundenparasit, lebt aber weiter auf dem liegenden Stamm und auch am Stock. Zur Klärung der Lebensweise, Verbreitung, Schädigung und wirtschaftlichen Bedeutung dieser Art sind noch weitere, eingehende Beobachtungen notwendig.

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Review of the Mycoflora of Hungary. Part V.*

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Ordo: Erysiphales

Familia: Erysiphaceae

Genus: Erysiphe DE CANDOLLE

Erysiphe aquilegiae DE CANDOLLE

Syn.: Erysiphe communis WALLR. ppe.; E. polygoni DC. emend. SALMON ppe. BOLLA 1. p. 53.; FUSS 1. VIII. p. 234., XVI. p. 25.; KALCHBRENNER 1. No. 543.; HAZSLINSZKY 1. p. 187.; FUSS 2. p. 462.; BÄUMLER 1. I. No. 309.; RICHTER 1.; BRESADOLA 1. No. 157.; BÄUMLER 2. III. No. 14.; MOESZ 12. col. 799.; GRESCHIK, HAZSLINSZKY, KMET, LINHART, MOESZ ap. MOESZ 16. p. 20.; UBRIZSY 2. p. 152.; TÓTH 2. p. 55.; UBRIZSY–VÖRÖS 1. p. 149.; CSORBA in UBRIZSY 5. II. p. 186.; HIRATA 1. p. 104., 105.

Erysiphe artemisiae (WALLR.) GREVILLE

Syn.: Erysiphe cichoracearum DC. ppe.; E. linkii LÉV. ppe.
KALCHBRENNER 1. No. 546.; HAZSLINSZKY 1. p. 184.; HAZSLINSZKY 2. p. 21.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. No. 312.; RICHTER 1.; BRESADOLA 1. No. 154.; BÄUMLER 2. III. No. 17.; SACCARDO, D. 1. No. 143.; HOLLÓS 1. p. 96.; HOLLÓS 7. p. 118.; BÄUMLER, GRESCHIK, HAZSLINSZKY, HOLLÓS, HOLUBY, KALCHBRENNER, KMET, MÁGOCSY-DIETZ, MOESZ, RICHTER, TAUSCHER ap. MOESZ 16. p. 21.; MOESZ, TAUSCHER ap. MOESZ 13. p. 53.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; UBRIZSY 3. p. 3.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 55.; VASS–TÓTH 2. p. 46.; SZIGETI–TÓTH 1. p. 17.; UBRIZSY–VÖRÖS 1. p. 150.; CSORBA in UBRIZSY 5. II. p. 186.

Erysiphe cichoracearum DE CANDOLLE emend. SALMON

Syn: Alphitomorpha communis var. cichoracearum WALLR.; Erysiphe communis FR. ppe.; E. varium FR. ppe.; E. compositarum DUBY ppe.; E. lamprocarpa (WALLR.) DUBY; E martii LÉV. ppe; Oidium ambrosiae THÜM.

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SCHULZER 2. p. 363.; HAZSLINSZKY 1. p. 184., 187.; HAZSLINSZKY 2. p. 21.; FUSS 2. p. 462.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. No. 308.; BÄUMLER 2. No. 13.; SIMONKAI 1.; MOESZ 2. No. 13.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 214.; PÁTER 1.; MOESZ 7. p. 6.; MOESZ 12. col. 799.; HOLLÓS 7. p. 118.; AUGUSTIN, BÄUMLER, BOROS, BUDAI, DEGEN, GREINICH, GYŐRFFY, KMET, MÁGOCSY-DIETZ, MOESZ, ROZSEMBERSZKY, TAUSCHER ap. MOESZ 16. p. 21.; AUGUSTIN, DEGEN, MOESZ, ROZSEMBERSZKY ap. MOESZ 13. p. 53.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; UBRIZSY 3. p. 3.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 55.; VASS 1. p. 49.; VASS-TÓTH 2. p. 46.; TIMÁR 1. p. 235.; VÖRÖS 9. p. 101.;UBRIZSY—VÖRÖS 1. p. 150.; CSORBA in UBRIZSY 5. II. p. 181.; HIRATA 1. p. 293., 303., 324., 325., 326., 327., 334., 336., 337., 340., 341., 342., 348., 354., 357., 358., 359., 360., 361., 363., 364., 367., 369., 370., 371., 372., 373., 374.

Erysiphe communis (WALLR.) LINK

Syn.: Erysiphe polygoni DC. emend. SALMON ppe.; E. martii LÉV. ppe.
FUSS 1. XVI. p. 25.; KALCHBRENNER 1. No. 543., 544.; HAZSLINSZKY 1. p. 186., 187.; HAZSLINSZKY 2. p. 21.; FUSS 2. p. 461., 462.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. No. 308., 313.; BRESADOLA 1. No. 156.; BÄUMLER 1. IV. p. 22. ad (309); HOLLÓS 1. p. 96.; RAPAICS 1. p. 215.; MOESZ 11. p. 96.; MOESZ 23. col. 819.; MOESZ 27. p. 145.; MOESZ 30.; BÄUMLER, BÁNYAI, BERNÁTSKY, BOROS, BUDAI, GRESCHIK, KALCHBRENNER, LINHART in Fung. hung. exs. No. 458., MÁGOCSY-DIETZ, MOESZ, RICHTER, SIMONKAI, SZEPESFALVY, TUZSON, ZSÁK, WIERZBICZKY ap. MOESZ 16. p. 22.; MÁGOCSY-DIETZ, RICHTER, SZEPESFALVI, TUZSON, ZSÁK ap. MOESZ 13. p. 53.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; UBRIZSY 3. p. 3.; TÓTH 2. p. 55.; VASS 1. p. 49.; TIMÁR 1. p. 235.; SZIGETI—TÓTH 1. p. 17.; TÓTH 10. p. 6.; UBRIZSY—VÖRÖS 1. p. 150.; CSORBA in UBRIZSY 5. II. p. 186.; HIRATA 1. p. 91., 92., 104., 116., 119., 121., 122., 123., 125., 127., 129., 130., 174., 176., 177., 178., 179., 180., 181., 182., 184., 185., 186., 188., 189., 190., 192., 195., 237., 238., 240., 243., 256., 263., 266., 318., 319., 320., 341.

Erysiphe convolvuli DE CANDOLLE

Syn.: Alphitomorpha communis δ convolvulacearum WALLR.; Erysibe communis var. convolvulacearum LINK; Erysiphe communis FR. ppe.; E. communis GREV. f. convolvuli POTEBNIA in JACZ.; E. convolvuli-sepii CAST.; E. polygoni DC. emend. SALMON ppe.
SCHULZER 2. p. 364.; FUSS 1. IV. p. 127.; KALCHBRENNER 1. No. 543.; SCHULZER 4. p. 173.; HAZSLINSZKY 1. p. 187.; HAZSLINSZKY 2. p. 22.; FUSS 2. p. 461.; BÄUMLER 1. No. 313.; RICHTER 1.; BÄUMLER 2. III. No. 14.; BÄUMLER, BUDAI, FILARSZKY, HAYNALD, HAZSLINSZKY, HUSZ, KMET, KÜMMERLE, LÁNYI, MÁGOCSY-DIETZ, MOESZ, SADLER ap. MOESZ 16. p. 24.; FILARSZKY, HUSZ, KÜMMERLE, MOESZ, SADLER ap. MOESZ 13. p. 53.;

UBRIZSY 2. p. 152.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 55.; TIMÁR 1. p. 235.; SZIGETI - TÓTH 1. p. 17.; UBRIZSY—VÖRÖS 1. p. 151.; CSORBA in UBRIZSY 5. II. p. 187.; HIRATA 1. p. 266.

Erysiphe cruchetiana BLUMER

Syn.: Erysiphe communis (WALLR.) LÉV. ppe.; E. communis f. ononidis FUCK.; E polygoni DC. emend. SALM. ppe.
FUSS 2. p. 461.; BÄUMLER 1. No. 309.; BUDAI, MOESZ, ZAHLBRUCKNER ap. MOESZ 16. p. 24.; MOESZ ap. MOESZ 13. p. 53.; TÓTH 2. p. 55.; UBRIZSY—VÖRÖS 1. p. 151.; CSORBA in UBRIZSY 5. II. p. 187.

Erysiphe depressa (WALLR.) SCHLECHTENDAHL

Syn.: Alphitomorpha depressa WALLR.; A. bardanae WALLR.; Erysiphe arctii GREV.; E. cichoracearum DC. emend. SALMON ppe.; E. cichoracearum f. bardanae JACZ.; E. communis m. carduacearum FR. ppe.; E. compositarum α lappae DUBY; E. montagnei LÉV.
BOLLA 1. p. 53.; KALCHBRENNER 1. No. 547.; HAZSLINSZKY 1. p. 186.; HAZSLINSZKY 7. p. 15., 22.; BÄUMLER 1. No. 308.; RICHTER 1.; BRESADOLA 1. No. 155.; HOLLÓS 1. p. 96.; HOLLÓS 7. p. 118.; BUDAI, GRESCHIK, KMET, KÜMMERLE, MÁRKUS, MÁGOCSY-DIETZ, MOESZ, PÓSCH, SCHNELLER, SZEPESFALVY ap. MOESZ 16. p. 24.; MOESZ, KÜMMERLE, SZEPESFALVY ap. MOESZ 13. p. 53.; UBRIZSY 2. p. 152.; TÓTH 2. p. 55.; TÓTH 10. p. 6.; UBRIZSY—VÖRÖS 1. p. 151.; CSORBA in UBRIZSY 5. II. p. 187.; HIRATA 1. p. 324., 363.

Erysiphe fischeri BLUMER

Syn.: Erysiphe cichoracearum DC. emend. SALMON ppe.
HOLLÓS 1. p. 96.; MOESZ 16. p. 25.; UBRIZSY—VÖRÖS 1. p. 152.; CSORBA in UBRIZSY 5. II. p. 187.; HIRATA 1. p. 369.

Erysiphe galeopsidis DE CANDOLLE

Syn.: Alphitomorpha communis var. labiatarum WALLR.; A. lamprocarpa WALLR. ppe.; A. labiatarum WALLR.; Erysiphe communis FR. ppe.; E. communis var. labiatarum LINK; E. communis δ labiatarum (LK.) DUBY; E. labiatarum CHEVALLIER; E. lamprocarpa SCHLECHT; E. lamprocarpa α galeopsidis (LK.) DUBY.
BOLLA 1. p. 53.; FUSS 1. IV. p. 127.; KALCHBRENNER 1. No. 545., 790.; HAZSLINSZKY 1. p. 184., 185.; HAZSLINSZKY 2. p. 21.; HAZSLINSZKY 5. p. 45.; FUSS 2. p. 460., 461.; BÄUMLER 2. No. 15.; BÄUMLER 1. IV. p. 22. (ad 310.); BRESADOLA 1. No. 155., 318.; MOESZ 2. No. 14.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 218.; HOLLÓS 7. p. 118.; BÄUMLER, BOLLA, BUDAI, GRESCHIK, HAZS-

LINSZKY, KALCHBRENNER, KMET, KÜMMERLE, LÁNYI, MÁGOCSY-DIETZ, MOESZ, PÁPAI, SCHNELLER, SZÉPLIGETI, TAUSCHER in Fung. eur. exs. No. 1738. in RABENHORST Fung. eur. exs. No. 1739., 1740., 1741., in Soc. dauphinoise No. 2329., et in THÜMEN Fung. austr. exs. No. 1041, 1042., ZAHLBRUCKNER, ZSÁK ap. MOESZ 16. p. 25.; MOESZ, SZÉPLIGETI, TAUSCHER, ZSÁK ap. MOESZ 13. p. 53.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 55.; VASS 1. p. 49.; TIMÁR 1. p. 235.; UBRIZSY—VÖRÖS 1. p. 152.; CSORBA in UBRIZSY 5. II. p. 187.; HIRATA 1. p. 278., 280., 281., 282., 283., 284., 287., 288., 291.

Erysiphe galii FUCKEL

Syn.: Erysiphe communis (WALLR.) FR. ppe.; E. martii auct.; E. polygoni DC. emend. SALMON ppe.
HOLLÓS 1. p. 96.; HOLLÓS 7. p. 118.; FILARSZKY, KMET ap. MOESZ 16. p. 26.; FILARSZKY ap. MOESZ 13. p. 53.; UBRIZSY—VÖRÖS 1. p. 152.; CSORBA in UBRIZSY 5. II. p. 187.

Erysiphe graminis DE CANDOLLE

Syn.: Alphitomorpha communis var. graminearum WALLR.; Erysibe communis var. graminum LINK; Erysiphe communis FR. ppe.
HAZSLINSZKY 1. p. 185.; FUSS 2. p. 461.; BÄUMLER 1. No. 311.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 215.; HOLLÓS 7. p. 118.; BÄUMLER, BUDAI, FLEISCHMANN, GRESCHIK, HAZSLINSZKY, HUSZ, KEREKES, LÁNYI, LENGYEL, LINHART in Fung. hung. exs. No. 80., MÁRKUS, MOESZ, PÓSCH, ZSÁK ap. MOESZ 16. p. 26; HUSZ, MOESZ, ZSÁK ap. MOESZ 13. p. 53.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; TÓTH 2. p. 55.; UBRIZSY—VÖRÖS 1. p. 152.; CSORBA in UBRIZSY 5. II. p. 178.; HIRATA 1. p. 42., 43., 44., 45., 49., 52., 56., 57., 58., 59., 60., 61., 62.

Erysiphe heraclei DE CANDOLLE

Syn.: Erysiphe angelicae FUSS; E. communis LINK ppe.; E. martii LÉV. ppe.; E. polygoni DC. emend. SALMON ppe.; E. umbelliferarum DE BARY ppe.
FUSS 1. XVI. p. 25.; KALCHBRENNER 1. No. 544.; HAZSLINSZKY 1. p. 186.; HAZSLINSZKY 2. p. 22.; FUSS 2. p. 462.; HAZSLINSZKY 3. p. 89.; BRESADOLA 1. No. 318.; SIMONKAI 1.; SACCARDO, D. 1. No. 144.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 215.; HOLLÓS 7. p. 118., 119.; BÄUMLER, BOLLA, BUDAI, ENDLICHER, FUSS in Herb. Norm. Transs. Cent. VII. No. 602., GRESCHIK, HUSZ, KMET, LAUDON, MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 79., MOESZ, RAPAICS, ZAHLBRUCKNER ap. MOESZ 16. p. 27.; HUSZ, MOESZ ap. MOESZ 13. p. 54.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; UBRIZSY 3. p. 3.; UBRIZSY 6. in SZÉKESSY p. 23.; UBRIZSY—VÖRÖS 1. p. 152.

Erysiphe horridula (WALLR.) LÉVEILLÉ

Syn.: Alphitomorpha cynoglossi WALLR.; A. horridula WALLR. ppe.; Erysiphe asperifoliorum GREV.; E. biocellata EHRENB.; E. cichoracearum DC. emend. SALMON ppe.; communis FR. ppe.; E. polygoni DC. emend. SALMON ppe. KALCHBRENNER 1. No. 542.; HAZSLINSZKY 1. p. 184., 188.; HAZSLINSZKY 2. p. 21.; BÄUMLER 1. No. 308.; BÄUMLER 2. No. 13.; MOESZ 2. No. 15.; HOLLÓS 1. p. 96.; MOESZ 11. p. 96.; MOESZ 31. p. 94.; BÄUMLER, BUDAI, DEGEN, FILARSZKY, GRESCHIK, KÜMMERLE, KMET, MÁGOCSY-DIETZ, MOESZ, SCHNELLER, SZÉPLIGETI ap. MOESZ 16. p. 28.; DEGEN, HAZSLINSZKY, MOESZ, SZÉPLIGETI ap. MOESZ 13. p. 54.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 152.; UBRIZSY 3. p. 3.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 55.; VASS 1. p. 49.; TIMÁR 1. p. 235.; UBRIZSY–VÖRÖS 1. p. 152.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 268., 269., 270., 271., 272., 273., 274., 275.

Erysiphe hyperici (WALLR.) FRIES

Syn.: Alphitomorpha communis var. hypericearum WALLR.; A. hyperici WALLR.; Erysiphe communis f. hyperici BRET. in JACZEWSKI; E. communis var. hypericorum RABENH.; E. communis α hypericearum FR.; E. martii LÉV. ppe.; E. martii f. hypericorum FUCK.; E. polygoni DC. emend. SALM. ppe. KALCHBRENNER 1. No. 544.; HAZSLINSZKY 1. p. 186.; FUSS 2. p. 461.; BOLLA, FUSS in Herb. Norm. Transs. Cent. IX. No. 804., GRESCHIK, KALCHBRENNER, KÜMMERLE ap. MOESZ 16. p. 29.; MOESZ ap. MOESZ 13. p. 54.; TÓTH 2. p. 56.; VASS 1. p. 49.; UBRIZSY–VÖRÖS 1. p. 153.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 117., 118.

Erysiphe labiatarum (WALLR.) CHEVALLIER

Syn.: Alphitomorpha labiatarum WALLR. ppe.; A. lamprocarpa WALLR. ppe.; Erysibe lamprocarpa SCHLECHT. ppe.; Erysiphe cichoracearum DC. emend. SALM. ppe.; E. communis var. labiatarum LINK ppe.; E. communis. r. labiatarum FR. ppe.
KALCHBRENNER 1. No. 545.; BÄUMLER 1. No. 310.; HOLLÓS 1. p. 96.; BLUMER 1. p. 272.; KALCHBRENNER, MOESZ ap. MOESZ 16. p. 29.; UBRIZSY–VÖRÖS 1. p. 153.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 278., 280., 283., 288.

Erysiphe lamprocarpa (WALLR.) DUBY

Syn.: Alphitomorpha lamprocarpa WALLR.; Erysibe circumfusa SCHLECHT.; E. lamprocarpa LINK ppe.; Erysiphe communis FR. ppe.; E. lamprocarpa (LK.) DUBY β plantaginis DUBY
FUSS 1. VIII. p. 233.; KALCHBRENNER 1. No. 545.; HAZSLINSZKY 1. p. 185.; HAZSLINSZKY 2. p. 21.; BÄUMLER 1. No. 308.; FUSS 2. p. 460.; BRESADOLA 1.

No. 155.; HOLLÓS 1. p. 96.; MOESZ 11. p. 96.; HOLLÓS 7. p. 118.; BUDAI, FUSS, GRESCHIK, HOLUBY, LAUDON, MÁGOCSY-DIETZ, MOESZ, PÓSCH, SCHNELLER, ZAHLBRUCKNER ap. MOESZ 16. p. 29.; UBRIZSY 2. p. 152.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 56.; VASS–TÓTH 1. p. 155.; VASS 1. p. 49.; VÖRÖS 9. p. 101.; SZIGETI–TÓTH 1. p. 18.; UBRIZSY–VÖRÖS 1. p. 153.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 311., 312.

Erysiphe martii LÉVEILLÉ s. str. BLUMER

Syn.: Alphitomorpha communis WALLR. ppe.; A. trifoliorum WALLR.; Erysibe communis var. leguminosarum LINK ppe.; Erysiphe polygoni DC. emend. SALM. ppe.; E. polygoni DC. emend. SALM. f. robiniae hispidae NANNIZZI; E. trifolii GREV. et E. lathryri GREV.; Microsphaera caraganae MAGN.; Oidium orbiculare NANNIZZI

FUSS 1. VIII. p. 234., XVI. p. 25.; KALCHBRENNER 1. No. 544.; HAZSLINSZKY 1. p. 186.; FUSS 2. p. 461.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. No. 313.; BÄUMLER 2. III. No. 18.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 215.; MOESZ 27. p. 145.; HOLLÓS 7. p. 118.; BUDAI, DEGEN, ENDLICHER, FUSS in Herb. Norm. Tr. Cent. IV. No. 305., GRESCHIK, HAZSLINSZKY, KMET, MÁGOCSI-DIETZ, MOESZ, RICHTER, ZAHLBRUCKNER ap. MOESZ 16. p. 29.; MOESZ, DEGEN ap. MOESZ 13. p. 54.; UBRIZSY 1. p. 7.; UBRIZSY 2. p. 153.; UBRIZSY 3. p. 3.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 56; VASS–TÓTH 1. p. 155.; VASS 1. p. 49.; TIMÁR 1. p. 235.; UBRIZSY–VÖRÖS 1. p. 153.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 171., 174., 186; TÓTHné 1. p. 172.

Erysiphe mayorii BLUMER

Syn.: Erysiphe communis WALLR. ppe.

HAZSLINSZKY 2. p. 22.; BUDAI, HOLUBY, MOESZ ap. MOESZ 16. p. 30.; UBRIZSY–VÖRÖS 1. p. 153.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 341.

Erysiphe montagnei LÉVEILLÉ

Syn.: Alphitomorpha communis o. compositarum WALLR. ppe.; Erysibe depressa var. carduorum DESM.; Erysiphe cichoracearum DC. emend. SALM. ppe.; E. cichoracearum DC. f. cirsii LASCH.; E. communis m. carduacearum FR. ppe.; E. communis var. cirsii LASCH.; E. compositarum γ cirsii DUBY; E. lamprocarpa LINK f. cirsii FUCKEL

KALCHBRENNER 1. No. 549.; HAZSLINSZKY 1. p. 184.; MOESZ 16. p. 30.; CSORBA in UBRIZSY 5. II. p. 188.; HIRATA 1. p. 324.

Erysiphe nitida (WALLR.) RABENHORST

Syn.: Alphitomorpha communis var. ranunculacearum WALLR. ppe.; A. nitida WALLR.; Erysiphe communis LÉV. ppe.; E. communis α ranunculacearum

Fr. ppe.; E. polygoni DC. emend. Salm. ppe.; E. ranunculi Cast.; E. ranunculi Grev.
Kalchbrenner 1. No. 543.; Hazslinszky 2. p. 22.; Fuss 2. p. 462.; Bäumler 1. No. 309.; Richter 1.; Moesz 12. col. 799.; Bäumler, Fuss in Herb. Norm. Transs. Cent. VI. No. 543.; Greschik, Győrffy, Hazslinszky, Holuby, Husz, Laudon, Linhart in Fung. hung. exs. No. 259., Moesz, Zahlbruckner ap. Moesz 16. p. 30.; Husz ap. Moesz 13. p. 54.; Tóth 2. p. 56.; Vass–Tóth 1. p. 156.; Vass 1. p. 49.; Ubrizsy–Vörös 1. p. 153.; Csorba in Ubrizsy 5. II. p. 188.; Hirata 1. p. 103., 106., 107., 108., 110., 111., 112.

Erysiphe pisi De Candolle

Syn.: Alphitomorpha pisi Wallr. ppe.; Erysiphe communis g. leguminosarum Fr. ppe.; E. macrosporus Martius; E. martii Lév. ppe.; E. polygoni DC. emend. Salm. ppe.
Kalchbrenner 1. No. 544.; Hazslinszky 1. p. 186.; Hazslinszky 2. p. 21.; Fuss 2. p. 461.; Bäumler 1. No. 313.; Richter 1.; Rapaics 1. p. 215.; Hollós 7. p. 118.; Bäumler, Fuss in Herb. Norm. Fl. Transs. No. 305.; Haynald, Kalchbrenner, Laudon, Mágocsy-Dietz, Páter, Sadler ap. Moesz 16. p. 31.; Moesz, Sadler ap. Moesz 13. p. 54.; Ubrizsy 1. p. 7.; Tóth 2. p. 56.; Ubrizsy–Vörös 1. p. 154.; Csorba in Ubrizsy 5. II. p. 189.; Hirata 1. p. 186.

Erysiphe polygoni De Candolle emend. Salmon

Syn.: Alphitomorpha communis β polygonearum Wallr.; Erysiphe communis f. acetosellae Thümen; E. communis f. rumicis Fuckel; E. communis f. polygonarum (Rabenh.) Jacz.; E. polygoni DC. emend. Salm. ppe.
Kalchbrenner 1. No. 543.; Fuss 1. XVI. p. 25.; Fuss 2. p. 461.; Bäumler 1. No. 309.; Richter 1.; Bäumler 2. III. No. 14.; Hollós 1. p. 96.; Hollós 7. p. 118.; Budai, Filarszky, Fuss in Herb. Norm. Transs. Cent. V. No. 405., Hazslinszky, Kmet, Lányi, Moesz, Tuzson in Krypt. exs. Vindob. No. 1427., Zahlbruckner ap. Moesz 16. p. 32.; Filarszky, Moesz, Tuzson, ap. Moesz 13. p. 54.; Ubrizsy 1. p. 7.; Ubrizsy 2. p. 153.; Tóth 2. p. 56.; Timár 1. p. 235.; Ubrizsy–Vörös 1. p. 154.; Csorba in Ubrizsy 5. II. p. 184.; Hirata 1. p. 93., 94., 95., 96.

Erysiphe salviae (Jacz.) Blumer

Syn.: E. galeopsidis DC. ppe.
Bäumler 1. No. 545.; Hollós 7. p. 118.; Kalchbrenner, Moesz ap. Moesz 16. p. 32.; Tóth 2. p. 56.; Ubrizsy–Vörös 1. p. 154.; Csorba in Ubrizsy 5. II. p. 189.; Hirata 1. p. 288., 289.

Erysiphe ulmariae DESMAZIÈRES

MOESZ ap. MOESZ 16. p. 32.; MOESZ ap. MOESZ 13. p. 54.; CSORBA in UBRIZSY 5. II. p. 189.

Erysiphe umbelliferarum DE BARY

Syn.: Erysiphe heraclei DC ppe.; E. communis LINK ppe.; E. martii LÉV ppe.; E. polygoni DC. ppe.
TÓTH 2. p. 56.; VASS 1. p. 49.; VASS – TÓTH 2. p. 46.; SZIGETI – TÓTH 1. p. 18.; TÓTH 10. p. 6.; CSORBA in UBRIZSY 5. II. p. 189.; HIRATA 1. p. 244., 245., 246., 247., 248., 249., 250., 251., 252.

Erysiphe urticae (WALLR.) KLOTZSCH

Syn.: Erysiphe martii LÉV. ppe.
SCHULZER 2. p. 364.; KALCHBRENNER 1. No. 544.; HAZSLINSZKY 1. p. 186.; UBRIZSY 3. p. 3.; UBRIZSY – VÖRÖS 1. p. 154.; CSORBA in UBRIZSY 5. II. p. 190.; HIRATA 1. p. 90.

Erysiphe verbasci (JACZ.) BLUMER

FUSS 1. XVI. p. 25.; FUSS 2. p. 460.; KALCHBRENNER, LÁNYI ap. MOESZ 16. p. 32.; HAZSLINSZKY, MOESZ ap. MOESZ 13. p. 54.; UBRIZSY – VÖRÖS 1. p. 154.; CSORBA in UBRIZSY 5. II. p. 190.; HIRATA 1. p. 303., 304.

Genus: Léveillula ARNAUD

Léveillula taurica (LÉV.) ARNAUD

Syn.: Erysiphe taurica LÉV.; E. depressa RABENH. ppe.; E. picridis CAST.; E. lappae CAST.; E. lanuginosa FUCKEL; E. saxaouli SOROKINE; E. alhagi SOROKINE; E. armata SOROKINE; E. pegani SOROKINE; E. lichenoides TRABUT et. SACC.; E. papilionacearum KOMAROV; E. lanata MAGN.; E. asterisci MAGN.; E. euphorbiae SPESCHNEW; E. acanthophylli SPESCHNEW; E. taurica LÉV. var. audina SPEG.; E. taurica LÉV. var. zygophylli MAIRE; E. ricini SPESCHNEW; Microsphaera bornmülleriana MAGN.; Oidium haplaphylli MAGN.; Oidiopsis sicula SCALIA; Oidiopsis taurica (LÉV.) SALM.; Ovulariopsis cistii JAAP.; Ovulariopsis teucrii JAAP.
HAZSLINSZKY 1. p. 185.; HAZSLINSZKY 2. p. 22.; HAZSLINSZKY 7. p. 15.; BÄUMLER 2. No. 14.; SIMONKAI 1.; SACCARDO, D. 1. No. 145.; HOLLÓS 1. p. 96.; HAZSLINSZKY, KMET, KÜMMERLE, LAUDON, MÁGOCSY-DIETZ in Krypt. exs. Vindob. No. 1426., MOESZ, SADLER ap. MOESZ 16. p. 33.; HAZSLINSZKY, KÜMMERLE, MOESZ, SADLER, ZSÁK ap. MOESZ 13. p. 55.; TÓTH 2. p. 56.; TIMÁR 1. p. 235.; TÓTH 10. p. 7.; UBRIZSY – VÖRÖS 1. p. 154.; CSORBA in UBRIZSY 5. II. p. 175.; HIRATA 1. p. 194., 227., 284., 292., 301., 304., 337.

Genus: Microsphaera LÉVEILLÉ

Microsphaera alphitoides GRIFFON et MAUBLANC

Syn.: Microsphaera abbreviata PECK; M. quercina (SCHW.) BURR.; M. alni (WALLR.) WINT. var. quercina NEGER; M. quercina auct. ppe.; M. abbreviata PECK. emend. SAVULESCU et SANDU-VILLE; Oidium quercina auct.; Oidium quercinum THÜM. var. gemmiparum FERRARIS; Oidium dubium JACZ.; Oidium alphitoides GRIFF. et. MAUBL.
RAPAICS 1. p. 218.; MOESZ 29. p. 36.; MOESZ 31. p. 94.; BÄUMLER, BUDAI, DEGEN, GÁYER, GREINICH, GRESCHIK, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ in Flora Hung. exs. No. 509., PÁTER, RAPAICS, SZILÁDY, VARGA, WAGNER ap. MOESZ 16. p. 33.; DEGEN, MÁGOCSY-DIETZ, MOESZ, SZILÁDY ap. MOESZ 13. p. 55.; UBRIZSY 1. p. 7.; KRENNER 1. p. 63.; PICBAUER 1. p. 4.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 56.; VASS—TÓTH 2. p. 46.; TIMÁR 1. p. 235.; VÖRÖS 9. p. 101.; UBRIZSY—VÖRÖS 1. p. 154.; CSORBA in UBRIZSY 5. II. p. 191.; HIRATA 1. p. 78., 81., 83.

Microsphaera alni (WALLR.) WINTER

Syn.: Alphitomorpha alni WALLR.; A. penicillata var. alni. WALLR.; Erysibe penicillata LK. ppe.; Erysiphe alni TUL.; Microsphaera alni (DC.) WINTER ppe.; Microsphaera alni (WALLR.) WINTER emend. SALM. ppe.
SACCARDO, P. A. 1. p. 117.; BÄUMLER in LINHART Fung. hung. exs. No. 486. et in REHM Ascomyceten No. 848., BOLLA, KALCHBRENNER, KMET ap. MOESZ 16. p. 34.; PICBAUER 2. p. 180.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 72., 73., 215.

Microsphaera berberidis (DC.) LÉVEILLÉ

Syn.: Erysiphe berberidis DC.; Alphitimorpha penicillata γ berberidis WALLR.; Erysibe penicillata var. berberidis LINK.; E. divaricata SCHLECHT.; Erysiphe penicillata var. berberidis FR.; Podosphaera berberidis (LÉV.) QUÉL.
KALCHBRENNER 1. No. 540.; HAZSLINSZKY 1. p. 183.; BÄUMLER 1. No. 302.; HOLLÓS 1. p. 97.; RAPAICS 1. p. 218.; MOESZ 27. p. 145.; MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 258., MOESZ, WAGNER ap. MOESZ 16. p. 34.; MÁGOCSY-DIETZ ap. MOESZ 13. p. 55.; UBRIZSY 1.p. 7.; UBRIZSY 3. p. 3.; TÓTH 2. p. 56.; TIMÁR 1. p. 235.; UBRIZSY—VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 194.; HIRATA 1. p. 114.

Microsphaera betae VANHA

Syn.: Erysiphe polygoni DC.; E. communis (WALLR.) LK. ppe.
UBRIZSY—VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 193.

Microsphaera divaricata (DC.) LÉV.

TÓTH 2. p. 56.; UBRIZSY – VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 217.

Microsphaera friesii LÉVEILLÉ

Syn.: Alphitomorpha penicillata δ rhamni catharticae SCHLECHT.; Erysibe penicillata var. rhamni catharticae LK.; Erysiphe penicillata c. rhamni cathartici FR.; Microsphaera alni (DC.) WINTER ppe.; M. alni (WALLR.) WINTER emend. SALM. ppe.; M. penicillata (WALLR.) LÉV. f. rhamni cathartici JACZ.

BOLLA 1. p. 54.; HAZSLINSZKY 1. p. 182.; RAPAICS 1. p. 218.; KALCHBRENNER ap. MOESZ 16. p. 34.; AUGUSTIN ap MOESZ 13. p. 55.; UBRIZSY 1. p. 7.; UBRIZSY – VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 217.

Microsphaera grossulariae (WALLR.) LÉVEILLÉ

Syn.: Alphitomorpha grossulariae WALLR.; A. penicillata β grossulariae WALLR.; Erysiphe grossulariae (LÉV.) DE BARY; E. penicillata β grossulariae FR.; Erysibe penicillata f. grossulariae LK.; Podosphaera grossulariae (LÉV.) QUÉL.

BOLLA 1. p. 54.; HAZSLINSZKY 1. p. 183.; BÄUMLER 1. No. 1515.; BRESADOLA 1. No. 152.; BÄUMLER 2. III. No. 12.; HOLLÓS 1. p. 97.; GRESCHIK, KMET, MOESZ, PÓSCH ap. MOESZ 16. p. 34.; MOESZ ap. MOESZ 13. p. 55.; UBRIZSY 1. p. 8.; UBRIZSY – VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 193.; HIRATA 1. p. 136.

Microsphaera guarinonii BRIOSI et CAVARA

Syn.: Erysiphe martii auct.; Trichocladia guarinonii (BR. et CAV.) ŠKORIČ; Trichocladia diffusa f. cytisi JACZ.

HAZSLINSZKY 1. p. 186.; SCHNELLER ap. MOESZ 16. p. 34.; CSORBA in UBRIZSY 5. II. p. 195.

Microsphaera hedwigii LÉVEILLÉ

Syn.: Erysiphe penicillata g. viburni lantanae FR.; Calocladia penicillata f. lantanae PASS.; Microsphaera alni (DC.) WINTER ppe.; M. penicillata (WALLR.) LÉV. f. viburni JACZ. ppe.

HAZSLINSZKY 1. p. 182.; BÄUMLER 1. No. 300.; HOLLÓS 7. p. 125.; BÄUMLER, BOLLA, LINHART in Fung. hung. exs. No. 257. ap. MOESZ 16. p. 35.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 316.

Microsphaera lonicerae (DC.) WINTER

Syn.: Alphitomorpha divaricata var. lonicerae SCHLECHT.; Erysibe divaricata var. lonicerae LINK; Erysiphe lonicerae DC.; E. penicillata BERK. ppe.; E. penicillata var. lonicerae FR.; Microsphaera ehrenbergii LÉV.; M. dubyi LÉV.
BOLLA 1. p. 54.; HAZSLINSZKY 1. p. 182., 183.; HAZSLINSZKY 7. p. 14.; BÄUMLER 1. No. 303., 306.; MOESZ 11. p. 97.; BÄUMLER in LINHART Fung. hung. exs. No. 357. et in REHM Ascomyceten No. 847., BOLLA, MOESZ ap. MOESZ 16. p. 35.; UBRIZSY 3. p. 4.; UBRIZSY–VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 313., 314., 315.

Microsphaera magnusii BLUMER

Syn.: Microsphaera alni (WALLR.) LÉV. emend. SALMON var. divaricata WALLR.; Microsphaera lonicerae auct.
BÄUMLER 2. III. No. 11.; BLUMER 1. p. 299.; GRESCHIK, KMET, ap. MOESZ 16. p. 35.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 217., 314., 315.

Microsphaera mougeotti LÉVEILLÉ

Syn.: Erysiphe mougeotti DE BARY; Microsphaera lycii (LASCH.) SACC. et ROUMEG.; Podosphaera mougeotti (LÉV.) QUÉL.
HAZSLINSZKY 1. p. 183.; BÄUMLER 1. No. 307.; BRESADOLA 1. No. 318.; HOLLÓS 1. p. 97.; HOLLÓS 7. p. 125.; GRESCHIK, MOESZ ap. MOESZ 16. p. 35.; MOESZ ap. MOESZ 13. p. 55.; UBRIZSY–VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 294.

Microsphaera syringae JACZEWSKI nec. MAGNUS

UBRIZSY–VÖRÖS 1. p. 155.

Microsphaera viburni (DUBY) BLUMER

Syn.: Alphitomorpha penicillata WALLR.; Erysiphe penicillata f. viburni opuli FR.; Microsphaera alni (DC.) WINT. ppe.; Microsphaera penicillata f. viburni JACZ. ppe.; Microsphaera (Calocladia) penicillata LÉV. ppe.
BÄUMLER 1. p. 22. (ad 300); MOESZ ap. MOESZ 16. p. 35.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 316.

Genus: Phyllactinia LÉVEILLÉ

Phyllactinia berberidis PALLA
SZIGETI–TÓTH 1. p. 19.

Phyllactinia hippophaës THÜMEN

Syn.: Erysiphe hippophaës REQUIEN; Phyllactinia guttata LÉV. f. hippophaës THÜMEN; Ph. corylea (PERS.) KARSTEN ppe.; Ph. suffulta (REBENT.) SACC. f. hyppophaës JACZ.; Ph. suffulta (REBENT.) SACC. var. angulata SALM.
BLUMER 1. p. 388.; LINHART in Fung. hung. exs. No. 455. ap. MOESZ 16. p. 35.; CSORBA in UBRIZSY 5. II. p. 178.

Phyllactinia mespili (CAST.) BLUMER

Syn.: Alphitomorpha lenticularis γ mespilorum WALLR.; Erysiphe amelanchieris CHAILLET; E. guttata var. mespili CAST.; E. oxyacanthae DC.; Phyllactinia guttata auct. ppe.; Ph. suffulta auct. ppe.; Ph. coryli auct. ppe.
HAZSLINSZKY 1. p. 180.; BÄUMLER 1. No. 296.; HOLLÓS 1. p. 97.; MOESZ 16. p. 35.; UBRIZSY–VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 178.; HIRATA 1. p. 142.

Phyllactinia roboris (GACHET) BLUMER

Syn.: Erysiphe roboris GACHET.; E. quercus MÉRAT; E. ilicis CASTAGNE; Phyllactinia schweinitzii LÉV.; Ph. suffulta f. quercina JACZ.
HAZSLINSZKY 1. p. 181.; BÄUMLER 1. No. 296.; BÄUMLER 2. III. No. 5.; HOLLÓS 1. p. 97.; GREINICH, KMET ap. MOESZ 16. p. 35.; UBRIZSY 1. p. 8.; UBRIZSY–VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 178.

Phyllactinia suffulta (REBENT.) SACCARDO

Syn.: Alphitomorpha guttata WALLR.; A. lenticularis WALLR.; Erysibe lenticularis RABENH.; E. lenticularis var. carpini DESMAZ.; Erysiphe coryli HEDWIG fil.; E. fraxini DC.; E. betulae DC.; E. orbiculatus EHRENBERG; E. alni DC.; E. varium et var. suffultum FR. ppe.; E. pachypus MARTIUS; E. guttata LINK; E. guttata FR.; E. detonsa FR.; E. mali DUBY; E. fogi DUBY; E. abnormis DUBY; E. oxyacanthae SECRETAN ppe.; E. pyri CAST.; E. cerasi CAST.; E. fungicola SCHULZER; Erysiphella carestiana SACC.; Phyllactinia guttata LÉV.; Ph. candollei LÉV.; Ph. corylea (PERS.) KARSTEN
BOLLA 1. p. 53.; SCHULZER 2. p. 362., 365., 366.; KALCHBRENNER 1. No. 534., 539.; SCHULZER 5. p. 58.; HAZSLINSZKY 1. p. 180., 181.; HAZSLINSZKY 2. p. 21.; BÄUMLER 1. No. 296., 305.; RICHTER 1.; BRESADOLA 1. No. 150.; BÄUMLER 2. III. No. 5.; SIMONKAI 1. p. 380.; HOLLÓS 1. p. 97.; MOESZ 11. p. 97.; BLUMER 1. p. 392., 393.; HOLLÓS 7. p. 127.; BÄUMLER in RABENHORST–WINTER Fung. eur. exs. No. 3047., 3048., 3647., et in LINHART Fung. hung. exs. No. 454., BOLLA, BUDAI, ENDLICHER, ENDREY, GÁYER, GREINICH, GRESCHIK, HAYNALD, HAZSLINSZKY, HOLUBY, HUSZ, KALCHBRENNER in RABENHORST Fung. eur. exs. No. 437., KÁLOVITS, KMET, LINHART in Fung. hung. exs. No. 255. I., II. et in REHM Ascomyceten No. 797., MÁGOCSY-DIETZ, MOESZ, NAGY, PÓSCH, SADLER, SCHNELLER, SCHULZER, SZÉPLIGETI ap. MOESZ 16. p. 36.; HAZSLINSZKY, MOESZ, SADLER, SZÉPLIGETI ap. MOESZ

13. p. 55.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; UBRIZSY 6. in SZÉKESSY p. 23.; TÓTH 2. p. 56.; VASS–TÓTH 2. p. 46.; SZIGETI–TÓTH 1. p. 19.; UBRIZSY –VÖRÖS 1. p. 155.; CSORBA in UBRIZSY 5. II. p. 176.; HIRATA 1. p. 66., 68., 72., 73., 75., 76., 78., 84., 110., 141., 153., 154., 160., 224., 242., 258., 260.

Genus: Podosphaera KUNZE

Podosphaera leucotricha (ELL. et EV.) SALMON

Syn.: Sphaerotheca castagnei LÉV.; S. leucotricha ELL. et EV.
BÄUMLER 1. No. 2132.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 216.; HUSZ ap. MOESZ 16. p. 38.; HUSZ, MOESZ ap. MOESZ 13. p. 55.; UBRIZSY 1. p. 8.; CSORBA 2. p. 1–7.; CSORBA 3. pp. 81–85.; CSORBA 4. p. 134.; UBRIZSY–VÖRÖS 1. p. 156.; CSORBA in UBRIZSY 5. II. p. 208.; HIRATA 1. p. 154.

Podosphaera myrtillina (SCHUB.) KUNZE et SCHMIDT

Syn.: Erysibe myrtillina RABENH.; Erysiphe myrtillina FRIES; Podosphaera kunzei LÉV.; P. oxyacanthae (DC.) DE BARY emend. SALMON ppe.; Sphaeria myrtillina SCHUBERT
BÄUMLER 1. No. 291.; BÄUMLER 2. III. p. 661., No. 1.; KMET, LINHART in Fung. hung. exs. No. 355. ap. MOESZ 16. p. 38.; HIRATA 1. p. 255.

Podosphaera oxyacanthae (DC.) DE BARY

Syn.: Alphitomorpha clandestina var. oxyacanthae WALLR.; A. clandestina WALLR.; A. oxyacanthae WALLR.; Erysiphe oxyacanthae DC.; E. clandestina FRIES; E. mali DUBY; Erysibe clandestina LINK; Podosphaera clandestina LÉV.; P. kunzei LÉV. ppe.; Erysiphe crataegi GROGN.
HAZSLINSZKY 1. p. 178.; HAZSLINSZKY 2. p. 20.; BÄUMLER 1. No. 292.; BLUMER 1. p. 136.; BÄUMLER in RABENHORST–WINTER Fung. eur. exs. No. 3042. et in REHM Ascomyceten No. 900. ap. MOESZ 16. p. 39.; MÁGOCSY-DIETZ ap. MOESZ 13. p. 55.; UBRIZSY 3. p. 4.; UBRIZSY–VÖRÖS 1. p. 156.; CSORBA in UBRIZSY 5. II. p. 212.; HIRATA 1. p. 141., 146.

Podosphaera tridactyla (WALLR.) DE BARY

Syn.: Alphitomorpha tridactyla WALLR.; Erysibe tridactyla RABENH.; Erysiphe tridactyla TUL.; E. bertoloni ROUMI; Podosphaera kunzei LÉV. ppe.; P. oxyacanthae (DC.) DE BARY var. tridactyla (WALLR.) SALM.; Oidium passerini BERTOLONI
BOLLA 1. p. 54.; KALCHBRENNER 1. No. 532.; HAZSLINSZKY 1. p. 178.; BÄUMLER 1. No. 293.; BÄUMLER 2. III. p. 661., No. 2.; HOLLÓS 7. p. 130.; BÄUMLER in REHM Ascomyceten No. 830., BOLLA, HUSZ, KMET, MOESZ,

ZIER ap. MOESZ 16. p. 39.; BERNÁTSKY, HUSZ, MOESZ ap. MOESZ 13. p. 56.; UBRIZSY 1. p. 8.; TÓTH 2. p. 56.; UBRIZSY—VÖRÖS 1. p. 156.; CSORBA in UBRIZSY 5. II. p. 212.; HIRATA 1. p. 149., 150., 151., 152.

Genus: Sphaerotheca LÉVEILLÉ

Sphaerotheca balsaminae WALLROTH

Syn.: Sphaerotheca castagnei LÉV. ppe.
HAZSLINSZKY 1. p. 179.; BÄUMLER 1. IV. p. 21. (ad. 294.); KMET, MOESZ, ZAHLBRUCKNER ap. MOESZ 16. p. 39.; UBRIZSY—VÖRÖS 1. p. 156.; CSORBA in UBRIZSY 5. II. p. 208.

Sphaerotheca epilobii (WALLR.) SACCARDO

Syn.: Erysibe epilobii LINK; Erysiphe macularis FR. ppe.; E. communis FR.; Torula epilobii CORDA; Podosphaera epilobii DE BARY; Sphaerotheca castagnei LÉV. ppe.; S. epilobii (LINK) SACC.; S. humuli (DC.) BURR. emend. SALM.
HAZSLIKSZKY 3. p. 89.; BÄUMLER 1. No. 1514.; BÄUMLER 2. III. p. 661., No. 3.; KMET, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 16. p. 39.; CSORBA in UBRIZSY 5. II. p. 208.; HIRATA 1. p. 239.

Sphaerotheca euphorbiae (CAST.) SALMON

Syn.: Botrytis euphorbiae CAST.; Erysiphe tomentosa OTTH; E. gigantasca SOROK. et THÜM.; Sphaerotheca tomentosa JACZ.; S. gigantasca (SOROK. et THÜM.) BÄUMLER; S. gigantasca (SOROK. et THÜM.) SCHROET.; S. mors-uvae (SCHWEIN.) BERK. emend. SALM. ppe.; Albigo tomentosa (OTTH) KUNZE BÄUMLER 1. No. 1110.; SACCARDO, D. 1. No. 141.; BLUMER 1. p. 98.; BÄUMLER in REHM Ascomyceten No. 1049. et in Krypt. exs. Vindob. No. 1917., Hedwigia p. 261., KMET ap. MOESZ 16. p. 39.; TÓTH 2. p. 56.; UBRIZSY—VÖRÖS 1. p. 156.; CSORBA in UBRIZSY 5. II. p. 208.; HIRATA 1. p. 204.

Sphaerotheca fugax PENZIG et SACCARDO

Syn.: Sphaerotheca humuli (DC.) BURR. emend. SALMON ppe.; S. macularis (WALLR.) MAGNUS f. geranii POTEBNIA in JACZ.
GRESCHIK ap. MOESZ 16. p. 40.; CSORBA in UBRIZSY 5. II. p. 208.; HIRATA 1. p. 197.

Sphaerotheca fuliginea (SCHLECHT.) SALMON

Syn.: Alphitomorpha fuliginea SCHLECHT.; A. communis WALLR. ppe.; A. fumosa WALLR.; Erysibe fuliginea LINK ppe.; Erysiphe fuliginea FR. ppe.; E. lamprocarpa β plantaginis (LINK) DUBY ppe.; E. lamprocarpa var.

melampyri MÉRAT; E. erigerontis canadensis LÉV.; E. plantaginis CAST.; E. xanthii CAST.; E. fuscata BERK. et CURT.; Podosphaera castagnei (LÉV.) DE BARY; Sphaerotheca castagnei LÉV.; S. detonsa (WEST.) KICKX.; S. microcarpa HAZSL.; S. drabae JUEL.; S. calendulae MALBRANCHE et ROUMEGUERE; S. humuli (DC.) SCHROETER ppe.; S. erigerontis OUDEM.; S. humuli (DC.) BURR. var. fuliginea (SCHLECHT.) SALM.; Albigo calendulae (MALBR. et ROUMEG.) KUNTZE

BOLLA 1. p. 53.; KALCHBRENNER 1. No. 548.; HAZSLINSZKY 1. p. 179.; HAZSLINSZKY 2. p. 20., 21.; HAZSLINSZKY 3. p. 89.; HAZSLINSZKY 7. p. 14.; BÄUMLER 1. No. 294.; HOLLÓS 1. p. 96.; MOESZ 11. p. 98.; MOESZ 23. col. 819.; MOESZ 27. p. 145.; BLUMER 1. p. 126.; HOLLÓS 7. p. 132.; BÄUMLER, BOLLA, BUDAI, FUSS, GYŐRFFY, HAZSLINSZKY, KALCHBRENNER, KMET, LAUDON, LINHART in Fung. hung. exs. No. 254., MÁGOCSY-DIETZ, MOESZ, PÉNZES, SZÉPLIGETI in LINHART Fung. hung. exs. No. 453., WAGNER ap. MOESZ 16. p. 40.; MÁGOCSY-DIETZ, MOESZ, PÉNZES, SZÉPLIGETI ap. MOESZ 13. p. 56.; UBRIZSY 1. p. 8.; TÓTH 2. p. 56.; VASS 1. p. 49.; VASS—TÓTH 2. p. 46.; VÖRÖS 9. p. 101.; UBRIZSY—VÖRÖS I. p. 156.; CSORBA in UBRIZSY 5. II. p. 204.; HIRATA 1. p. 213., 296., 301., 302., 307., 312., 331., 332., 333., 347., 348., 349., 368., 372., 374., 376., 377.

Sphaerotheca fusca (FRIES) BLUMER

Syn.: Erysiphe fusca FR.; Alphitomorpha lamprocarpa *β* balsaminae WALLR.; Sphaerotheca castagnei LÉV. ppe.; S. pannosa BONORDEN; S. humuli (DC.) BURR. var. fuliginea (SCHLECHT.) SALM. ppe.; Podosphaera castagnei (LÉV.) DE BARY ppe.

HAZSLINSZKY 1. p. 179.; HAZSLINSZKY 2. p. 20.; MOESZ 12. col. 81.; GRESCHIK, KÜMMERLE ap. MOESZ 16. p. 41.; TÓTH 2. p. 56.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 208.; HIRATA 1. p. 368.

Sphaerotheca humuli (DC.) BURRILL

Syn.: Sphaerotheca humuli (DC.) BURR. emend. SALM. ppe.; S. castagnei LÉV.; S. macularis MAGNUS f. humuli (LÉV.) JACZEWSKI; S. epilobii (LINK) SACC.; Erysibe macularis var. humuli FICINUS et SCHUBERT; Erysiphe humuli DC.; E. macularis FRIES; Albigo humuli (EHRENB.) KUNTZE

ENDLICHER 1. No. 120.; KALCHBRENNER 1. No. 533.; HAZSLINSZKY 1. p. 179.; HAZSLINSZKY 7. p. 14.; BÄUMLER 1. No. 294.; RICHTER 1.; SACCARDO. D. 1. No. 140.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 218.; BÄUMLER, BUDAI, ENDLICHER, HAZSLINSZKY, HOLLÓS, HOLUBY, HUSZ, KALCHBRENNER, KMET, LÁNYI, MÁRTON, MOESZ, PÁTER, PÓSCH, RICHTER, ZAHLBRUCKNER ap. MOESZ 16. p. 41.; MOESZ ap. MOESZ 13. p. 56.; UBRIZSY 1. p. 8.; PICBAUER 1. p. 4.; PICBAUER 2. p. 180.; UBRIZSY 3. p. 4.; TÓTH 2. p. 56.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 203.; HIRATA 1. p. 89.

Sphaerotheca macularis (WALLR.) JACZEWSKI

Syn.: Mucor erysiphe L. ppe.; Sclerotium erysiphe PERS. ppe.; Albigo macularis EHRENB.: A. humuli (EHRENB.) KUNTZE ppe.; Alphitomorpha macularis WALLR. ppe.; A. clandestina β aphanis WALLR.; A. aphanis WALLR.; A. macularis WALLR. ppe.; A. horridula WALLR. γ spiraeacearum et δ dryadearum; Erysibe macularis SCHLECHT.; E. potentillae LIBERT.; Erysiphe macularis GREV.; E. communis FR. ppe.; E. alchemillae DUBY; E. horridula RABENH. ppe.; E. macularis c. poterii RABENH.; Sphaerotheca castagnei LÉV. ppe.; S. humuli (DC.) BURR. ppe.; S. anserinae WARNSTORF; Oidium fragariae HARZ
KALCHBRENNER 1. No. 533.; HAZSLINSZKY 1. p. 179.; RICHTER 1.; BÄUMLER 1. No. 294.; GRESCHIK, KMET, MÁRTON ap. MOESZ 16. p. 41.; VÖRÖS 9. p. 101; TÓTH 2. p. 56.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 208.; HIRATA 1. p. 138., 143., 145., 146., 160.

Sphaerotheca mors-uvae (SCHWEINITZ) BERKELEY

Syn.: Erysiphe mors-uvae SCHW.; Albigo mors-uvae (SCHW.) KUNTZE
BÄUMLER 1. No. 1739.; SCHILBERSZKY 1.; KERN 1.; MOESZ 12.; MOESZ 24. p. 219.; MOESZ 26.; RAPAICS 1. p. 216.; MOESZ 30.; BÄUMLER, FILARSZKY, GREINICH, GRESCHIK, GYŐRFFY, HOLLÓS, HUSZ, KERN, KOVÁCS, KRIKOVSZKY, MOESZ in Krypt. exs. Vindob. No. 1813.; RŐMER, SCHILBERSZKY, SZABÓ ap. MOESZ 16. p. 41.; MOESZ ap. MOESZ 13. p. 57.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 201.; HIRATA 1. p. 135.

Sphaerotheca pannosa (WALLR.) LÉVEILLÉ

Syn.: Alphitomorpha pannosa WALLR.; Erysibe pannosa SCHLECHT., Erysiphe pannosa FRIES; Podosphaera pannosa DE BARY var. *rosae* WORONICHIN
KALCHBRENNER 1. No. 541.; HAZSLINSZKY 1. p. 178.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. No. 295.; BRESADOLA 1. No. 319.; BÄUMLER 2. III. No. 4.; HOLLÓS 1. p. 96.; RAPAICS I. p. 218.; BÄUMLER, DEGEN, GRESCHIK, HUSZ, KMET, LINHART in Fung. hung. exs. No. 354/a., MÁGOCSY-DIETZ, MOESZ, PÁTER ap. MOESZ 16. p. 42.; DEGEN, MOESZ ap. MOESZ 13. p. 57.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; TÓTH 2. p. 56.; VASS—TÓTH 2. p. 46.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 204.; HIRATA 1. p. 155., 156., 157.

var. *persicae* WORONICHIN

RAPAICS 1. p. 217.; CSORBA 1. p. 110.; BUDAI, DOBOS, HUSZ, LINHART in Fung. hung. exs. No. 354/b., MOESZ, PÓSCH ap. MOESZ 16. p. 42.; MOESZ ap. MOESZ 13. p. 57.; UBRIZSY 1. p. 8.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 204.; HIRATA 1. p. 151.

Sphaerotheca sanguisorbae (DC.) BLUMER

Syn.: Alphitomorpha ferruginea SCHLECHT.; A. macularis y. poterii WALLR.; Albigo humuli (EHRENB.) KUNTZE ppe.; Erysibe poterii LINK.; E. fuliginea LINK ppe.; Erysiphe fuliginea FR. ppe.; E. ferruginea FR.; Sphaerotheca castagnei LÉV. ppe.; S. humuli (DC.) BURRILL ppe.; S. macularis f. sanguisorbae RABENH. in JACZ.
HAZSLINSZKY 1. p. 179.; MOESZ ap. MOESZ 16. p. 42.; MOESZ ap. MOESZ 13. p. 57.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 208.

Genus: Trichocladia (DE BARY) NEGER

Trichocladia astragali (DC.) NEGER

Syn.: Calocladia holosericea (LÉV.) FUCK.; Microsphaera astragali (DC.) TREV.; M. astragali (DC.) SACC.
KALCHBRENNER 1. No. 538.; HAZSLINSZKY 1. p. 186.; BÄUMLER 1. No. 301., 313.; RICHTER 1.; BÄUMLER 2. III. No. 9.; BRESADOLA 1. No. 153.; MOESZ 2. No. 16.; HOLLÓS 1. p. 96.; RAPAICS 1. p. 218.; HOLLÓS 7. p. 118., 125.; BÄUMLER, BUDAI, GRESCHIK, KÜMMERLE, MÁGOCSY-DIETZ, MOESZ, PÉTERFI, ZAHLBRUCKNER ap. MOESZ 16. p. 43.; KÜMMERLE, MOESZ, ap. MOESZ 13. p. 58.; UBRIZSY 1. p. 8.; UBRIZSY—VÖRÖS 1. p. 157.; LEHOCZKY 1. p. 236.; TÓTH 2. p. 56.; VASS—TÓTH 1. p. 156.; VASS 1. p. 49.; VASS—TÓTH 2. p. 46.; HIRATA 1. p. 164.; CSORBA in UBRIZSY 5. II. p. 194.

Trichocladia bäumleri (MAGN.) NEGER

Syn.: Microsphaera bäumleri MAGN.
HAZSLINSZKY 1. p. 186.; BÄUMLER 1. No. 313.; MOESZ 12. col. 801.; BUDAI, KMET, KÜMMERLE, MÁGOCSY-DIETZ in Krypt. exs. Vindob. No. 1147., MOESZ ap. MOESZ 16. p. 43.; UBRIZSY 1. p. 8.; PICBAUER 1. p. 4.; VASS—TÓTH 1. p. 156.; VASS 1. p. 50.; VASS—TÓTH 2. p. 46.; CSORBA in UBRIZSY 5. II. p. 194. HIRATA 1. p. 192., 194.

Trichocladia evonymi (DC.) NEGER

Syn.: Microsphaera evonymi SACC.; Calocladia comata LÉV.
BOLLA 1. p. 53.; KALCHBRENNER 1. No. 537.; HAZSLINSZKY 7. p. 14.; BÄUMLER 1. No. 304.; BÄUMLER 2. III. No. 10.; HOLLÓS 1. p. 97.; RAPAICS 1. p. 218.; MOESZ 11. p. 98.; MOESZ 27. p. 145.; MOESZ 30. p. 97.; HOLLÓS 7. p. 125.; BÄUMLER, FUSS, GRESCHIK, HAMBALKÓ, HAZSLINSZKY, KÁLOVICS, KMET, LINHART in Fung. hung. exs. No. 76.; MÁGOCSY-DIETZ, MOESZ, SZABÓ, WAGNER ap. MOESZ 16. p. 43.; HAMBALKÓ, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 58.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; TÓTH 2. p. 56.; VASS—TÓTH 2. p. 46.; UBRIZSY—VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 215., 216.

Trichocladia robiniae TSCHERMIENKA

UBRIZSY–VÖRÖS 1. p. 157.

Trichocladia tortilis (WALLR.) NEGER

Syn.: Erysiphe tortilis LINK; E. tortilis (WALLR.) FR.
HAZSLINSZKY 1. p. 186.; BÄUMLER 1. No. 314.; BÄUMLER 2. III. No. 19.; GRESCHIK, KMET, MOESZ in Flora hung. exs. No. 207. ap. MOESZ 16. p. 44.; MOESZ ap. MOESZ 13. p. 58.; UBRIZSY 3. p. 4.; TÓTH 2. p. 56.; UBRIZSY–VÖRÖS 1. p. 157.; CSORBA in UBRIZSY 5. II. p. 189.; HIRATA 1. p. 242.

Genus: Uncinula LÉVEILLÉ

Uncinula aceris (DC.) SACCARDO

Syn.: Mucor erysiphe L. ppe.; Alphitomorpha bicornis WALLR.; Erysibe bicornis LINK; Erysiphe bicornis FR. ppe.; E. aceris DC.; E. varium FR.; Uncinula bicornis LÉV.
KALCHBRENNER 1. No. 535.; FUSS 1. XVI. p. 25.; SCHULZER 2. p. 369.; FUSS 2. p. 462.; BÄUMLER 1. No. 297.; BÄUMLER 2. III. No. 6.; MOESZ 2. No. 17.; HOLLÓS 1. p. 97.; RAPAICS 1. p. 218.; HOLLÓS 7. p. 134.; BÄUMLER, BOLLA, FILARSZKY, GREINICH, GRESCHIK, GYŐRFFY, HAZSLINSZKY, KMET, LINHART in Fung. hung. exs. No. 256., MÁGOCSY-DIETZ in LINHART Fung. hung. exs. No. 77., MOESZ, SCHNELLER, SCHULZER, SZÉPLIGETI, ZAHLBRUCKNER ap. MOESZ 16. p. 44.; KRENNER, LINHART, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 13. p. 58.; UBRIZSY 1. p. 8.; KRENNER 1. p. 63.; TÓTH 2. p. 56.; VASS 1. p. 50.; VASS–TÓTH 2. p. 46.; UBRIZSY–VÖRÖS 1. p. 158.; CSORBA in UBRIZSY 5. II. p. 200.; HIRATA 1. p. 209., 211.

Uncinula clandestina (BIV. BERN.) SCHROETER

Syn.: Alphitomorpha adunca γ ulmorum WALLR.; Erysibe adunca var. ulmorum LINK; Erysiphe clandestina BIV. BERN.; E. adunca FR. ppe.; E. ulmi CAST.; E. adunca var. ulmorum DUR. et MONT.; Uncinula bivonae LÉV.; U. clandestina f. japonica HENNINGS
HOLLÓS 1. p. 97.; CSORBA in UBRIZSY 5. II. p. 200.; HIRATA 1. p. 87.

Uncinula necator (SCHWEIN.) BURRILL

Syn.: Erysiphe necator SCHWEIN.; E. tuckeri BERK.; Erysibe tuckeri (BERK.) SCHROETER; Oidium tuckeri auct.; Sphaerotheca castagnei var. vitis FUCKEL; Uncinula americana HOWE; U. spiralis BERK. et CURT.; U. subfusa BERK. et CURT.; U. spiralis var. racemosum THÜM.; U. ampelopsidis PECK.; U. actinidiae MIYABE
FUSS 2. p. 432.; BÄUMLER 1. No. 1516.; BERNÁTSKY, MESZÉNA in LINHART

Fung. hung. exs. No. 356. ap. MOESZ 16. p. 44.; BERNÁTSKY, MOESZ ap. MOESZ 13. p. 58.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; CSORBA in UBRIZSY 5. II. p. 195.; HIRATA 1. p. 219.

Uncinula prunastri (DC.) SACCARDO

Syn.: Alphitomorpha prunastri WALLR.; A. adunca var. prunastri WALLR.; Erysibe adunca var. prunastri LINK; Erysiphe adunca var. prunastri FR.; E. prunastri DC.; Uncinula wallrothii LÉV.
KALCHBRENNER 1. No. 536.; SCHULZER 2. p. 368.; HAZSLINSZKY 1. p. 181.; HAZSLINSZKY 2. p. 21.; FUSS 2. p. 462.; BÄUMLER 1. No. 298.; BÄUMLER 2. III. No. 7.; HOLLÓS 1. p. 97.; RAPAICS 1. p. 218.; KMET, MÁGOCSY-DIETZ ap. MOESZ 16. p. 45.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; TÓTH 2. p. 56.; UBRIZSY—VÖRÖS 1. p. 158.; CSORBA in UBRIZSY 5. II. p. 200.; HIRATA 1. p. 150., 152.

Uncinula salicis (DC.) WINTER

Syn.: Alphitomorpha adunca WALLR.; A. obtusata SCHLECHT.; A. adunca α amentacearum WALLR.; Erysibe adunca var. populi et E. obtusata LINK; Erysiphe salicis DC.; E. populi DC.; E. adunca GREV.; E. adunca a. populi, b. salicis FR.; E. capreae (DC.) DUBY; E. populi PATOUILLARD; Uncinula adunca LÉV.; U. columbiana SELBY; U. salicis var. epilobii VESTERBREN; U. conidiigena COCCONI
BOLLA 1. p. 54.; KALCHBRENNER 1. No. 536.; SCHULZER 2. p. 369.; HAZSLINSZKY 1. p. 181.; FUSS 2. p. 462.; BÄUMLER 1. No. 299.; RICHTER 1.; BRESADOLA 1. No. 151.; BÄUMLER 2. III. No. 8.; SACCARDO, D. 1. No. 192.; HOLLÓS 1. p. 97.; RAPAICS 1. p. 218.; MOESZ 11. p. 98.; MOESZ 12. col. 801.; BÁNYAI, FILARSZKY, GRESCHIK, HAYNALD, KALCHBRENNER, KISS, KMET, LINHART in Fung. hung. exs. No. 78., MÁGOCSY-DIETZ, MOESZ, PÉTERFI, ZAHLBRUCKNER, ZSÁK ap. MOESZ 16. p. 46.; MOESZ ap. MOESZ 13. p. 58.; UBRIZSY 1. p. 8.; UBRIZSY 3. p. 4.; VASS 1. p. 50.; UBRIZSY—VÖRÖS 1. p. 158.; CSORBA in UBRIZSY 5. II. p. 200.; HIRATA 1. p. 67., 68., 69., 70., 71.

Oidium LINK

Oidium begoniae PUTTEMANS

UBRIZSY—VÖRÖS 1. p. 158.

Oidium evonymi-japonici (ARC.) SACCARDO

Syn.: Oidium leucoconium DESM. f. evonymi japonici ARCHANGELI
SIMONKAI, MOESZ ap. MOESZ 16. p. 46.; UBRIZSY 3. p. 5.; UBRIZSY—VÖRÖS 1. p. 159.; HIRATA 1. p. 215.

Oidium helianthi auct.

UBRIZSY—VÖRÖS 1. p. 159.

Oidium lini ŠKORIČ

UBRIZSY 3. p. 5.; UBRIZSY—VÖRÖS 1. p. 159.; HIRATA 1. p. 200.

Oidium solani auct.

MOESZ ap. MOESZ 16. p. 46.; UBRIZSY—VÖRÖS 1. p. 159.; HIRATA 1. p. 298.

Oidium verbenae THÜMEN et BOLLE

MÁGOCSY-DIETZ ap. MOESZ 16. p. 46.; UBRIZSY—VÖRÖS 1. p. 159.

Oidium vincae (SAVUL. et SANDU VILLE) UBRIZSY

UBRIZSY—VÖRÖS 1. p. 159.

Oidium spec. div.

KALCHBRENNER 1. No. 533.; HAZSLINSZKY 1. p. 179.; FUSS 2. p. 432.; HOLLÓS 7. p. 132.; AUGUSTIN, GÁYER, KÜMMERLE, MOESZ ap. MOESZ 16. p. 46.; UBRIZSY 3. p. 5.; UBRIZSY—VÖRÖS 1. p. 159.; HIRATA 1. p. 200., 230., 231., 232., 233., 234., 298., 306., 321.

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Addenda ad Part IV.

Puccinia pelargonii-zonalis DORIDGE

FOLK 1. p. 6.; FOLK 2. p. 441—443.; UBRIZSY-VÖRÖS 2. p. 340.; UBRIZSY in litt.

Uromyces nerviphilus (GROG) HOTS.

TÓTHNÉ 1. p. 173.

Aecidium crambes MOESZ

MEGYASZÓ, Balatonkenese, UBRIZSY in litt.

FOLK GY. 1. Új betegség: a muskátlirozsda. Kertészet és Szőlészet, XV. 1966. 5. p. 6.

FOLK GY. 2. A muskátli rozsdabetegségének hazai előfordulása. XVII. Növényvédelmi Tudományos Értekezlet, II. Bp. 1967. p. 441—443.

TÓTHNÉ, ZAHORECZ E. 1. Pillangósvirágú takarmánynövények parazita mikrogombái Gödöllőn. Agrártud. Egyet. Közlem. Bp. 1967. pp. 171—177.

UBRIZSY G. and VÖRÖS J. 2. Mezőgazdasági Mykologia. Akad. Kiadó, Bp. 1968. pp. 577.

Preliminary Reports

Outbreak of the Black Cutworm (*Scotia ipsilon* Hufn.) in Hungary and Comments on Migration of Adults

By

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The damages of *Scotia ipsilon* Hufn., though a commonly known moth species in Hungary, were first observed in July, 1965. Damages were caused in maize, beans and cabbages (but less on cucumbers), sown subsequently to the flood in the inundational area of the Körös. The appearance, without prior gradational phenomena, of the damages of *Scotia ipsilon* in Central Europe can easily be explained by the assumed or occasionally proven migrations of the adults.

The Black Cutworm (*Scotia ipsilon* Hufn. (= *Agrotis ypsilon* Rott.)) is generally distributed in Hungary, as witnessed by both earlier (ABAFI-AIGNER, 1907) and recent data (KOVÁCS, 1953, 1956). Despite this fact, however, it had as yet not appeared as a pest. Among the most prominent Noctuid pests of the past two decades in Hungary, we find the names of primarily *Scotia segetum* Schiff., *Euxoa temera* Hbn. and *Rhyacia ravida* Schiff., having caused grave damages especially in 1948–55 (JERMY, 1950). They were followed later by *Heliothis maritima* Grasl., the damages of which were observed first in 1954, formerly not or hardly known as a pest in Hungary (NAGY, 1957).

As valid for Central Europe, experience agrees that despite the general appearance during almost the entire vegetational period and the occasional swarming of *Scotia ipsilon* damages caused by the larvae are relatively rare events. In the last forty years, for instance, it was recorded as a pest from Poland (WORONIECKA-SIEMASZKO, 1928), Switzerland (THOMANN, 1944), and chiefly Germany (GESSNER, 1928; MÜLLER, 1949; NOLTE and FRITSCHE, 1953; SCHATZ, 1953; WACHTENDORF, 1955). Under warmer climatic zones, its damages are more significant and frequent (MÜLLER, 1953).

The first damages caused in Hungary happened in rather special circumstances. Owing to the high water levels of the east Hungarian rivers in 1965, also the river Körös left its restrictive banks. The agricultural fields of the inundational area were flooded during 10–25 June, so that sowing had to be repeated after the retreat of the waters.

The first signs of damage were observed on 19 July, followed by a significant loss of foliage and even of plants on 23 July (Table 1). On this date, the larvae were about 1–3 cm large, thus in the second half of their larval develop-

ment. The adults developed from the specimens reared further in the laboratory in August and then the specific identification was also made.

For the vegetables listed above, damages had generally levelled out at the above values, since the area was subsequently and successfully treated by application of molasses bait containing Lindane (Hungaria L2) with the exception of cabbage, which was treated obviously too late.



Fig. 1. Germinating maize plants, damages by the larvae of *Scotia ipsilon* Hufn. (Photo: B. Nagy)

On the other hand, the large maize stand, cultivated as usual in more extensive conditions, was left unprotected and untreated, with the result that it was practically destructed by the larvae (Fig. 1).

Beside the cultures of the flooded area, no similar damages had been recorded from the neighbourhood. Thus MÜLLER's statement (1953) that the ovipositing adults favour wet soils in the wake of inundations seems to be corroborated by observations in Hungary. Incidentally, NIKOLOVA (1961) observed the damages of the species in similarly low regions, near river courses, in Bulgaria.

On the basis of the data of the light trap network operating in Hungary, it can be established that the adults of *Scotia ipsilon* fly continuously, generally

Table 1

The rate of damages caused by larvae of *Scotia ipsilon* Hufn. in the inundated fields of the Körös area on 23 July, 1965.

Stand	Height of plants in cm	Abundancy of larvae, ind/m²	Loss of foliage in %	Loss of plants in %
Zea mais	3— 8	5—10	(10—20)	90
Brassica oleracea	5—10			80
Phaseolus vulgaris	3—10	20—40	20—40	10—20
Cucumis sativus	2— 4	?	5—10	1—2
Weed zone (along road)	5—25	30—40	?	?

from May to November (KOVÁCS, 1960—1965). From the year 1964, preceding the damages here recorded, VOJNITS (1966) mentions the capture of 116 individuals by the light trap network; the specimens appeared from June till 12 November. A further exemplar was attracted by the light on 19 February (South Hungary).

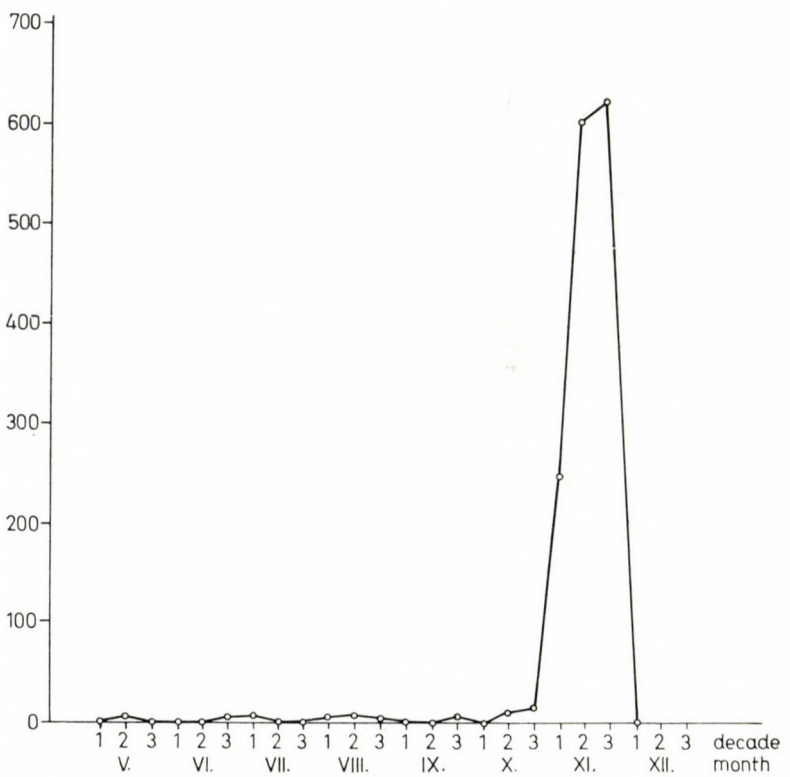


Fig. 2. *Scotia ipsilon* Hufn. exemplars captured by a light trap in East Hungary (Mikepércs) in 1963 (KOVÁCS, 1960—65)

Earlier authors have already commented on the fact that the adults of the black cutworm migrate in the southern parts of Europe (WOLFSBERGER, 1953; etc.). A migration towards the Mediterranean area in the autumn, and another northbound one in the spring, seem to be well established. RIVNAY (1964), however, showed their migration also within the Mediterranean area. It is the winter in Central Europe, and the extreme warming up and desiccation of the soil in the Mediterranean area, which interrupt the contiguous development of the generations, so that these factors may result in the migration of the moths. In both cases, of course, the main factor will act through the vegetation itself.

To investigate the periode and rate of migration in Hungary no detailed and comprehensive analysis has as yet been made, though it were greatly facilitated by the still functioning 41 light traps of the nationwide network (KOVÁCS, 1962). In this connection, we refer to merely a single datum (KOVÁCS, 1960—1965), namely that one of the traps in East Hungary (Mikepércs) captured 1267 *Scotia ipsilon* specimens during November, 1963, assumably correlatable with the migrations discussed above.

It seems to us that the unexpected damages of *Scotia ipsilon*, appearing without any special local build-up of the population, might be easily explained by the migratory habits of this species.

*

We have to express our gratitude to the Plant Protection Station at Tarhos, namely to J. KOMLÓDI, who called our attention to the damages and who also collaborated in the local surveys.

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Sorosporium cenchri Henn. var. *levis* n. var. a New Smut Fungus from Hungary*

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The spores of a *Sorosporium* sp. occurred on sandbur in Hungary are smooth walled. On the basis of this character the name *Sorosporium cenchri* Henn. var. *levis* n. var. is proposed.

Introduction

The common sandbur [*Cenchrus pauciflorus* Benth. (=*C. tribuloides* L.)] is a comparatively new weed plant in Hungary. It was introduced to the country presumably between 1935 and 1940. Distribution of the weed is localized to a certain area of the Great Hungarian Plain. No fungal or other diseases on this host have been observed in Hungary until 1967.

During the course of a weed control experiment in 1967, however, a smut disease of sandbur was observed.

Description of the Pathogen

Inflorescences of the infected plants were consumed by the dark-brown to black mass of the spores, covered by an evanescent, yellowish-brown peridium (Fig. 1). Spores were united in balls, which contained no mycelial elements. Therefore, the pathogen was undoubtedly a species of *Sorosporium*.

According to FISCHER (1953) the main characteristics of the single well known *Sorosporium* species from the host plant *Cenchrus*, *Sorosporium cenchri* Henn. [= *S. syntherismae* (Peck) Farl.] are as follows: "Spores globose, subglobose to elongate, olive brown, abundantly and minutely echinulate, $9.5-13\ \mu$ in diameter." "Spore balls more or less evanescent, globose to oblong, dark brown, $35-70\times70-105\ \mu$."

Since the spores of the *Sorosporium* species collected in Hungary are definitely smooth walled, it represents a new variety:

* Appearance of this fungus was announced for the first time in the book Agricultural Mycology (Ubrizsy and Vörös, 1968.).



Fig. 1. Symptoms of *Sorosporium cenchri* var. *levis* on *Cenchrus pauciflorus*



Fig. 2. Spore balls of *Sorosporium cenchri* var. *levis*



Fig. 3. A single spore ball of *Sorosporium cenchri*. Note the fitting together of the multangular spores

Sorosporium cenchri Henn. var. levis n. var.

Spore balls (Figs 2 and 3) are dark brown to black, their shapes are usually irregular or ellipsoidal, $53-88 \times 44-62\ \mu$. They are sometimes globose, and in this case $44-62\ \mu$ in diameter.

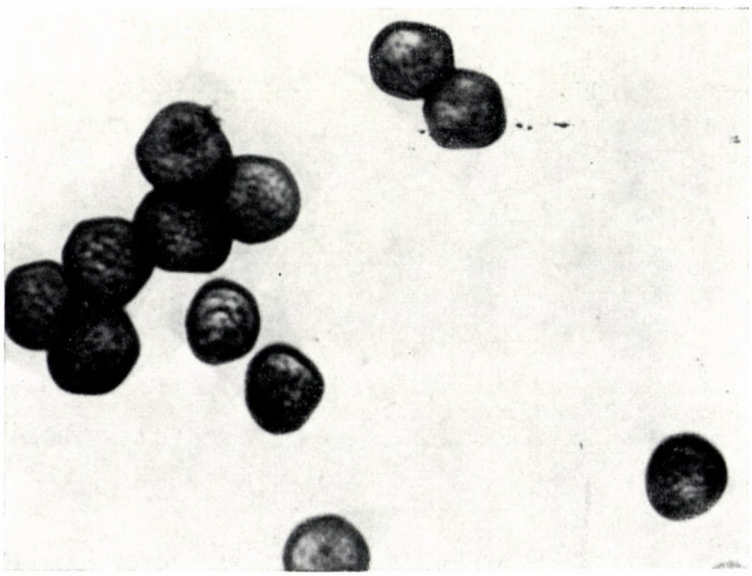


Fig. 4. Isolated spores of *Sorosporium cenchri* var. *levis*

Spores are never really globose. They are characteristically rounded multangular, or sometimes irregular (Fig. 4). Measurements of the spores are generally $10.1-13.5\times7.8-11.2\ \mu$. The roughly globular spores are $10.1-11.5\ (-13.5)\ \mu$ in diameter. The spore wall is absolutely smooth, however, the internal content of the spore is granular, especially in transmitted light.

Hab.: In the inflorescences of *Cenchrus pauciflorus*, Kecskemét–Talfája, Hungary. (Leg.: Dr. G. UBRIZSY, September 27, 1967).

Discussion

During the past three decades a number of *Sorosporium* species have been described from the host plant *Cenchrus*. *Sorosporium brefeldianum* Fischer et Holton [= *S. cenchri* (Brefeld) Zundel, non *S. cenchri* Henn.] has quite different characters (FISCHER and HOLTON, 1957). The description of two additional *Sorosporium* species from *Cenchrus* (*S. myosuroides* Hirschhorn and *S. catharticum* R. Maire) from Argentina and from Africa, respectively, are unfortunately not available for us.

Investigations carried out by MARTIN (1943) revealed the possibility of the production of interspecific hybrids between *Sorosporium cenchri (= S. syntherismae)* and *Sphacelotheca panici-miliacei (= Sphacelotheca destruens)*. According to MARTIN's results, however, the heritability of the verrucose spore wall character was clearly dominant in the *Sorosporium cenchri* (verrucose spore wall) X *Sphacelotheca panici-miliacei* (smooth spore wall) hybrids. Therefore, the development of the new, smooth walled *Sorosporium* variety in *Cenchrus* in Hungary by means of hybridization is – at least – very questionable.

At present no biological or genetical data are available concerning the development of the new *Sorosporium* variety, however, new results in the future will help to clear up the taxonomical position of the fungus described here.

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Planctomyces from Vietnam

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In January 1966, in Hanoi, Vietnam, in the detached backwater of the Red River, in the very filthy water two kinds of *Planctomyces* were living at the time of algal bloom. One of them *P. Békefii* was found in two morphotypes. The arms of one type are very thin, those of the other one are thicker. In the algal bloom 81 taxons took part, *P. Békefii* were present in 6 per cent, quantitatively. *P. guttaeformis* is much more rare. They were present also in the benthos sample, taken from the place of algal bloom. Apart from them, number of algae reached up to 113. Here *P. Békefii* was also the most frequent, but it is much rare, than in the algal bloom.

In January 1966, in Hanoi, Vietnam, in the detached backwater of the Red River, a long lasting algal bloom developed in the very filthy water of the lake. The lake is in the city, it is surrounded by living-houses, and dirty water runs into the lake from the houses. In algal bloom and in water samples, originated from the alga-covered lake bottom of algal-bloom I identified 130 sorts of micro-organisms, from which two belonged to the genus *Planctomyces*.

In the algal bloom 81 taxons took part. From among the plancton fungi it is the *Planctomyces Békefii* which is the most frequent. I identified 115 sorts of organisms in the benthos, both organisms occur here too, but much scarcely; here *P. Békefii* lived in a higher number. In the algal bloom it was rather conspicuous that the two plancton fungi were present in 6 per cent quantitatively.

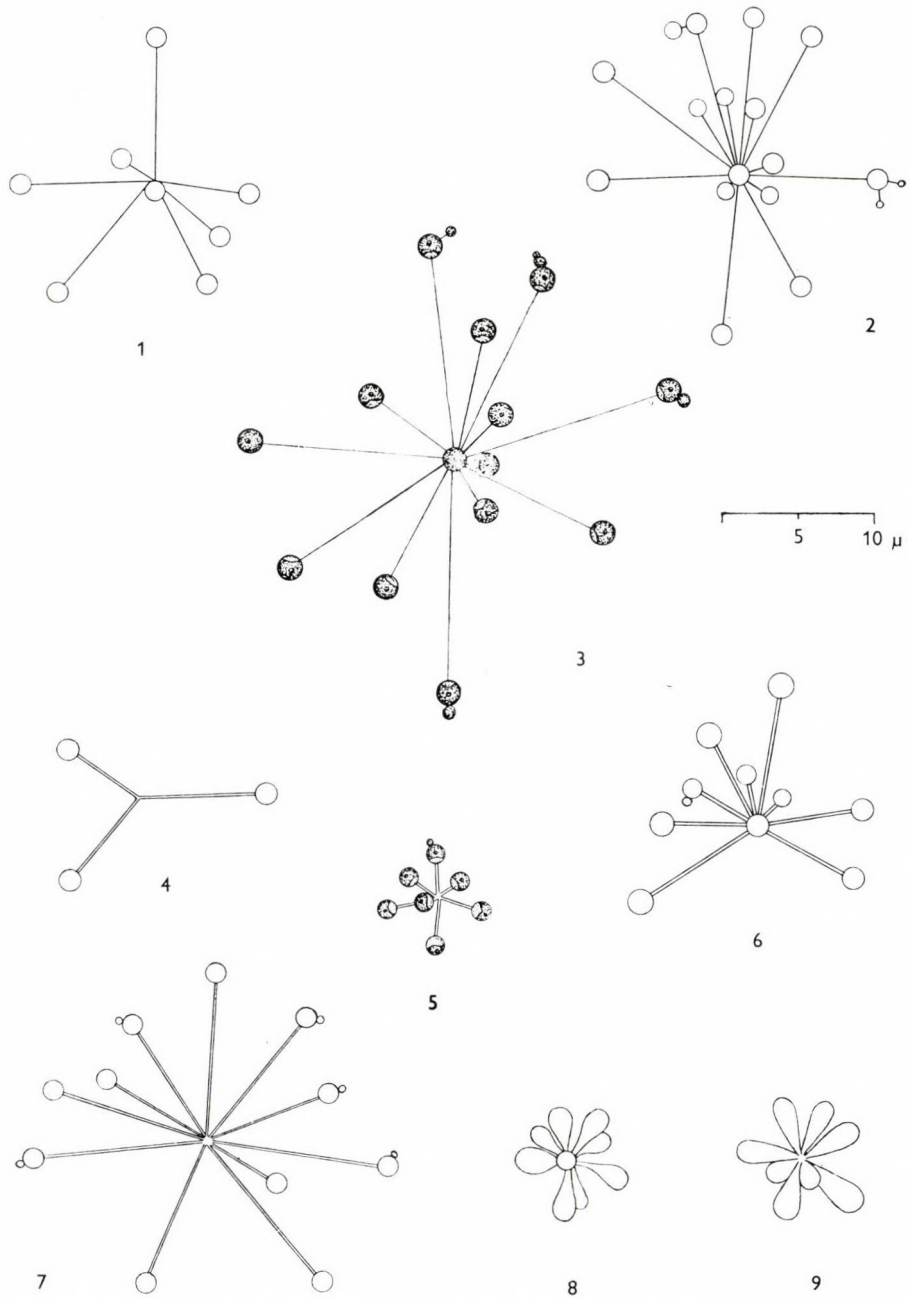
1. Planctomyces Békefii Gim.

Figs 1–7

It has two morphotypes. One of them is characterized by very thin sticks, the length of which can reach 10.5 μ. Diameter of the developed spherical cells (spores, conidia) is 1.1–1.3 μ. In the colony the number of the arms can vary between 3–14 (Fig. 1–3).

In case of the other morphotype arms are thicker, their length is 3–10.3 μ. Diameter of the cells at the arm ends is 1.3–1.5 μ, in developed stage. Number of the arms amounts to 3–11. The arms of both types are straight (Fig. 4–7).

I observed these morphotypes in water-basins in India too. Colonies, with thin arms, observed in Hanoi are identical with colonies observed in India, only



Figs 1—3. *Planctomyces Békefii* I. morphotype
Figs 4—7. *Planctomyces Békefii* II. morphotype
Figs 8—9. *Planctomyces guttaeformis*

the diameter of the developed cells is smaller. (Their diameter is 1.3—1.5 μ in India). Colonies with thinner arms have smaller dimensions in India, the length of the arms is about 4 μ. These types occur in Hanoi too, but colonies with longer sticks can be found in a higher number. But the diameter of the developed cells is somewhat bigger. (In India it is 1—1.3 μ).

There is living a third morphotype in India. Arms holding the cells are the longest, their length can reach 16.3 μ, they are never straight, they are always curved or undulating. Diameter of the developed cells is 1.3—1.5 μ.

2. *Planctomyces guttaeformis* Hortob.

Figs 8—9

The length of the drop-form arms is 3.5—4 μ. The number of the arms in one colony can reach 9. The width of the arms at the widest part is about 2 μ; they join with their thick end to each other.

For the first time it was described as *Planctomyces Stranskae* WAWRIK by HEYNIG in 1961 (HEYNIG, 1961, p. 411, Fig. i—1), but it is not identical with that. In case of *Planctomyces Stranskae* WAWRIK the number of the cells is 5—11 per colonies; the length of the cells is 2.5—3 μ, width is 1 μ. On the other hand organisms described by WAWRIK as *Planctomyces Stranskae* and *P. subulatus* in 1952, cannot be accepted as new species, because they belong to the forma specialis of *P. Békefii* Gim., according to general opinion (FOTT—KOMAREK, 1960; SKUJA, 1964; HORTOBÁGYI, 1966). That is why I considered to describe as *P. guttaeformis* the organism, observed in the fish pond at Buzsák but wrongly determined by HEYNIG in 1965 (HORTOBÁGYI, 1966, p. 113, Fig. 12—17).

I found the same organism in water-basins in India too. Their cell dimension is maximum 4 μ, their width is maximum 1.7 μ. The number of cells can reach 8 in one colony.

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Antifeeding Effect of Some Substituted Phenoxy Compounds on Chewing and Sucking Phytophagous Insects

by

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In a previous paper (JERMY and MATOLCSY, 1967) it has been demonstrated that some phenoxy-ethanol and phenoxy-acetic acid type compounds showed strong antifeeding action.

In order to study some structure-activity correlations within this group and to find out the structural limits and conditions of this action, further compounds have been tested.

Materials and Method

The tests included compounds, insect species (adults), and plants shown in Table 1. With the three coleopterous species, *L. decemlineata*, *T. dilaticollis* and *C. macula-alba* the feeding tests were carried out and evaluated in the same way as described by JERMY and MATOLCSY (1967). (Number of replicates : 4.) The inhibitory effect on the oviposition of *C. macula-alba* in young poppy capsules was evaluated by comparing the number of eggs deposited into the treated and untreated (control) capsules. (Number of replicates : 4.) With the two leafhopper species, *E. plebejus* and *A. bicinctus*, the rejective effect has been measured by the frequency of the presence of leafhoppers on the treated leaf discs as compared to the untreated ones. Readings were made in one to two hours' intervals. (Number of replicates : 4.)

Results

The results are summarized in Table 1. No unambiguous conclusions can be drawn from these data since the sequence of effectivity of the individual derivatives is different for the different insect species tested, i. e. there is a considerable species-specificity in regard to susceptibility to this type of compounds. Some general tendencies become apparent, however, the most striking of which is the fact that the inhibitory effect is mostly related to the substituents on the aromatic ring, while the O-alkyl part of the molecule seems to play a subordinate role in this respect. Halogen-substitution seems to be a decisive factor, and the 2,4,6-tribromo-

Table 1

Antifeeding, antioviposition, and repellent effect of some alkyl-aryl-ethers

Compounds	Antifeeding			Anti-oviposition	Repellent	
	effect on					
	Leptinotarsa decemlineata Say on potato leaf discs	*Tanymecus dilaticollis* Gyll h. on corn leaf discs	*Ceutorrhynchus macula-alba* Hbst. on poppy leaf discs	*Ceutorrhynchus macula-alba* Hbst. on poppy capsules	*Aphrodes bicinctus* Schrk. on red clover leaf discs	*Eucelis plebejus* Fall. on red clover leaf discs
4-chlorophenoxy-ethanol	+++*	+++	+++	+	+++*	+++*
4-methoxyphenoxy--ethanol	0	+++	0	++	+++*	+++*
3,5-dichlorophenoxyacetic acid	+	+++	++	+++*	+	+
3,5-dichlorophenoxy-acetamide	+	+++*	+++*	+++*	+++	+
3,5-dichlorophenoxy-N-diethylacetamide	0	+++	+	+++*	++	+
2,4,6-trichlorophenyl-methyl-ether	+++*	+++	+	++	+++* p	+++* p
2,4,6-trichlorophenyl-ethyl-ether	+++	+++	+	—	++ p	++ p
2,4,6-trichlorophenoxy-ethanol	+++*	+++	++	+++*	++ p	+++* p
2,4,6-trichlorophenoxy-acetic acid	+++*	+++	0	+	+++* pp	+++ pp
α-(2,4,6-trichlorophenoxy)-propionic acid	+++*	+++	+++	++	+++* p	+++ p
2,4,6-tribromophenoxy-ethanol	+++*	+++*	++	+++*	+++* p	+++* p

Note. +++ = no feeding, no oviposition, no presence (strongest inhibition)
+++ = feeding, presence less than 20% of the control
++ = feeding, presence 20 to 50% of the control, few eggs
+ = feeding, presence 50 to 80% of the control
0 = feeding, presence more than 80% of the control, many eggs (no inhibition)
p and pp = weak and strong phytotoxic effect

substitution results in a more effective phenoxy-ethanol than the 2,4,6-trichloro-substitution. Among the different chloro-substituted derivatives those of 4-chloro- and 2,4,6-trichloro-substitution are highly effective, while the 3,5-dichloro-derivatives show less effectivity.

These findings have reaffirmed our former conclusions (JERMY and MATOLCSY, 1967) according to which 2,4,6-trichloro-phenoxy-ethanol and 2,4,6-trichlorophenoxy acetic acid seem to be the most promising antifeedants of the phenoxy-group as these two compounds fulfill the structural requirements of translocability within the plant and of lack of phytotoxic (hormon-like) activity (WAIN, 1957).

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Chenopodium-Arten und Varietäten als neue Testpflanzen für das Kartoffel-Y- und Kartoffel-X-Virus

Von

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Die verschiedenen *Chenopodium*-Arten als virophile Wirtspflanzen eignen sich zum Nachweis für eine Anzahl von Pflanzenviren. Bei der Identifizierung einiger kartoffelpathogener Viren wurden in erster Linie von den *Chenopodium*-Arten, *Ch. amaranticolor* Coste et Reyn., *Ch. album* L. und *Ch. quinoa* Willd. verwendet (HOLLINGS, 1956; BODE, 1966; VULIČ und HUNNIUS, 1967). In September 1967 an »The 6th Conference of the Czechoslovak Plant Virologists« berichteten wir kürzlich, dass verschiedenen *Chenopodium*-Arten und Varietäten – ausser *Ch. murale* L. – gegenüber Kartoffel-Y-Virus (KYV) anfällig sind (HORVÁTH, 1968).*

In eigenen Untersuchungen wurde der Frage nachgegangen, inwieweit sich das KYV und das Kartoffel-X-Virus (KXV) mit Hilfe von verschiedenen *Chenopodium*-Arten und Varietäten nachweisen lassen. Es wurden acht *Chenopodium*-Arten [*Ch. botrys* L., *Ch. capitatum* L. (Ascherson), *Ch. foetidum* Schrad., *Ch. giganteum* Don., *Ch. glaucum* L., *Ch. murale* L., *Ch. opulifolium* Schrad. und *Ch. quinoa* Willd.] und zwei *Chenopodium*-Varietäten (*Ch. quinoa* Willd. var. *rubescens* und *Ch. quinoa* Willd. var. *viridescens*) mit vier Stämmen des KYV und mit dem sog. X^G-Stamm des KXV (HORVÁTH und BECZNER, 1968) infiziert. Die zu den Versuchen verwendeten KYV-Stämme wurden bereits in früheren Arbeiten beschrieben. Ihre Reaktion bei verschiedenen Pflanzenarten ist teilweise auch mitgeteilt worden (HORVÁTH, 1966a; b; 1967a; b; c; 1969). Die Beimpfungen wurden mit unverdünntem Pressaft unter Verwendung von Karborund mittels Glasspateln vorgenommen.

Alle genannten *Chenopodium*-Arten und Varietäten – ausser *Ch. murale* L. beim KYV – zeigten Symptome der Primärinfektion (Abb. 1 und 2). Etwa 4–6 Tage nach der Beimpfung kamen folgende Symptome vor: chlorotische, rundliche, kleine, graue, nekrotische Primärläsionen und vorzeitiges Absterben der Blätter. Systemische Infektion und auch charakteristische Symptomdifferenzen zwischen verschiedenen Stämmen des KYV und KXV und *Chenopodium*-Arten und Varie-

* Unter der Druckfertigung unserer Symposiumbericht und dieser Arbeit teilte SCHMELZER (1967) mit, dass *Ch. foetidum* Schrad. und *Ch. quinoa* Willd. anfällig und *Ch. murale* L. gegenüber dem Kartoffel-Y-Virus unanfällig sind.

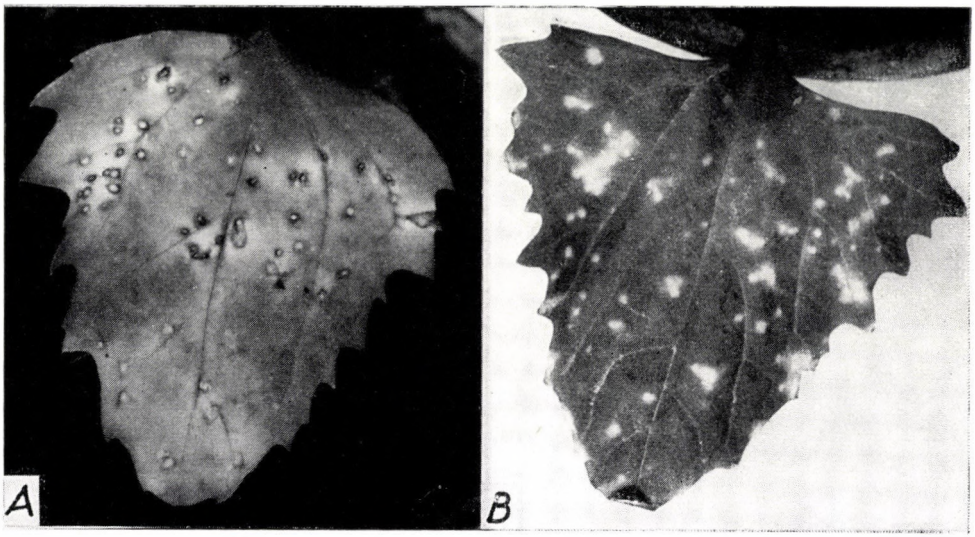


Abb. 1. Symptome des Kartoffel-Y-Virus an *Chenopodium giganteum* Don. (A) und *Chenopodium capitatum* L. (Ascherson) (B)

täten konnten wir nicht feststellen. Die Pflanzen von *Ch. murale* L. widerstanden der Infektion des KYV; sie blieben in den Untersuchungen symptomlos.

In einigen Arbeiten (z. B. KLINKOWSKI und KEGLER, 1962; BODE und VOGEL, 1965) verliefen die Infektionen mit KYV auf *Ch. quinoa* Willd. symptomlos (immun), während in unseren früheren Untersuchungen (HORVÁTH, 1962; 1964) und neuerdings in den Experimenten von DELGADO-SANCHEZ und GROGAN (1966) das KYV neun Tage nach der Beimpfung chlorotische Lokalläsionen hervorrief. Nach unseren früheren Ergebnissen und den von DELGADO-SANCHEZ und Mitarb. mitgeteilten Daten konnten wir nochmals im vorigen Jahre feststellen, dass nicht nur bei *Ch. quinoa* Willd., sondern auch bei den *Chenopodium*-Varietäten *(Ch. quinoa* Willd. var. *rubescens* und *Ch. quinoa* Willd. var. *viridescens)* eine typische, lokale Anfälligkeit gegen KYV besteht.

Nach den Ergebnissen eigener Untersuchungen (HORVÁTH, 1962; 1964) und anderer Autoren (KLINKOWSKI und KEGLER, 1962; UPRETI und NAGAICH, 1963; LINNASALMI, 1964; BODE und VOGEL, 1965 und andere) reagiert *Ch. quinoa* Willd. mit Primärläsionen gegen das KXV. Die im vorigen Jahre wiederholt in den eigenen Untersuchungen erzielten Ergebnisse stehen in Übereinstimmung mit den aus der Literatur bekannten Angaben. Neulich konnten wir feststellen, dass die zwei *Chenopodium*-Varietäten *(Ch. quinoa* Willd. var. *rubescens* und *Ch. quinoa* Willd. var. *viridescens)* auch gegen das KXV mit lokaler Symptombildung reagierten.

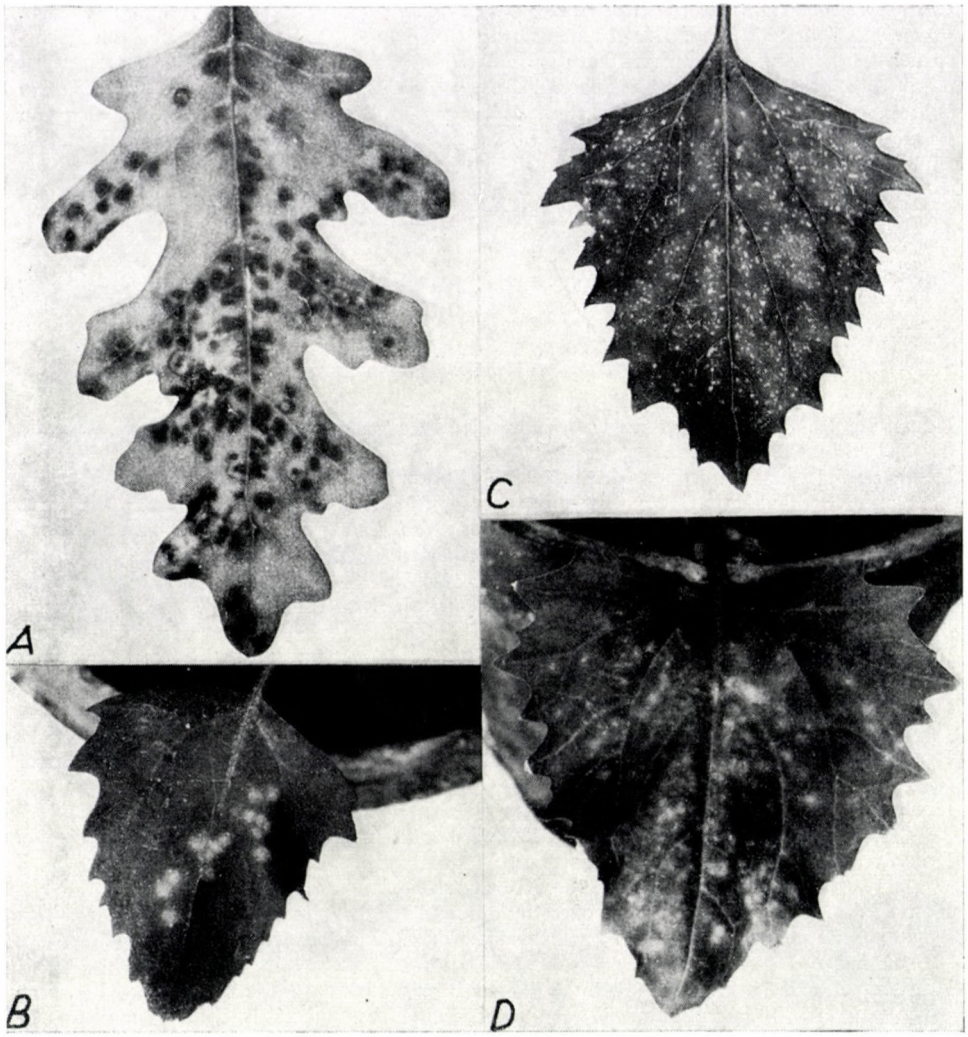


Abb. 2. Symptome des Kartoffel-X-Virus an verschiedenen *Chenopodium*-Arten. A: *Chenopodium foetidum* Schrad., B: *Chenopodium giganteum* Don., C: *Chenopodium murale* L. und D: *Chenopodium capitatum* L. (Ascherson)

*

Fräulein É. Tarcsi sei für ihre Mitarbeit bei der technischen Durchführung der Versuche und Frau O. Kassai für die Photoarbeiten herzlich gedankt.

Anmerkung bei der Korrektur: Nach der Drucklegung kamen uns zwei Arbeiten von B. B. Nagaich und G. C. Upreti (Indian Potato J. *7*, 90—94, 1965) und N. Ghena (An. Prot. Pl. *4*, 89—99, 1966) zur Kenntnis. Nagaich und Upreti stellten fest, dass *Ch. ficifolium* Sm., *Ch. murale* L., *Ch. mittalae* L., *Ch. opulifolium* Schrad. gegen KXV und KYV, und

Ch. ambrosioides L., *Ch. capitatum* L., *Ch. foetidum* Schrad. und *Ch. foliosum* Moench. gegen KXV anfällig sind. Die Ergebnisse an *Ch. murale* L. mit KYV stehen im Widerspruch mit meinen Arbeiten und mit den Feststellung von SCHMELZER (1967). GHENA weist darauf hin, dass *Ch. opulifolium* Schrad., *Ch. glaucum* L., *Ch. botrys* L. und *Ch. foetidum* Schrad. gegen KYV lokal anfällig sind. Es ist erstaunlich, dass in den Experimenten von GHENA die Pflanzen von *Datura metel* L. lokal anfällig gegen KYV waren.

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Book Review

The Dynamic Role of Molecular Constituents in Plant Parasite Interaction. Proceedings of a conference held May 15–21, 1966 at Gamagori, Japan. Edited by C. J. MIROCHA and I. URITANI. The American Phytopath. Society, Inc. St. Paul, Minnesota. 1967. Pp. X + 372.

The publication of the proceedings of this conference is the result of the cooperative effort of scientists of Japan and the United States in trying to solve the problems of the complex host-parasite relationships on a molecular level.

The text is divided into the following chapters: Changes in cytoplasm and cell wall material. Solute and water relations during infection. Metabolic shifts during infection. Toxins of host and parasite in the infection process. Biochemistry of virus infection. Protein metabolism in the host. Altogether 21 papers are published in this volume on different subjects. The form of the book is very attractive and contains a considerable amount of valuable contributions to the biochemistry and physiology of plant diseases. Authors and titles of papers are as follows:

S. AKAI, M. FUKUTOMI, N. ISHIDA and H. KUNOH: An anatomical approach to the mechanism of fungal infections in plants. W. R. BUSHNELL: Symptom development in mildewed and rusted tissues. T. TANI: The relation of soft rot caused by pathogenic fungi to pectic enzyme production by the host. D. F. BATEMAN: Alteration of cell wall components during pathogenesis by *Rhizoctonia solani*. R. D. DURBIN: Obligate parasites: Effect on the movement of solutes and water. A. E. DIMOND: Physiology of wilt disease. C. J. MIROCHA and P. D. RICK: Carbon dioxide fixation in the dark as a nutritional factor in parasitism. J. M. DALY: Some metabolic consequences of infection by obligate parasites. K. TOMIYAMA, R. SAKAI, T. SAKUMA and N. ISHIZAKA: The role of polyphenols in the defence reaction in plants induced by infection. J. KUC: Shifts in oxidative metabolism during pathogenesis. K. TAMARI, N. OGASAWARA and J. KAJI: Biochemical response of plants to toxins produced by the rice blast fungus. R. P. SCHEFFER and R. B. PRINGLE: Pathogen-produced determinants of disease and their effects on host plants. H. OKU: Role of parasite enzymes and toxins in development of characteristic symptoms in plant disease. T. MISAWA and S. KATO: Changes in nuclear contents of host cells infected by virus. T. HIRAI and T. TAKAHASHI: Mitochondrial activities of detached tobacco leaves infected with tobacco mosaic virus — The possible source of energy for virus multiplication. W. N. TAKAHASHI: The biochemistry of plant virus infection. N. SUZUKI: Some properties of the double-stranded RNA of rice dwarf virus. M. E. DEVAY, W. C. SCHNATHORST and M. S. FODA: Common antigens and host-parasite interactions. T. AKAZAWA and L. RAMAKRISHNAN: Change in chloroplast proteins of the rice plant infected by the blast fungus, *Piricularia oryzae*. I. URITANI, T. ASAHAI, T. MINAMIKAWA, H. HYODO, K. OSHIMA and M. KOJIMA: The relation of metabolic changes in infected

plants to changes in enzymatic activity. M. A. STAHMANN: Influence of host-parasite interactions on proteins, enzymes, and resistance.

Following each paper a very valuable text on discussion is published which helps in understanding questions and problems more deeply. This volume is greatly recommended for everyone who is interested in plant pathology on a molecular level.

Z. KIRÁLY

Transactions of XVII. Hungarian Scientific Conference for Plant Protection, Vol. I–II, Budapest 1967. Publication of Hungarian Society for Agriculture.

The variegated and from scientific point of view very interesting texts of lectures held on the XVII. Scientific Conference for Plant Protection was published by the Hungarian Society for Agriculture and by the Company "Agrotröszt" in two big volumes, on 679 pages. By doing this, the two institutions have rendered a substantial service to the professional propaganda, whose aim is to popularize the scientific results in the plant protection practice. The well got-up book, printed with "rotaprint" system, contains not only the texts in Hungarian, but gives also summaries in English or German. So the contents of 103 lectures are easily accessible not only for the Hungarian general public interested in plant protection, but also for people from abroad, as the summaries give ample information about the problems of Hungarian plant protection and about the new results achieved. Special emphasis was laid upon the questions in connection with plant protection of stone fruits, on problems connected with pesticide residues and on complex plant protection systems of alfalfa and other leguminous crops. From the chapters of general plant protection, the problem of technical minimums in field crop protection and the necessity of an up-to-date complex plant protection system of horticultural plants (vegetables) were discussed. In connection with weed control research, data about new and more efficient herbicides and combinations are presented in the volume.

From the interesting phytopathological papers those dealing with virus research and the use of new systemic fungicides against mildews may be mentioned; the zoological sections have dealt mainly with soil desinfectants, prevention of damage caused by game, with biology of new insect pests and, finally, with new methods of the autocidal (sterile male) technique.

It may be stated that the already traditional annual Scientific Conferences for Plant Protection and their publications present every year a broad-scale review of results achieved in the Hungarian plant protection and are of great help both to the subsequent research work and to the agricultural production.

The publication may be ordered from the Hungarian Society for Agriculture or from the Research Institute for Plant Protection.

G. UBRIZSY

S. TERÉNYI, GY. JOSEPOVITS and GY. MATOLCSY: Növényvédelmi Kémia (Plant Protection Chemistry). Academy Publishing House (Akadémiai Kiadó) 1967.

Since many decades there has been in the Hungarian agricultural literature a considerable need of a good review summarizing the most recent results of plant protection chemistry. This gap was filled up by the fine work of 462 pages, written by three outstanding specialists, which comparises and treats all the problems of chemical plant protection.

In the general part of the book the reader may get a thorough information about the significance of plant protection, the various methods of control and, above all, about the importance of chemical control. In the following chapters the main active materials, the methods of formulation and modes of action of pesticides are dealt with. In the special part of the book the fungicides and bactericides are treated thoroughly, then the seed dressing materials, preparates containing copper and other anorganic elements, anorganic sulphur compounds and fungicides with eradicant action are discussed, followed by the curative and systemic fungicides. This chapter ends with the discussion of problems related to antibiotics and phytocides. From the zoocidal materials mainly the insecticides, acaricides, nematocides, molluscicides, rodenticides and other preparates are dealt with. The herbicides are treated in two big groups, the ones with contact action and the ones showing translocation phenomena and systemic action; the authors present here an excellent summary of most recent domestic and foreign results.

At the end of the volume other materials used in plant protection are discussed and among the traditional pesticides the reader may find accounts of insect attractants and repellents, chemosterilants and, finally, of growth regulators and defoliants.

By reading the book and literature cited, it is obvious that the authors mastered their chapters and not only compiled the foreign literature data but incorporated their own results and the ones achieved in other Hungarian research institutions as well. So their work became an indispensable book of reference both for scientists working in pesticide synthesis and research of mode of action and for graduate teaching too. At the same time the plant protection practice cannot dispense with this work, which gives a scientific and most complete synopsis of the extremely differentiated and complex material of up-to-date plant protection chemistry.

Some minor deficiencies have to be mentioned though, as the missing of retardants (CCC, amino-succinic acid etc.) and that of some basic Hungarian literature data.

The authors achieved a pioneer and most honourable work by publishing their "Plant Protection Chemistry" which may give directions for a long time ahead both to chemists, biologists and agricultural experts.

G. UBRIZSY

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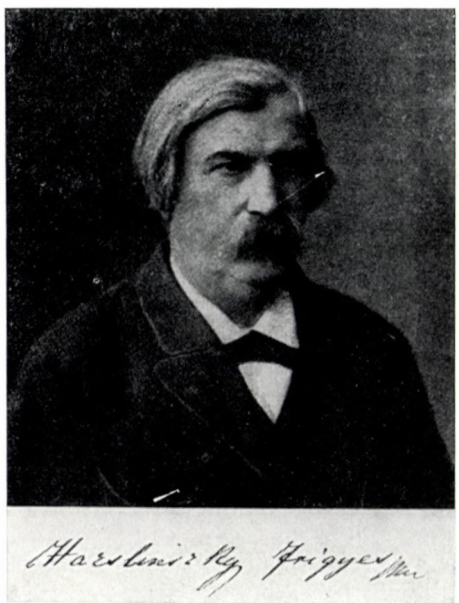
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150th Anniversary of Frigyes Hazslinszky

By

G. UBRIZSY

Research Institute for Plant Protection, Budapest



Mycological research, as is well known, began in Europe with the works of Charles de L'Ecluse (Clusius) in Western-Hungary (Pannonia) (UBRIZSY, 1967). These investigations were continued by J. A. SCOPOLI, I. LUMNITZER, I. ENDLICHER in Hungary in the 18th century. Studies aimed at surveying the total flora of fungi and vegetation of historical Hungary (Carpathian-basin) commenced only in the 19th century, with the extensive investigations of three outstanding mycologists: ISTVÁN SCHULZER of Müggenburg, KÁROLY KALCHBRENNER and FRIGYES HAZSLINSZKY.

From among these, the life-work of ÁGOST FRIGYES HAZSLINSZKY (1818—1896) (Fig. 1) is outstanding for its thoroughness and comprehensive classification of fungi. It was he, who established the Hungarian mycological floristic, system-

atic and plantgeographic research. This was raised to the European level mainly by the works of LÁSZLÓ HOLLÓS (1859—1940) and GUSZTÁV MOESZ (1873—1946). In this way Hungary became renowned for such mycological research all over Europe.

The difficult and thankless pioneering work can be attributed mainly to FRIGYES HAZSLINSZKY, who — in addition to fungi — carried out investigations on algology, lichenology and bryology. He laid the foundations for algology, lichenology and bryology in Hungary with the books, that were written during his monumental life-work. For this reason SÁNDOR MÁGÓCSY-DIETZ in his necrology on HAZSLINSZKY (1889) wrote that "he was the KITAIBEL of the Hungarian cryptogamic flora" and one can find the similar epitaph on his grave in Eperjes (Presov) that was engraved on the forgotten grave of KITAIBEL in Budapest: "Gaude Hungaria quae talem tulisti."

A botanist of remarkable talents, he was born in Késmárk (Kezmarok) on 6th January 1818, of an old Hungarian Protestant craftsman family. (The family originated from Hazslin, and their name was originally SOLTÉSZ, later KEVE.) His mother, ZSUZSANNA KUCHTA, was an adopted daughter of the BADÁNYI family in Szepesség. He completed his primary and secondary studies in Késmárk, and went to Debrecen for one year to perfect his knowledge of the Hungarian language (his homelanguage was German). As a private teacher of the KARSA family he studied law at Sárospatak (1838—39), then he broke off his studies. He studied Evangelical theology in Késmárk, and he obtained a certificate of his college studies in Hungarian language in 1841. Considering himself to be inadequately grounded in natural sciences, especially in chemistry, he enrolled in the high school in Debrecen (1841—43). Here he continued his earlier studies on botany, improving his knowledge in this field, from the big classifying works of the contemporary botanists (ENDLICHER, WAHLENBERG, DIÓSZEGI—FAZEKAS, LINNÉ etc.). His first botanical publications at this time were ("Novum systema regni vegetabilium" or rather "Javaslatok a magyar botanikai terminologia tárgyában" 1844.). He presented these to the Grand Session of the Hungarian physicians and scientists in Temesvár (Timisoara), where the publications were accepted. Being dissatisfied with his studies in Debrecen, he sold his valuable books, and took a journeyman examination on saddlering and wheelwright, to avoid any difficulties in crossing the Austrian border. He went to Vienna and studied chemistry and paleontology at the University of Technical Sciences of Vienna, in 1844—45. He also conducted investigations at the university on plant-physiology, his experiments drawing such wide-spread interest that he intended to apply for the job of honorary lecturer. At this time he received an investigation from home and on the encouragement of his friends — FERENC PULSZKY, PÁL SZIRMAI — members of the great reform generation — he cut short his career abroad, and returned to Eperjes, to accept the ordinary public school teacher's position in mathematics and natural sciences in the college of Eperjes (20th April, 1846). He remained faithful to the alma mater in Eperjes, becoming the director of the college and later the director of the secondary school several times. Here he married TEREZIA PUTZ in 1846. All his life was

dedicated to teaching and botanical research because as he wrote: "The country is poor, very poor, because people cannot comprehend that only the superiority of sciences can conquer the mixture of nationalities." His scientific and pedagogic life-work was led by his love for his country and nation, all the more, because he had to overcome enormous difficulties in the course of his scientific researches. He had to above all eliminate ". . . the insurmountable indifference, which hinders the scientist. This indifference is unrecognized by the leaders of the nation and they do not care for curing this." (A letter to LAJOS JURÁNYI, 1873.) On a call to his patriotism he took part in the war of independence, fighting with his colleagues and students in the battle at Kassa (Kosice) which ended with a disaster. His colleague JÓZSEF SZILVÁSSI died beside him, then — horrified by the war — he turned back to his family and to the alma mater. He continued his life uneventfully. At the age of 78, on the 50th anniversary of his teaching and scientific work, his students and colleagues commemorated his work. He looked back with pride and contentment to the long life that was led and supported by the steady struggle and inspiration.

FRIGYES HAZSLINSZKY began to collect and examine the phanerogamyc flora of the High Tatra (Visoky Tatry) in his student years, he published the works "Eperjes and its surroundings", later "Flora of County Sáros". He travelled over Hungary (Carpathian Basin) with his friend KÁROLY MAUKSCH already in 1841, he returned from the Dalmatian coast with enormous amount of plants. On the stimulation of his students he compiled and wrote the first book on the flora of the Hungarian highland ("Északi magyarhon iránya, 1864"), which was published as "Magyarhon edényes növényeinek füvészeti kézikönyve", comprising all the Hungarian flora. His interest was soon attracted by the cryptogamic flora and vegetation, that was almost entirely neglected in Hungary. He published his monograph "Magyarország és társországai moszatviránya i" already in 1867, in which he could reach only the *Diatomaceae*, but the book comprised the data of earlier research works. (I. LUMNITZER enumerated 10 algae in his work "Flora Posoniensis", but HAZSLINSZKY wrote up the rich collection of A. GRUNOW and SÁNDOR MÁRKUS.) His bryologic studies were presented by his other essays ("Északi Magyarország lombosmohái", "A Kárpátok májmohái", etc.). He published the results of his lichenological collections and investigations already in 1870 as "Adatok Magyarhon zuzmóirányához" which was followed by the monograph "A Magyar birodalom zuzmóflórája", published by the Society of Natural Sciences in 1884. This book would not have been finished without the aid of data obtained by his student, HUGÓ LOJKA (1844—1887) and without the help of ÁRMIN KNAPP in Slavonia. He continued his plant-geographic investigations too and on the basis of data, and observations, collected in the course of his research, he wrote his floristic works "A borsai Pietroz iránya" (1865) and "Tokaj-Hegyalja iránya" (1865).

HAZSLINSZKY's mycological research held a central position in his life. The most travelled Hungarian mycologist, he collected in the whole country. He employed in his monographs not only his own studies, but the data of his students

and other scientists. Though he worked in the age of mycology (see: the big taxonomical life-work of the TULASNE-brothers, DE BARY, O. BREFELD), he worked alone, for this reason he was doubtful about the polymorphism of fungi, the heterogeneity of rusts, the connection between the change of progenies of the conidial and the perfect spore-forms and the experimental (culture) investigations on the relation of the different spore generations. Once he spoke out against the culture method, which led to epoch-making discoveries otherwise ("Eine antijordanische Species." Öst. Bot. Zeitschr. 1880). His view of species was rather conservative, he tended more towards the "big species" than the specialized "small species", which meant the modern trends in the sphere of rusts and smuts for example. On account of these one can find only few new species in his extensive life-work (altogether 100 new plant species were nominated and described by him), because he insisted on the old, classic species or rather on the so called groups of species. (This caused great difficulties to his successors, e.g. to GUSZTÁV MOESZ.) He did not deal with plant pathology but published works on some economically important pathogen fungi (e.g. "Über Pleospora und Puccinia des Spargels", 1864; "Die Sphärien des Lyciums", 1865; "Die Sphärien der Rose", 1870; "Über den Farbstoff des Nussschwammes, Polyporus hispidus", 1870) were all published in the ÖBZ.

As the first result of his assiduity and of his comprehensive work on the taxonomy of fungi, he published a work on mycology in the Journal of the Society of Natural Sciences (Math. Term. Tud. Közl.) in 1861: "Eperjes viránya fényporlói vagy Stilbospórái." Later he published his works in English, German, Austrian etc. journals, such as the Greville, Abhandlungen d.-bot. Vereins d. Provinz Brandenburg and in the ÖBZ mentioned above. From his monographs the outstanding ones are: "Magyarhon hasgombái *(Gasteromycetes)*" 1875; "Magyarhon üszöggombái és ragyái *(Ustilaginales, Uredinales)*" 1876, both were published in the Journal of the Society of Natural Sciences (Math. Term. Tud. Közl.), moreover "Magyarhon myxogasterei (Myxomycetes)" Eperjes, 1877, "Rendhagyó kőgombák (Discomycetes), 1881, "Magyarhon és társországainak szabályos Discomycetjei (Discomycetes)", 1885, "Magyarhon és társországainak Sphaeriái" 1893, all of these were published in the Journal of the Society of Natural Sciences (Math. Term. Tud. Közl.). He published his work "A honi peronospora-félék *(Peronosporales)*" in 1893 in the Booklets of Natural History (Természetrajzi Füzetek).

Parallel with the research of microscopic fungi he devoted enormous time and effort to the study of macro-fungi, although his colleagues I. SCHULZER and KÁROLY KALCHBRENNER were also engaged in this field. He debated with ISTVÁN SCHULZER, who described many new species too quickly, because HAZSLINSZKY thought that the "production of synonyms" was exaggerated (many fungi, written by SCHULZER proved to be synonyms in the subsequent researches) (see the article of HAZSLINSZKY on this as "Hymenomycetologisches" in the ÖBZ, in 1881). The full summary of his macromycologic research which at the same time was his most comprehensive and important work was published in 1895: "Magyarhon és

társországainak húsos gombái *(Hymenomycetes)*" (Journal of the Society of Natural Sciences: Math. Term. Tud. Közl. XXVI.). He was not satisfied with simple floristic statements of species or lists. He tried to relate the spread of macrofungi (chorology) with geographic factors, soil conditions, climatic effects etc., on the basis of the phytogeographic view, which developed after the work of DE CANDOLLE—A. KERNER. In spite of the fact, that he could not finish his work in this respect, the mycochorologic essay published by him was nevertheless an epoch-making work in this subject: "A magyar-honi lemez-gombák *(Agaricini)* elterjedése" was published in the Journal of the Society of Natural Sciences (Math. Term. Tud. Közl.) in 1890. This work dealt with the plant-geographic situation of the group *Agaricales* in Hungary.

The scientific result of his productive, assiduous life was the altogether 118 monographies and bigger studies of scientific demand. His work was followed with great attention by his numerous foreign colleagues. Thus it is obvious that he was appreciated by his students and followers, which is characterized by the list of the new species that were named after HAZSLINSZKY: 1 alga, 17 fungi, 3 lichens, 1 moss, 4 flowering species and 5 fossils. A whole generation of eminent Hungarian botanists was educated by him (e.g. LAJOS JURÁNYI, JÁNOS FÁBRY, HUGÓ LOJKA, LAJOS SIMONKAI, SÁNDOR MÁGÓCSY-DIETZ, ALADÁR RICHTER, LÁSZLÓ HOLLÓS, MÁRTON PÉTERFFY, etc.), these people respected him as their master all the time. As an appreciation of his merits and his scientific activity he was honoured in Hungary, later abroad. He was a corresponding member of the Hungarian Academy of Sciences already in 1863 (his inaugural lecture was held on the "System of Lichens"), becoming an ordinary member of the Scientific Academy in 1886. He was elected honorary member of the Society of Natural Sciences and of the Société Mycologique de France in Paris in 1889. At the end of his life in 1890, the king affirmed his ancient nobility, and the "of Hazslin" title of nobility was given to him and his successors.

The collection of his flowering and vascular cryptogam plants, consisting of about 11 200 species was given to the National Museum (at present in the Botanical Collection of the Museum of Natural Sciences) together with his collection of fungi. The collection of duplicates of fungi was presented to the Botanical Institute of the University in Budapest.

Born 150 years ago, he established with his great investigations not only the sure and broad foundations of the Hungarian mycological researches, but also set the trends and mode of investigations of the cryptogam flora and vegetation. It is justifiable therefore to pay our respects to HAZSLINSZKY and appreciation of his great efforts.

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Role of Epidermis in Necrotic Lesion Formation on Leaves of *Nicotiana glutinosa* by Tobacco Mosaic Virus and Its Nucleic Acid

By

A. YAMAGUCHI and T. SHIMOMURA

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When TMV was rubbed on the lower surface of *N. glutinosa* leaves from which the lower epidermis had been removed, a few necrotic lesions appeared on the stripped area. They appeared a little bit later than those on epidermis-attached area and less coloured than normal lesions. When TMV was rubbed on the upper surface from which the lower epidermis had been partly removed, as many lesions were produced on areas devoid of epidermis as on the attached area. Lesion numbers produced on stripped areas from which the lower epidermis was removed 20 hours after rub-inoculation on the surface, are the same as those on the attached area. TMV-RNA behaved similarly to whole TMV in respect of lesion formation on stripped areas.

TMV introduced directly into parenchymatous tissue of *N. tabacum* var. Bright Yellow could initiate infection and multiplied considerably, though the virus titer was less than that of epidermis-attached discs.

From these results we suggested that the epidermis is necessary for necrotization of leaf cells although naked parenchymatous cells sustain virus establishment and reproduction.

Introduction

In leaves plant virus is introduced to the epidermal cells through slight wounds that were made by rubbing the leaf surface with or without Carborundum. KONTAXIS (1961) suggested that the presence of epidermal tissue was required for necrotic lesion formation in leaves of *Nicotiana glutinosa* by tobacco mosaic virus (TMV) infection, because of his failure of necrotic lesion production on leaf areas devoid of epidermal tissue. In contrast, DIJKSTRA (1964) and YAMAGUCHI and SHIMOMURA (1964) could obtain a few necrotic lesions on leaf areas from which the epidermis was peeled away. This paper aims to elucidate the role of epidermal tissue in necrotic lesion formation by virus infection.

Materials and Methods

Plant material

Nicotiana glutinosa L. and *N. tabacum* L. var. Bright Yellow plants were used as local lesion and systemic hosts for TMV, respectively. The lower epidermis was partly peeled away from the detached leaves. Inoculum was added by rubbing

either on the lower or the upper surface. For protecting the leaf area devoid of epidermis from drying, the detached leaves are incubated: 1. in Petri dishes in which there is moist filter paper, 2. in Petri dishes in which there is plain agar, and sometimes 3. whole plant with leaves from which the lower epidermis was peeled are placed in a moist chamber covered with a Glass Bell Jar. Best results were obtained by using method 2. Petri dishes were placed in the chamber under fluorescent light at about 25°C.

Virus

Tobacco mosaic virus, ordinary strain, purified by differential centrifugation was used. TMV-RNA was prepared by the method of FRAENKEL-CONRAT et. al. (1961).

Results

Local lesion formation in N. glutinosa leaves with whole TMV

1. *Formation of necrotic lesions in leaf areas devoid of epidermis*

Epidermis of lower surface was peeled away partly and TMV (0.1 or 1 mg/ml) was applied to the surface by using various ways. No lesions were produced when the inoculum was poured on or infiltrated *in vacuo* in the stripped area. A few lesions, however, appeared when the surface was rubbed with cotton pad swabbed in the TMV solution. When adding Carborundum, a little bit more lesions appeared.

2. *Comparison of lesion number of the stripped area with that of the attached area*

Epidermis of lower surface was partly peeled away. The surface was rubbed gently with cotton pad swabbed in TMV solution with 600 mesh Carborundum. The inoculated leaves were incubated in three ways as has been described. Results are shown in Table 1. A few lesions appeared on the area devoid of epidermis, while abundant lesions appeared on the attached area. Lesions produced were counted and number was converted on the basis of the same leaf area for both epidermis-stripped and attached area. Necrotic lesions on stripped areas are apt to form on small lateral veins and to enlarge along them. They became visible a little bit later than normal lesions and their colour was grayish brown compared with the dark brown on normal area.

3. *Effect of adding Bentonite to the inoculum on lesion number*

Since naked parenchymatous cells contact with the inoculum directly, it is naturally supposed that the given inoculum may be destroyed by leaf ribonuclease before establishment on leaf cell. Three to five mg per leaf of Bentonite were added to TMV inoculum. Number of necrotic lesions obtained was 8 and 22 in stripped area while 455 and 150 on normal area, respectively, in two replications. We could not see great improvement in the number of necrotic lesions on stripped areas by adding Bentonite.

Table 1

The ability of necrotic lesion formation in stripped areas of *N. glutinosa* leaves from which the lower epidermis was removed and was inoculated on the surface with tobacco mosaic virus

Expt. No.	Concentration of TMV (mg/ml)	No. of leaves	No. of lesions produced on*	
			Epidermis-stripped area	Epidermis-attached area
1	1	3	1	23
2	1	3	2	abundant
3	1	4	3	57
4	1	5	2	45
5	1	6	2	176
6	1	6	1	214
7	1	9	66	234
8	1	6	0	283
9	0.1		0	abundant
10	0.1		18	268
11	0.1		22	160
12	0.1		0	219
13	0.1		2	312

* No. of lesions produced was based on the same area for both epidermis-stripped and attached areas.

Table 2

The ability of necrotic lesion formation in stripped areas of *N. glutinosa* leaves from which the lower epidermis was removed and was inoculated on the upper surface with tobacco mosaic virus

Expt. No.	Concentration of TMV (mg/ml)	No. of leaves	No. of lesions produced on*	
			Epidermis-stripped area	Epidermis-attached area
1	1	6	73	242
2	1	5	197	453
3	0.1		167	236
4	0.1		253	220
5	0.1		91	101

* No. of lesions produced was based on the same area for both epidermis-stripped and attached areas.

4. *Lesion formation on the upper surface of leaves from which lower epidermis had been peeled away*

Epidermis of the lower surface was peeled away partly in a mosaic pattern. TMV was rub-inoculated on the upper surface. Many necrotic lesions appeared

on the leaf area of the upper surface corresponding with the leaf area from which the lower epidermis was peeled away as well as the normal area. The comparison of lesion numbers in both areas is shown in Table 2.

5. *Lesion formation on stripped areas of N. glutinosa leaves from which inoculated-epidermis was peeled away 20 hours after inoculation*

Lower surface of leaves were inoculated with TMV (0.1 mg/ml). After 20 hours, the epidermis of this surface was peeled away. As many lesions as in the epidermis-attached area were produced in the stripped area (Fig. 1). Results are shown in Table 3.

Table 3

The ability of necrotic lesion formation in stripped areas of *N. glutinosa* leaves from which the lower epidermis was removed 20 hours after inoculation on the lower surface with tobacco mosaic virus

Expt. No.	Concentration of TMV (mg/ml)	No. of leaves	No. of lesions produced on*	
			Epidermis-stripped area	Epidermis-attached area
1	0.1	6	644	630
2	0.1	6	1041	1123
3	0.1	6	578	573

* No. of lesions produced was based on the same area for both epidermis-stripped and attached areas.

Table 4

The ability of necrotic lesion formation in stripped areas of *N. glutinosa* leaves from which the lower epidermis was removed and was inoculated on the surface with nucleic acid isolated from tobacco mosaic virus (TMV-RNA)

Expt. No.	Concentration of TMV-RNA (mg/ml)	No. of leaves	No. of lesions produced on*	
			Epidermis-stripped area	Epidermis-attached area
1	1.2	4	7	31
2	1.2	6	16	82
3	1.2	6	4	16
4	0.6	8	2	15
5	1.0	10	23	295
6	1.7	6	2	354
7	0.5	10	6	93
8	1.0	6	0	24

* No. of lesions produced was based on the same area for both epidermis-stripped and attached areas.

Local lesion formation in N. glutinosa leaves with TMV-RNA

One of the reasons for the production of less necrotic lesions by whole TMV on the leaf area devoid of epidermis may be as follows. The virus that was introduced into the epidermal cell uncoat there and naked nucleic acid infect parenchymatous cells that layered under epidermal cells. If this occurs parenchymatous



Fig. 1. Necrotic local lesions produced on epidermis-stripped areas of *N. glutinosa* leaf from which the lower epidermis was partly removed 20 hours after inoculation of TMV

cells might require naked RNA from TMV. The following experiments were performed to ascertain this point.

Epidermis of lower surface was peeled away partly and TMV-RNA was inoculated on the surface by rubbing with Carborundum. Number of necrotic lesions produced on stripped area did not increase markedly by inoculation of TMV-RNA comparing with that of whole TMV (Table 4). When lower epidermis

was peeled and inoculated on upper surface, 43 lesions were formed on leaf area of upper surface from which lower epidermis was peeled away, while 93 lesions on normal area. When lower epidermis was peeled away 20 hours after inoculation on lower surface, 52 and 138 lesions were obtained for epidermis-stripped and attached area, respectively. All circumstances in lesion production were the same with inoculation by whole TMV.

TMV reproduction in N. tabacum leaf areas devoid of epidermis

Another reason for less production of necrotic lesions on the stripped area of *N. glutinosa* leaves may be considered as follows: Naked parenchymatous tissues from which epidermal layer is removed cannot sustain virus multiplication by rubbing inoculation because of their heavy damage. To ascertain this, the following experiments were done. Part of the lower epidermis of *N. tabacum* var. Bright Yellow, systemic host for TMV, was peeled away and TMV (0.1 mg/ml) was rubbed on the surface with Carborundum. Epidermis-free and attached discs were punched and incubated in moist Petri plates under fluorescent light at 25°C for 5 days. Recovery test of the virus from both leaf discs was done by bioassay using *N. glutinosa*. Results are shown in Table 5. Virus content in epidermis-free leaf discs was less than that in epidermis-attached leaf discs. It is clear, however, that infection did occur and a considerable amount of virus multiplied within epidermis-free leaf discs.

Table 5

The ability of virus reproduction in stripped area of *N. tabacum* var. Bright Yellow leaves from which the lower epidermis was removed and was inoculated on the surface with tobacco mosaic virus

Expt. No.	TMV titer* in	
	Epidermis-stripped area	Epidermis-attached area
1	24.9	57.8
2	14.3	58.0
3	11.1	99.0
4	34.6	107.0
5	22.3	29.5

* Lower epidermis of Bright Yellow leaves was partly removed. After rub-inoculation with TMV (0.1 mg/ml) on the surface, discs from epidermis-stripped and attached areas were punched and incubated under fluorescent light at 25°C. After 5 days, these discs were homogenized and assayed the virus content multiplied in the discs by inoculation of leaves of *N. glutinosa*. Figures are the number of lesions produced on *N. glutinosa* leaf area of 10 cm^2.

Discussion

KONTAXIS (1961) suggested that epidermal tissue may be necessary for the necrotic lesion formation. This suggestion is derived from his results that no necrotic lesions developed on leaf parts devoid of the upper epidermis following inoculation of the stripped area. However, DIJKSTRA (1964) as well as we could obtain necrotic lesions on the stripped area. The number of the necrotic lesions produced on the stripped area was much less than that of the attached area for rubbing inoculation with Carborundum. The ratio of lesion numbers on attached area to stripped area is summarized in Table 6. When the lower epidermis was removed and inoculated on its surface, the ratio was 16.6 and 15.1 for inoculum of TMV and TMV-RNA, respectively. In contrast to this, when the upper surface was inoculated from which lower epidermis had been removed the ratio was 1.6 and 2.2, and when the lower epidermis was removed 20 hours after inoculation of the same surface the ratio was 1.0 and 2.6. These figures show clearly that marked reduction in lesion number could be prevented when the upper epidermis of a leaf from which the lower epidermis had been removed, was rub-inoculated or when inoculated epidermis remained for 20 hours after inoculation.

Table 6

The comparison of the ability of necrotic lesion formation in epidermis-stripped and attached areas of *N. glutinosa* leaves by TMV and TMV-RNA (summarized data)

Experimental methods	Inoculum	
	TMV	TMV-RNA
When inoculum was rubbed on the lower surface of leaves from which the lower epidermis had been removed	16.6*	15.1
When inoculum was rubbed on the upper surface of leaves from which the lower epidermis had been removed	1.6	2.2
When the lower epidermis was removed 20 hours after rubbing inoculation on the lower surface of leaves	1.0	2.6

* The figure shows the ratio of the number of necrotic lesions produced on the epidermis-attached area of *N. glutinosa* leaves to those on stripped area.

A possibility of the difficulty in lesion formation might be the harmful effect of the stripping procedure (DIJKSTRA, 1964; YAMAGUCHI and SHIMOMURA, 1964). However, the successful multiplication of the virus within stripped area of *N. tabacum* may suggest that the introduction of the virus into parenchymatous cell and subsequent multiplication could occur even in stripped area despite of low efficiency caused by considerable damage of peeled cells. Another possibility might be differential distribution of phenolic substances such as chlorogenic acid between epidermal and parenchymatous tissue. The less darkened lesions in stripped area suggested this possibility, though we have not yet examined it.

Thus, the process of necrotic lesion formation within epidermis-lacking area may be traced as follows. When epidermal strips are peeled away, most parenchymatous cells are too damaged to be able to sustain virus reproduction anymore. Some cells, however, remain acceptable for the virus. The virus that was introduced directly into parenchymatous cells multiplies within them. Soon after the infection process commences, cell necrotization begins. Epidermal tissue must play a role in this process although its nature is not yet clear. This assumption may be strengthened by the following observation. The appearance of necrotic lesions on the stripped area is delayed a few days as compared to the normal area. The number of necrotic lesions on stripped area increases gradually up to 8 days after inoculation, while on normal area get its maximum number within 4 days. This may be explained that initiation of necrotization process in stripped area requires association with the epidermis of opposite side. So, it might take time to reach virus to this side where virus multiply and cooperate for necrotization. When the lower surface was peeled and inoculated on the upper surface or when lower epidermis was removed 20 hours after inoculation, lesion appearance on both attached and stripped areas was almost at the same time. Moreover, the results of experiments of 20 hours later peeling might show that the necrotization process begins during early stage of virus multiplication. In conclusion, we might say that parenchymatous cells permit virus reproduction but they need cooperation with epidermal cells for their necrotization.

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The Control of Potato Degeneration by the Glass-Tube Test

By

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The author tried to establish correlations between the positive (red colour) and negative reaction observed in potato tubers and their degeneration.

The reaction was affected by the storage temperature. If stored at 1—5°C, by the end of winter the number of tubers showing negative reaction increased by 11%. In tubers stored at 10—12°C the increase amounted to 64%.

Between harvest and planting time the change of reaction varied according to size group. In the large and medium tubers the increase of the number of negative tubers amounted to 10% in the average, in the small tubers it reached 44%.

There was a certain correlation between the reaction of the parent tuber and that of the clone generation. In the clone generation derived from positive parent tuber, the proportion of positive tubers to negative ones was 11 : 1. In contrast, in the clone generation of a negative parent tuber the proportion was 2 1/2 : 1. When testing the hills containing the yield of positive parent tubers a larger proportion of purely positive hills was found than in the hills of negative parent tubers.

The type of reaction changed in the same variety depending on whether it was planted in the spring or during the summer.

The yield of negative seed tubers was 27% less than that of positive seed tubers.

The smallest yield was observed in the negative progenies of negative tubers, where the number of tubers was 45% less and the weight 87% less than that of the positive tubers.

Out of the methods used to study the degree of degeneration in potatoes only the biuret reaction showed closer correlation with the GT.

Introduction

We refer, first of all to the paper (SZIRMAI, 1960, 1963) published in 1960 in which the underlying ideas of the glass-tube test are described and the correlations, on the basis of the tests carried out with the aid of this method, are established.

In this work it was possible to separate with the aid of positive and negative reactions the vital seed tubers of higher polyphenoloxidase activity and lesser virus infection, on the one hand, and the avital tubers of reduced polyphenoloxidase activity and more intense infection, on the other. In recent years on the basis of these observations we tried to establish correlations, mostly in field experiments, between the reaction of glass-tube-tested potato tubers and their yield,

between the two reaction types (plus or minus) of the clonal generation and their respective yields and between the tubers thus separated and certain symptoms characteristic of degeneration.

Materials and Methods

As known from the previous publication the glass-tube (GT) testing consists in inserting a short glass-tube into the tuber, and introducing in this tube 0.1 ml normal sheep serum, containing phenol. The serum reacts with the juice of the tuber accumulating in the tube from the damaged tissues. The reaction is considered positive if a red ring is formed on the surface of the serum. The reaction is negative when no ring is formed.

A strong reaction is indicated by $^{++}$, a weak reaction by $^{+}$ and the absence of ring formation by^{-}.

The potatoes, mainly "Gülbaba" variety, were separated by the described method, and the length and weight of 100 tubers were established in both groups. The potatoes were graded in 6 classes according to length between 1 cm and 12 cm. Results are related to the positive reaction, since the tubers belonging to this group were of better quality.

The tubers separated on the basis of the glass-tube reaction were planted and the clonal generation was again tested in the same way.

The same material was tested with other methods used to establish degeneration in potatoes, as follows: the copper-test according to BECHHOLD (BECHHOLD and ERBE, 1932), Fehling's test (SCHUSTER, 1956), Igel—Lange test (IGEL and LANGE, 1955), potassium permanganate test (SCHUSTER, 1962), the biuret reaction (FRIEDRICH, 1938), UV-test (MCLEAN and KREUTZER, 1944).

Results were obtained from the study of 2600 tubers and expressed in percentage.

Results

It was established that the groups separated on the basis of glass-tube testing were not consistent but underwent modifications under the effect of the environment. During longer experimental periods the group showing positive reaction shifted toward negative, or was transformed completely. The opposite, a shift toward the positive reaction was never observed.

For the change in reaction various factors may be responsible: the storage temperature, the effect of storage from autumn to spring, the type of reaction shown by the parent tuber, the scale relations of the tuber.

Conditions of storage

The potato tubers were strongly affected by the storage conditions. There was a great difference between storage in a cellar at 10 to 12°C, or outside in a cool place (between 1 and 5°C). When stored in a warm place most of the tubers

lost the favourable positive reaction. The potatoes stored outdoors became negative to 11%, those stored in the cellar to 64%. Thus the relative shift to negative amounted to 53% (Table 1).

Table 1

The GT reaction of the tuber as affected by storage temperature

Time of test	Storage temperature				Relative shift toward negative reaction
	1–5°C		10–12°C		
	+	–	+	–	
Autumn (October)	79	21	79	21	
Spring (February)	68	32	11	89	→ 53 %

Changes occurring between harvesting and planting

There was a shift in the reaction observable between harvesting and planting. As mentioned a substantial change occurred. The change was most spectacular in the smaller size tubers. The worst the conditions of storage were the greater was the change that occurred. From autumn to spring under favourable "cool" storage conditions the reduction of positive reaction in the tubers amounted to about 10%, in the larger size groups (8—11 cm and 6—8 cm group). However, the situation was different in the group of small tubers (3—5 cm). The difference in the size in relation to reaction manifested itself already at the time of digging up. Among the smaller tubers about 5% more showed negative reaction, than among the larger ones (Table 2).

Table 2

The shift of reaction between harvest and planting time as a function of tuber size

Time of test	Large 8–11 cm		Medium 6–8 cm		Small 3–5 cm		Relative shift toward negative reaction
	+	–	+	–	+	–	
Autumn (October)	80	20	82	18	76	24	
Spring (February)	72	28	72	28	32	68	
						44	→ 34 %

The effect of the parent tuber

The tendency of the reactions shifted also in relation to the reaction of the parent tuber. In other words the progeny were affected by the character of the parent tuber. The reaction type of the clonal generation was studied as a function

of the type of the parent generation. Apart from the diagnostic aspect this problem has a genetic aspect as well. In Table 3 the reaction type of the clonal generation

Table 3

The clone generation classified according to their positive or negative character as established by GT

Parent tubers	++		+		−	
Clonal generation related to potato variety	+	−	+	−	+	−
"Gülbaba" I	87	13	79	21	70	30
"Gülbaba" II	83	17	80	10	40	60
"Rózsa"	98	2	94	6	85	15
"Somogyi sárga" I	96	4	92	8	80	20
"Somogyi sárga" II	92	8	84	16	81	19
Appr. proportions	11 : 1		7 : 1		2 1/2 : 1	

is shown as a function of the reaction type of the parent generation in three potato varieties. ("Gülbaba" I and II, "Rózsa" and "Somogyi sárga" I and II.) In this experiment the majority of tubers coming from positively reacting parent tubers showed positive reaction and only about 2 to 17% became negative. The clonal generation of negative parent tubers is also mixed. We would have expected to find in the majority of cases negative tubers, however, the majority was positive. At the same time the proportion of negative tubers was higher than in the clonal generation from positive parent tubers (15 to 60%). The group of lesser positive activity, marked +, occupies an intermediate position (6—21%). Thus the proportion of positive and negative tubers in the clonal generation is 11 : 1, 7 : 1, 2 1/2 : 1 in relation to double cross, single cross and negative parent tubers, respectively.

Thus the reaction type of the parent tubers does not determine that of the clonal generation, it determines only the tendency.

It is interesting to investigate the distribution in one clone. No direct correlation could be observed. In some of the hills the reaction type of the parent tuber prevailed, in others, however, tubers of mixed character were found.

An interesting regularity could be observed in the distribution of the tubers within a single hill (Table 4). The clonal generation in hills of positive parent tubers was more often positive, than negative.

Double cross, single cross and negative tubers, 100 each, were planted in the field. The clonal generation was examined by glass-tube test (GT) to establish correlation between the character of parent and clonal generations.

The proportion of single plants yielding only positive or negative tubers in relation to the character of the parent tuber was the following: 5 : 1 : 1, 2 1/2 : : 2 : 1, and 1 : 1 : 1.

Table 4

The distribution of reaction type per hill of positive or negative parent tubers, respectively

Reaction in the parent tuber	Reaction in the clonal generation per hill			Proportion of categories according to reaction
	++	±	−	
+ +	70	15	15	5 : 1 : 1
+	48	34	18	2 1/2 : 2 : 1
—	32	34	34	1 : 1 : 1

± Mixed hills containing tubers showing positive and negative reaction.

Investigations were carried out with tubers after longer storage periods. It was established that sprouted tubers rapidly lost their positive character.

In the majority of cases the tubers with thread sprouts and the aged tubers showed negative reaction.

The type of characteristic reaction may change within the same test material or even in the same tuber, as seen from the following experiment.

In 1965 a uniform GT positive seed material of the "Epoca" potato variety was used. A part of the tubers was planted early April. The yield was harvested in August and proved to be of positive character. The other half was kept till the middle of summer below ground where the temperature rose from the initial 5°C to 22°C. These tubers showed all negative reaction. They were divided in two lots. One half was planted early July. The second half was stored in a refrigerator at 5°C for another two months. The potatoes lifted in the first half of November showed positive reaction, whereas the lot kept under refrigeration was all negative.

Thus it was proven that the clonal generation may be of positive character even if the parent tubers turned negative. However, the parent tubers, once turned negative, become irreversible. The compound causing the colour reaction (polyphenoloxidase) seems to be inactivated and cannot be reactivated in the same tuber, however, in the clonal generation it might be freshly formed.

When tubers were examined the parent tuber proved to be negative while the filial tuber at the same time positive.

Crop characteristics as a function of the type of reaction

The distribution of yield according to length of tuber and output was then examined as a function of the reaction type.

For these experiments tubers of uniform character were chosen. Their length varied between 8.5 and 9 cm and the weight of each tuber from 75 to 80 gr. The material was tested by the GT prior to planting. The distribution of positive and negative entities was accidentally 50%. From every one of the three groups (+ +,

+ and —) 50 tubers were planted. The yield was then classified according to tuber length in order to be able to relate the results to the reaction type.

In these investigations from the previously introduced six grades only three were taken into account (3 to 6).

As seen in Table 5 the uniform character of the parent tubers changed in the clone generation in several respects, particularly in relation to tuber weight. The distribution according to type of reaction did not change to a great extent, however, the yield decreased from 22.1 kg/hill or 21.9 kg/hill to 16.2 kg/hill in the groups of high, low and no polyphenoloxidase activity, respectively. Thus in the group of negative character the yield amounted to 27% less than in the group of high activity. The low yield derived mainly from inproportionate reduction in the number of large size tubers (grades 5 and 8). In accordance with the decrease in yield the average weight of tubers decreased also in the group showing negative reaction.

Table 5

The classification of tubers according to length and weight in relation to the result of glass-tube test

Yield	Class. according to size	The reaction in the parent tuber			Decrease of yield
		++	+	−	
50 parent tubers, each were chosen according to reaction	6	18	18	12	
	5	109	105	85	
	4	131	141	161	
	3	118	102	114	
Total number of tubers		376	366	372	
Weight, kg		22.1	21.9	16.2	→ 27%
Average tuber weight, g		58	60	43	
GT negative reaction (%)		15	→ 27	→ 37	

Classes of size: 6 = 12 cm, 5 = 8—11 cm, 4 = 6—8 cm, 3 = 3—5 cm.

It was interesting to note that with decreasing enzyme activity the potato yield decreased.

In the second experimental year the crop derived from positive and negative parent tubers was examined (Table 6). 100 tubers both of positive and negative reaction were planted and the yield per hill was examined by the GT method. The yield was then classified according to tuber length into 4 classes so as to be able to see the frequency of the smallest sizes.

It was established that in the negative clonal generation of the negative parent tubers the number of tubers was 45% less.

There was no substantial change as regards classes according to size. However, the crop weight decreased uniformly with the reduction of activity. The

Table 6

The classification of the clonal generation, according to length, size and weight as a function of GT positive, or negative parent tuber

Clonal generation	Class. according to size	Parent tuber				Decrease of yield
		+		−		
		+	−	+	−	
The yield per hill of positive and negative parent tubers, 100 each	4	160	50	30	0	
	3	220	180	170	170	
	2	30	90	60	60	
	1	10	0	0	0	
Total number of tubers		420	320	260	230	→ 45 %
Weight, kg		9.0	4.0	2.5	1.2	→ 87 %
Average tuber weight, g		22	13	9	5	

Classes of size: 4 = 6—8 cm, 3 = 3—5 cm, 2 = 1—2 cm, 1 = 1 cm.

decrease in weight amounted to 87% in the negative yield of negative parent tubers. This experiment was carried out under unfavourable conditions, thus the yield was lower than the average. This circumstance might have influenced also the extensive drop in yield of negative tubers.

Correlations between reaction type and degeneration

The GT method did not prove suitable to differentiate between virus infected and uninfected individual tubers, thus it could not be used for virus diagnosis in the laboratory. However, in mass tests there was a differentiation according to reaction type in various potato varieties.

None the less the differences were not absolutely consequent. When field experiments were evaluated the results were mixed. In certain years and varieties the correlation between GT reaction and virus infection was nearly significant whereas in others the correlation was loose.

When other methods for the establishment of the grade of degeneration were used, as mentioned above, the results in general were negative. A certain correspondence was found however, between the biuret reaction and the GT reaction. This amounted to 90% in the case of positive glass-tube reaction and to 70% in the case of negative reaction. In a detailed experiment the samples giving positive biuret reaction amounted to 84% of the samples found positive with glass-tube test, and 76% of the GT negative samples proved negative with biuret test (Table 7). Next best results were obtained with the Kaho-test, where the agreement was 50 and 60%, respectively. Similar results were found with the Igel—Lange reaction. The results of other methods could not be evaluated because of the extensive deviation.

Conclusions

From the experiments several useful conclusions may be drawn. The most desirable, namely, to find a safe method for virus diagnosis, was not achieved, however, various physiological phenomena, hitherto unknown were correlated. The glass-tube test gives a qualitative as well as a quantitative evaluation since the intensity of colour is characteristic of the enzyme activity.

Table 7

The correlation between the biuret reaction and positive and negative GT reaction in tubers

Sample	Biuret+ / GT+	Biuret− / GT−
1	8/10	7/10
2	9/10	7/10
4	6/10	9/10
5	9/10	7/10
Correspondence	84%	76%

The denominator contains the number of tubers investigated by GT, and the numerator those by biuret reaction.

A definite correlation was established between the vitality of the tuber (in the field) the size of tuber and the yield, on one hand, and the positive glass-tube reaction, on the other. Similarly correlation was established between the negative glass-tube test and the opposite characteristics. The shift of the positive reaction in the direction of the negative was accompanied by a shift toward unfavourable characteristics.

The existence of a genetic correlation is shown by the fact that the reaction of the clone generation corresponded mostly with that of the parent tuber, sometimes a completely uniformly positive or negative clone was found in a single hill. The distribution may be characterized by a definite proportion.

For the changes relative to environmental factors the reduction, disappearance or transformation of a compound present in the tuber seems to be responsible.

From the environmental effects that of temperature was investigated. The results correspond to those found by other research-workers, studying the same

subject (LIN CH'WAN-KWANG, TIEN PO, 1961; NECHYPORCHUK, 1965; SZIRMAI, 1951; TIZIO, 1952).

Although no direct relation could be established between the glass-tube test and virus infection, the fact that negative reaction was much more frequent in small tubers, further, as seen in earlier experiments (SZIRMAI, 1950), in the older, degenerated varieties, seems to prove the presence of certain correlations.

However, the investigation of parent and filial tubers points to the contrary, since the filial tuber of a negative parent was positive, whereas in reality the filial tuber of a virus infected tuber is usually also virus infected.

Similarly, the change of positive reaction to negative in the Epoca variety during storage seems to disprove the correlation between virus infection and reaction type.

Probably by extending the experiments to follow the phytopathological process of potato degeneration with the GT would elucidate the correlation of the virus infection on the one hand and physiological changes, on the other.

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Ein Beitrag zur Verwendung des *Solanum demissum* Bastards A6 als Testpflanze für den Nachweis des Kartoffel-Y-Virus

Von

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22 aus verschiedenen Ländern stammende Kartoffel-Y-Virusisolate, von denen 16 in die normale Stammgruppe (Y^N), zwei in die Gruppe der Tabakrippenbräunestämme (Y^R), drei in die Gruppe anomale Stämme (Y^{An}) und einer in die Stammgruppe C (Y^C) gehörten, wurden in zwei Untersuchungen auf ihre Nachweismöglichkeit mittels des *Solanum demissum* A6-Blatt-Testes geprüft.

Es konnten Unterschiede in der Form der Läsionsausbildung, der Schnelligkeit des Auftretens der Läsionen und der Sicherheit des Virusnachweises gefunden werden. Es zeigte sich, dass der A6-Blatt-Test nicht auf alle geprüften Kartoffel-Y-Virusisolate ansprach.

Einleitung

Seit KÖHLER (1953) aus einer Kreuzung von *Solanum demissum* Lindl. × *Solanum tuberosum* L. cv. *Aquila* die auf einige Pflanzenviren gut ansprechende Hybride A6 fand, wird diese Pflanze in zunehmendem Masse zum Nachweis des Kartoffel-Y-Virus (*potato virus* Y) benützt (BODE, 1959, 1963; ARENZ und VULIČ, 1961; KELLER und BÉRCES, 1962; HORVÁTH, 1962, 1964; MÜNSTER und CORNU, 1962; WENZL, 1963, 1967; HEIN und BARTELS, 1963; DE BOKX, 1963; MICZYŃSKI, 1965; NOHEJL, 1966 und andere).

Die Einzelpflanze ergibt durch ständigen Neuaustrieb aus den Achselsprossen ausreichend Testblätter, von denen jedes gesunde und frische Blatt im Schalentest zum Virusnachweis einer zu untersuchenden Probe dienen kann. Lediglich in ungünstiger Jahreszeit bereitet die Anzucht einige Schwierigkeiten, die aber durch den Einsatz von Zusatzbeleuchtung und die Verwendung *physiologisch* jüngeren Pflanzgutes (BATENBURG, 1965) zu umgehen sind. Durch die Möglichkeit der Wiederverwendung von negativ reagierenden A6-Testblättern für eine zweite Inokulation (HUNNIUS et al., 1964) ergibt sich eine hohe Blattausbeute pro A6-Pflanze.

Die Technik des A6-Blatt-Testes wurde wiederholt in der Literatur beschrieben (KÖHLER, 1953; BODE und BERCKS, 1959; DE BOKX und HIDDEMA, 1961; VULIČ und ARENZ, 1963; ZIMMERMAN-GRIES und NITZANY, 1963; STAUDERER,

1963; De Bokx, 1964; Münster, 1965; Scheller, 1965; Keller und Bérces, 1966 und andere*).

Der Grad der Sicherheit des Testes hängt zweifellos von der Viruskonzentration der zu untersuchenden Probe, der Bereitschaft der Testpflanze und den äusseren Bedingungen der Durchführung des Nachweises ab. In vorliegender Arbeit interessierte die Frage, ob auch das Kartoffel-Y-Virusisolat einen Einfluss auf die Nachweismöglichkeit ausübt.

Material und Methoden

Nach Köhler (1953) sollen alle von ihm benutzten Kartoffel-Y-Virusstämme mit deutlichen Symptomen auf A6 reagieren. Zur Klärung dieser Frage wurden im September 1964 im Institut für Pflanzenzüchtung Gross-Lüsewitz (DDR) Abreibungen mit 22 Kartoffel-Y-Virusisolaten (vgl. Horváth, 1966a, b, 1967a, b) auf *Solanum demissum* A6 ausgeführt. Als Virusquelle dienten jeweils zwei Pflanzen von *Nicotiana tabacum* L. cv. *Samsun*, die zur ersten Untersuchung (am 17. 9. 1964) fünf Wochen zuvor; zur zweiten Untersuchung (am 29. 9. 1964) sechs Wochen zuvor mit dem jeweiligen isolierten Kartoffel-Y-Virusisolat infiziert wurden. Grundsätzlich wurden alle Blätter der zwei Tabakpflanzen im Mörser zerrieben. Je zehn gesunde Blätter von A6-Pflanzen, die systemisch mit Kartoffel-X-Virus (*potato virus* X) verseucht waren, wurden mit Karborund bestäubt (Maschenweite 500), mit je fünf Tropfen des Kartoffel-Y-virushaltigen Presssaftes und einem Tropfen Phosphatpufferlösung (pH 7) versetzt und vorsichtig mit einem Glasreiber abgerieben. Nach der Inokulation wurden die Testblätter unter Leitungswasser abgespült und im ersten Versuch in die im Institut üblichen Kästen, im zweiten Versuch in Petrischalen, wie sie Köhler (1953) und Horváth (1964) beschrieben, eingelegt. Beide Versuche wurden in den Vormittagsstunden durchgeführt.

Die Kästen, wie auch die Petrischalen, verblieben in einem Raum bei konstant +24°C und unter Dauerlicht von 1000 bis 1500 Lux. Im Laufe des Testes musste den Kästen Wasser zugefügt werden. Vom dritten Tag nach der Inokulation erfolgte die Auszählung der sichtbaren Läsionen auf den Blättern.

Ergebnisse

Wie aus Tabelle 1 zu ersehen, erfolgte die Läsionsausbildung unterschiedlich rasch. Während einige Isolate unter den gegebenen Bedingungen bereits 4—5 Tage nach der Inokulation auf nahezu allen Blättern Befall zeigten, verzögerte sich die Symptomausbildung der mit anderen Isolaten beimpften Blätter zum Teil erheblich.

* Während der Drucklegung erschien eine ausführliche Arbeit von S. Bérces und E. R. Keller (Eur. Potato J. *11*, 117—133, 1968), auf die leider nur hingewiesen werden kann.

Tabelle 1

Anzahl der *Solanum demissum* A6-Testblätter mit Läsionen nach Inokulation mit verschiedenen Kartoffel-Y-Virusisolaten in Tagen nach der Abreibung

Kartoffel-Y-Virus-isolat	Stamm-gruppe[1]	1. Test						2. Test									
		Tag nach Abreibung															
		4.	5.	6.	7.	8.	9.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
CSW	Y^N	0	1	1	1	1	1	7	10	10	10	10	10	10	10	10	10
EP	Y^C	0	0	0	1	1	3	0	0	2	3	5	7	8	8	9	9
Epe	Y^{An}	7	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10
PVY-R	Y^N	0	0	0	0	1	1	1	2	3	3	3	3	3	3	3	3
PVY-L	Y^N	8	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10
PVY-N	Y^N	10	10	10	10	10	10	9	10	10	10	10	10	10	10	10	10
PVY-W	Y^N	3	3	3	3	8	8	4	10	10	10	10	10	10	10	10	10
M 3	Y^R	3	5	5	6	9	9	0	0	1	3	5	6	9	10	10	10
Lü 85	Y^{An}	9	10	10	10	10	10	8	10	10	10	10	10	10	10	10	10
Bie	Y^N	9	9	9	10	10	10	4	10	10	10	10	10	10	10	10	10
Von	Y^N	6	9	9	9	10	10	0	4	6	6	6	8	8	8	9	9
Gie	Y^R	9	10	10	10	10	10	5	10	10	10	10	10	10	10	10	10
PK	Y^N	0	0	0	1	2	2	0	1	1	1	1	1	1	1	1	1
Ine	Y^N	5	5	5	8	10	10	0	4	8	9	9	9	9	9	9	9
UM	Y^N	2	4	5	7	10	10	0	0	2	4	5	8	8	8	8	8
BdN	Y^N	0	0	0	1	4	5	0	2	2	2	2	3	3	3	3	3
Adg 43	Y^N	2	5	8	10	10	10	0	2	3	4	5	6	8	8	8	8
Rs 188	Y^{An}	7	10	10	10	10	10	0	3	5	9	9	9	9	9	9	9
Lü 72	Y^N	0	0	1	3	3	3	0	0	0	0	0	0	0	0	0	0
Lü 86	Y^N	8	8	8	8	10	10	0	4	7	9	10	10	10	10	10	10
PVY-LL	Y^N	9	10	10	10	10	10	Nicht untersucht									
PVY-P	Y^N	10	10	10	10	10	10	Nicht untersucht									

[1] Y^N : Normalstamm des Kartoffel-Y-Virus
Y^R : Tabakrippenbräunestamm des Kartoffel-Y-Virus
Y^C : C-Stamm des Kartoffel-Y-Virus
Y^{An}: Anomaler Stamm des Kartoffel-Y-Virus

Die Sicherheit des Virusnachweises war nicht bei allen Kartoffel-Y-Virusisolaten gegeben (Tab. 2). Von den 22 untersuchten Isolaten konnten nur 16 mit genügender Sicherheit (d. h. mind. 90%) nachgewiesen werden; zwei Isolate reagierten ziemlich unsicher, bei vier weiteren Isolaten war der Nachweis über A6 sehr fraglich.

Auffällig war, dass nicht bei allen Isolaten in den beiden Versuchsreihen annähernde Übereinstimmung bestand. Die Ursachen hierfür konnten noch nicht geklärt werden.

ZIMMERMAN-GRIES und NITZANY (1963) fanden bei einigen Kartoffel-Y-Virusisolaten keine Lokalläsionen, wenn sie die Testblätter in einem Temperaturbereich von 21°C bis 27°C Raumtemperatur aufbewahrten. Bessere Ergebnisse erhielten sie bei Temperaturen von 19°C bis 22°C. Eigene Versuche in Temperaturkammern brachten bei 20°C keine Verbesserung gegenüber 24°C.

Tabelle 2

Sicherheit des Virusnachweises mittels *Solanum demissum* A6-Test bei Inokulation mit verschiedenen Kartoffel-Y-Virusisolaten

Kartoffel-Y-Virus-isolat	Anzahl der positiv reagierenden Blätter in %			Nachweis-möglichkeit
	1. Inokulation	2. Inokulation	$\bar{x}$	
CSW	10	100	55	unsicher
EP	30	90	60	unsicher
Epe	100	100	100	sicher
PVY-R	10	30	20	sehr unsicher
PVY-L	100	100	100	sicher
PVY-N	100	100	100	sicher
PVY-W	80	100	90	ziemlich sicher
M 3	90	100	95	sicher
Lü 85	100	100	100	sicher
Bie	100	100	100	sicher
Von	100	90	95	sicher
Gie	100	100	100	sicher
PK	20	10	15	sehr unsicher
Ine	100	90	95	sicher
UM	100	80	90	ziemlich sicher
BdN	50	30	40	sehr unsicher
Adg 43	100	80	90	ziemlich sicher
Rs 188	100	90	95	sicher
Lü 72	30	0	15	sehr unsicher
Lü 86	100	100	100	sicher
PVY-LL	100	Nicht untersucht	100	sicher
PVY-P	100	Nicht untersucht	100	sicher

Es ist anzunehmen, dass die Intensität der bei der *Solanum demissum* A6-Hybride beobachteten nekrotischen Reaktionen einerseits durch die Konzentration der Virusisolate, anderseits durch Faktoren, die die Empfänglichkeit der Wirtspflanze beeinflussen, erklärt werden kann.

Zur Klärung der Frage, ob die Änderung der Intensität der beobachteten nekrotischen Reaktion von der parallelen Änderung der Viruskonzentration abhängig ist, bedarf es noch weiterer Untersuchungen. Infolge technischer Gründe war eine Wiederholung des gleichen Versuches im Frühjahr und Sommer nicht möglich, um zu prüfen, ob jahreszeitlich Unterschiede in der Symptomausbildung durch die Kartoffel-Y-Virusisolate vorkommen.

Die Grösse der Läsionen, ihre Form und die Anzahl pro Blatt waren nicht einheitlich (s. Abb. 1—3). Überwiegend zeigten sich mehr oder weniger stark ausgeprägte kreisförmige Nekrosen. Das Gewebe in der Mitte der Ringe blieb erhalten. Teilweise bildeten sich ringförmige Flecken mit nekrotischem Kern, wobei sich die Nekrosen in den Nerven weiter fortsetzten. Eine Vermischung mit anderen Virusarten dürfte auf Grund der Trennungsarbeiten (Horváth, 1967c) ausgeschlossen sein.

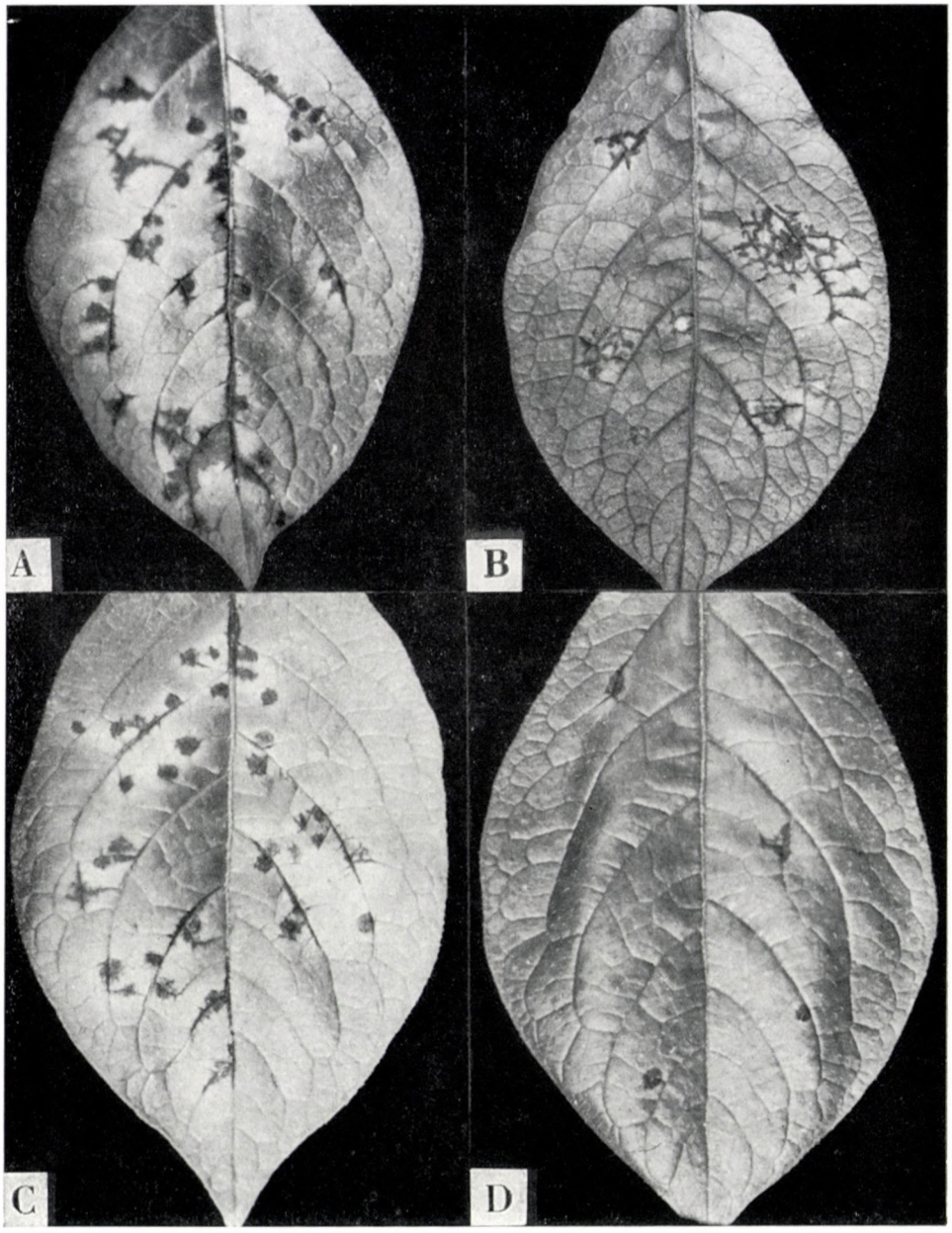


Abb. 1. Symptomausbildung auf A6-Testblättern — inokuliert mit den Kartoffel-Y-Virusisolaten. A: CSW, B: EP, C: Epe und D: PVY-R. Aufgenommen am 9. Tag nach der Inokulation

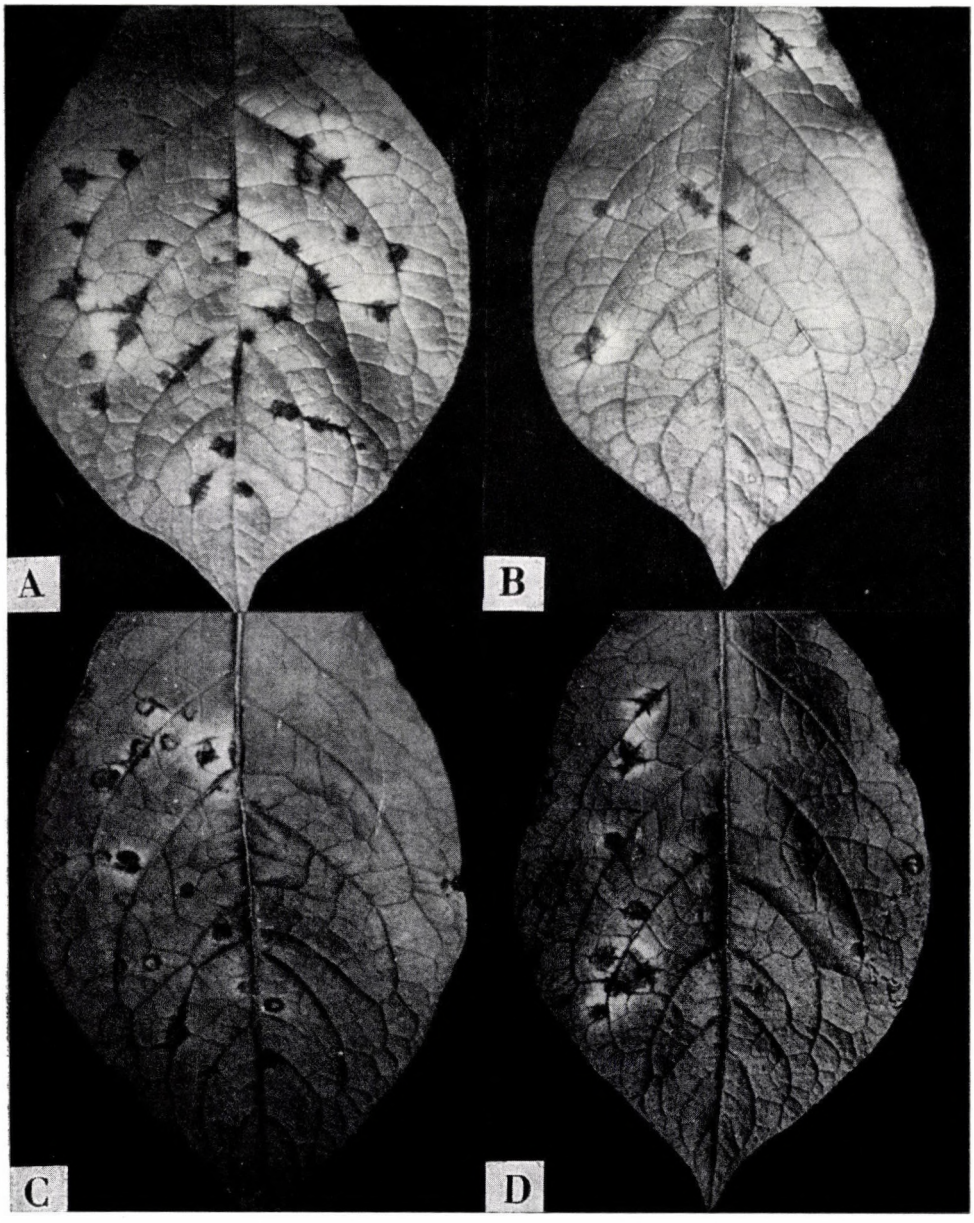


Abb. 2. Symptomausbildung auf A6-Testblättern — inokuliert mit den Kartoffel-Y-Virusisolaten. A: PVY-L, B: PVY-N, C: PVY-W und D: Lü 85. Aufgenommen am 9. Tag nach der Inokulation

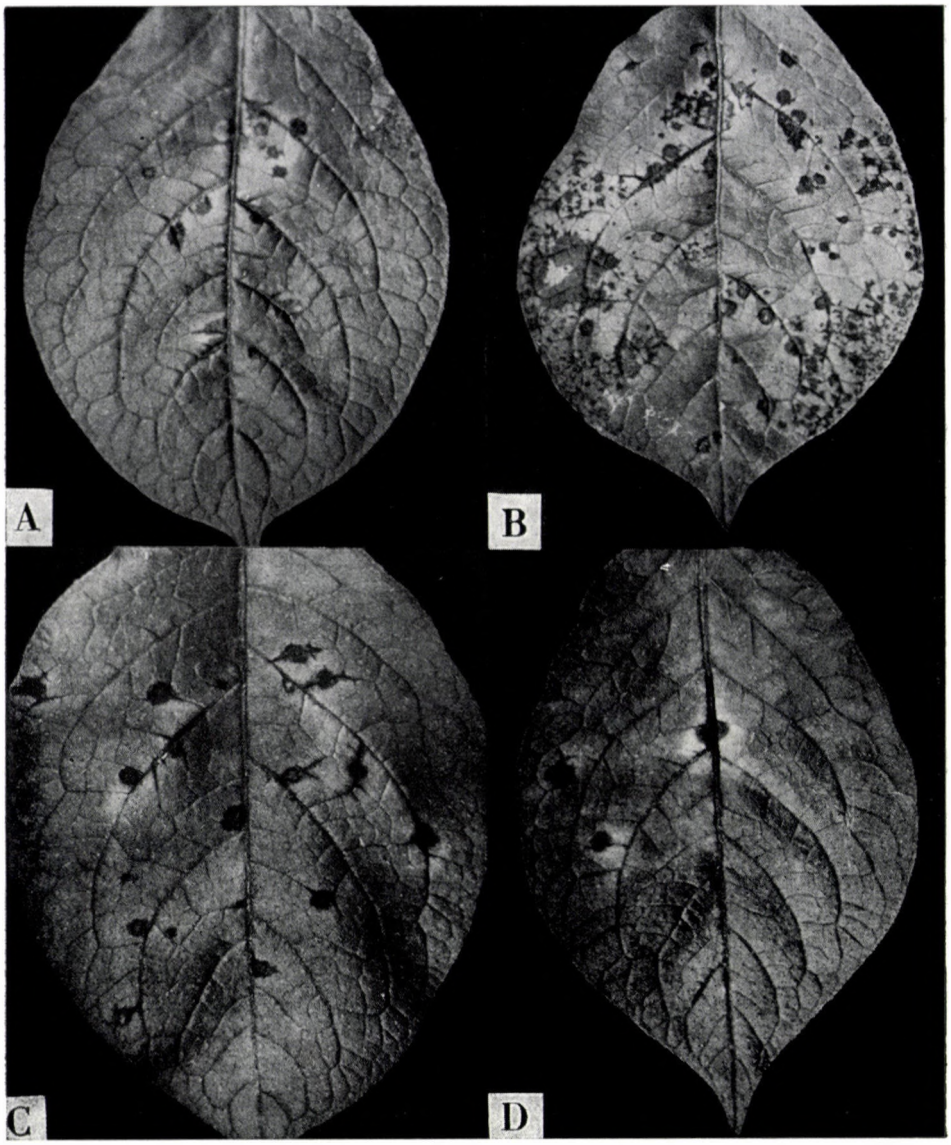


Abb. 3. Symptomausbildung auf A6-Testblättern — inokuliert mit den Kartoffel-Y-Virusisolaten. A: Ine, B: Lü 86, C: Von und D: Gie. Aufgenommen am 9. Tag nach der Inokulation

Diskussion

Die in den vorliegenden Untersuchungen gefundenen unterschiedlichen Zeitspannen in der Läsionsausbildung stimmten mit Feststellungen von KÖHLER (1953) überein. Eine Differenzierung zwischen Kartoffel-A-Virus (*potato virus* A) und Kartoffel-Y-Virus wird hierdurch fraglich. Zur Differentialdiagnose sollten andere Testpflanzen mit verwendet werden. Möglicherweise bietet die von WENZL (1963) vorgeschlagene Methode, nur eine Hälfte des Testblattes zu beimpfen und auf Grund der Überwanderung in die nicht inokulierte Blatthälfte in Verbindung mit entstandenen Sekundärläsionen die Entscheidung zu treffen, einen Lösungsweg. Eine Differenzierung der verschiedenen Kartoffel-Y-Virusisolate an Hand der entstehenden Läsionen dürfte ebenso fraglich sein.

Die Erkenntnis von ARENZ und HUNNIUS (1958), HUNNIUS (1965) und BEEMSTER (1965), dass die Rippenbräunestämme des Kartoffel-Y-Virus sich wesentlich schneller in der Kartoffelpflanze ausbreiten als die normalen Stämme, liess die Vermutung entstehen, eine ähnliche Erscheinung im A6-Blatt-Test zu finden. Dem widersprechen aber die Ergebnisse des Kartoffel-Y-Virusisolates M 3, bei dem sich eine merklich verzögerte Läsionsausbildung gegenüber z. B. PVY-L oder PVY-N zeigte.

Während KÖHLER (1953) fand, dass der A6-Bastard gegen alle von ihm geprüften Kartoffel-Y-Virusstämme mit der Ausbildung deutlich sichtbarer Läsionen reagierte, konnte in eigenen Versuchen bei bestimmten Isolaten (PVY-R und Lü 72) kein eindeutiger Nachweis geführt werden. Auch von DMOCHOWSKI (mündl. Mitteilung) wird berichtet, dass bei der Kartoffelsorte *Giewont* der Y-Virusnachweis mit der Testpflanze *Solanum demissum* »Y« (Sdy) durchgeführt wird, da der A6-Blatt-Test nicht sicher sei.

Die vergleichenden Ergebnisse, die wir bei der symptomatologischen Untersuchung der mit der *Solanum demissum* A6-Hybride genetisch verwandten Kartoffelsorten — aus denen die Kartoffel-Y-Virusisolate stammten — fanden, stimmten nicht mit den Beobachtungen von MICZYŃSKI (1965) überein. Ein solches Isolat (Lü 72), das an einer homologen Kartoffelsorte schwere Symptome ausgelöst hatte, zeigte auf den Blättern der A6-Hybride nur schwache Symptome und auch die Nachweisbarkeit war sehr unsicher. Das Isolat Rs 188 war an der homologen Kartoffelsorte symptomlos, während es an den Blättern der A6-Hybride starke Symptome auslöste.

Die Einschränkung der Verwendbarkeit des A6-Bastards wird dadurch gemildert, dass offensichtlich die gefährlicheren Tabakrippenbräunestämme des Kartoffel-Y-Virus (M 3, Gie) und anomalen Stämme des Kartoffel-Y-Virus (Epe, Lü 85 und Rs 188) mit ausreichender Sicherheit fassbar sind (vgl. Tabelle 2).

*

Für die technische Assistenz während der Untersuchungen möchten wir Frau K. MÖLLER danken.

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The Occurrence of a Bacterium sp. Belonging to the *Pseudomonas syringae* van Hall Group on *Sorghum* spp.

By

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An important leaf disease of *Sorghum* spp. is caused by a bacterium belonging to the *Pseudomonas syringae* van Hall group. The symptoms of the disease have been known for long ago in Hungary, however, the pathogen responsible for it has not been exactly identified as yet. The pathogen was isolated from leaf spots of Sudan grass and sorghum. The pathogenicity of the bacterium was proven by its ability to induce hypersensitive reaction in incompatible plants and typical symptoms on the original host plants. Its biochemical properties corresponded to those of *Bacterium (Pseudomonas) holci* as was described by KENDRICK (1926). It was established on the basis of serological tests that the strains isolated from sorghum are identical with those isolated from Sudan grass, however, they differ in their antigenic structure from the *P. syringae* isolated from lilac.

Introduction

The economical value of cultivated *Sorghum* species is reduced by diseases attacking these crops from year to year. A wide range of diseases of viral, fungal, and bacterial origin appear on sorghums (TARR, 1962).

The identification of pathogens on the basis of symptoms is extremely difficult under field conditions because various *Sorghum* species react uniformly by red or later by brown discolouration, to the attack of both different pathogens and pests. Because of the similarity of the symptoms the diseases caused by various bacteria cannot be safely differentiated on the basis of symptomatology.

The red spot symptoms have long been known in Hungary (KERN, 1931; KRENNER, 1929, 1941; SZIRMAI, 1950), however, the pathogen has not yet been identified. The results contained in this paper constitute the first description of the pathological and biochemical properties of bacteria belonging to the *P. syringae* group which occur on *Sorghum* species.

Symptoms

The spots on the leaf caused by the bacterium are dark green and water-soaked then turn to red and later becoming dry. Their center is light coloured with a striking red ring. On certain sorghum and Sudan grass varieties there is a brown

discolouration. Sometimes the center of the spot does not become light coloured and the whole spot retains a uniform red or brown colour. The lesions are round, elliptical, or occasionally elongated and irregular (Figs 1 and 2). Marginal infection appears as a necrotic streak along leaf edges. The infection begins on the lower



Fig. 1. The natural symptoms of the disease on the leaves of *Sorghum bicolor* (L.) Moench

leaves, however, under favourable conditions it spreads to the upper ones. Under serious attacks the lesions cover the larger part of the leaf surface sometimes killing the whole leaf. Similar symptoms may sometimes be observed on the leaf sheath, stem and branches of the head. Production of bacterial exudate has not been observed.

Isolation of the bacterium and its pathogenicity

The pathogen was isolated from leaf spots. After the washing of the leaf surfaces inoculum was taken from the spots and it was plated on broth agar medium. After incubation at 28°C for 48 hours bacterial colonies appeared on the surface of the plates. Different types of bacterial colonies were isolated. In order to select the pathogenic strains the KLEMENT's method (1963) was used. This method is based on the fact that most phytopathogenic pseudomonads induce the hypersensitive reaction in incompatible host-parasite combinations, which manifests itself in a rapid necrosis within 24 hours (KLEMENT and LOVREKOVICH, 1961). Four

strains originating from *Sorghum bicolor* (L.) Moench (C-1, C-2, C-3, C-4) and two strains isolated from *Sorghum sudanense* (Piper) Stapf. (SZ-1, SZ-2) were able to induce the hypersensitive reaction in tobacco leaf. These strains were the subject of further investigations.



Fig. 2. Disease symptoms on the leaves of *Sorghum sudanense* (Piper) Stapf. leaves

Identification of the bacterium

After 3 days all the pathogen strains formed colonies of 3 mm in diameter lentiform, circular, smooth, greyish white and transparent. Under the microscope the bacteria appeared as rods, Gram-negative and motile.

To establish the biochemical properties of the bacterium the usual methods were applied (DOWSON, 1957). The strains isolated from sorghum and Sudan grass behaved uniformly in the biochemical tests. They rapidly liquefied gelatine. In litmus milk they gave alkaline reaction, followed by peptonization. In pepton-water ammonia was produced and hydrogen sulphide was not. In tryptophan broth indol was not produced. Nitrate was not reduced to nitrite and starch was not hydrolyzed.

Of the carbon compounds, which were added to a synthetic nutrient broth, acid was formed in the case of D,L-arabinose, galactose, saccharose, mannose and mannitol. Lactose, maltose, raffinose, salicin were not utilized.

Serological investigations

The strains isolated from sorghum and Sudan grass behaved uniformly in morphological, pathological and biochemical investigations. To confirm their identity the antigen structure of bacteria was also investigated. One strain isolated from sorghum (C-1) and another from Sudan grass (SZ-1) were used as test organisms for the serological investigations. In order to establish their relationship to *P. syringae* they were compared to a strain of *P. syringae* (strain No. 524 from the National Collection of Plant Pathogenic Bacteria, England) isolated from lilac.

To gain samples of immune serum, rabbits were injected intravenously 5 times in every three days, using 1, 2, 2, 3 and 4 ml of inocula, respectively. The cultures of the C-1, SZ-1 and 524 strains incubated for 24 hours, were suspended in sterile normal saline solution and used as inocula. Bacterial suspensions contained 10^9 to 10^{10} living cells per ml. The agglutination test was carried out by the method of MACKIE and MCCARTNEY (1959). The agglutination tubes were kept in water bath at 37°C for two hours and then at room temperature overnight.

Table 1

Agglutination test with non-treated bacteria

Serum	Bacterial strain	Dilutions of the serum									
		1 : 20	1 : 40	1 : 80	1 : 160	1 : 320	1 : 640	1:1280	1:2560	1:5120	C
C-1	C-1	+	+	+	+	+	+	+	+	+	—
	SZ-1	+	+	+	+	+	+	+	+	—	—
	524	+	+	—	—	—	—	—	—	—	—
SZ-1	C-1	+	+	+	+	+	+	+	+	—	—
	SZ-1	+	+	+	+	+	+	+	+	+	—
	524	—	—	—	—	—	—	—	—	—	—
524	C-1	+	+	+	+	+	—	—	—	—	—
	SZ-1	+	+	+	+	+	+	—	—	—	—
	524	+	+	+	+	+	+	+	+	+	—

As is seen in Table 1 the strain isolated from Sudan grass (SZ-1) and that from sorghum (C-1) produce agglutination of the similar titer with the serum of the other, as with their own. However, *P. syringae* 524 strain (isolated from lilac) did not produce agglutination with sera of strains C-1 and SZ-1. Strains C-1 and SZ-1 gave agglutination of a lower titer with the serum of strain 524. It was shown that the antigen components of bacteria isolated from sorghum and Sudan grass are identical and their antigenic structure is similar but not identical with that of *P. syringae* 524.

It was shown by LOVREKOVICH and KLEMENT (1961) that when a bacterial suspension boiled for 10 minutes at 100°C was used as antigen for the agglutination test the thermostabile antigens of *P. tabaci* gave more specific reaction, than

the non treated, thermolabile antigens. Therefore, the agglutination test was carried out with heat-treated antigens as well. The results of these experiments are shown in Table 2. Here again, the thermostabile antigen components of strains C-1 and SZ-1 were identical, however, different from the antigenic structure of *P. syringae* 524 strain.

Table 2

Agglutination test with heat-treated bacteria

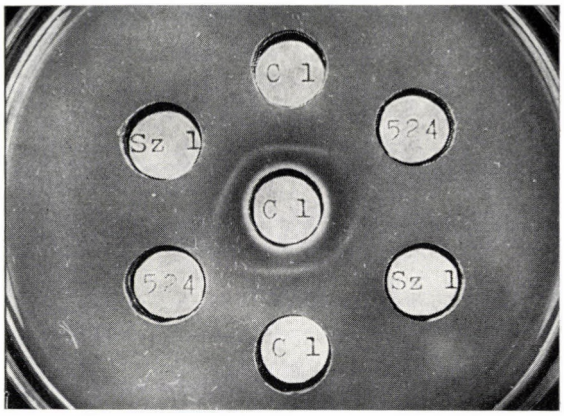
Serum	Bacterial strain	Dilutions of the serum							
		1 : 20	1 : 40	1 : 80	1 : 160	1 : 320	1 : 640	1:1280	C
C-1	C-1	+	+	+	+	+	±	—	—
	SZ-1	+	+	+	+	+	±	—	—
	524	—	—	—	—	—	—	—	—
SZ-1	C-1	+	+	+	+	+	±	—	—
	SZ-1	+	+	+	+	±	—	—	—
	524	—	—	—	—	—	—	—	—
524	C-1	—	—	—	—	—	—	—	—
	SZ-1	—	—	—	—	—	—	—	—
	524	+	+	+	+	+	+	—	—

The microprecipitin test gave similar results. In these experiments the agar-gel-technique (OUCHTERLONY, 1958) was applied. A bacterial suspension of 10^{11} cell/ml density was treated for 1 hour under a pressure of 1 atm. 15 ml 1.5% washed agar dissolved in physiological saline containing 0.025% merthiolate was poured into the Petri dishes. The antiserum was put in the center well of the agar-plate and the antigens in the wells around it. The diameter of the wells was 8 mm. During diffusion the plate was kept at room temperature for 1—2 days and then assayed. The bacteria isolated from sorghum and Sudan grass contained identical precipitinogens. This was concluded from same precipitin bands. However, the immune serum of the strain 524 precipitated only its own antigen and did not precipitate those of the bacteria isolated from sorghum and Sudan grass (Figures 3, 4, 5).

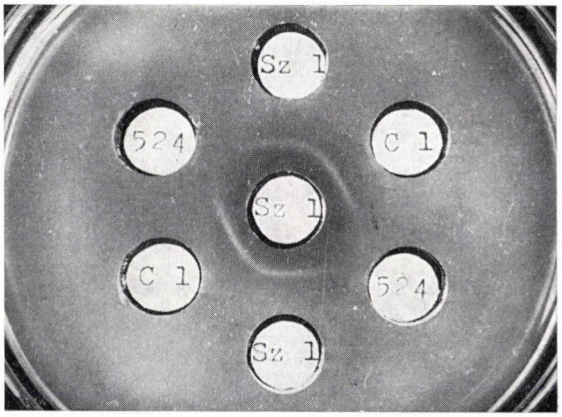
Thus the serological test applied in our investigations proved unambiguously the identity of bacteria isolated from sorghum and Sudan grass. On the other hand these strains possess an antigenic structure distinct from that of *P. syringae* 524.

Artificial inoculations

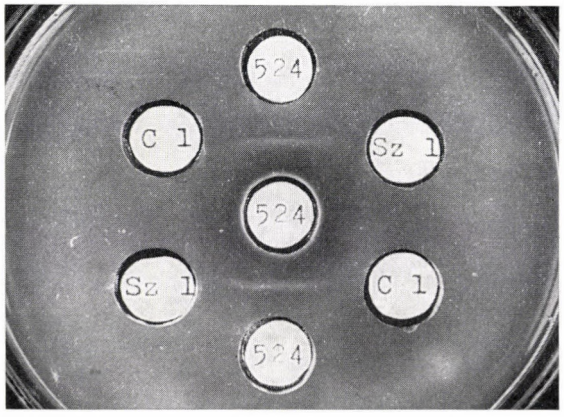
In these experiments all the strains were used (C-1, C-2, C-3, C-4, SZ-1, SZ-2, 524). The individual plants of *Sorghum bicolor* (L.) Moench and *Sorghum sudanense* (Piper) Stapf. were grown in the green house and were inoculated with bacterial suspensions prepared from a 24-hour slant agar culture. According to one of the inoculation methods the surface of the leaves was rubbed by Celite and



3



4



5

Figs 3, 4, and 5. Cross reaction between the strains isolated from sorghum (C-1), Sudan grass (SZ-1) and lilac (524), respectively. The serum is applied in the center and the antigens around it

the bacterial suspension was sprayed onto the leaves (Fig. 6). Another method consisted of pricking the leaves with a needle dipped in the bacterial suspension. The typical symptoms appeared on the plants treated by pricking after 3 days, whereas on the sprayed leaves after 5 days. The symptoms using the spraying method are

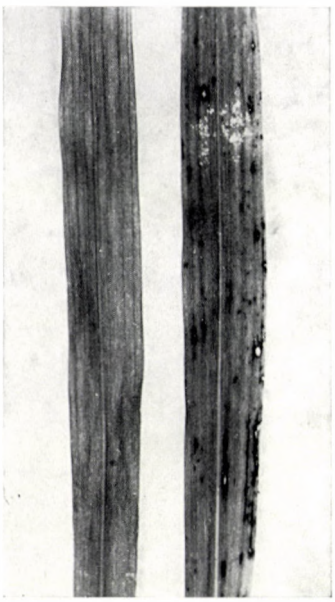


Fig. 6. The symptoms induced by artificial inoculation on young sorghum leaves

shown in Figure 6. There was no difference in the pathogenicity of the strains, isolated from sorghum and Sudan grass. The symptoms of *P. syringae* No. 524 was milder (Fig. 7), as compared to the strains from sorghum and Sudan grass.

Discussion

Leaf spotting of various *Sorghum* species may be caused by the following three bacterial species: *Pseudomonas andropogoni* (Erwin F. Smith) Stapp; *Xanthomonas holcicola* (Elliott) Starr and Burkholder and *Pseudomonas syringae* van Hall. KENDRICK (1926) described a bacterium isolated from corn and *Sorghum* spp. as *Bacterium* (Pseudomonas) *holci*. ELLIOTT (1951) considered *P. syringae* as identical with *P. holci*. In the literature pertinent to the question both species names are used for the bacteria isolated from sorghum and Sudan grass (KOVACHEWSKI, 1931; PANIC and ARSENIJEVIC, 1966; PASTUCHENKO, 1961; TARR, 1962; UBRIZSY, 1965).



Fig. 7. The symptoms obtained by stubbing the leaves with *P. syringae* strains isolated from sorghum (C-1), Sudan grass (SZ-1) and lilac (524), respectively. [Control (K) is on the left]

The biochemical properties of the bacteria isolated by the author from sorghum and Sudan grass are near identical to those of the strain described by KENDRICK as *P. holci.*

The occurrence of *P. syringae* was described on numerous trees and herbaceous plants. However, the bacteria isolated from various hosts are not uniform in their pathological and biochemical properties. LOVREKOVICH, KLEMENT and DOWSON (1963) compared the antigenic structure of a number of *P. syringae* strains. They differentiated eight serological groups but did not find correlation between serotypes and pathotypes. *P. syringae* and *P. mors-prunorum* Wormald isolated from tree plants form well defined groups (CROSSE, 1966), but we cannot realize this on herbaceous plants.

The antigenic structure of the bacteria isolated from sorghum and Sudan grass proved to be identical in the test carried out by the author, however, it differed from that of *P. syringae* 524 isolated from lilac.

The taxonomy of the bacteria belonging to the *P. syringae* group which were isolated from herbaceous plants is not definitively determined. For the final determination further comparative biochemical, pathological, serological and phage-sensitivity tests have to be carried out.

At present, we have to consider the bacterium pathogenic to sorghum and Sudan grass as a member of the *P. syringae* group.

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The Tarragon Rust Disease (*Puccinia dracunculina* Fahrend.) and the Method of Its Control

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Damage caused by rust infection in tarragon has been investigated. As a result of infection a decrease in the utilizable materials of the crop takes place. On the basis of the results of small plot experiments, pilot-plant experiments and large-scale farming, it was demonstrated that it is possible to control rust infection with different dithiocarbamates.

Materials and Methods

Tarragon rust *(Puccinia dracunculina)* causes heavy losses (more than 50 per cent crop loss) in Hungary. Therefore, it was urgently necessary to work out a suitable method of control of rust infections. Investigation with tarragon rust began in the autumn of 1964 at the nursery of the Research Institute for Medical Plants. The rust infection has been carried out by inoculations with a spore suspension, to supply the investigations continuously with fresh material.

On the basis of the preliminary investigations field experiments were conducted in the tarragon cultivation of the Darányipuszta State Farm, where — as a result of the damage caused by the rust in the recent years — only 40% of the plants were present. However, plant-density on the experimental plots was uniform.

The size of the small-plots was 10.5 m^2, the treatments were repeated 4 times. Medium-size experiments were carried out on a field of 2000 m^2. The small plots were sprayed with a knapsack sprayer, in the medium-size experiment a "Rapid-tox II" machine was applied, in both cases with 1000 liter/ha water.

The cultivation and the harvest have been carried out on the same way both on the experimental plots and on the large-scale fields. After measuring the yield the volatile oil was distilled from the green plants.

The gas chromatographic investigation of the volatile oil was made with a CHROM I type machine of the Research Institute for Medical Plants. Investigations on the residues were made in the National Research Institute for Feeding Science.

In the experimental years rainfall was higher than the average during the growing season.

The first small plot experiment was carried out in the spring of 1965, at the Darányipuszta State Farm. For the first cut the treatments were started on the 3rd

June — too late — and till the first cut, which was carried out on 28th June, 3 treatments were taken. As the treatments begun too late it was impossible to gain suitable results concerning the first cut, but it was possible to find out the optimum amount of the spray volume (1000 liter/ha), as well as some pre-selection was taken regarding fungicides.

For the second cut treatments started just after the first cut, and till the second cut six treatments were carried out. On the experimental plots the second cut was taken on 21st October, before the second cut — on 15th September — samples were taken to determine the decrease of the utilizable plant material. At this investigation more than 3000 leaves were sorted.

On the basis of the favourable results of 1965 the experiment has been continued in 1966, when more recent fungicides were applied.The sprayings were made in every 10 or 20 days. In the growing season 11 and 8 sprays were carried out in the intervals mentioned above.

In the medium size experiment plants were sprayed 10 times.

Results

Identification of the fungus

In the investigations — which started in the autumn of 1964 and were taken simultaneously with the field experiments — healthy tarragon plants were inoculated with spores which were isolated from rust infected tarragon plants. At the same time measure was taken to determine the feature of the spores. It was found that the disease was caused by the tarragon rust *(Puccinia dracunculina* Fahrend.) in every case. This fungus has various synonyms in literature: *Puccinia absinthii* DC., *Puccinia Artemisiae* Fuckel-Fuss., *Uredo Artemisiae* Rabenh.-Fuss. and *Puccinia tanaceti* DC. (MÜHLE, 1956; SAVULESCU, 1953; UBRIZSY, 1966).

The pathogenic fungus, which damaged the tarragon plants at Daránypuszta, will be referred to in this paper as tarragon rust, this — in every case — is *Puccinia dracunculina* Fahrend.

The tarragon rust is an attoecic fungus, with a two-celled teleutospore, the aecidia and the spermogenic spore-form is missing in its development (SAVULESCU, 1963; UBRIZSY, 1966).

According to the investigations, the form of the *uredospores* of the tarragon rust are generally smooth, ellipsoid, their colour changes from straw-yellow to light-brown. In one uredo colony several thousand spores are covered with a blister-form inflated epidermis, this later splits open to scatter the spores. Thickness of the wall of the spores is 1—2 μ. The uredospores appear generally at the beginning of the summer on the lower side of the tarragon leaves, at first on the lower leaves, they gradually turn pale, yellow around the rust-colonies; later the whole leaf withers and falls.

The steady dark brown *teleutospore* colonies of the tarragon rust appear on the lower side of the leaves at the end of the summer. These colonies are overwintering. According to the investigations the teleutospores are rather compact, their form is similar to a mace, they are two-celled and settled on a long, central originated stick. The thickness of the spore wall is 2—8 μ. The extreme sizes of the teleutospores were 41—55 × 20—29 μ, as mean values. The mean value of the size of the teleutospores was 48 × 25 μ. The sporeholding sticks are entirely light-coloured; their length changes between 39—93 μ, their width changes between 6—10 μ.

Both the uredospores and the teleutospores can be found on the lower side of the leaves and on the stem of the tarragon plants. Vigorous uredospores were found on dry plants in the early spring too, consequently, the rust can overwinter by uredospores.

According to the investigations temperature above 18°C and humidity above 65% was extremely favourable for sporulation.

Small-plot and medium-size experiments

The effect of the rust on the metabolism of the host-plant is in strict connection with the caused damage (POZSÁR and KIRÁLY, 1959; SHAW, 1964).

In the small-plot experiments carried out in 1965 decrease of the utilizable material content was investigated on the basis of samples gathered during the growing season and in the harvest. Results of the investigations can be found in Tables 1 and 2.

Table 1

Decrease of the useful plant material after 15 days of the appearance of the rust

Treatments		Dry leaves	Partially dry leaves	Leaves with moderate withering	Healthy leaves
		Number	Number	Number	Number
Control		58.0%	16.0%	4.0%	22.0%
Hortocritt	0.3%	0.5%	4.2%	1.8%	93.5%
Organil-66	0.3%	0.3%	1.9%	2.8%	95.0%
Melprex	0.3%	0.7%	5.3%	8.0%	86.0%
SD	0.1%	10.97	6.52	6.17	10.00

In addition to the very good results which can be seen in the Tables, it was found in the spring of 1966 that the treated tarragon plants of the experimental plots overwintered well, sprouted vigorously, grew 10—20 cm higher, flowered

Table 2

Decrease of the utilizable plant material in the plants damaged by the tarragon rust, at harvest

Treatments		Balding in percentage of the length of the stem	From the total fresh weight			Oil content % of the samples from the green weight*
			weight of the stem	weight of dry leaves	weight of the green leaves	
Control		90%	55.5%	15.9%	28.6%	0.25%
Hortocritt	0.3%	17%	26.9%	0.3%	72.8%	0.68%
Organil-66	0.3%	19%	29.4%	0.6%	70.0%	0.62%
Melprex	0.3%	13%	22.5%	0.2%	77.3%	0.78%
SD	0.1%	18.9	15.2	7.5	11.1	—

* In the case of the volatile oil % the number of the samples was not sufficient to count SD.

6—9 days earlier than the plants on the untreated plots. A part of the crop in the untreated plots died out again during the winter. The development of the over-wintered untreated plants was rather slow, compared with the treated ones.

Similar good results were gained in the small-plot experiments, carried out in 1966. The result is summarized in Table 3. It can be seen from Table 3 that,

Table 3

Result of the small-plot experiment in 1966

Treatments			Annual yield		In the average of the two cuts	
			green plant, kg/ha	volatile oil, kg/ha	balding	average weight of one plant, dkg
Siaprit Act.	0.3%	10*	2.020	6.69	14.9	14.34
Siaprit Act.	0.3%	20*	2.453	7.07	19.1	14.84
Hortocritt	0.3%	10	2.809	10.16	13.7	15.76
Hortocritt	0.3%	20	1.819	6.58	19.9	13.22
Organil-66	0.3%	10	3.132	9.87	13.8	16.25
Organil-66	0.3%	20	2.055	7.21	18.7	15.41
Zineb	0.3%	10	2.484	9.74	14.16	15.38
Zineb	0.3%	20	1.794	6.37	20.0	12.72
Vitigran	0.6%	10	1.894	7.05	22.6	13.83
Control I.		—	1.063	1.44	90.7	4.21
Control II.		—	941	1.44	84.0	4.44
SD	0.1%		498	—**	13.8	4.62

* 10 = sprayed per 10 days, 20 = sprayed per 20 days.

** In the case of the volatile oil yield the number of the samples was not sufficient to count SD.

similarly to the previous year, the tendency of the experiment justified the effectiveness of the treatments applied.

In Table 4 results of the medium size experiment can be found. To compare these figures, the yields of the Darányipuszta State Farm in 1965, 1966 and 1967 are reported. It can be seen from the Table, that the effectiveness of the large-scale protection was more than two times higher. In addition the protection promoted the normal development of the plants and the ripening of the shoots.

Table 4

Average results of the medium-size experiment and large-scale cultivation

	Yield in 1965		Yield in 1966		Yield in 1967	
	green plant, kg/ha	volatile oil, kg/ha	green plant, kg/ha	volatile oil, kg/ha	green plant, kg/ha	volatile oil kg/ha
Large-scale cultivation without protection	889	2.12	1414	5.75	—	—
Medium-size experiment treated with Hortocritt 0.3	—	—	3636	11.45	—	—
Large-scale cultivation with the suggested method of protection*	—	—	—	—	4431	22.28

* Plant decay caused by the rust was filled up at the early spring and there were about 30% more plants compared to the number of the plants in previous years on the same place.

Investigation on the quality of the volatile oil

With the suggested method of protection the quality of the volatile oil — gained from plants which were protected from the rust and cut in time — was suitable in all respects.

Gas chromatographic investigations showed that there was no considerable difference in the presence of the components and in their total quantity in the case of the volatile oils, gained from untreated plants and from plants treated with different fungicides. The oils uniformly contained about 70% estragole as a main component, as well as α-pinene, β-pinene and limonene, nearly in the same quantity.

Investigations on the residues showed uniformly that in the volatile oil undecomposed residues could not be found. The contact of copper and zinc in the volatile oils corresponded to the contamination which used to be found in other oils, too.

Discussion

The systemic small-plot experiments, medium-size experiments, and the results of the large-scale cultivation in 1967, proved that tarragon plants can be protected from the tarragon rust by spraying with a 0.3% Organil-66, Hortocritt, Zineb fungicides (600 lit./ha). The protection must be commenced about 2—3 weeks after the beginning of the growing season, and plants should be sprayed in every 10—15 days. Late treatments, reduction of the proposed volume and of the proposed concentration may decrease significantly the efficiency of the treatments.

Weedy tarragon field offer a suitable biological condition for the sporulation of the fungus.

Economical investigations and calculations showed that with the control method proposed in this paper the net yield increased more than the double.

As the action of the dithiocarbamate-type fungicides is rather short especially during some meteorological and microclimatic conditions, it is important to search for fungicides, with similar good effect, but with substantially longer action. In the meantime it is practical to use Hortocritt or Organil-66. These fungicides afforded better protection in the experiments than Zineb, which is used in the large-scale farming up to the present.

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Die wachsende Bedeutung der pflanzenhygienischen (phytohygienischen) Anschauung

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Die im weiten Sinne aufgefasste Phytopathologie bildet die wissenschaftliche Grundlage nicht nur für die Pflanzenpathologie, sondern auch für die Pflanzenhygiene (Phytohygiene) und daher auch für den praktischen Pflanzenschutz. BRAUN (1956, 1962—1963) und MÜHLE (1961, 1962, 1967), die in ihren Studien die Aufteilung der Pflanzenhygiene bzw. der Pflanzenheilkunde (Phytiatrie, Phytomedizin) behandeln, fassen die tierischen Schädlinge neben der allgemeinen Pflanzenpathologie und Epidemiologie im Sachkreis der Phytotraumatologie zusammen. Die Infektionskrankheiten erregenden Mikroben (Viren, Bakterien, Sporozoen und Pilze) werden dagegen — nach ihrer Meinung — mitsamt den tierischen Parasiten (z. B. Gallen bildende Endoparasiten, Nematoden usw.) bzw. den parasitischen Pflanzen (z. B. parasitische Blütenpflanzen) der Phytoparasitologie zugewiesen und auch dort behandelt. Auf diesen Grundlagen beruht die Bekämpfungslehre (Phytophylakologie), die Phytotherapie (Chemo- und Biotherapie) und die Phytopharmazie, die mitsamt der Praxis im *integralen Pflanzenschutz* als grösserer Einheit miteinander im Zusammenhang stehen (UBRIZSY, 1964, 1966, 1967), s. Tabelle 1.

Die Pflanzenpathologie (Phytopathologie) ist seit dem Erscheinen des Werkes von J. BERKELEY (Vegetable pathology, 1847) bzw. des Buches JULIUS KÜHNES (Die Krankheiten der Pflanzen, 1858) in aller Welt eine selbständige und auf einem hohen Niveau kultivierte Wissenschaft, für die im Laufe der Zeiten auf allen höheren Agrarlehranstalten ein gesonderter Lehrstuhl errichtet worden ist. Mit der Zeit folgte auf den Lehrstuhl für Pflanzenpathologie auch die Organisierung eines Lehrstuhls der angewandten oder landwirtschaftlichen Insektenkunde. Infolge der schnellen Zunahme und Differenzierung der Fachkenntnisse sowie des zunehmenden Auftretens der Pflanzenkrankheiten, Pflanzenschädlinge und Unkräuter, dem eine verstärkte Schadenerregung folgte, *konnte der früher* im Rahmen der beiden Lehrstühle unternommene theoretische und praktische *höhere Unterricht des sogenannten »Pflanzenschutzes« weder im Ausland noch in Ungarn die an ihn gestellten vielfachen Aufgaben lösen*, da es nicht gelang, für die Produktionsbetriebe gut vorbereitete Fachleute laufend auszubilden. Im Ausland entwickelte sich daher an den Hochschulen oder Universitäten für Gartenbau oder Agronomie überall eine *selbständige Fakultät des Pflanzenschutzes*, deshalb wurde es auch in Ungarn

nötig, an der Universität für Agrarwissenschaften zu Gödöllő im Rahmen einer 1jährigen Fortbildung die Ausbildung der sogenannten Fachingenieure für Pflanzenschutz zu organisieren. Die letztere Massnahme bedeutet aber nur eine vorläufige Lösung.

Da die meisten Probleme des praktischen Pflanzenschutzes vor allem hygienischer Natur sind, ist es verständlich, dass schon vor 30 bis 40 Jahren zeitgemässe Bestrebungen zur Erarbeitung einer praktischen Pflanzenhygiene (Phytohygiene), anstelle des im engeren Sinne genommenen Pflanzenschutzes (Phytophylakologie), erfolgten. Zu dieser Zeit wurden auf zeitgemässen Grundlagen die ersten Forschungsinstitute und Versuchsstationen organisiert. Im Ausland wird dieses Fachgebiet in wissenschaftlicher Beziehung auch heute noch als Pflanzenhygiene (Phytohygiene) betrachtet, deshalb trägt z. B. die Gesellschaft der französischen Fachleute der Pflanzenpathologie folgenden Titel: Gesellschaft für Phytiatrie und Phytopharmazie. In Westdeutschland ist die Vereinigung deutscher Pflanzenärzte schon seit nahezu zwei Jahrzehnten tätig, und der Ausdruck Pflanzenschutz wird nur für die Lösung praktischer Aufgaben vorbehalten (REINMUTH, 1956; MAYER, 1959 usw.).

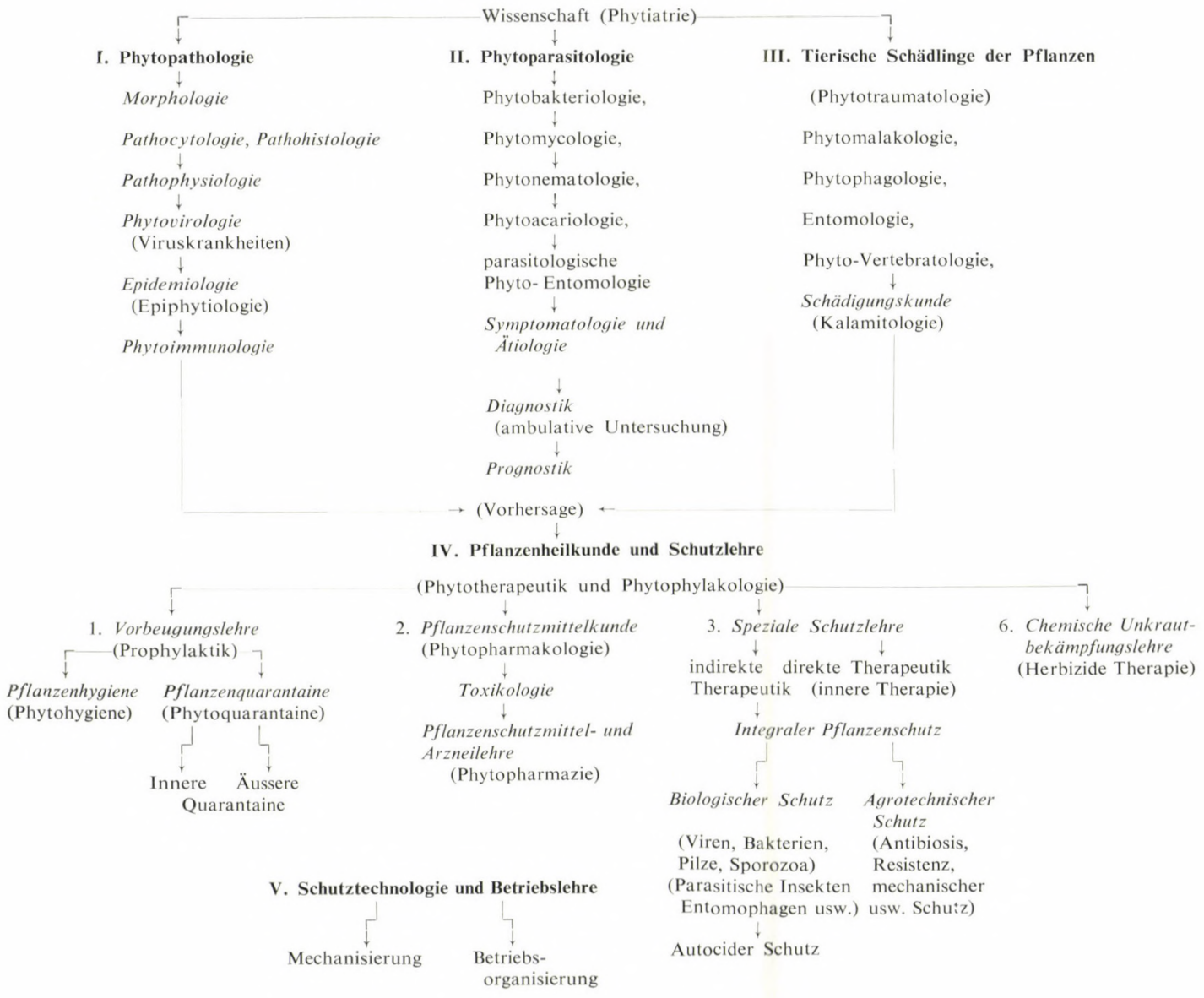
Auch in Ungarn wurde der vorherige Pflanzenschutz in ein Pflanzengesundheitswesen umorganisiert; das Zentralinstitut arbeitete als ein Institut für Pflanzenhygiene. Die zunehmende Bedeutung unseres Fachgebietes in der Gegenwart, die starke Differenzierung der Fachkenntnisse, die weite Verbreitung des chemischen Pflanzenschutzes sowie der indirekten bzw. direkten Ektotherapie, die wissenschaftliche Klärung der Krankheitserscheinungen und Seuchen (Epiphytien), die zunehmende Bedeutung der Vorbeugung (Prävention) sowie das Vordringen des komplexen bzw. *integrierten Pflanzenschutzes* — der im zeitgemässen Gesundheitsschutz der Kulturpflanzen von der Prävention bis zur Therapie und von der Agrotechnik bis zu den biologischen Schutzmassnahmen allerlei Mittel einsetzt — machen es nötig, dass man sich mit einem zunehmenden Interesse an die Frage der zeitgemässen pflanzenhygienischen Anschauung und ihres praktischen Ausbaus wendet.

Der Pflanzenschutz bzw. die Pflanzenhygiene ist ein sich ausbreitendes Fachgebiet, das an ökonomischer Bedeutung Jahr für Jahr gewinnt, da es auch ein wichtiger Faktor der Intensivierung und i. allg. der Produktionsintensität ist. Je grösser die Produktionsintensität, um so bedeutender ist die zeitgemässe Bekämpfung der Krankheiten und Schädlinge! Bei uns ist es allbekannt, dass das Schadenausmass der verschiedenen Pflanzenkrankheiten, Schädlinge und Unkräuter jährlich etwa 8 bis 10 Milliarden Forint beträgt, und dass sich die Kosten ihrer praktischen Bekämpfung auf 1,5 Milliarden Forint/Jahr belaufen; diese Schadensummen könnten bedeutend gesenkt werden bzw. der Kostenfaktor des Pflanzenschutzes könnte wesentlich ermässigt werden, wenn der Praxis entsprechend ausgebildete Pflanzenschutz-Spezialisten zur Verfügung stehen würden.

Im dritten Fünfjahrplan richtet sich die in der Landwirtschaft geplante Entwicklung nach *der Intensivierung*. Unter intensiven Verhältnissen liegt das »sine qua non« der quantitativen Ertragsergebnisse, vor allem aber auch der qualitativen

Tabelle 1

Die Gliederung der pflanzenhygienischen (bzw. pflanzenärztlichen)



Produktion in der Zeitgemässheit und im hohen technologischen Stand des Pflanzenschutzes bzw. der Pflanzenhygiene. Die meisten Produktionsaufwände des Pflanzenbaus (z. B. der Einsatz von Handelsdüngern, gezüchteten Pflanzen, der Bewässerung usw.) fliessen nur dann zurück, wenn die Kulturen vor Krankheiten und Schädlingen geschützt werden, das heisst wenn im Produktionsbetrieb der betriebliche Pflanzenschutz einen hohen Stand erreicht. Ein zeitgemässer und entsprechend programmierter Pflanzenschutz ist deshalb so sehr erwünscht, da der Anteil der rückkehrenden Kosten sowie die Rückkehrzeit ausserordentlich günstig sind. Ein jeder in den Pflanzenschutz investierte Forint kehrt bei uns 4- bis 5malig zurück.

Die Chemisierung dringt nicht nur in der landwirtschaftlichen Produktion, sondern *in unserem ganzen Leben vor*; mit dem chemischen Pflanzenschutz kommen nicht nur die Beschäftigten der Produktionsbetriebe, sondern auch die Konsumenten in eine enge Beziehung. Einer der wichtigsten Faktoren des in aller Welt geführten Kampfes um mehr Lebensmittel und Erhöhung des Lebensstandes ist die Erhöhung der Produktivität in der Landwirtschaft, wobei der Mechanisierung und Chemisierung eine ausschlaggebende Bedeutung zukommt. Es ist aber allbekannt, dass infolge des intensiven chemischen Pflanzenschutzes die Rückstände der giftigen Pflanzenschutzmittel ein stets schwierigeres Problem bedeuten. Einige weisen darauf hin, dass die Menschheit heute vor einem besonderen Dilemma steht: es ist ihr die Wahl zwischen dem zunehmenden Hunger infolge der Übervölkerung oder der Lebensmittelvergiftung infolge der Rückstände an chemischen Mitteln geboten. Wie GRANHALL (1963) betont, spielt sich dieses Drama vorrangig auf dem Gebiete des Pflanzenschutzes ab, da vor allem der Pflanzenschutz bzw. die Pflanzenhygiene dafür verantwortlich sind, die Ernte möglichst restlos der menschlichen Ernährung zuzuführen, nicht aber den Krankheiten, Schädlingen und Unkräutern zur Beute lassen.

Ohne die indirekte und direkte Wirkung der im chemischen Pflanzenschutz zur Bekämpfung der Krankheiten, Schädlinge und Unkräuter der Kulturpflanzen eingesetzten human-toxischen Stoffe unterschätzen zu wollen und ohne den schädlichen Einfluss der einzelnen Pflanzenschutzmittel auf die natürlichen Lebensgemeinschaften, nützlichen Insekten und insektenfressenden Vögel, auf Wild und Fische bzw. auf die Fruchtbarkeit und die Kleinlebewesen des Bodens unbeachtet zu lassen, soll auf einige solche allgemeine Erscheinungen der Landwirtschaft der Welt hingewiesen werden, die trotz aller derzeit bestehenden Schwierigkeiten die Nötigkeit der Chemisierung der landwirtschaftlichen Arbeiten, vor allem aber der Verstärkung des chemischen Pflanzenschutzes befürworten. Eine solche allgemeine Tendenz ist die Verknappung der Arbeitskräfte in allen entwickelten Industrieländern, die eine möglichst intensive Mechanisierung und Chemisierung erfordert; ferner der steigende Qualitätsanspruch des Marktes (und besonders der ausländischen Märkte) wie auch der Konsumenten gegenüber den gekauften Erzeugnissen, weiters eine bedeutende Mässigung der Produktionskosten und i. allg. der Selbstkosten. Der chemische Schutz stellt den schnellsten und meistens billigsten Pflanzenschutz sowie die wirksamste Erfüllung der pflanzenhygienischen Aufgaben

dar, wofür die Industrialisierung der landwirtschaftlichen Produktionsarbeit auch weiterhin unverändert mit einem weitgehenden Einsatz verschiedener Chemikalien eng verbunden sein wird. Dies beweisen auch die folgenden Angaben. Während 1961 in Ungarn die Zahl der zugelassenen und im Handel erhältlichen Pflanzenschutzmittel 96 betrug, waren es 1962 — 125, 1963 — 136, 1968 schon 223. Zugleich waren es in der BRD 1080 i. J. 1962, 1090 i. J. 1963 und schon über 1100 i. J. 1967. In den USA betrug die Zahl der zugelassenen Mittel 9072 i. J. 1961, 9487 i. J. 1963 und beläuft sich derzeitig auf über 9500.

Von seiten der Betriebspraxis stellt der weitgehende Einsatz der gefährlichen Pflanzenschutzmittel auch das unumgänglich gewordene Problem der Erweiterung der Facharbeiterbildung im Pflanzenschutz. Die Zahl der betrieblichen Pflanzenschutzmaschinen stieg nähmlich parallel mit der Förderung des Pflanzenschutzes von 2000 St. des Jahres 1964 auf 8500 St. im Jahre 1966 und wird bis 1970 wahrscheinlich 12 000 St. erreichen. Der Aufwand an Pflanzenschutzmitteln wird 1970 voraussichtlich 100 000 Tonnen überschreiten und einen Umschlagswert von 1 bis 1,3 Milliarden Tonnen betragen. Unter Beachtung des Anlaufs an Maschinen und chemischen Mitteln bis 1970 soll binnen drei Jahren die Ausbildung von 7000 Facharbeitern bzw. angelernten Arbeitern gelöst werden. Diese Zielsetzung kann bei dem derzeitigen Tempo des Unterrichts nicht realisiert werden, da statt der planmässigen Ausbildung der vorgesehenen 2500 Personen jährlich nur 700 bis 800 Personen ausgebildet werden (Szöllősi, 1967). Obwohl der Aufwand an Chemikalien und der Einsatz von Pflanzenschutzmaschinen einen so grossen Produktionswert darstellten, verfügt nur jeder dritte Produktionsbetrieb über einen Pflanzenschutzfachmann, aber nur über einen mit Facharbeiterausbildung. Dagegen stehen in allen Produktionsbetrieben eine Reihe von Mechanisierungsfachleuten zur Verfügung (Maschineningenieure, Traktoristen, Monteure usw.), wobei die gesamten Maschinenamortisationskosten der Landwirtschaft, bezogen auf einen Produktionszyklus, 1,9 Milliarden Forint betragen.

In aller Welt befasst man sich mit dem Problem der Pflanzenschutzmittelrückstände und der Karenzzeiten; sogar internationale Organisationen, wie die FAO, EPPO, WHO und andere, haben sich dessen angenommen. In dieser Hinsicht ist die Bestimmung jener minimalen Menge der verschiedenen Pflanzenschutzmittel akuter und chronischer Toxizität am wichtigsten, die als annehmbare tägliche Dosis (als sogenannter Toleranzwert) vom Menschen sein ganzes Leben lang verzehrt werden kann, ohne zu einer nachweisbaren Schädigung zu führen. In allen Staaten mit einer entwickelten Landwirtschaft wurden Massnahmen über die an Lebens- und Futtermitteln duldbaren, das heisst, maximal zugelassenen Rückstandsmengen (Toleranzwerte) getroffen, oder ist man zumindest überall, wo die Methode der Toleranzwerte noch nicht angewandt werden kann, daran bestrebt, beim Einsatz von human oder tierisch toxischer Pflanzenschutzmitteln eine Karenz, das heisst Wartezeit, anzuwenden. Es könnte natürlich nur begrüsst werden, wenn man auch für die einzelnen Pflanzenschutzmittel sogenannte Spezifikationen, das heisst solche Zusammenfassungen herstellen würde, die die chemischen und physikalischen Eigenschaften der Wirkstoffe, ihre Wir-

kungsart und mikroanalytischen Methoden enthalten würden, ähnlich den Veröffentlichungen der WHO über die Arzneimittel gegen humane Infektionskrankheiten.

Die einzig mögliche Weise der Beseitigung der sachlichen Fehler und Mangelhaftigkeiten im praktischen Pflanzenschutz ist die Einbürgerung und Verbreitung der zeitgemässen pflanzenhygienischen Anschauung im Kreise der Fachleute, aber auch überall in der landwirtschaftlichen Produktionsarbeit. Solange nicht in aller Welt die heute kaum umgängliche institutionelle Organisierung der pflanzenärztlichen Ausbildung und der Pflanzenheilkunde erfolgen wird, soll wenigstens eine moderne Auffassung der Pflanzenhygiene bzw. eine richtige hygienische Praxis ausgebildet werden, die nicht nur aus einer Prophylaxis der Krankheiten und Schädlinge sowie aus der Übereinstimmung agrotechnischer, agrobiologischer, mechanischer, physischer und chemischer Methoden besteht, sondern auch die externen oder internen Verfahren der Heilbehandlung anwendet. Die pflanzenhygienische Anschauung soll auf grundlegenden phytopharmazeutischen, pathophysiologischen und biochemischen Grundlagen beruhen. Auf Grund der modernen und umfassenden pflanzenpathologischen bzw. -hygienischen Anschauung werden die Entwicklungsmöglichkeiten des in der Praxis durchführbaren effektiven Pflanzenschutzes in den folgenden Richtungen gesehen:

1. In der eingehenden biochemischen Kenntnis der Stoffwechselverhältnisse der Krankheitserreger sowie der Wirt–Parasit-Beziehung können die Mittel und Verfahren des chemischen Pflanzenschutzes (z. B. die Anwendung von Antibiotika, systematischen Fungiziden usw.) mit grösserer Sicherheit und bewusster erwählt werden.

2. Nach der Klärung des pflanzenphysiologischen und biochemischen Hintergrundes der Resistenz-Züchtung werden durch die bewusste und fachmässige Auswahl und Erzeugung von resistenten Individuen, Stämmen usw. resistente Pflanzensorten für die Produktion gesichert.

3. In der genaueren Kenntnis von Infektion, Erkrankung, Krankheitsablauf und Bionomie der Schädlinge kann durch die chemische Beeinflussung der Stoffwechselvorgänge eine künstliche Resistenz in den Kulturpflanzen entwickelt werden.

4. Durch eine gründliche Erschliessung der Biozönosen und der Synökologie der tierischen Schädlinge und der Unkräuter, durch die Einführung von selektiven, superselektiven und systemischen Mitteln sowie von Schutzmitteln repellenter und attraktanter Natur können auch die Interessen des integralen Pflanzenschutzes weitgehend berücksichtigt werden.

5. Durch die sinnvolle Erwählung und Beeinflussung der Umweltbedingungen können für die Kulturpflanzen solche günstigen Verhältnisse geschaffen werden, die eine Vorbeugung der Krankheiten und Schädlinge ermöglichen.

Solange nicht die Fachingenieure, Brigadeführer und Facharbeiter des Pflanzenschutzes, aber auch alle Kader damit im klaren sind, dass der Schutz der Kulturpflanzen bzw. die Sicherung grösserer Ernteerträge nur *mit Hilfe komplexer Methoden* auf Grund einer richtigen biozönologischen Auffassung durch eine

weitgehende Sicherung der pflanzenhygienischen Belange möglich ist, können in der fachgemässen Führung und erfolgreichen Realisierung des chemischen Schutzes keine entscheidenden Fortschritte erwartet werden. *Am dringendsten soll für die Erlernung der ätiologischen, toxikologischen, arbeitshygienischen Grundlagen im Rahmen eines organisierten Unterrichts und für die Verbreitung der wissenswerten toxikologischen Kenntnisse in bezug auf die Pflanzenschutzmittel bei den Arbeitern der Produktionsbetriebe gesorgt werden.* Allein durch die Übermittlung der Fachkenntnisse kann die erfolgreiche Verbreitung des chemischen Pflanzenschutzes in der Praxis und seine störungslose Anwendung erhofft werden. Im Komplex der Schutzmassnahmen können nur das zu erwählende richtige Pflanzenschutzmittel, der erwünschte Behandlungszeitpunkt, die nötige minimale Dosis, eine möglichst minimale Zahl der Anwendung bzw. der Spritzungen, eine Einschränkung der Missbräuche der giftigen Pflanzenschutzmittel auf einen möglichst engen Kreis, i. allg. die Sicherung eines *sinnvollen und harmonischen Schutzes* die Verwirklichung all jener Aufgaben fördern, die vor dem Pflanzenschutz bzw. der Pflanzenhygiene stehen. Die schädlichen Pflanzenschutzmittelrückstände, die Gefahren der akuten und chronischen Toxizität, der Anspruch zur Verhütung der chemischen Vergiftung natürlicher Lebensgemeinschaften, aber über all diesen hinweg die sinnvolle Senkung des Kostenfaktors des Pflanzenschutzes lassen es erwünschen, dass man den Pflanzenschutz bzw. die Pflanzenhygiene als eine komplexe Tätigkeit betrachte, als einen vornehmlich technologischen Zweig der wissenschaftlichen Wirtschaft bzw. der landwirtschaftlichen Produktion, der im Produktionsbetrieb nur von einem gut ausgebildeten Fachmann geleitet werden kann. Auch im Fachgebiete der Pflanzenhygiene ist das Zeitalter der Scharlatane und Quacksalber verstrichen, da die Pflanzenpathologie auf exakten, sicheren wissenschaftlichen Grundlagen beruht.

Die zeitgemässe pflanzenhygienische Anschauung verleiht den Fachleuten, aber auch den praktischen Produzenten nicht nur eine motivierte, toxikologische, ätiologische, i. allg. phytopharmazeutische Konzeption der Pflanzenschutzmittel als Gifte, sondern sichert zugleich auch die Lösung der praktischen Aufgaben auf einer grundsätzlich wissenschaftlichen Basis. Diese *wissenschaftliche Basis* beruht einerseits auf der biozönotischen Auffassung des integralen Pflanzenschutzes, andererseits auf der die Prävention und auf der die Therapie umfassende Bestrebung der Phytohygiene.

Aus dem bisher gesagten geht schon hervor, dass durch die Einführung der pflanzenhygienischen Anschauung nicht nur der praktische Pflanzenschutz, sondern auch *die Forschung* des Pflanzenschutzes bzw. *der Phytiatrie* revolutionäre Änderungen erlebte, indem an die Stelle der früheren, oberflächlichen biotechnischen Methoden und Anschauungen die auf tiefen wissenschaftlichen Grundlagen beruhende, naturnahe phytiatrische Anschauung trat, sowie die ausgedehnten Forschungen, die die Grundlage der ersten bilden und die sich nach der Klärung jener Zusammenhänge richten, die zwischen den einzelnen Schädlingen und Krankheiten und den Kulturpflanzen als Wirtspflanzen sowie den ökologischen Faktoren bestehen.

Gegen den Einsatz der Pflanzenschutzmittel werden — wie schon erwähnt — folgende Einwände gemacht: die Pflanzenschutzmittelrückstände gefährden die Gesundheit der Konsumenten, im Mangel selektiver Wirkungen werden auch nützliche Insekten umgebracht, die Handhabung der Mittel führt zu akuten oder chronischen arbeitshygienischen Leiden. Verfasser und DUFRENOY (1962) sind der Meinung, dass keiner der Einwände stichhaltig ist, da bei einer richtigen Anwendung, Auswahl der Schutzmittel und bei einer umsichtigen Behandlung die zugelassenen Toleranzen der Rückstände mit einer sehr grossen Sicherheit bestimmt wurden und diese daher keine Gefahr bedeuten; die neueren organischen Phosphorverbindungen und Karbamate sind systemischer Natur und in ihrer Wirkung hochselektiv. Schliesslich können die arbeitshygienischen Gefahren durch ein genaues Einhalten der Gebrauchsanweisungen auch bei den Mitteln stark akuter Toxizität gänzlich vermieden werden. Zugleich kann durch den vielseitigen, das heisst integralen Pflanzenschutz die Gefahr der Vergiftung der Biozönosen vermieden werden.

Heute gibt es im Bereiche des Pflanzenschutzes bzw. der Pflanzenhygiene keine universalen Verfahren und Mittel mehr. Im betrieblichen Pflanzenschutz soll es überall den örtlichen Verhältnissen und lokalen Biozönosen entsprechend entwickelte vertikale, nach unten gerichtete Schutzsysteme geben, die vom Fachingenieur des Pflanzenschutzbetriebs — mit der Zeit vielleicht vom Pflanzenarzt — erarbeitet und gelenkt werden sollen. Die Aufgabe des Fachmannes der Pflanzenhygiene besteht daher in der Aufschlüsselung des im Lande gültigen horizontalen Pflanzenschutzes zu einem *vertikalen Pflanzenschutz* im Produktionsbetrieb. Unsere Pflanzenhygiene bzw. unser Pflanzenschutz darf nur auf der Landesebene horizontaler Richtung sein, soll aber im Produktionsbetrieb in vertikaler Richtung wirken. Der chemische Pflanzenschutz soll daher nur dann angewandt werden, wenn es der Ertrag erfordert, anders gesagt, der Schutz soll mit solchen Mitteln und Gaben erfolgen, die in den Biozönosen die mindesten Störungen verursachen und deren chronische Toxizität weder den Menschen noch die Nutztiere beim Verzehren der Produkte gefährden. Unser Ziel soll nicht das Erreichen totaler Wirkungen sein, wir sollen uns mit einer solchen Beschränkung der schadenerregenden Populationen, Schädlinge oder Unkräuter begnügen, die praktisch den Schutz der Ernte bedeutet. Dieser betriebliche Pflanzenschutz vertikaler Natur soll sich getrost auf die lokalen Insekten- und Krankheitsprognosen und auf Grund der Bodenverunkrautung auf die Vorsage des Unkräuterschadens stützen. Wir sollen ferner innerhalb des gegebenen Betriebes das die Schädlinge und Krankheitserreger intensiv beeinflussende Bioklima, die Agrobiozönose selbst sowie ihre Struktur kennenlernen; der betriebliche Pflanzenschutz soll den örtlichen Verhältnissen entsprechend maximal verfeinert werden. Dieser vertikale Pflanzenschutz soll bis zu den Pflanzensorten, im Obstbau sogar bis zum Einzelbaum, dringen und soll die resistenten, repellenten usw. Sorteneigenschaften, das heisst die reiche Schatzkammer der Antibiose ausnutzen. Der zeitgemässe integrale Pflanzenschutz ist daher ein solcher komplexer Pflanzenschutz, der auf der eingehenden Kenntnis der örtlichen Naturverhältnisse (auch der Agrobiozönose), auf der weitgehenden Ausnutzung der Agrotechnik und der Antibiose sowie auf den Angaben der

Populationsdynamik und der Prognose beruht und womöglich mit selektiven und superselektiven Mitteln arbeitet.

Zur praktischen Lösung all dieser Aufgaben kommt der gut ausgebildete Pflanzenarzt in Betracht. Der Aufgabenkreis eines Pflanzenarztes (sei er an einen Produktionsbetrieb verpflichtet oder in der pflanzenhygienischen Organisation tätig) wäre folgendermassen gegliedert: ambulative Untersuchung (die Diagnostik von Krankheitserregern, Schädlingen und Krankheiten an eingesandten Mustern; die ätiologische Prüfung und Diagnostik der Krankheitserscheinung im Freiland), in wichtigeren und komplizierteren Fällen eine klinische Untersuchung (auf allen Pflanzenschutzstationen bzw. Forschungsinstituten und im zentralen Quarantänenlaboratorium pathologisch-anatomische und pathophysiologische Untersuchungen, Infektionstest-Untersuchungen usw.) sowie Beratung; in den Produktionsbetrieben die Führung der Schlagstammbücher in pflanzenhygienischer Beziehung, Erarbeitung lokaler Prognosen, Planung und Durchführung präventiver, protektiver, extinktiver und kurativer Massnahmen; die Erarbeitung und Differenzierung des komplexen Systems der Pflanzenhygiene und der Agrotechnik innerhalb eines Betriebes oder einer Region – im allgemeinen *die Erarbeitung und Bestimmung von vertikalen, tiefgreifenden pflanzenhygienischen Methoden für die Region bzw. den Betrieb und sogar den* Betriebszweig und *Betriebsteil* anstatt des gegenwärtigen horizontalen Pflanzenschutzes. Ein jeder Pflanzenarzt würde von Zeit zu Zeit in der Gesellschaft für Pflanzenärzte des Agrarwissenschaftsvereins über den Fortschritt des Faches einen Überblick bekommen und müsste alle vier Jahre an der verbindlich vorgeschriebenen Hochschulfortbildung teilnehmen. Es stehen Fachzeitschriften zur Verfügung (Acta Phytopathologica Ac. Sc. Hung.; Növényvédelem: Pflanzenschutz), die zwischen den Fachleuten der Forschung, der Verwaltung und der Betriebspraxis enge Beziehungen herstellen.

Wir sind der Meinung, dass die Organisierung einer zeitgemässen Pflanzenärzteausbildung nach dem Beispiel der traditionsreichen und weltberühmten ungarischen tierärztlichen Ausbildung *die Aufgabe unserer Generation ist*, da wir auf diesem Gebiete nicht ohne Präzedenzien und anerkennungswürdigen Traditionen stehen (denken wir nur an die Tätigkeit von GYÖRGY LINHART, GYULA ISTVÁNFFY, KÁROLY SCHILBERSZKY, JÓZSEF JABLONOWSZKI, BÉLA HUSZ u. a.). In dieser Beziehung soll daran erinnert werden, dass Verfasser schon 1942 als junger Assistent an der Erarbeitung des vom seitdem verstorbenen Universitätsprofessors Dr. FERENC KÖVESSI ebenfalls über die Pflanzenärzteausbildung geplanten Memorandums teilnahm. Die Realisierung des Entwurfs wurde durch die Ereignisse 1944 verhindert, obwohl dieser schon damals ein fertiger Entschluss der Landwirtschaftlichen Fakultät der Technischen Hochschule und des damaligen Landwirtschaftsministeriums war. Es ist daher unbestreitbar, dass wenn man vor 25 Jahren die Organisierung der ungarischen Pflanzenärzteausbildung für zeitgemäss hielt, so diese Ausbildung 25 Jahre später, nach einer so mächtigen Entwicklung revolutionärer Natur, wie es die Chemisierung der Landwirtschaft bedeutet, nicht nur unverändert aktuell blieb, sondern *es würde sogar ein grosser Fehler sein, ihre Realisierung* weiter zu hinauszuzögern.

Es sei erlaubt, die Begründung der Ausbildung von Pflanzenschutzspezialisten in Ungarn noch mit dem folgenden zu untermauern.

Unsere tierärztliche Ausbildung richtet sich auf die zeitgemässe hygienische Versorgung nur von einigen Nutztieren. Die durch die Tiermedizin und Tierheilkunde jährlich geretteten Werte erreichen nicht einmal annähernd die Grössenordnung, die mittels der praktischen Pflanzenhygiene durch gründlich ausgebildete Pflanzenschutzspezialisten (sogenannte »Pflanzenärzte«) gerettet werden könnten, da diese auch *heute schon jährlich 4 bis 5 Milliarden Forint betragen.* Wir müssen uns auf die Angaben von SHAW (1964) berufen, nach denen sich in den USA der gesamte tierhygienische Schaden auf 1,8 Milliarden Dollar jährlich, der gesamte pflanzenhygienische Schaden aber auf 8,1 Milliarden Dollar beläuft. Zugleich müssen sich aber die Abhandlungen des Pflanzenschutzes nicht mit der Pathologie und Parasitologie bzw. Hygiene und Heilkunde einiger Nutztiere, sondern mit zahlreichen Krankheitserregern, physiologischen und sonstigen Krankheiten sowie tierischen Schädlingen einer Anzahl von Kulturpflanzen des Garten- und Ackerbaus und von Zierpflanzen befassen, was umfangsmässig die Rahmen der tierärztlichen und in gewisser Beziehung auch die der menschenärztlichen Wissenschaften weit überschreitet. Die wichtigeren Kulturpflanzen Ungarns haben nämlich heute *365 tierische Schädlinge*, über *300 pathogene Pilze*, etwa *20 virose Krankheiten*, ferner *100 bis 120 bakterielle Erkrankungen* sowie zahlreiche physiologische Krankheiten. Man muss aber auch mit einer bedeutenden Schadenerregung von parasitischen Blütenpflanzen und raumparasitischen Unkräutern rechnen. Der durch die Unkräuter verursachte Schaden überschreitet jährlich 3 Milliarden Forint, darum ist *der höhere Unterricht* des am meisten zeitgemässen Schutzverfahrens, *der Theorie und Praxis der chemischen Unkrautbekämpfung heute schon unentbehrlich.* Ebenfalls fehlt bei uns der organisierte Unterricht der in den entwickelten landwirtschaftlichen Betrieben stets unentbehrlicheren *Agrochemiker* (der mit den Kenntnissen der Pflanzenschutzmittel und Mineraldünger, das heisst mit der Chemisierung der Landwirtschaft vertrauten Spezialisten). Dieser Unterricht könnte ins Lehrprogramm der Fakultät bzw. des Faches des Pflanzenschutzes gut eingegliedert werden.

In der Kenntnis der Bedeutung des Pflanzenschutzes bzw. der Pflanzenhygiene in aller Welt bedeutet der Gedanke der Organisierung der Ausbildung von Pflanzenschutzspezialisten in Ungarn weder einen Vorsprung noch eine etwa schädliche Utopie. Im Gegenteil! Das im Verlag der UNESCO erschienene Werk von Pierre AUGER: »Current trends in scientific research« (1964) widmet z. B. dem Pflanzenschutz einen grösseren Umfang als dem Fachgebiete Crop production, das den Pflanzenbau, den Gartenbau und die Pflanzenzüchtung umfasst. Die Problematik der Pflanzenhygiene und ihre wissenschaftliche Grundlage ist daher grösser und breiter als die derzeit bekannte Problematik der Agrotechnik, des Gartenbaues usw. insgesamt. In der BRD sind 23 Forschungsinstitute des Pflanzenschutzes tätig, ausser den spezialen Instituten von über 15 Pflanzenschutzmittelwerken und den Lehrstuhlinstituten der Hochschulen, die auf einem sehr hohen Niveau arbeiten. Aber eine ähnliche Lage finden wir auch in den USA, in England,

in Frankreich bzw. im Rahmen der RGW-Staaten in der Sowjetunion, in Bulgarien, in der DDR usw.

Die ausgebildeten *Pflanzenschutzspezialisten* (Pflanzenärzte) würden teils in den Produktionsbetrieben, teils in der pflanzenhygienischen Verwaltungsorganisation bzw. in der pflanzenhygienischen Forschung und Lehre *angestellt* werden. Allein in den Produktionsbetrieben benötigt man schon heute 2000 praktizierende Pflanzenschutzspezialisten, zugleich besteht der Anspruch auf weitere 200 bis 300 Pflanzenschutzspezialisten zur Ergänzung des Fachpersonals der gegenwärtigen Pflanzenschutzstationen (20 St.) und zur Sicherung des Nachwuchses und der Kaderergänzung an Pflanzenschutzinspektoren, die bei den Komitaten und Kreisen schon tätig sind. In den meisten Fachmittelschulen, technischen Schulen und sogar an den Hochschulen fehlen die Spezialisten der Pflanzenhygiene als Lehrpersonal. Es wartet daher auch im Bereiche des Fachlehrernachschubes eine wichtige Rolle auf die spezialisierte pflanzenhygienische Ausbildung. Solange auf der unteren und mittleren Stufe die Spezialisierung klar durchgeführt wird (es gibt Ausbildungsstätten für Facharbeiter und Brigadeführer des Pflanzenschutzes, ferner eine technische Schule und Oberschule für den Pflanzenschutz), wurde auf der oberen Stufe die Spezialisierung des Pflanzenschutzes bzw. der Pflanzenhygiene noch nicht durchgeführt, was aber einen Missstand hervorruft (es gibt schon zahlreiche Fälle dafür), dass nähmlich in den Produktionsbetrieben *die auf unterer oder mittlerer Stufe ausgebildeten Facharbeiter, Brigadeführer usw. des Pflanzenschutzes höhere Fachkenntnisse besitzen als der Agronom oder Oberagronom allgemeiner Ausbildung*, was zu unerträglichen fachlichen und sonstigen Widersprüchen führt.

Zur zeitgemässen Lösung all dieser Zielsetzungen bietet sich ein einziger Weg, und zwar die institutionelle Entwicklung der gehobenen Spezialistenbildung im Pflanzenschutz (eventuell der pflanzenärztlichen Bildung) im Rahmen einer höheren Agrarlehranstalt. Ich bin der Meinung, dass die Universität für Gartenbauwissenschaften (Budapest), deren Begründung bevorsteht, für dieses Ziel am besten geeignet wäre, da ihre zentrale Lage, ihr Positionsvorteil sowie ihre örtlichen Energien, Beziehungen zu den Fachinstitutionen (z. B. zu dem Forschungsinstitut für Pflanzenschutz, dem Forschungsinstitut für Agrochemie, Pflanzenschutzdienst u. a.) sowie die Tatsache, dass sich in der Gartenbauproduktion die Betriebsaufgaben des Pflanzenschutzes auf 40 bis 60% der gesamten Produktionstätigkeit belaufen, und daher dieses Fachgebiet im Bereiche der Gartenbauproduktion mit den grössten Ansprüchen auftritt, gänzlich dafür sprechen, dass die neue 5jährige *Fakultät für Pflanzenschutz* (Pflanzenhygiene) bzw. das neue *Fach* an dieser Universität organisiert werden soll.

Es kommt natürlich auch die Universität für Agrarwissenschaften zu Gödöllő in Frage, wo derzeit die Fachingenieurausbildung des Pflanzenschutzes im Rahmen eines einjährigen Lehrganges vorgenommen wird. »Bei dieser Ausbildungsform soll jedoch betont werden, dass die derzeitige Fachingenieurausbildung für den Pflanzenschutz eigentlich keine Spezialisten erzieht, sondern die Fortbildung der Agraringenieure sichert, die zu den allgemeinen agronomischen

Kenntnissen während eines Studienjahres auch den Pflanzenschutz dazulernen« (SZÖLLŐSI, 1967).

Während des 5jährigen Universitätsstudiums und den angeschlossenen praktischen Beschäftigungen würde nicht nur eine enge Ausbildung für den Pflanzenschutz erfolgen, sondern auch die Ausbildung der Studierenden in der landwirtschaftlichen Chemie (Chemotechnik und Agrochemotechnik), was eine *gehobene agrochemische Ausbildung* bedeutet. Das würde nicht weniger bedeuten, als dass der in den Staatsgütern derzeit so sehr entbehrte Agrochemiker-Fachingenieur in den Kenntnissen der Pflanzenschutzmittel, Mineraldünger, Regulatorstoffe usw. in entsprechender Form eine eingehende und zeitgemässe Ausbildung bekommen würde. Nur durch eine solche anspruchsvolle wissenschaftliche und Facharbeiterausbildung grosser Tragweite könnte die planmässige, folgerichtige Erziehung und Einstellung zur Arbeit der in den Produktionsbetrieben derzeit schon unentbehrlichen Spezialisten des Pflanzenschutzes (der Pflanzenhygiene) gesichert werden. Ohne dies könnten die moderne Anschauung der Pflanzenhygiene, die intensive Chemisierung der landwirtschaftlichen Produktion, die Erhöhung der Ertragssicherheit und i. allg. die gehobene Agrotechnik auch in der Zukunft nicht restlos realisiert werden.

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Die Porlinge Ungarns und ihre phytopathologische Bedeutung

(Polypori Hungariae)
III. Teil*

Von

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Im III. Teil der Abhandlung werden die Gattungen *Heteroporus* (Lanzi) Donk, *Placodes* Quél., *Ischnoderma* Karst. em. Murr., *Phaeolus* Pat. und *Anisomyces* Pilát der Familie Polyporaceae behandelt.

Heteroporus (Lanzi) Donk

Der Fruchtkörper besteht gewöhnlich aus einem Busch mehrerer, fächerartig zusammengesetzter Hüte. Stiel ± ausgeprägt, zentral oder exzentrisch. Trama weisslich bis licht eichenholzfarbig, zweiteilig; obere Schicht weich, schwammig; untere Schicht zähfaserig, härtend. Röhrensubstanz vom Trama verschieden, Poren spalten sich schnell, Öffnungen labyrinthartig. Sporen hyalin, kugelig-ellipsoid, 4—6,5 × 3—5 μ; häufig Konidien.

In Ungarn kommt eine Art vor:

Heteroporus biennis (Bull.) Lanzi

(Abb. 1)

Die synonymen Namen: Polyporus biennis (Bull.) — *Daedalea biennis* (Bull.) Fr. — *Abortiporus biennis* (Bull.) Sing. — *Sistotrema rufescens* (Pers.) — *Daedalea rufescens* Pers. — *Polyporus rufescens* (Pers.) Fr. — *Polyporus heteroporus* Fr. — *Ceriomyces terrestris* Schulzer, in Verh. zool.-bot. Ges. 1874. — *Daedalea polymorpha* Schulzer, in Oest. Bot. Zeitschrift, 1880. u. a.

Abbildung: SCHULZER 1869, p. 723. — SCHULZER, 1874, p. 452. — SCHULZER 1880, Taf. I. — ? BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, 45/5 Taf.

Literatur: SCHULZER (1869) fand den Pilz bei Kömlőd im Imsós-Wald neben einem Eichenstock im Humus. Nach der sehr schönen Zeichnung kann man die Art zweifellos identifizieren. In seinen späteren Verlautbarungen beschreibt er die-

* I. Teil erschien in Az Erd. és Faip. Egy. Tud. Közl., 1965. 1—2.
II. Teil erschien in Acta Phytopathologica, 1968. Vol. 3. No. 2. Pp. 221—239.

sen sehr verschiedentlichen Schwamm (1874) als eine geschlechtlose, Conidiensporen erzeugende Art *(Ceriomyces terrestris)*, und später (1880) als eine neue Art *(Daedalea polymorpha)*.

IGMÁNDY (1958) erwähnt zwei Fundorte bei Sopron.

Herbarium: Bélapátfalva (HE., Csapó) — Budapest (HE., Kalmár, Marton) — Sopron (HE., Halupa, Lenky, Molnár) — Sümeg (HMN., Pieschel).

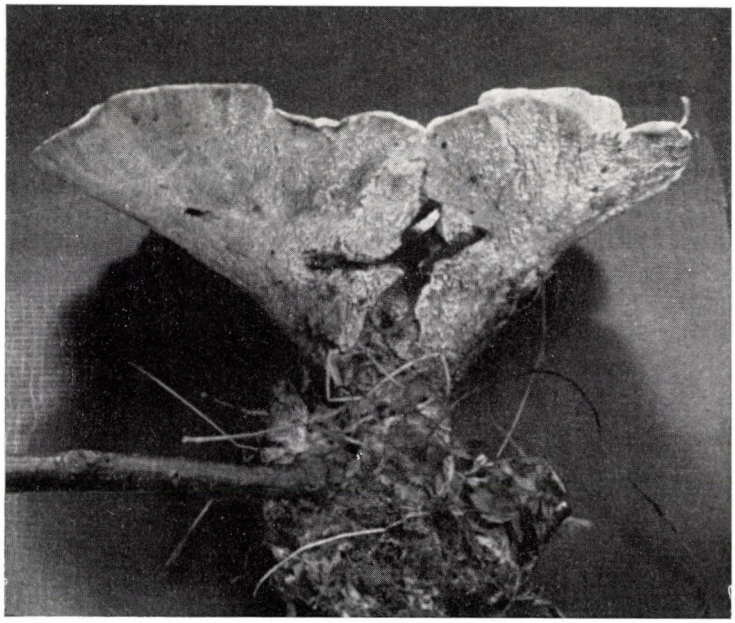


Abb. 1. *Heteroporus biennis* (Bull.) Lanzi. Sopron

Vorkommen und phytopathologische Bedeutung: Seltene Art. In Laubwäldern (Buchen, Stiel- und Traubeneiche) auf liegendem Holz und auf Wurzeln als Saprophyt. Verursacht Weissfäule. Da dieser Schwamm selten vorkommt, hat er keine wirtschaftliche Bedeutung.

Placodes Quél

Der Fruchtkörper sitzt seitlich auf, ist rund-, nieren- oder zungenartig, an der Basis stielartig verdünnt; seine glatte, einheitliche Oberfläche ist von einer abschälbaren, pergamentartigen, von der Trama gut abgesonderten Haut bedeckt. Trama kreideweiss-weisslich; im frischen Zustand saftig, käseartig; ausgetrocknet härtend, zäh. Röhrensubstanz einschichtig, von der Trama sich deutlich absondernd. Öffnung der Röhrchen ± regelrecht rund, ausgetrocknet sich spaltend.



Abb. 2. *Placodes betulinus* (Bull.) Quél. an *Betula*. Csipkerek

Sporen zylindrisch-ellipsoid oder allantoid, hyalin. Sporenstaub weiss. Auf Laubholz *(Betula, Quercus)* vorkommend.

In Ungarn sind 2 Arten bekannt.

Schlüssel zur Bestimmung der Arten

1a Trama verfärbt sich nicht in Ferrichloridlösung; Sporen allantoid 4.0—5,0 × × 1,0—1,5 μ; Fruchtkörper auf Birken *(Betula)*:

1. P. betulinus

1b Trama verfärbt sich in Ferrichloridlösung auf bräunlichgrau; Sporen zylindrisch-ellipsoid 7,0—8,5 × 2,0—3,0 μ; Fruchtkörper auf Eichen *(Quercus)*:

2. P. quercinus

1. Placodes betulinus (Bull.) Quél

(Abb. 2)

Die synonymen Namen: Polyporus betulinus (Bull.) Fr. — *Piptoporus betulinus* (Bull.) Karst. — *Ungulina betulina* (Bull.) Pat. — *Placoderma betulinum* (Bull.) Hennig u. a.

Abbildung: Schulzer, 1869, p. 785. — Bánhegyi, Bohus, Kalmár, Ubrizsy, 1953, Taf. 57/3. — Gyarmati, Igmándy, Pagony, 1964, Abb. 63. — Ubrizsy, 1965, II. p. 344. — Haracsi, 1966, B., p. 136.

Literatur: SCHULZER (1869) bezeichnet diesen Porling als eine in ganz Ungarn vorkommende Art. SZEMERE (1930) erwähnt zwei Fundorte in Transdanubien: Pamuk (SZEMERE) und Brennbergbánya (BREUER). Auf das häufige Vorkommen weist SZEMERES folgende Bemerkung: »Unsere Jungen haben ihn rund ausgeholt und innerlich benetzt, mit einem dünnen Faden eingesponnen, und so erzeugten Sie einst einen guten Ball und in Transdanubien auch noch heutzutage.« FEHÉR und BESSENYEI (1933) erwähnt Soproner, UBRIZSY (1941) Nyírséger (Bátorliget, Fény) Vorkommen; IGMÁNDY (1958) erwähnt mehrere Fundorte bei Sopron und bespricht den durch diesen Schwamm in Birkenwaldungen verursachten Schaden.

Herbarium: Bakonyszombathely (HE., Stubnya) — Csipkerek (HE., Igmándy—Pagony) — Körmend (HE., Igmándy—Pagony) — Nádasd (HE., Kiss—Pagony) — Ófehértó (HE., Igmándy) — Sopron (HE., Igmándy) — Sopronkövesd (HE., Pagony).

Vorkommen und phytopathologische Bedeutung: Dieser Schwamm ist der treue Gefährte der bei uns überall vorkommenden Warzenbirke (*Betula pendula* Roth) und der nur vereinzelten Haarbirke (*B. pubescens* Ehrh.). Häufige Art, stellenweise massenhaft auftretend. Auf eingehenden oder abgestorbenen, stehenden, liegenden oder geworfenen Stämmen, auf dicken Ästen, Stöcken. Typischer Parasit auf schon geschwächter Wirtpflanze, lebt weiter als Saprophyt auf der abgestorbenen organischen Masse. Infiziert durch Wunden. In reinen Birkenbeständen und Birkengruppen sind die durch Schneedruck verursachten Wunden offene Tore für die Infektion. Der Schwamm verursacht Rotfäule; die Zersetzung geht schnell vor sich. Der technische Schaden ist bedeutend.

2. Placodes quercinus (Schrad.) Quél

Die synonymen Namen: Polyporus quercinus (Schrad.) Fr. — *Ungulina quercina* (Schrad.) Pat. — *Piptoporus quercinus* (Schrad.) Pil. — *Polyporus suberosus* Krombh. — *Caloporus fuscopellis* Quél. — *Polyporus phalliodorus* Schulzer, Mpt., p. 780. 1869. — *Polyporus cadaverinus* Schulzer, in Kalchbrenner, Icon., p. 55. 1877. — ? *Polyporus flabellatus* Schulz. et Bres., in Schulzer, Neue Pilz-Species, Hedwigia, p. 145., 1885 u. a.

Abbildung: SCHULZER, 1869, p. 781. — KALCHBRENNER, 1877, XXXV. Taf. Fig. 1. — BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Taf. 57/2.

Literatur: KALCHBRENNER (1873/77) beruft sich auf die in SCHULZERS (1869) Handschrift, Seite 780 beschriebenen *Polyporus phalliodorus*, gibt aber falsch die Seite an und benutzt eine andere Benennung *(Polyp. cadaverinus)*. — HAZSLINSZKY (1895) berichtet über das Vorkommen im Komitat Vas (gefunden durch MÁRTON) ohne nähere Angaben.

Herbarium: Sopron (HE., Igmándy).

Vorkommen und phytopathologische Bedeutung: Sehr seltene Art. Auf Eichenstock und Eichenholz. Da der Schwamm selten vorkommt, hat er keine wirtschaftliche Bedeutung.

Ischnoderma Karst. em. Murr.

Fruchtkörper sitzt seitlich, bei der Basis oft auf die Unterlage herablaufend, einzeln, oder aus mehreren Hüten zusammengestellt. Obere Fläche ist mit einer dünnen, braunen, mit schwarzen Kreisen gefärbten, radial gerunzelten, harzigen Schicht überzogen. Trama von der Röhrenschicht abgesondert, frisch saftig, weiss, später härtend, korkig, bräunend. Poren sehr klein, rund, Porenschicht weisslich, nach Berührung schnell grau-braunend. Sporen hyalin, zylindrisch, gebogen, 5—6 × 1,5 μ. Auf Laub- und Nadelbäumen.

In Ungarn, aber in ganz Europa durch eine einzige Art vertreten.

Ischnoderma resinosum (Fr.) Karst

Die synonymen Namen: Polyporus resinosus Fr. — *Polyporus fuliginosus* (Scop.) Fr. — *Polyporus fuscus* (Pers.) Lloyd. — *Polyporus benzoinus* (Wahl.) Fr. — *Polyporus morosus* Kalchbrenner, Oest. Bot. Zeitschrft. 1869. — ? *Polyporus gibbus* Schulzer, Mpt. p. 772. 1869.

Abbildung: ? SCHULZER, 1869, p. 775, fig. 1. — KALCHBRENNER, 1873/77, Taf. XXXVI, fig. 1. — BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Taf. 57/4.

Literatur: HOLLÓS (1933) sammelte den Schwamm auf Wurzelstöcken in der Umgebung von Szekszárd. SZEMERE (1930) erwähnt zwei Fundorte: Nagytétény (auf Pfirsichbaum, gesammelt durch SCHILBERSZKY) und Brennbergbánya (auf Nadelholz, gesammelt durch BREUER). IGMÁNDY (1958) gibt einen Fundort an in Sopron, erwähnt diesen Pilz auch bei den Porlingen der Buche (1964), doch ohne Fundortangabe.

Herbarium: Budapest (HE., Miskó) — Miskolc, Jávorkút (HE., Igmándy) — Sopron (HE., Haracsi, Koronky) — Szilvásvárad (HE., Cerny—Igmándy) — Tormafölde (HE., Igmándy) — Uzsa (HE., Varga).

Vorkommen und phytopathologische Bedeutung: Fichte und Tanne *(Picea* und *Abies)* und die Rotbuche *(Fagus)* sind die Wirtpflanzen dieser Art. Kommt in den Anpflanzungen und in den autochthonen Beständen nur selten vor. Meist nur auf Stöcken oder auf liegenden Stämmen. Verursacht Weissfäule.

Phaeolus Pat

Der Fruchtkörper ist blumenstraussartig, meistens mit einem zentrischen stielartigen Auslauf (auf dem Boden oder auf der Schnittfläche der Stöcke) oder seitlich sitzend, konsolenartig (am Stamm); frisch weich, das Fleisch nass, trocken schwammig, brüchig. Die voneinander gut abgesonderten Trama und Röhrenschicht sind meistens auffallend gefärbt: gelb, rostbraun, ziegelrot u. ä. Im Hymenium fehlen die Stacheln. Fruchtkörper einjährig, Röhren in einer Schicht. Poren

von rund bis labyrinthisch, die Wand oft gespalten und gezähnt. Sporenstaub weiss, Sporen hyalin, elliptisch. In Ungarn 3 Arten. Im westlichen Grenzgebiet kann man eventuell auch den *Phaeolus fibrillosus* antreffen. Deshalb wurde auch diese Art in den Bestimmungsschlüssel aufgenommen.

Schlüssel zur Bestimmung der Arten

1a Der Fruchtkörper besteht aus mehreren ± abgesonderten Hüten, gleicht einem Strauss, Durchmesser 10—40 cm, mit einem zentrischen stielartigen Ansatz; am Boden (auf Wurzeln) und auf der Schnittfläche der Stöcke; anfangs orangegelb, später rostbraun, Oberfläche filzig, dann glatt; Röhrenschicht olivbraun, später rostbraun, Poren unregelmässig, labyrinthartig, Sporen ellipsoid, 5—8 × 3,5—5 μ. In Nadelwaldungen auf Wurzeln, Stöcken und Nadelstreu: *1. Ph. schweinitzii*

1b Fruchtkörper andersgestaltet, meist seitlich sitzend, konsolenförmig 2

2a Fruchtkörper besteht aus mehreren, eng übereinander, seitlich sitzenden, konsolenförmigen Hüten; sonstige Merkmale wie im Punkt 1a: *1. Ph. schweinitzii*

2b Die Merkmale sind andere 3

3a Fruchtkörper seitlich, manchmal auf die Basis herablaufend, lehmgelb, -braun; wird durch die Einwirkung alkalischer Lösung gleich lilafarbig; Sporen elliptisch 2,5—4,5 × 2,0—3,5 μ. Auf Laubbäumen: *2. Ph. rutilans*

3b Fruchtkörper wird durch die Einwirkung alkalischer Lösung nicht lilafarbig 4

4a Fruchtkörper nur 2—6 cm, konsolenförmig oder verflacht zurückgekrempelt; charakteristisch orange-, ziegelrot; Sporen ellipsoid, 3—6,5 × 2—3,5 μ (nach PILÁT). Auf Stöcken und Wurzeln der Fichte, Tanne usw. *Ph. fibrillosus (Karst.) B. et G.*

4b Fruchtkörper mit einem Durchmesser von 15—30 cm ± zusammengewachsenen Hüten und seitlich sitzend, auffallend orange-safrangelb; Sporen elliptisch, 4,0—5,5 × 2,5—3,5 μ. Auf Eichen: *3. Ph. croceus*

1. Phaeolus schweinitzii (Fr.) Pat

(Abb. 3)

Die synonymen Namen: Polyporus schweinitzii Fr. — *Hapalopilus schweinitzii* (Fr.) Donk. — *Phaeolus sistotremoides* (Alb. et Schwein.) Murr. — *Labirinthia truncata* Schulzer, Mpt. p. 712, 1869.

Abbildung: SCHULZER, 1869, p. 173. — BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Taf. 48/2. — IGMÁNDY, 1958, p. 122. — GYŐRFI, 1963, Abb. 104. — UBRIZSY, 1965, II. p. 346.

Literatur: IGMÁNDY (1958) erwähnt viele Fundorte aus Sopron.



Abb. 3. *Phaeolus schweinitzii* (Fr.) Pat. am Boden des *Larix*-Bestandes. Sopron



Abb. 6. *Phaeolus croceus* (Pers.) Pat. an *Quercus robur*. Sitke

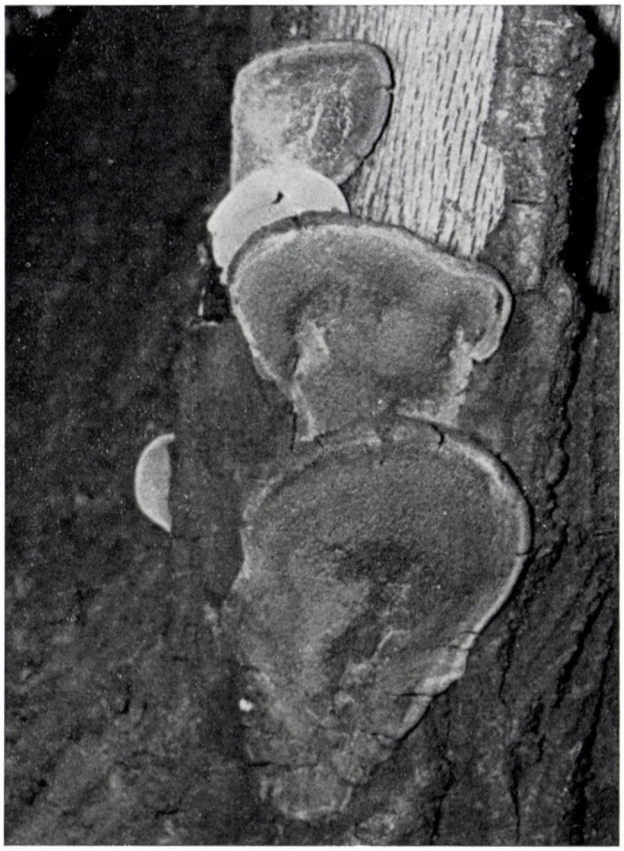


Abb. 4. *Phaeolus rutilans* (Pers.) Pat. an *Quercus cerris*. Lovasberény

Herbarium: Budapest (HMN., Babosné-Véssey) — Körmend (HE., Igmándy—Pagony) — Nádasd (HE., Igmándy—Pagony) — Pölöske (HE., Igmándy) — Sopron (HE., HMN., Babosné, Nernhardt, Haracsi, Igmándy u. n. m.).

Vorkommen und phytopathologische Bedeutung: Der Schwamm wurde bisher meistens in den westtransdanubischen Nadelwaldungen wahrgenommen (Komitate: Győr-Sopron, Vas und Zala) und auch daselbst gesammelt. Hier aber ist er auf einigen kleineren Flächen eine gemeine Art. Vornehmlich auf dem Wurzelkopf der *Picea*, *Larix* und *Pinus*, seltener auf der *Abies*, aber auch vom Stock etwas entfernt, auf verdeckten oder freistehenden Wurzeln, manchmal auf dem Stamm in verschiedener Höhe. Er ist einer der gefährlichsten Stockfäulepilze der Nadelbäume, der hauptsächlich durch die Wunden des Stocks und der Wurzeln die Bäume infiziert. Die befallenen Bäume fallen sehr oft dem Winde zum Opfer. Wo der Pilz massenhaft vorkommt, verursacht er grossen Schaden, dieser

wird aber oft — unrichtig — dem *Fomes annosus* zugeschrieben. In alten Lärchenbeständen und in geharzten Weissföhrenwäldern wurde seine grosse Schädigung aufgefunden; die vom Stock ausgehende Fäule drang sehr hoch in den Stamm empor. Erzeugt Rotfäule. Das würfelig zersetzte, rotbraun verfärbte Holz riecht intensiv nach Terpentin.

2. Phaeolus rutilans (Pers.) Pat

(Abb. 4)

Die synonymen Namen: Polyporus rutilans (Pers.) Fr. — *Polystictus rutilans* (Pers.) Bieg. et Guill. — *Hapalopilus rutilans* (Pers.) Murr. — *Polyporus nidulans* Fr. u. a.

Abbildung: BÁNHEGYI, BOHUS, KALMÁR, UBRIZSY, 1953, Tafel 48/3.

Literatur: SCHULZER (1857) fand den Schwamm in Ost- und Südungarn, wo er auf abgefallenen Buchen- und Eichenästen wuchs. HOLLÓS (1913, 1933) hat ihn zwischen der Donau und Theiss an mehreren Stellen (Kecskemét, Nyárlőrinc, Pusztavacs), in Transdanubien bei Szekszárd gesammelt. BOHUS (1939) erwähnt sein Vorkommen im Vértes-Gebirge (Vérteskozma), MOESZ (1942) bei Budapest, IGMÁNDY (1958) hingegen in der Umgebung von Sopron.

Herbarium: Bakonybánk (HE., Igmándy) — Bajna (HE., Igmándy) — Balatonendréd (HMN., Mágócsy-Dietz) — Budapest (HMN., HE., Bohus, Kalmár, Moesz) — Bükk-hegység (HMN., Bohus—Babosné) — Császár (HE., Igmándy—Varga) — Cserénfa (HE., Igmándy) — Csókakő (HMN., Bohus) — Galgamácsa (HE., Igmándy) — Gérce (HE., Igmándy) — Gyöngyössolymos (HE., Nagy) — Győrszentmárton (HE., Igmándy) — Ispánk (HE., Pagony) — Iván (HE., Apt, Igmándy) — Kaposgyarmat (HE., Igmándy) — Kerecsend (HE., Igmándy) — Mátraháza (HMN., Moesz) — Mecsek-Geb. (HE., Koronky) — Pári (HE., Igmándy) — Pilismarót (HE., Stubnya) — Pölöske (HE., Igmándy) — Réde (HE., Igmándy) — Sitke (HE., Igmándy—Pagony) — Sopron (HE., Deák, Igmándy usw.) — Szekszárd (HMN., Hollós) — Tamási (HE., Igmándy) — Ugod (HE., Igmándy, Kovács).

Vorkommen und phytopathologische Bedeutung: Aus den Angaben des Herbariums ist erkennbar, dass der Schwamm in Ungarn überall vorkommt. Zwar fehlen im Herbarium Exemplare aus dem östlichen Teile des Landes, aber nur darum, weil diese Teile floristisch noch nicht erschlossen sind. Dieser Schwamm ist häufiger in den Eichen- und Zerreichenbeständen des Hügellandes und der Vorgebirge. Da ist er überall, wenn auch nur vereinzelt, zu finden. Vorerst kommt er auf Eichen (*Quercus sessiliflora* Salisb., *robur* L., *pubescens* Will.), Zerreichen (*Quercus cerris* L.), dann Weissbuchen *(Carpinus)*, Buchen *(Fagus)*, Linden *(Tilia)*, Birken *(Betula)*, aber auch auf der Vogelkirsche (*Prunus avium* L.) vor. In der Umgebung von Sopron hatte man ihn einmal auch auf der Föhre (*Pinus silvestris* L.) beobachtet.

Der Porling ist ein typischer Saprophyt. Lebt auf vertrockneten Ästen am Baum oder auf dem Boden, seltener auf Stöcken, manchmal als Wundenparasit



Abb. 5. *Phaeolus croceus* (Pers.) Pat. Das Fleisch des Fruchtkörpers charakteristisch gestreift. Sitke



Abb. 7. *Anisomyces odoratus* (Wulf.) Pilát auf Bullen der *Picea*. Sopron

auf dem Stamm von *Prunus avium* L. Verursacht Weissfäule. Auf den Bäumen mit farbigem Kern *(Quercus)* lokalisiert sich die Fäule nur auf dem Splint. Da dieser Saprophyt hauptsächlich auf Dünnholz gedeiht, ist der verursachte Schaden nicht beachtenswert.

3. *Phaeolus croceus (Pers.) Pat*

(Abb. 5, 6)

Die synonymen Namen: Polyporus croceus (Pers.) Pat. — *Hapalopilus croceus* (Pers.) Donk. u. a.

Herbarium: Sitke (HE., Haracsi, Igmándy, Pagony).

Vorkommen und phytopathologische Bedeutung: Diese in ganz Europa verbreitete, aber überall sehr seltene Art kommt in den Weissbuchen-Linden-Stieleichenbeständen der Niederung der Raab vor. Auf einem, der hier noch stehenden, viele Jahrhunderte alten Stieleichenbaum (*Quercus robur* L.) konnte man jahrelang einen sehr grossen ($30 \times 15 \times 12$ cm), safrangelb-farbigen Fruchtkörper auffinden. Dieser ganz seltene Pilz verursacht die Rotfäule des Kernes.

Anisomyces Pilát

Fruchtkörper seitlich sitzend, von langer Dauer, aber auch perennierend. Röhren entweder gar nicht oder nur verwaschen schichtig. Trama zäh, korkartig. Substanz von Porenwand und von Trama ähnlich, als wären die Röhren in das Fleisch hineingebohrt. Farbe vom Trama und Röhrensubstanz rostbraun, Röhren innen weiss-staubig. Poren rund bis labyrinthartig. Sporenstaub weiss, Spora hyalin, zylindrisch. Auf Nadelbäumen vorkommend.

In Ungarn kommt eine Art vor:

Anisomyces odoratus (Wulf.) Pilát

(Abb. 7)

Die synonymen Namen: Polyporus odoratus (Wulf.) Fr. — *Trametes odorata* (Wulf.) Fr. — *Osmoporus odoratus* (Wulf.) Singer u. n. w.

Abbildung: Bánhegyi, Bohus, Kalmár, Ubrizsy, 1953, Taf. 52/5.

Literatur: Igmándy (1958) erwähnt mehrere Fundorte bei Sopron.

Herbarium: Baktalórántháza (HE., Igmándy) — Bárna (HE., Igmándy) — Diósjenő (HE., Rumszauer) — Inke (HE., Igmándy) — Marcaltő (HE., Csötönyi) — Miskolc, Jávorkút (HE., Igmándy) — Sopron (HE., Igmándy, Juharos a. n. m.).

Vorkommen und phytopathologische Bedeutung: Hauptwirtpflanze des Porlings ist bei uns die Fichte *(Picea)*, auf der Föhre (*Pinus silvestris* L.) ist er sehr selten. Wächst vornehmlich auf Fichtenstock und hauptsächlich in Gegenden, wo dieser Baum seit langer Zeit angepflanzt wird — dort ist aber dieser Schwamm keine seltene Art (zum Beispiel in der Umgebung von Sopron). Man findet ihn aber auch in Parkanlagen und im Walde, wo die Fichte nur einzeln oder in Grup-

pen steht (Baktalórántháza, Marcaltő). Der Fruchtkörper erscheint hauptsächlich im dichten Fichtenbestand, im Kahlschlag dagegen selten. Rotfäuleerreger. Die jungen Fruchtkörper und die eine neue Porenschicht erzeugenden Fruchtkörper riechen intensiv nach Fenchel. Das zersetzte Holz hat den gleichen charakteristischen Fenchelgeruch. Durch das Zersetzen der zurückgelassenen Stöcke ist der Schwamm aus biozönotischem Gesichtspunkt ein nützlicher Saprophyt.

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A Study on the Biological Activity of Isoelectronic Systems

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The isoelectronic compounds pyridine-2-thiol-N-oxide (I) and 2-fluoro-thiophenol (II) were tested as model substances for fungistatic action in order to examine whether isoelectronicity can serve as a basis for similar biological properties. While pyridine-2-thiol-N-oxide, known to be a potent fungicide, proved to be highly active, the activity of 2-fluoro-thiophenol was poor, indicating that isoelectronic compounds or groups, respectively, are not interchangeable as regards to their biological action.

Analogies between the infrared and Raman spectra of pyrazine-di-N-oxide and p-difluorobenzene as well as between those of pyridine-N-oxide and monofluorobenzene have been reported by SZŐKE, VARSÁNYI and BAITZ (1967a, 1967b). The similarities in the parameters within these two pairs of compounds are due to the fact that the N-oxide and the corresponding fluoro derivatives have isoelectronic structures. The criteria of isoelectronicity have been formulated by BENT (1966) as follows: the same number of electrons and heavy atoms and agreement between the bond diagrams, and as a consequence, similar electronic structures and similar heavy atom geometries. As for the characteristic N—O and C—F bonds (diatomic molecules) the interatomic distances, the reduced masses, the mean values of electronegativities and effective nuclear charges are similar:

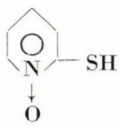
$R(NO) = 1.151$ Å	$R(CF) = 1.271$ Å
$\mu_{NO} = 0.134$	$\mu_{CF} = 0.136$
$(e_N e_O)^{1/2} = 3.24$	$(e_C e_F)^{1/2} = 3.16$
$(Z_N^* Z_O^*)^{1/2} = 4.22$	$(Z_C^* Z_F^*)^{1/2} = 4.12$

There is a great similarity also in the bond orders of N—O and C—F, the —I effects being nearly equal and the free electron pairs of O and F atoms interact similarly with the π bond system of the aromatic ring (SZŐKE, VARSÁNYI and BAITZ, 1967a).

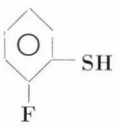
As these characteristics are known to be decisive factors of biological activity, it was tempting to test whether isoelectronic molecules can be expected to show similarities also in their biological properties.

Pyridine-2-thiol-N-oxide (I) and 2-fluoro-thiophenol (II) were chosen as

model substances as they can be regarded as isoelectronic compounds and the former is known to be a potent fungicide, its activity being extensively studied by SIJPESTEIJN et al. (SIJPESTEIJN, ROMBOUTS, VAN ANDEL and DEKKER, 1958; ROMBOUTS and SIJPESTEIJN, 1958).



I



II

Pyridine-2-thiol-N-oxide has been submitted by Olin Mathieson Chemical Corporation, New York; 2-fluoro-thiophenol has been prepared according to SHARGHI and LALESARI (1963) (University of Teheran) and submitted by the authors. The fungistatic action of the compounds was tested as sodium salts on two fungal species, using the "spray" and "bath" techniques described by BÁNKI et al. (1966). The results are presented in Table 1.

Table 1

Fungistatic activity of pyridine-2-thiol-N-oxide and 2-fluoro-thiophenol on two fungal species. — Concentration of the test substance in % required for ED_{50}

	Pyridine-2-thiol-N-oxide	2-Fluoro-thiophenol
Alternaria tenuis		
inhibition of germination		
spray	$5 \cdot 10^{-6}$	2
bath	$7 \cdot 10^{-7}$	$5 \cdot 10^{-2}$
inhibition of growth		
spray	$5 \cdot 10^{-6}$	1
bath	$2 \cdot 10^{-8}$	10^{-2}
Aspergillus niger		
inhibition of germination		
spray	$3 \cdot 10^{-5}$	1
bath	$9 \cdot 10^{-8}$	$3 \cdot 10^{-1}$
inhibition of growth		
spray	$3 \cdot 10^{-5}$	1
bath	$9 \cdot 10^{-8}$	$3 \cdot 10^{-1}$

The experimental data gave further evidence of the potent fungistatic action of pyridine-2-thiol-N-oxide, while the isoelectronic 2-fluoro-thiophenol proved to be practically inactive.

Although no generalization should be made from a single pair of compounds, this negative result indicates that isoelectronicity cannot serve as structural basis

for common biological properties, however, other factors not included in the concept of isoelectronicity must be dominating in this respect.

This is in contrast with the biological role of some other features of molecular structure, e.g. with the possibility of interchange of isosteric groups without significant alteration of biological activity.

The main reason of the failure of isoelectronicity in designing of equieffective compounds might find its explanation in the fact that isoelectronic compounds may show great differences in polarity, a factor of importance in biological behaviour.

Further studies are needed in order to examine whether isoelectronicity can serve as basis for designing of antimetabolites.

*

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The Effects of Various Fruit Species on the Development and Diapause of the Plum Fruit Moth (*Grapholitha funebrana* Tr., *Lepidoptera:Tortricidae*)

By

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The well-known hosts of the plum fruit moth (*Grapholitha funebrana* Tr.) are the plum and, in Hungary, the blackthorn. Diapause studies were made with respect to other hosts reported in literature. The number of larval molts, in the fruits was determined by the number and size of the cast-off cephalic capsules.

Following the control hosts (plum and greengage), the lowest mortality during development has been found in apple. On ripe cherry, however, mortality of 43 per cent was observed. In the fruit species listed above, the larvae had four instars and the onset of diapause depended on the duration of daily illumination. Concerning unripe cherry, it was probably the quality of food and not the light period which affected the commencement of diapause. In all other fruit species, mortality was above 50 per cent. In the green nut variety studied, no plum fruit moths had successfully developed.

Introduction

The development of *Gr. funebrana* Tr. in plum is well-known in its entire area of distribution (BOVEY, 1937) and appears therein on the pest-lists of nearly all countries, including also in Hungary. Its occurrence in other fruit species is probably explainable by the effects of olfactory attractance (DESEŐ, 1967b).

Literature lists the following hosts:

It was found in sloe by KOSTROVSKY in Turkestan (1914), by UVAROV near Stauropol (1914) and by VINOKUROV in Yereva (1916). Its presence in this host is commonly known also in Hungary (DESEŐ, 1967a,b).

MOERICKE (1960) reports on damages caused to sour-cherries. According to this author, only one generation of the plum moth develops in sour-cherry; the observed rate of infestation was 2—4 per cent. UBRIZSY—REICHART (1958), as well as SYTENKO (1960), also record sour-cherry as one of the hosts of the plum fruit moth in the maritime provinces of the Soviet Union.

Cherry as a food-plant is listed by KOSTROVSKY (1914) in Turkestan and by SCHOYEN (1917, 1919) in Norway, while ECKSTEIN in Germany (1933) explicitly defines it as "Pflaumen und Kirschenmade". According to WIESMAN (1936), failing a plum crop the first brood developes in cherry; infections of 3—5 per cent were observed. Cherry as a host of the plum moth is also mentioned by MIELLER in

Germany (1939), and by DANILEVSKY (1958), who found it in this fruit everywhere from the Transbalkan to the maritime provinces. GLOWACKI (1966) submits an accurate description of the deformations on the fruit caused by the development of the larvae.

Apricot was listed by SACHAROV in Astrakhan (1914), by an anonymous writer in Turin (1916, 1916, 1920), by BÖHM in Austria (1948), and by UBRIZSY—REICHART in Hungary (1958); DANILEVSKY found it ranging from the Transbalkan to the maritime provinces (1958), TIHONOV in Samarkand (1962), and OL'KHOVSKAVA-BURKOVA in the Ukraine (1963). According to BALÁS (1966), it occurs but rarely on apricots in Hungary.

Peach as host was reported by KOSTROVSKY in Turkestan (1914), by BÖHM in Austria (1948), by UBRIZSY—REICHART in Hungary (1958), and by OL'KHOVSKAVA-BURKOVA in the Ukraine (1963). BALÁS, again, states that the pest occurs seldom in peaches (1966) in Hungary.

VITKOVSKY found it in nut in Bessarabia (1913), and PLOTNIKOV in Turkestan (1914).

RADETZKY reports the plum moth from apple in Turkestan (1913), and VERESCHAGIN—VERESCHAGINA (1957) even contend that the first brood infests the apple and it is only the second generation which attacks plum. According to these authors, there are no plum fruit moths in plum tree stands isolated from apple orchards. (In our laboratory experiments, apple is regularly used as the host of the plum moth.)

Within a given habitat, the fate of insect populations is primarily determined by the host plants available. For the plum moth, the formation and the subsequent fate of the host crop is the primary factor on which the survival of the population depends. It is common knowledge that, in Hungary, plum trees do not invariably yield fruit. The commonest factor causing an early fruit-fall is the damage attributable to the plum sawflies (*Hoplocampa minuta* Christ., and *H. flava* L.). The small, fallen fruits, discarded owing to sawfly activity, are lost as possible sites for the oviposition and development of larvae of the plum fruit moth. In times of heavy infections by *Hoplocampa* spp., nearly 100 per cent of the crop may fall from the trees. With respect to the literature data submitted above, the problem of the future fate of the plum fruit moth population inhabiting a like orchard comes automatically to the fore. To obtain a clearer picture of the factors involved, we have investigated the fruit species which might serve as hosts, in want of a plum crop, for the development of the plum fruit moth. We have attempted in our experiments to receive information on the following problems:

1. Which are the common fruit species suitable for the complete development of the plum moth in Hungary? — 2. What is the mortality rate during development in various fruits? — 3. Is there a difference in the numbers of larval molts and the sizes of head capsules per fruit species? — 4. Do the different host qualities affect the diapause of the plum fruit moth? — 5. Does a given fruit species modify the inducing effect of the photoperiod on diapause?

The following fruit species had been examined: unripe (green) and ripe (red) cherry; half ripened sour-cherry; unripe (green) and ripe (yellow) apricot; unripe (green) peach; unripe (green) Jonathan apple; unripe (green) winter pear; ripe (concurrently with barley) summer pear; unripe (green) nut. Greengage and Besztercei plum variety were used for control. The above fruit species occur frequently in gardens and orchards in Hungary, hence they are available as secondary, in want of the primary, hosts for the plum moth.

Material and Methods

Eggs deposited on the Besztercei plum variety placed in wooden frame containers covered by gauze and kept in the laboratory at room temperature had been individually cut off, together with some square millimeters of the underlying plum skin, and glued by rye flour-paste onto the fruit to be examined. (The detailed description of the egg-transference by the gluing rearing method is given in SÁRINGER's (1967) paper.) Fruits bearing eggs were placed in glass dishes. For facilitating the pupation of full grown larvae, pieces of corrugated paper were placed between the fruits. The glass containers were covered by cellophane. Twenty-five fruits of each fruit species were used in the experiments. The rearing containers were put in boxes and exposed to various photoperiods in a constant temperature chamber at 23°C ($\pm$1.7). It was shown in earlier experiments (SÁRINGER, 1967) that whereas a daily light period shorter than 14 hours induces diapause in the plum moth, an illumination of 17 hours results in development without diapause. In the present experiments we have therefore applied two short-days (12 and 14 hours) and one long-day (17 hours). By this arrangement, the effects, resulting from various photoperiods and host species, influencing diapause could be concurrently studied.

At the end of summer the diapausing larvae were placed on the table of an insectarium outdoors. They have spent the winter there, and the hatching of adults was also observed there in the spring.

The fruits in which the larvae developed were stored in 4 per cent formol, then cut open under the microscope and exuvial head capsules in the tunnels counted. From their numbers and breadth, inferences were drawn on the number of molts. Measurements characteristic of the various stages of molting were already known from earlier investigations (DESEŐ, 1966).

Results and Discussion

The summarized results are shown in Table 1. From the data the following inferences per fruit species can be made:

Apple (variety Jonathan, unripe, green). Following the plum variety Besztercei and the greengage, as primary hosts, the developing plum fruit moth larvae

Table 1

Development of the plum fruit moth (Grapholitha funebrana Tr.) in several fruit species

Fruit	Conditions of rearing	Number of generation	Commencement of experiment	First adult hatching in 1966	Last adult hatching in 1966	First adult hatching in 1967	Last adult hatching in 1967	Percentage of hatched adults* in 1966	Percentage of hatched adults* in 1967	Percentage larval and pupal mortality	Mean larval and pupal mortality
Plum ("Besztercei" variety, control)	23° 17h	summer II.	VI. 30	VII. 29	VIII. 3	—	—	100	0	0	0
	12h	,,	,,	—	—	V. 6	V. 10	—	100	0	
Greengage (control)	23° 17h	summer I.	VI. 11	VII. 7	IX. 3	V. 11	V. 11	83.3	16.7	20.0	25.2
	12h	,,	,,	—	—	V. 3	V. 13	0	100	31.9	
Apple ("Jonathan")	23° 17h	summer I.	VI. 18	VII. 23	VII. 23	—	—	100	0	50.0	
	14h	,,	,,	—	—	V. 9	V. 10	0	100	25.0	32.5
(unripe, green)	12h	,,	,,	—	—	V. 9	V. 30	0	100	22.7	
Cherry	23° 17h	summer I.	VI. 18	VII. 16	VII. 25	V. 11	V. 11	40.0	60.0	59.1	
(ripe, red)	14h	,,	,,	—	—	V. 8	VI. 13	0	100	20.9	43.0
	12h	,,	,,	—	—	V. 8	V. 9	0	100	49.2	
Sour-cherry	23° 17h	summer I.	VI. 3	VII. 1	VII. 1	—	—	100	0	66.7	
(half ripened)	14h	,,	VI. 7	—	—	V. 1	V. 8	0	100	52.3	57.3
	12h	,,	VI. 10	—	—	V. 6	V. 7	0	100	53.0	

Cherry (unripe, green)	23° 17^h	summer I.	VI. 7	—	—	V. 6	V. 8	0	100	78.6	
	14^h	,,	,,	—	—	V. 1	V. 16	0	100	61.3	62.5
	12^h	,,	,,	—	—	V. 5	V. 8	0	100	47.6	
Apricot (unripe, green)	23° 17^h	summer I.	VI. 17	VII. 17	VII. 17	—	—	100	0	83.4	80.0
	12^h	,,	,,	—	—	V. 11	VI. 14	0	100	76.7	
Summer pear (ripe)	23° 17^h	summer I.	VI. 18	—	—	—	—	—	—	100	
	14^h	,,	,,	—	—	V. 8	V. 11	0	100	74.5	80.4
	12^h	,,	,,	—	—	V. 8	V. 13	0	100	66.7	
Peach (unripe, green)	23° 17^h	summer I.	VI. 18	—	—	—	—	—	—	100	
	14^h	,,	,,	—	—	—	—	—	—	100	88.9
	12^h	,,	,,	—	—	V. 9	V. 9	—	100	66.7	
Apricot (ripe)	23° 17^h	summer II.	VI. 30	—	—	—	—	—	—	100	93.7
	12^h	,,	,,	—	—	V. 7	V. 9	0	100	87.5	
Pear (unripe, green)	23° 17^h	summer I.	VI. 18	—	—	—	—	—	—	100	
	14^h	,,	,,	—	—	—	—	—	—	100	96.4
	12^h	,,	,,	—	—	V. 9	V. 11	0	100	89.2	
Walnut (unripe, green)	23° 17^h	summer I.	VI. 18	—	—	—	—	—	—	100	
	14^h	,,	,,	—	—	—	—	—	—	100	100
	12^h	,,	,,	—	—	—	—	—	—	100	

* Without respecting mortality during larval or pupal stage.

had the lowest rate of mortality in apple. The rescuing role of apple with regard to the plum fruit moth population was first experienced in practice in 1967. In a mixed orchard, nearly 100 per cent of the plum fruit crop was cast owing to the damages caused by *Hoplocampa* species. A rather considerable amount (5—8 per cent) of plum fruit moth adults had hatched from the apples gathered in the orchard 15—30 July for the sake of collecting codling moth larvae. In 1966, a year when there was hardly any *Hoplocampa* damages in the same orchard, only occasional plum fruit moth adults were reared from apples. The number of larval molts developing in apple was four, and the size of the head capsules also agreed with the data obtained from those developing in plum fruits.

Cherry (unripe, green). Though with a significant mortality, larvae developed under all three photoperiodic exposures. The cherry fruits had to a certain extent modified, as related to the control, the photoperiodic effect, since diapause was induced also at long-day light period (17 hours). This phenomenon requires further study. The number of molts and the size of the head-capsules agree with the data obtained from larvae developing in the control host.

Cherry (ripe, red). Development had a lower rate of mortality than in unripe cherries. The effects of photoperiodicity agreed with those experienced in the control breedings, that is, it was not the quality of the fruit but the light period which determined the onset of diapause. The number of larval molts and the size of the head capsules again agreed with those obtained from larvae developing in plum fruits.

Sour-cherry (half ripened). The larvae developed with 50 per cent mortality. The effect of the photoperiod on diapause was similar to that found in the control. The development of the larvae did not differ from that in the control hosts.

Apricot (unripe, green). The larvae developed under a significant mortality. There were no modifying photoperiodic effects against those found in the control.

Apricot (ripe, yellow). Mortality was higher than in the unripe apricots. Photoperiodic effects agreed with those of the control.

Peach (unripe, green). The plum fruit moth develops only with high mortality rate in this fruit.

Summer pear (ripening concurrently with barley). The mortality is lower than in the case of the winter pear. Photoperiodic effects are similar to those of the control.

Winter pear (unripe, green). The plum fruit moth developed with a very high mortality in this fruit. The winter pear did not modify the effects of the photoperiod as compared to the control.

We had no occasion to establish the number of molts and the size of the head capsules for larvae developing in apricots, summer pears, peaches, and winter pears.

Nut (unripe, green). No possibility of development was given, as the L_1 larvae were unable to penetrate the fruit.

Considering mortality rates occurring during the larval and pupal stages, mortality rate lower than 50 per cent was found in the case of two fruit species only: in unripe apples and ripe cherries.

Table 1 shows, besides the possibility of development of the plum fruit moth, also a precedence of the fruit species examined. It is to be emphasized, however, that only certain varieties of the given fruit species had been studied, and it is quite possible that the plum fruit moth may develop in some other varieties of the examined fruit species, as it was observed with respect to diverse varieties of peach by BOVEY in 1937.

Conclusions

1. Of the fruit species examined, mortality rates below 50 per cent of the developing larvae were found, except for the plum variety Besztercei and the greengage only in the unripe Jonathan variety of apple and in ripe cherry.

2. The differences in quality of the various fruit species have no remarkable effects on the diapause of the plum moth.

3. The larval diapause of the plum fruit moth is decisively determined by the photoperiod.

4. In the examined Besztercei plum variety, in the greengage, in the unripe Jonathan variety of apple, in ripe and unripe cherry, and in the sour-cherry variety studied, the number of larval molts was found to be four and there were no differences in the size of the exuvial head capsules of the larval instars in the various fruit species.

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* RAE = Review of applied Entomology.

Sexual Attractiveness of Virgin Plum Fruit Moth, *Grapholitha funebrana* Tr. *(Lepidoptera: Tortricidae)* Females Examined by ^{32}P Labelled Males

By

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Authors studied the attraction of males by virgin females of *G. funebrana* in the field. Males marked by ^{32}P isotope were released. Traps containing virgin females were placed at points of concentric rings of different radii around the site of release of the males. According to the results, the rate of males captured by the traps containing virgin females showed no proportional decrease by distance.

No difference was found in the behaviour, duration of life, and mating ability of unmarked males and those marked with ^{32}P.

Detailed data were obtained on the optimal height of traps, the daily flight periods of males, as well as the relationship between age and attractiveness of the virgin females.

Introduction

During the last ten years many a word was said on the injurious effects of pesticides on the living world. Starting from the sadly deleterious findings, research workers, active in the field of plant protection, endeavoured in recent years to work out methods by the application of which crops could be grown free of pesticide residues with a hopefully concomitant decrease in injuries of the biocenoses. Among the many new approaches, the present paper submits the results of our experiments made for the sake of elaborating a method to collect male insects by the sex attractants of virgin females.

Entomologists have for a long time been aware of the attraction exerted of the sexes on one other, but the possibilities of using this characteristic in plant protection was recognized only in the early thirties by STELLWAG (cited by LHOSTE, 1962). He recommended that virgin females be placed in closed wire cages to have the males lured to them. Practical attempts were also soon made for the attraction of males of certain moth species. However, the results of the experiments forced the investigators to the nearly uniform statement that this method could find no wide-ranging practical application until the attractant substance is not produced synthetically.

Attention has currently turned to this method again and every effort is made, e.g. in the United States of America, for the synthetical production of the attractant substances (cf. JACOBSON's comprehensive work, 1965).

In our opinion, even until the synthetic mass production of the relevant substances is solved, it ought to be clarified, for as many pests as possible, which sex (male or female) attracts the other one, and to what rate could a reduction of the population by sex attractants be effected. *G. funebrana* is one among the species of which we know nothing in this respect. We have found in 1966, by preliminary field observations, that males can be gathered by the use of freshly hatched virgin

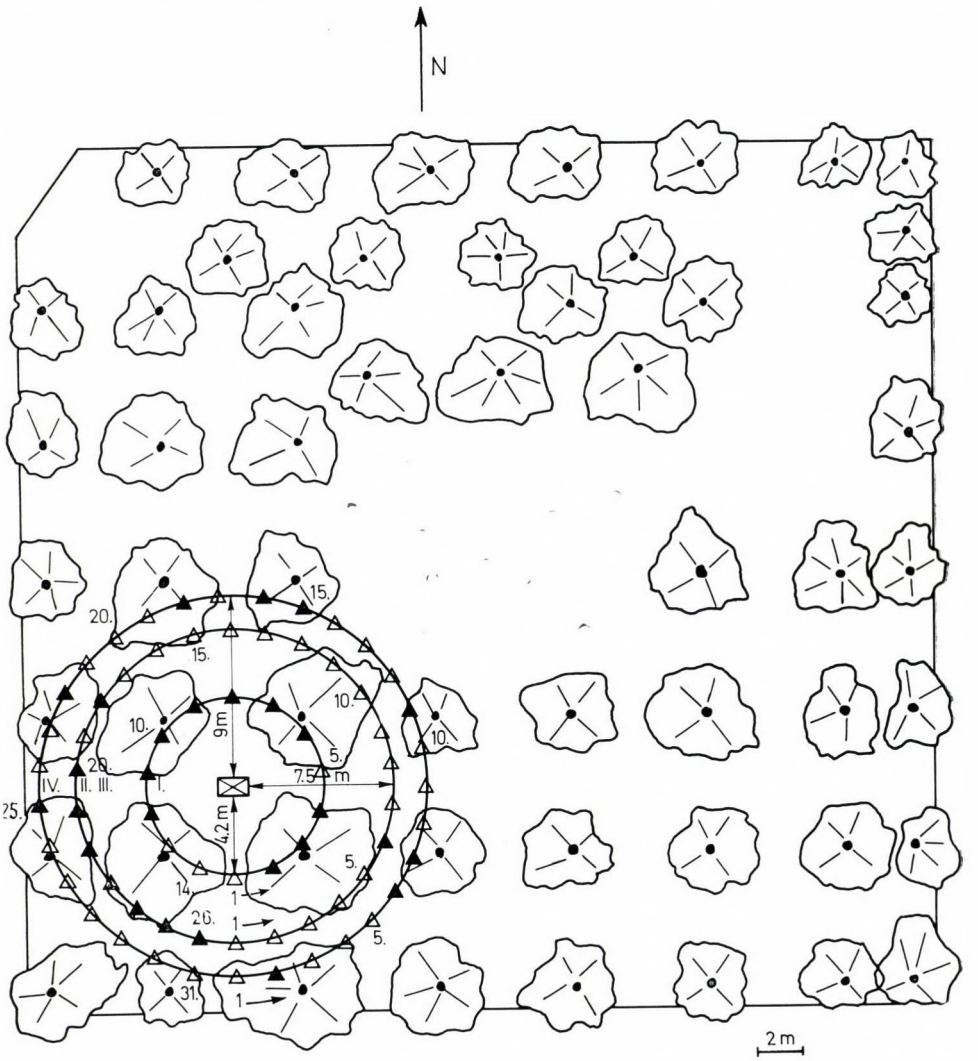


Fig. 1. Sketch map of the plum orchard. Point of release of the males ×. Traps containing virgin females (△) arranged in circles in experiments I—IV; traps capturing males designated as ▲

females in the first 5—8 days of their life. In these experiments, our aim was to determine the flight periods of subsequent generations. Since our results appeared to be favourable with respect to the effects of attraction, we have decided to launch a series of experiments in 1967.

The main purpose of these experiments was to establish the capturable rate of realized males by traps containing virgin females, the traps being placed in various distances from the release point. By this, informations were sought for on the attractiveness of traps baited with virgin females and on the dispersion pattern of the males.

Material and Methods

1. To mark the males to be realized, a honey-water solution of H_3PO_4 containing ^{32}P had been used. The honey-water solution was of about 1 per cent concentration. Various amounts of the solution were transferred by pipette into 14 cm Petri dishes lined above and below with filter paper. Adults moving around and sucking up the fluid get labelled. The Petri dishes were kept closed for 10—15 minutes, then opened on a (1 m high) table in the plum orchard (in the site marked in Fig. 1). A part of the adults took to wings at once, while another part still lingered for a while in the dishes. Some moths left the filter paper only after 20 hours!

The amounts of the fluid and the data of activity per ml used in the experiments are given in Table 1.

Table 1

Amount of fluid labelled with ^{32}P and activity data per ml

No. of experiment	Amount of fluid soaked in filter paper (ml)	Activity per ml in n Ci
I.	8	0.08
II.	3	0.24
III.	4	85.73
IV.	3	8.64

The traps containing the virgin females were placed along circles of different radii as measured from the site of release. Table 2 contains the data referring to the number of traps, the distance measured from the release point, as well as those of the height of traps measured from ground level.

Experiments were carried out during the mating period on four occasions: experiment I on 2 June, experiment II on 8 June, experiment III on 13 July, and experiment IV on 21 July.

2. The behaviour of unmarked males and those marked by ^{32}P was concurrently studied in $20 \times 20 \times 20$ cm wooden-frame insectaries, covered by gauze in the isotope laboratory. These investigations covered the life-span and mating

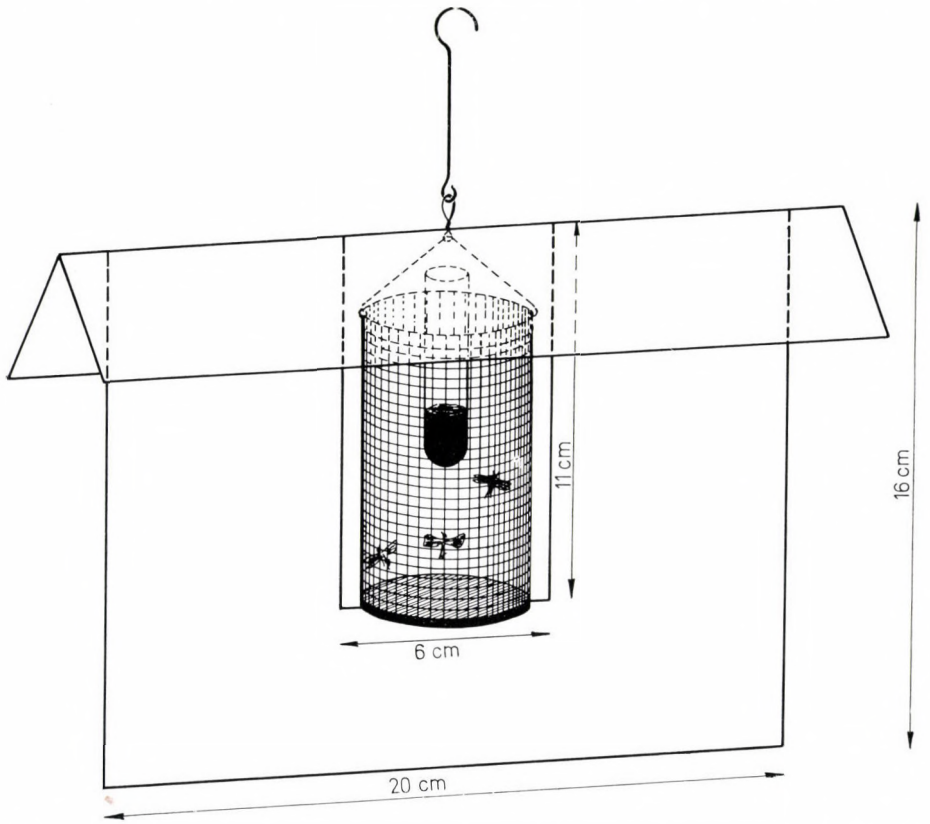


Fig. 2. Jermy's sticking trap. Wire cylinder containing virgin females of *G. funebrana*

ability of the males, and the egg production of the females paired with them. To establish egg production, 5 green plums were daily hung up in each insectary.

3. The traps containing the virgin females (Fig. 2) were made, on Dr. T. JERMY's suggestions, from 0.5 mm tin plates. The freshly hatched virgin females were placed in the cylindrical wire cage affixed to the centre of the trap, — three females to each trap. A glass tube was introduced, its end closed by a cotton plug, from above into the wire cage. Some honey was smeared on the cotton and drops of tap-water introduced daily into the tube; the honey-water served for the feeding of the females. Both sides of the trap was coated with the concentrated substance "Poly-kil"; its active agent being 75 per cent indopol polybutene, used against Metatetranychus ulmi, and manufactured by the Berk Chemicals Co.,

Table 2

Arrangement of traps in the experiments

No. of experiments	Number of traps	Mean distance of traps in m from point of release of males	Smallest	Greatest	Mean	Distance of neighbouring traps in m
			height of traps in m above ground			
I.	14	4.2 ± 0.4	1.20	2.88	2.38	1.8
II.	26	7.5 ± 0.6	1.0	3.12	2.25	1.8
III.	26	7.5 ± 0.6	1.0	3.12	2.25	1.8
IV.	31	9.0 ± 0.3	1.20	2.88	1.82	1.8

Baker St., London W. 1. In field conditions, the substance retained its stickiness for 3—4 weeks. The males approaching the virgin females stuck to the gluey matter; they were collected in every 24 hours and examined in the isotope laboratory. Those few females which died during the experiment were in the meantime supplemented by freshly hatched virgin females (about 5—47 per cent had to be replaced during the various experiments). The location of the traps in the four experiments is shown in Fig. 1.

4. The adults necessary for the experiments, were obtained from laboratory cultures. The basic stock of our cultures was represented by mature larvae hiding for hibernation into sackcloth bands fastened to the trunk of plum trees on the first days of August in the preceding year. The larvae hibernated in the free, then, at the beginning of April, the diapausing larvae were cut, together with their cocoons, from the cloth bands and put singly into glass vials stopped by cotton plugs. Isolated breeding prevented the copulation of the freshly hatched females.

By the time of the flight period of the hibernated brood, the virgin females were available by this method. At the time of the summer generation, laboratory breeding furnished further insects. The detailed description of the egg-transplantation and gluing method has been published the essence of mass laboratory rearing in a previous paper (SÁRINGER, 1967). In the laboratory cultures, the larvae pupated in corrugated paper strips, hence the pupae were in this case cut from the strips and reared isolated.

5. To study the optimal height of the traps, we have placed in our experimental area (Fig. 1) three traps each at heights of 1.2—1.5 m, 2.0—2.5 m and 2.8—3.2 m above the ground, in the crown of a 12-year old greengage tree near the edge of the canopy and with 3 virgin females per trap. (The crown of the tree began 1.2 m above the ground, its top ending at a height of 3.5 m. The shape of the crown was roughly sphaerical, its widest diameter 3.9 m.) The males were taken from the traps and counted daily.

6. To study the flight periods of the males, one trap each, containing 3 freshly hatched virgin females, was hung at a height of 2.0—2.3 m on four Besztercei variety plum trees, each 12 years old, removed at a distance of 6 m from each other and standing in an east-west row. The traps were hung on the trees at 3 p.m.

Every 3—9 hours and for 3 days, we have examined the traps (also during the nights) and the captured males counted.

7. All meteorological data taken into consideration during the experiments were obtained from the Meteorological Station at Keszthely; the instruments of the Station are placed about 1.5 km to the south of the experimental orchard.

Results and Discussion

1. *The behaviour of marked and unmarked males in the laboratory*

No differences could be observed in the behaviour, life-span and mating ability of the unmarked males and those marked with ^{32}P. Their life-span fluctuated in laboratory conditions between 3—18 days. Neither did the egg production of females mated with marked and control males show any essential difference. The egg production of 27 females confined with 33 marked males was 74 as related to one female for a 10-day period of oviposition in the first half of June. And 33 females, brought together with 40 unmarked males, also laid 61 eggs per female during the 10-day period.

Of the 22 observed copulations of the marked males, 3 happened in the forenoon and all others in the late afternoon hours. During the same period, we have observed 14 pairings of the control males, one in the forenoon and thirteen also in the late afternoon hours.

Table 3

Data of labelled males released and captured by traps

No. of experiments	Time of experiment	Number of released labelled males	Number of captured males	Number of captured labelled males	Number of captured labelled males in percentage of released labelled males
I.	VI. $2,10^h$	69	26	24	34.9
II.	VI. $8,10^h$	31	7	5	16.1
III.	VII. $13,11^h$	26	7	5	19.2
IV.	VII. $21,10^h$	55	17	9	16.3

2. *Investigations on the released marked males*

Table 3 contains the experimental results with regard to the marked males released in the field. Tables 4 to 7 show the activity data of the males captured by the traps during the four experiments.

Since the activity of the adults of most moth species is also largely influenced by meteorological conditions, we have shown in Fig. 3 the data of the daily

average temperature, temperature at 7 p.m., daily precipitation, daily average wind velocity as well as wind direction at 7 p.m. It is commonly known that moth adults become active during the twilight and early evening hours, hence also the data measured at 7 p.m. were included in Figure 3.

As shown by the data of Table 3, the traps placed in a circle with a 4.2 m radius, captured twice as much males in experiment I than in all other ones. There may be two probable explanations: either the distance of the traps from the point of release of the marked males or the weather conditions prevailing during the several experiments.

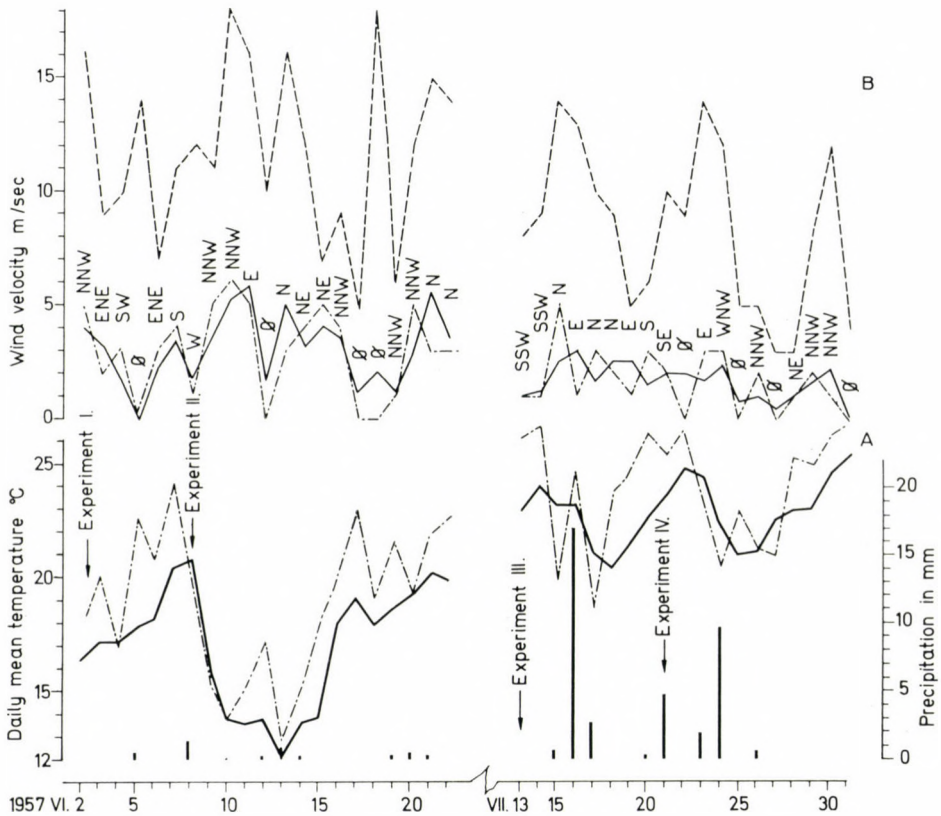


Fig. 3. Meteorological data measured during the experiments. A = Daily mean temperature in C° (——), temperature at 7 p.m. (·–·–·–·), and daily precipitation in mm (vertical line); B = Daily mean of wind velocity in m/sec (——), wind velocity measured at 7 p.m. (·–·–·–·), and maximum daily value of wind velocity (-----). Capital letters denote wind direction established at 7 p.m.

In experiment II, the small numbers, as related to those of experiment I, of the captured marked males and informations on weather conditions imply that the lower activity of the males was probably due to the sudden decrease in temperature. Namely on the second day of experiment II temperature fell to 20.9°C, with a further decrease during the next days. When temperature values had again approached 20°C, 8—10 days had elapsed since the day of release. And at that

Table 4

Captured males and their activity data in experiment I

Date	Serial number of trap	Number of captured males	Number of captured labelled males	Activity value per male (cpm)*
VI. 3	2.	2	2	10; 9
	4.	1	1	8
	6.	1	1	15
	8.	1	—	—
	9.	3	3	23; 14; 13
	11.	3	3	18; 17; 15
	12.	2	2	9; 17
VI. 5	3.	3	3	15; 10; 11
	7.	2	2	6; 10
	8.	4	4	8; 5; 10; 17
	9.	1	—	—
VI. 7	2.	1	1	18
	3.	2	2	9; 24
VI. 3—8		26	24	

* Activity was determined by automatic energy selective scintillation (type "Gamma" NK 108). Measuring method: time preselection; error: 20 per cent.

Table 5

Captured males and their activity data in experiment II

Date	Serial number of trap	Number of captured males	Number of captured labelled males	Activity value per male (cpm)*
VI. 12	6.	2	2	38; 46
	20.	1	—	—
	21.	2	2	34; 42
VI. 17	18.	1	1	15
	20.	1	—	—
VI. 12—17		7	5	

* Error: 10 per cent.

time the attraction of the virgin females had already diminished, according to experiments, to the minimum. Apart from temperature, wind velocities were also unfavourable for the flight of the males during experiment II.

Similarly to experiment II, about half as much males have again been recaptured during experiments III and IV than in experiment I. Temperature and wind velocity data fail to explain here the half amounts of recaptures since, in these respects, conditions were considerably more favourable than during experiment I.

Table 6

Captured males and their activity data in experiment III

Data	Serial number of trap	Number of captured males	Number of captured labelled males	Activity value per male (cpm)*
VII. 17	11.	1	1	2414
	21.	2	2	765; 953
	22.	1	1	311
	24.	1	—	—
	25.	1	1	103
	26.	1	—	—
VII. 13—17		7	5	

* Error: 1 per cent.

Table 7

Captured males and their activity data in experiment IV

Data	Serial number of trap	Number of captured males	Number of captured labelled males	Activity value per male (cpm)*
VII. 24	7.	1	1	25
	11.	1	1	86
	15.	1	1	1245
	16.	1	1	667
	18.	3	3	242; 4142; 828
	22.	1	—	—
	25.	2	2	244; 4264
VII. 28	2.	2	—	—
	6.	1	—	—
	15.	1	—	—
	16.	1	—	—
	22.	2	—	—
VII. 24—28		17	9	

* Error: 1 per cent.

As is to be seen in Figure 3, average temperature was above 20°C during the period of both experiments, and the mean of wind velocity hardly exceeded 4 m/sec, hence this value was also more favourable than during experiment I. Only precipitation was significantly higher than in experiment I, but this, again, fell during the night and forenoon hours and could hardly have had any disturbing effects during the flight period of the males.

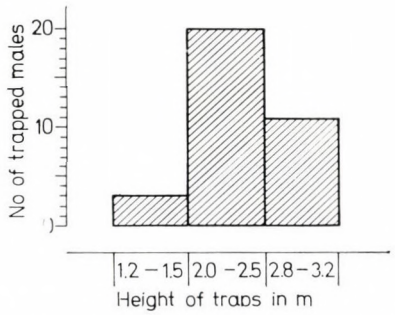


Fig. 4. Height of traps above ground and number of captured males

Figure 3 shows also wind directions measured at 7 p.m., for the sake of studying connections between wind direction and the number of males captured by the traps (cf. the location of the traps in Figure 1). No correlation could be shown between wind direction at 7 p.m. and the number of males captured by the traps during the experiments. This does not mean, however, that wind direction might not influence an easier discovery of the virgin females, since the sex attractants of the female, by which the male starts towards her, spreads surely quicker by wind; it was the alternation of rapid changes and shifts in wind direction what made the demonstration of connection unfeasible in our case.

The results of the four experiments indicate that the number of captured males does not decrease proportionally with the distance in the examined space, and this could be attributed to either the attraction of the females extending to at least the investigated distance or the dispersion of the males beyond it.

3. *Investigations concerning the optimum location as to height of the traps*

Figure 4 shows the data of the experiments made for the purpose of establishing the optimum height of the traps above ground level. According to our data, traps placed at 1.2—1.5 m above the ground captured 3 males, those at 2.0—2.5 m 20 males, while those near the top of the canopy at 2.8—3.2 m, 11 males. Accordingly, we may state that, in a plum tree of a canopy size and shape as described, most males occurred at a height of 2.0—2.5 m, that is, in the upper third of the canopy. The importance of these data lies in the circumstance of our having released the males among plum trees of these very canopy size and age, we have arranged the traps also later at these heights in order to capture as many males as possible. One should bear it in mind that this optimum height of the traps applies to trees of the investigated canopy shape and size, and it cannot be general-

ized as the size of the canopy of younger trees are naturally different. It follows therefore that if a decimation of plum moth populations by traps containing virgin females in a given plum orchard be attained, it should first be ascertained at which canopy level the activity of the males is the highest.

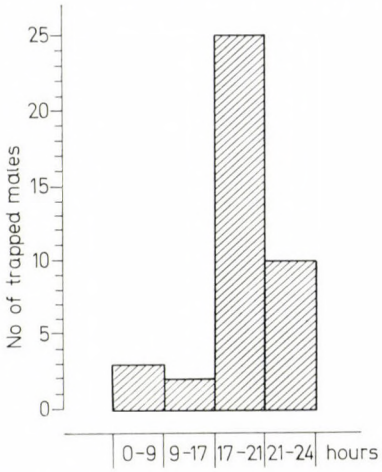


Fig. 5. Daily flight periods of males

4. *Investigations concerning the flight period of the males*

Figure 5 shows the captures in the four traps placed (between 8—16 May, 1967) for the purpose of observing the flight period of the males. It is quite clear from Figure 5 that the traps caught more than 50 per cent of the males between 5—9 p.m. Significantly less were caught in the forenoon and during the night. The inference that the most intensive flight of the males falls between 5—9 p.m. in the first half of May is wholly justified.

5. *Investigations concerning the relationship between the attraction and age of the virgin females*

This relationship was studied in the experiment carried out for the purpose of investigating the flight period of the males. Results are given in Figure 6. As is to be seen, the trap captured 5 males on the day following its exposure, then 2 on the second day, 3 on the third day, none on the fourth, fifth, and sixth days, and 1 each on the seventh and eighth days. The number of males captured by the traps shows that attraction is intense for a few days (1—3) after hatching, then it decreases rapidly but still exists even on the day of death.

6. *Investigations concerning the connection between the number of captured males and temperature measured at 7 p.m.*

Figure 7 shows the results of experiments made with 9 traps for 8 days in the first half of May. It is to be seen that most males were captured at temperatures above 20°C.

Conclusions

The following conclusions may be drawn from the results of the experiments:

1. In the case of *G. funebrana* it is the females which attract the males. By placing freshly hatched virgin females into suitable traps, the male population can be reduced in a given plum orchard.

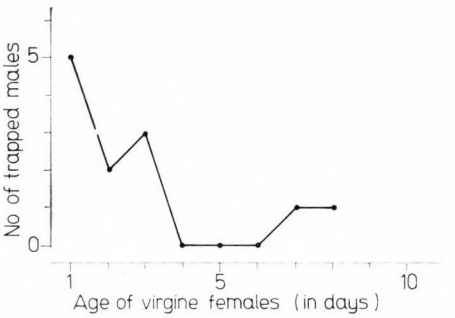


Fig. 6. Relationship between age and attraction of females

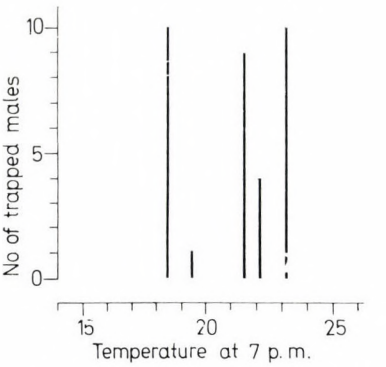


Fig. 7. Relationship between temperature and number of trapped males

2. No differences were observable, in insectaries in the laboratory, with respect to behaviour, life-span and mating ability between unmarked males and those marked with ^{32}P. The inference is justified therefore that behaviour of marked males, released in the field, also agrees with that of the males of natural population.

3. The activity of the males is the highest at a height of 2.0—2.5 m above ground, with respect to a 12-year old, 3.5 m high, Ringló variety plum tree of nearly sphaerical canopy, hence in an orchard showing corresponding data the traps containing virgin females are to be placed at this height.

4. By the marking method using ^{32}P, the ethology of *G. funebrana* can be studied also in the field.

5. The number of males captured by traps containing virgin females does not decrease proportionally with the distance from the point of release; according to our experiments, it is still significant at a distance of 9 m.

6. The most intensive flight period of the male population is between 5—9 p.m. in the first half of May.

7. The attraction of the virgin females on the males is intense during the first three days after hatching, then it rapidly decreases but is still extent even on the day of death (the eighth day).

8. The amount of males captured by the traps is related to temperature; the traps captured more males at temperatures above 20°C than below it.

*

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Book Review

GÁBOR UBRIZSY and JÓZSEF VÖRÖS: *Mezőgazdaságí Mykologia* (Agricultural Mycology). Pp. 1—576. Akadémiai Kiadó, Budapest, 1967. In Hungarian with contents lists in English, German and Russian. 247 figures, 17 × 24 cm, cloth. Forints 110.—

This book presents principles of mycology with special reference to plant pathogenic fungi as well as fungi having importance in soil science and forestry. It is written primarily for research workers of the field of mycology, however, university students or graduate students interested in agricultural mycology or plant pathology can use this book as well.

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The authors have given a very good interpretation of the state of the experimental and other findings in this field. The style is clear and the text is readable. This book is greatly recommended for plant pathologists, university students as well as other research workers of the field of agricultural mycology.

Z. K.

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Effect of Quality and Quantity of Light on Survival, Growth and TMV-content of Tobacco Cells in vitro

By

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Viability was compared of normal with tobacco mosaic virus (TMV)-infected tobacco cells in microcultures after continuous exposure to cool white, incandescent, red or green light for 6, 12, 18, 24 and 48 hours. Observations were made on 400—600 cells immediately after exposure and after a dark period of 48 hours. After continuous cool white light for 48 hours, 36—38% of cells of both types survived; exposures of 6, 12, 18 and 24 hours gave higher rates of survival. Respectively, 97% and 96% of TMV-infected and normal cells survived exposure to incandescent light. Red light progressively reduced the viability of cells exposed. Cells from TMV-infected callus survived slightly more (86%) than healthy cells (81%) after 48 hours. After 8 days in green light, 65% of the TMV-infected cells and 68% of healthy cells survived. Counts taken after a 48 hour dark period following the exposure to each quality of light showed survivals comparable to those taken immediately after exposure. The unexposed control cells after 48 hours in the dark showed 99% viability. Wet weight of TMV-infected tissue grown in cool white light was reduced during the first 4 days, but increased during an additional 4-day exposure. TMV-infected cells exposed to green light showed an initial lag period of two days and a steady increase in wet weight up to six days; thereafter growth rate decreased. Unexposed tissue showed increased growth throughout the 8 day exposure. The TMV-infectivity of cells exposed to cool white light steadily increased throughout the 8 days; while unexposed control cells showed an initial decrease in TMV-infectivity for the first 4 days, and thereafter a continued increase. The virus infectivity of the cells exposed to green light was higher than that of unexposed control cells at each exposure interval.

Introduction

Extensive study has been made of the effects of light on growth and metabolism of whole plants, but the role of light in growth and differentiation of higher plant cells and tissues *in vitro* has received less attention. Excised roots and callus cultures from stem, roots and other plant parts are grown either in the light or in the dark (see GAUTHERET, 1959).

Growth, differentiation and chlorophyll development of callus tissues were influenced by the quality and quantity of light (VENKATESWARAN, 1965; VASIL and HILDEBRANDT, 1966; FUKAMI and HILDEBRANDT, 1967; NAKATA and HILDEBRANDT, 1967; CHEN and VENKATESWARAN, 1968).

Various aspects of callus growth and tobacco ring spot virus activity were also influenced by light (REINERT and KASPERBAUER, 1966).

Prolonged microscopic observations of plant cells in microcultures (JONES *et al.* 1960) continuously exposed the cells to a light source. Whether this type of illumination had any effect on survival and division of healthy living cells or cells infected with tobacco mosaic virus (TMV) was of basic interest in clarifying the interaction of the cell-virus-light system. The quantity and quality of light was also of interest since chloroplasts may play an important part in virus synthesis in plant cells. This paper reports studies of survival of healthy and growth and TMV infectivity of TMV-infected tobacco callus exposed to cool white, incandescent, red or green light.

Materials and Methods

Healthy and TMV-infected tobacco *(Nicotiana tabacum* variety Havana 38) callus tissues were used in these studies. The two stock cultures maintained on solid or liquid C- or D-media (HILDEBRANDT, 1962) in 150 ml prescription bottles with plastic caps were transferred every three weeks to fresh medium and incubated in a dark room maintained at 26° C and 30% relative humidity.

Microcultures were prepared for treatment and observation of the isolated cells. Two week old callus tissue was transferred from solid media to 40 ml of liquid C medium in prescription bottles and was incubated on a reciprocating shaker in the dark. After four to six days of growth in liquid culture, cells were picked with sterile micropipettes under a dissecting microscope from petri plates containing 2 ml of 4 to 6 day old shake cultures and 20 ml of fresh liquid C medium. Tobacco cells from liquid culture were placed in a microculture chamber; four to five microcultures were grouped to obtain about 200 cells. The microcultures in sterile petri plates were exposed to the red, green or cool white light for specific times in a Brunswick Psycrotherm incubator at 28° C. After exposure, the microcultures were examined with a phase-contrast microscope, and then incubated in the dark for 48 hours at 26° C, after which they were again observed.

Cinemoid plastic filters (KLEIN, 1963) were used with cool white fluorescent lamps to obtain the different wave lengths of visible light. The Cinemoid No. 24 green filter transmitted between 450 to 585 mμ with a peak at 510 mμ; the Cinemoid No. 14 ruby filter transmitted 575 to 700 mμ with a peak at 650 mμ. Four 100 W lamps were used for incandescent light.

The growth rate of callus in 40 ml of liquid C medium under different light conditions was studied. After callus was weighed aseptically, one gram (wet weight) of callus tissue was transferred to each bottle. Half the number of bottles were wrapped individually in aluminium foil to prevent exposure to light. Three of each of the covered and uncovered bottles were removed from the shaker after time intervals of 1, 2, 4, 6 and 8 days, and the wet weight of tissue was determined after the liquid was filtered off.

TMV infectivity in the callus cells was determined by grinding the tissue in the liquid medium and inoculating the homogenate on half leaves of 2 month old tobacco *(N. tabacum* × *N. glutinosa)* plants. Exposed and unexposed tissues from three bottles were used for each test after 0 to 192 hours of exposure. The other half of the leaf was inoculated with a 1 : 10,000 dilution of standard extract of TMV from systematically infected tobacco plants.

Results

Response of cells of different ages to exposure to cool white light: Normal callus tissue is a mixture of cells of different ages and different stages of cellular development. To understand the possible differences in reaction of cells to light at different stages of their development, cells from 4 day old liquid cultures consisting mainly of young cells and cells from 8 day liquid cultures with mature and aged cells were exposed to 1500 ft-c cool white light for 6, 12, 18, 24, or 48 hours (Table 1). After 48 hours exposure to continuous cool white light 50% of young cells survived, but only 36% of aged cells survived the same exposure.

Table 1

Per cent survival of healthy, young and old tobacco cells in microculture exposed to cool white light
(1500 ft-c; 28°C)[1]

Exposure time	After exposure	48 hr. after exposure	Unexposed control
Young cells			
6 hours	97.0	96.0	100.0
12 hours	86.5	85.5	100.0
18 hours	80.0	78.5	99.5
24 hours	70.6	68.6	99.0
48 hours	52.2	50.2	98.5
Old cells			
6 hours	99.5	99.5	100.0
12 hours	94.5	94.5	100.0
18 hours	81.7	80.8	99.5
24 hours	49.3	49.3	99.5
48 hours	41.5	35.5	99.5

[1] Average of 400 cells from 2 experiments

Effect of quality of light on survival of healthy and TMV-infected cells: TMV-infected and healthy tobacco cells were exposed to continuous cool white light 1200 ft-c for 6, 12, 18, 24 and 48 hours at 28°C. Three experiments involving a total of 600 cells for each period were used for the study. Continuous light for 6 hours produced no detectable morphological changes in either healthy or TMV-infected tobacco cells. Longer exposure to cool white light reduced survival of both healthy and TMV-infected cells more than did other qualities of light. Only 36 and 38%, respectively, of the two types of cells survived after continuous light for 48 hours (Table 2). Exposures of 12, 18 and 24 hours gave

Table 2

Per cent survival of TMV-infected and healthy tobacco cells in microculture exposed to cool white light (1200 ft-c)[1]

Exposure time	After exposure	48 hr. after exposure	Unexposed control
TMV-infected cells			
6 hours	93.8	92.8	100.0
12 hours	65.0	62.1	99.0
18 hours	55.2	53.1	99.0
24 hours	45.3	45.4	99.0
48 hours	38.4	37.1	99.0
Healthy cells			
6 hours	94.9	93.2	100.0
12 hours	76.8	74.3	99.0
18 hours	67.5	59.2	99.0
24 hours	57.0	56.9	99.0
48 hours	38.0	35.9	99.0

[1] Average of 600 cells from 3 experiments

intermediate ranges of survival. Counts taken after a 48 hour dark period gave results comparable to the one taken immediately after exposure. In both types of cells 99% survived in unexposed control cells grown in the dark. The TMV-infected cells exposed to 12, 18 and 24 hours showed slightly lower rates of survival than the healthy cells.

The effect of light from the light source of the phase contrast microscope on the living tobacco cells in microculture is of interest. Light transmission studies (Table 6) were made with the transformer and diaphragm at various settings. There was a maximum of 120 ft-c with the diaphragm wide open and no filter. The light intensity was reduced to 20 ft-c when passed through a slide microculture.

Table 3

Per cent survival of TMV-infected and healthy tobacco cells in microculture exposed to incandescent light (600 ft-c)[1]

Exposure time	After exposure	48 hr. after exposure	Unexposed control
TMV-infected cells			
6 hours	97.7	97.7	100.0
12 hours	98.0	96.7	100.0
18 hours	98.2	94.2	99.5
24 hours	98.5	98.0	99.3
48 hours	96.7	96.7	99.1
Healthy cells			
6 hours	100.0	99.7	100.0
12 hours	99.7	99.2	100.0
18 hours	99.2	98.5	100.0
24 hours	96.8	96.5	100.0
48 hours	96.0	95.5	100.0

[1] Average of 400 cells from 2 experiments

Incandescent light weakly affected survival of cells (Table 3). After 48 hours exposure to 600 ft-c incandescent light, 97 and 96%, respectively, of TMV-infected and healthy cells survived. The effect of incandescent light on cells exposed for 6, 12, 18 and 24 hours was also slight. Microscopic examinations did not reveal any changes in cytoplasmic, nuclear or organelle detail after the different exposures.

Red light (600 ft-c) progressively reduced the survival of cells exposed for 6, 12, 18, 24 and 48 hours (Table 4). TMV-infected cells showed 86% and healthy cells 81% survival after 48 hours exposure. Unexposed control cells after 48 hours showed 99% survival.

Cell survival was determined in cultures exposed to green light for 1, 2, 4, and 8 days. The prolonged exposure to green light (600 ft-c), like that with cool white light, severely affected survival of both TMV-infected and healthy tobacco cells when compared with unexposed cells' survival (Table 5). Sixty-five and sixty-eight per cent, respectively, of TMV-infected and healthy cells survived after 8 days in green light. The dark grown controls of both types showed 99% survival.

Table 4

Per cent survival of TMV-infected and healthy tobacco cells in microculture exposed to red light
(600 ft-c; 28°C)[1]

Exposure time	After exposure	48 hr. after exposure	Unexposed control
TMV-infected cells			
6 hours	97.8	97.8	100.0
12 hours	96.5	95.2	100.0
18 hours	95.1	93.8	99.5
24 hours	88.4	87.4	99.2
48 hours	85.8	85.0	99.2
Healthy cells			
6 hours	98.5	98.3	100.0
12 hours	97.3	95.8	100.0
18 hours	93.3	92.5	99.8
24 hours	91.1	90.0	99.7
48 hours	80.7	79.2	99.7

[1] Average of 600 cells from 3 experiments

Table 5

Per cent survival of TMV-infected and healthy tobacco cells in microculture exposed to green light
(600 ft-c; 28° C)[1]

Exposure time (Days)	After exposure	48 hr. after exposure	Unexposed control
TMV-infected cells			
1	92.4	90.4	100.0
2	84.1	81.8	99.0
4	78.9	77.0	99.0
8	65.2	53.0	98.7
Healthy cells			
1	92.8	86.1	100.0
2	81.4	79.0	100.0
4	81.4	76.8	99.5
8	67.6	67.1	99.0

[1] Average of 600 cells from 3 experiments

Table 6

Light transmission through the Zeiss phase-contrast microscope.[1]
Lamp: Carl Zeiss: 380177: 6 v. 15 W. Phase: I

Trans-former volts	Without slide		With slide	
	No filter ft-c	Green filter ft-c	No filter ft-c	Green filter ft-c
6	10	5	9	5
6	14	5	12	5
8	22	5	20	5
	Diaphragm: 20 (Maximum)			
5	45	10	45	10
6	70	13	70	13
8	120	20	120	20
	Through Microculture-Diaphragm: 6 (Optimum)			
5			10	5
6			13	5
8			20	5

[1]Light readings in ft-c using G. E. type 213 light meter

Effect of quality of light on TMV synthesis: Prolonged illumination with cool white and green light was studied with respect to tissue growth and TMV synthesis in TMV-infected callus tissue in liquid shake cultures exposed for 1, 2, 4, 6, and 8 days. After a lag period of two days the tissues exposed to cool white light showed an increase in wet weight up to eight days (Fig. 1). The un-unexposed tissue showed a more gradual increase in growth throughout the 8 days exposure. The virus contents of the unexposed cells were lower than that in exposed cells at each exposure interval except at 6 days. The unexposed cells showed a gradual increase in growth, but the exposed cells had a marked increase after 4 days exposure period. There was a corresponding increase in virus activity of both tissues. However, virus infectivity was greater in the exposed cells corresponding to the increased weight.

In cultures exposed to green light (Fig. 2) cell growth increased up to 6 days, but was slightly less for 2 days. The virus infectivity was again greater in cultures following the exposure for 6 and 8 days. Unexposed tissue showed the greatest wet weight after 6 days of the growth period, but a slight decrease the last 2 days. The virus activity of the unexposed cells decreased during the first four days exposure to white light and then progressively increased through 8 days.

Discussion

The difference in the rates of growth and virus contents of exposed and unexposed cells suggests an interrelationship of cellular metabolism and virus multiplication. Quality of light and duration of exposure influenced survival of TMV-infected and healthy tobacco cells grown *in vitro*. Prolonged (2–8 days) exposure was important. Cells of both types showed tolerance to exposure to incandescent and red light, while continuous exposure to cool white and green light had adverse effects on survival. It was of interest that when exposed and unexposed cells increased in growth there were corresponding increases in the virus infectivities. The sharp increase in growth of exposed cells (Fig. 1) after 4 days was followed by a sharp increase in virus infectivity.

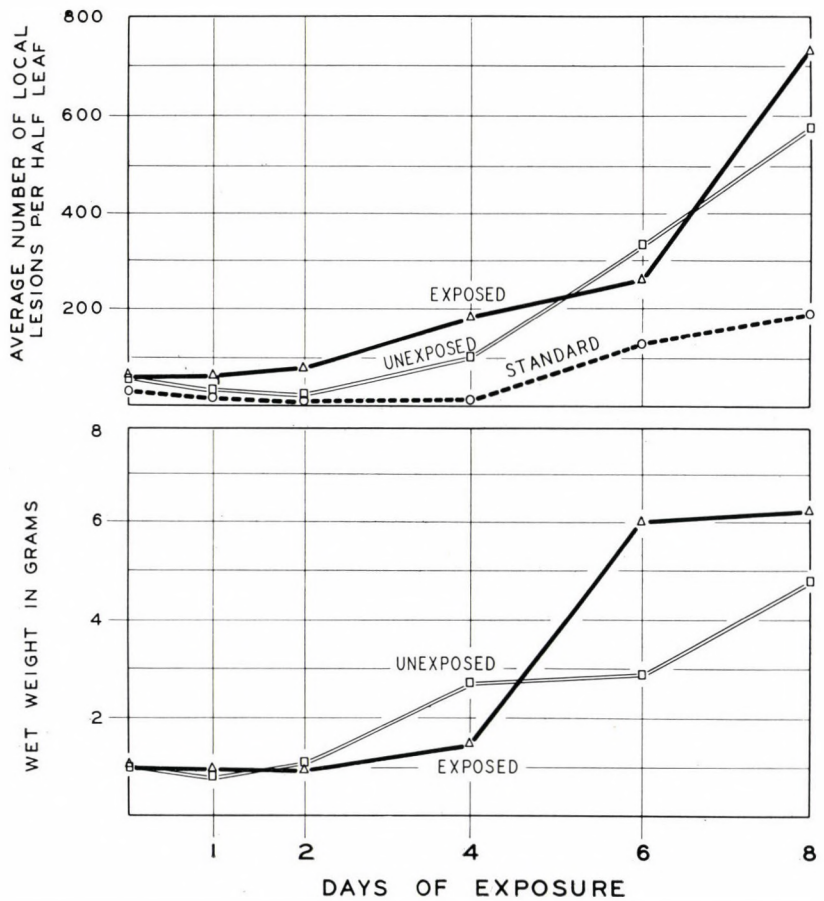


Fig. 1. Effect of cool white light on growth of tobacco cells and TMV-infectivity

Measurement of the maximum transmitted incandescent light falling on the microcultures from the microscope illumination incandescent lamp was much less (120 ft-c) than the 600 ft-c incandescent illumination used to study the reaction of the cells. These data, results on survival and observations of the exposed and unexposed cells, suggested that the incandescent light intensities normally used for microscopic observations were not harmful to the cells.

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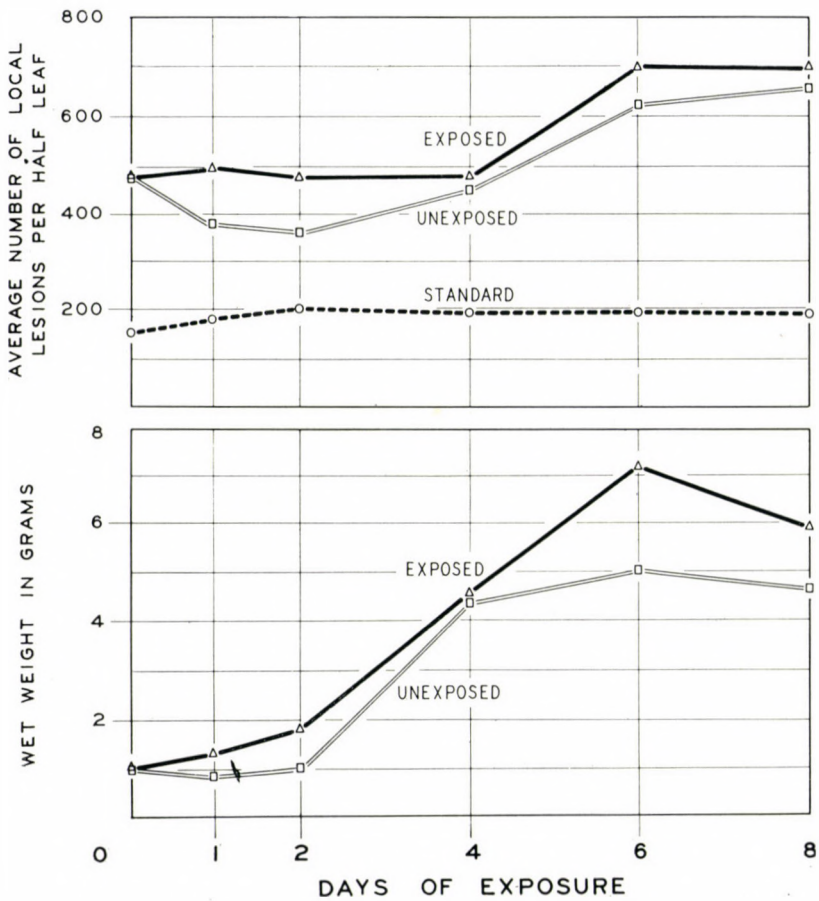


Fig. 2. Effect of green light on growth of tobacco cells and TMV-infectivity

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Correlation between the Cytokinin-like Activity and the Inhibitory Action on TMV Infection of Some Pyrimidine Antimetabolites

By

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Out of 31 potential pyrimidine antimetabolites 2-thiouracil, 6-azauracil, 6-azacytosine, 6-azathymine, 6-methyluracil, β-ureidocrotonic acid ethylester and ethylsemicarbazidoacetate hydrochloride proved to be active inhibitors of tobacco mosaic virus infection. 6-Methyluracil, β-ureidocrotonic acid ethylester (as a possible precursor of methyluracil) and ethyl-semicarbazidoacetate hydrochloride (as a possible precursor of azauracil) revealed characteristic cytokinin activity. It is suggested that the cytokinin activity of these three antimetabolites is in relation with their suppressive action on virus infection.

Introduction

Several analogues of the natural pyrimidine nucleobases are known to be effective against animal and plant viruses, their activities probably being based on their action as nucleic acid antimetabolites. 2-Thiouracil and 6-azauracil are mentioned as the most active members of this group. On the other hand, it was demonstrated that cytokinins, which may be regarded as purine analogues, exert an inhibitory action on tobacco mosaic virus (TMV) infection indirectly by stimulation nucleic acid and protein synthesis of the host plant (KIRÁLY, EL HAMMADY and POZSÁR, 1968). Recently, it has also been shown (POZSÁR and MATOLCSY, 1968) that some pyrimidine base analogues are able to stimulate the incorporation of uracil and adenine into the insoluble fraction of bean leaves.

In the present study several nucleobase analogues and related derivatives have been tested for their action on TMV infection in order to draw some conclusions concerning structure-activity relationship and mechanism of action. Some of the compounds tested can be regarded as thio- or aza-analogues of the intermediate products of uracil biosynthesis and as a consequence as potential precursors of 2-thiouracil and 6-azauracil, respectively. Our investigations were extended to some nucleobase-analogues which were found by ŠKODA et al. (1962) as the most effective ones among 125 tested compounds against *Escherichia coli*.

Materials and Methods

The compounds tested were as follows:

Potential precursors of 2-thiouracil:

β-Thioureidopropionic acid, 5,6-dihydro-2-thiouracil, 2-thioorotic acid, thio-Δ-hydantoin acetic acid.

Analogues and partly potential precursors of 6-azauracil:

Semicarbazido-acetic acid, ethyl-semicarbazidoacetate-hydrochloride, glyoxylic acid semicarbazone, α-ketoglutaric acid semicarbazone, dihydro-6-azauracil, 2-thio-6-azauracil, 4-thio-6-azauracil, 2,4-dithio-6-azauracil, 2-methylthio--6-azauracil, 5-bromo-6-azauracil, 6-azauracil-5-carboxylic acid, β-(6-azauracil--5-yl)-propionic acid, 2-methylthio-3-methyl-4-thio-6-azauracil, 6-azathymine, 2-thio-6-azathymine, 6-azacytosine, urazole(3,5-dioxyl-1,2,4-triazole).

5-Aza-derivatives:

Formyl-biuret, 5-azauracil, 5-azacytosine, 5-azauracil-6-carboxylic acid.

Analogues of uracil:

β-Ureidocrotonic acid ethylester, dihydro-uracil, 6-methyluracil, 4-thiouracil. 2-Thiouracil, 6-azauracil and kinetin were used as control substances.

Most of these compounds were synthesized by us by known methods; others were kindly submitted by Dr. J. GUT and Dr. A. PÍSKALA from the Institute of Organic Chemistry and Biochemistry of the Czechoslovak Academy of Sciences, Prague and by Dr. V. ŠKARIC from the Institute Ruder Boškovic, Zagreb.

Half leaves of tobacco (*Nicotiana tabacum* var. Xanthi) and french bean (*Phaseolus vulgaris* var. Pinto) were brushed daily with the compounds to be tested and with water, respectively, for 6 successive days. After the treatments the whole leaves were inoculated with tobacco mosaic virus (TMV) strain U-1. Local lesions appeared 2–3 days after inoculation and were counted.

Assays for cytokinin activity were made by the chlorophyll retention test (OSBORNE and MCCALLA, 1961): 2 cm sections of first leaves of barely were floated on the test solutions and held in darkness for 4–5 days.The leaf tissues were then extracted with a 80 per cent ethanol. Green color was evaluated by measuring the optical density of the extracts at 665 mμ using a photometer.

The methods of measuring the rate of incorporation of precursors of protein and nucleic acid have been described in an earlier paper (KIRÁLY, EL HAMMADY and POZSÁR, 1968). This procedure was followed also in the present investigation.

Results and Discussion

Tables 1 and 2 as well as Fig. 1 show the effect of different compounds on the appearance of local lesions induced by TMV in detached and attached leaves of Xanthi tobacco. In addition to their suppressive effect on local lesion number (in other words, their effect on the susceptibility of the host), 6-methyluracil, β-ureidocrotonic acid ethylester and ethyl-semicarbazidoacetate hydrochloride revealed characteristic cytokinin-like activity. The cytokinin effect was evaluated by the chlorophyll retention test and by measuring the incorporation of precursors of protein and nucleic acids into the acid insoluble fraction of leaf tissues pretreated with these compounds (Tables 3 and 4). These tests have

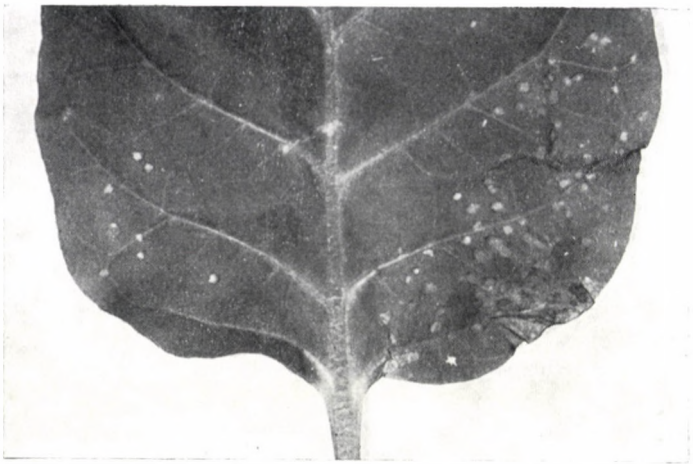


Fig. 1. Action of 6-methyluracil on TMV infection. The left half of the tobacco leaf was treated with a 100 ppm solution of 6-methyluracil, the right half with water prior to inoculation



Fig. 2. Senescence retarding and growth-promoting action of 6-methyluracil. The left half of the Pinto bean leaf was treated with a 100 ppm solution of 6-methyluracil, the right half with water

shown that the three compounds mentioned above act as senescence inhibitors (Fig. 2). Half leaves of beans treated with these substances remained green for a longer period than the control half leaves. As a further symptom of the cytokinin action, a considerable enlargement of the treated half leaves could be observed (Fig. 2.). These results indicate that there is a correlation between virus inhibition and cytokinin activity of 6-methyluracil, β-ureidocrotonic acid and ethyl-semicarbazidoacetate hydrochloride, as is the case with kinetin and other cytokinins (KIRÁLY, EL HAMMADY and POZSÁR, 1968). This provides further evidence of the common biochemical mechanism of different types of cytokinins which increases virus resistance of the host by increasing its juvenility.

As both uracil and thymine (5-methyluracil) are inactive in the virus inhibition, the cytokinin and virus inhibitory activity of 6-methyluracil must be regarded as a function of the methyl-substitution on carbon atom 6. At present no reasonable theory can be brought forward concerning the background of this finding nevertheless some characteristic features of the molecular structure

Table 1

The action of some purine and pyrimidine derivatives on infection induced by TMV in floating leaf discs of Xanthi tobacco

Treatment	Local lesion number/100 discs	Per cent inhibition
Control (H_2O)	4400	—
Kinetin (50 ppm)	1500	65
2-Thiouracil (10 ppm)	910	80
6-Methyluracil (100 ppm)	2409	46
β-Ureidocrotonic acid ethylester (100 ppm)	3340	25
6-Azauracil (100 ppm)	2820	36
Ethyl-semicarbazidoacetate hydrochloride (100 ppm)	3420	23
6-Azacytosine (100 ppm)	2730	39
6-Azathymine (100 ppm)	2610	41

might be considered. As compared to uracil the introduction of the 6-methyl-group displays a steric effect as well as a + I and a Nathan-Baker-effect, this last resulting in a delocalized molecular orbital comprising "quasi π-electrons" of the methyl-group and π-electrons of the uracil-molecule. As compared to thymine the 6-methyl-substitution results, in addition to the alteration of the

Table 2

The action of some purine and pyrimidine derivatives on infection induced by TMV in attached half leaves of Xanthi tobacco

Treatment	No. of local lesions on half leaves		Per cent inhibition
	Control	Treated	
Kinetin (100 ppm)	96	27	72
2-Thiouracil (10 ppm)	112	20	82
6-Methyluracil (100 ppm)	110	66	40
β-Ureidocrotonic acid ethylester (100 ppm)	149	66	56
6-Azauracil (100 ppm)	83	40	52
Ethyl-semicarbazidoacetate hydrochloride (100 ppm)	96	42	55
6-Azacytosine (100 ppm)	125	57	54

Table 3

The activity of some purine and pyrimidine derivatives in the chlorophyll retention test (Chlorophyll concentration was determined by measuring the optical densities of extracts of barley leaves floated on the test solutions. Optical density was expressed as extinction at 665 mμ)

Treatment	Extinction at 665 mμ	Per cent
Control (H_2O)	0.13	100
Kinetin (50 ppm)	0.41	315
6-Methyluracil (100 ppm)	0.22	169
β-Ureidocrotonic acid ethylester (100 ppm)	0.29	223
Ethyl-semicarbazidoacetate hydrochloride (100 ppm)	0.24	184

molecular shape, in a shift of the methyl-group from carbon atom 5 having a relatively high π-electron-density to carbon atom 6 with a lower density value, as calculated by SCF LCAO MO method (Ladik and Appel, 1962).

The similar effect of β-ureidocrotonic acid ethylester can be attributed either to the *per se* activity of the unchanged molecule or to its enzymic cyclisation into the active 6-methyluracil, according to equation

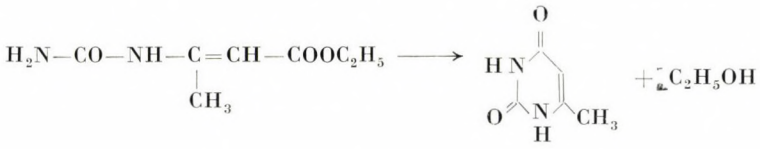


Table 4

The effect of benzyladenine and some pyrimidine antimetabolites and precursors on the incorporation of S^{35}-cysteine and C^{14}-adenine into the acid insoluble fraction of Xanthi tobacco (Half leaves of Xanthi tobacco were treated once a day for 6 days with water (as control) and with the compound to be tested, then leaf discs from both half leaves were floated on the labelled cysteine and adenine, respectively)

Treatment	S^{35}-Cysteine		C^{14}-Adenine	
	Count per minute/100 mg	Per cent	Count per minute/100 mg	Per cent
Control (H_2O)	2432	100	283	100
6-Methyluracil (100 ppm)	5702	234	349	123
Control (H_2O)	2673	100	238	100
β-Ureidocrotonic acid ethylester (100 ppm)	3578	134	287	121
Control (H_2O)	2348	100	265	100
Ethyl-semicarbazidoacetate hydrochloride (100 ppm)	4183	178	362	137
Control (H_2O)	—	—	262	100
Benzyladenine (30 ppm)	—	—	454	173

No similar conclusion can be drawn for semicarbazido-acetic acid ethylester showing a marked cytokinin and antiviral activity. This compound could be regarded as a potential precursor of dihydro-6-azauracil. However, this latter compound proved to be inactive thus making an enzymic cyclization improbable.

Further studies are in progress on uracil-derivatives bearing substituents with different steric and/or electronic effects in order to elucidate structural requirements and mechanism of action.

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Evaluation of 6-Methyluracil and β-Uraminocrotonic Acid Ethylester against Tobacco Mosaic Virus under Field Conditions

By

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Treatments with 6-methyluracil and β-uraminocrotonic acid ethylester suppressed the symptoms and increased the yield of TMV infected tomato plants.

The concept of host-parasite relation generally used in plant pathology demands new interpretation in connection with virus diseases. The biosynthesis of the virus-particles responsible for infectious genetic information is based on the same pathways as the metabolism of the normal proteins and nucleic acids in the cells of the host plant. The virus-infected cell forms a new biological entity also in the genetical sense and an interference necessary for killing the parasite results also in the destruction of the host organism.

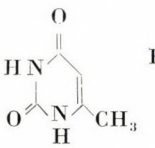
Due to this fact most of the promising control methods used against virus diseases are of preventive character, such as agrotechnical methods, use of inhibitors of infection, chemical control of vectors responsible for the spread of the viruses, resistance-breeding, etc. All therapeutical methods (heat and chemotherapy) are of theoretical rather than of practical interest. No unambiguous results have been attained either by the antibiotic approach.

New possibilities in chemotherapy emerged from the use of substituted purine and pyrimidine bases (nucleobase analogues). 2-Thiouracil revealed a fair virus-inhibitory activity (COMMONER and MERCER, 1951; 1952; MATTHEWS, 1956) sufficient for practical purposes, but its phytotoxic action precluded the possibility of practical application (MILINKÓ, 1961).

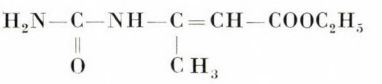
Correlation between virus-susceptibility and the intensity of protein metabolism has been reported recently (KIRÁLY, EL HAMMADY and POZSÁR, 1968). Based on this finding the increase of the resistance by chemical means seems to be the most promising method for the control of virus diseases. Substances with a kinetin-effect, such as kinetin, benzyladenine and benzimidazole, revealed a marked virus-inhibiting effect (SCHLEGEL and RAWLINS, 1954; KIRÁLY and SZIRMAI, 1964; POZSÁR and KIRÁLY, 1965; 1966; ALDWINCKLE and SELMAN, 1967; POZSÁR, EL HAMMADY and KIRÁLY, 1967), their practical use is limited however by their high cost.

Based on the promising laboratory and glasshouse results obtained with 6-methyluracil (I) and with its potential precursor β-uraminocrotonic acid ethylester (II) (MATOLCSY, EL HAMMADY and KIRÁLY, 1968), the evaluation of these compounds under field conditions seemed to be justified. Due to the simple

method of preparation and the low cost of the starting materials an eventual practical application can be expected to be economical.



I



II

Materials and Methods

Both test compounds were prepared according to the method of DONLEAVY and KISE (1963). Tomato plant of the variety "Kecskeméti merevszárú" were grown in glasshouse and treated through seven days daily with a 200 ppm. aqueous solution of the substances. Half of the seedlings were treated before, half after the infection carried out with tobacco mosaic virus (TMV) U_1 strain, maintained on Samsun tobacco.

9 days after inoculation the seedlings were transplanted to plots. Each of the plots included 40 plants and a four-fold replication was used. Virus-infected untreated as well as uninfected untreated plants served as control.

Results

Due to unfavourable weather conditions in 1968 the development of the plants was poor, many stocks died and the yield was low. The high virus infection resulted a marked underdevelopment of the diseased plants and heavy mosaic- and leaf-deformation symptoms appeared both on the treated as well as on the untreated plants.

As seen on the table, treatment with 6-methyluracil and to a lesser degree with β-uraminocrotonic acid ethylester effected a marked diminishing of the symptoms and an increase of the yield, but the results were behind those of the untreated control.

Treatment	Yield kg/100 stocks
6-Methyluracil	
treatment before infection	25.0
treatment after infection	24.5
β-Uraminocrotonic acid ethylester	
treatment before infection	15.0
treatment after infection	9.5
Untreated infected control	9.6
Untreated uninfected control	90.0

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Virusbefall an Magnolien (*Magnolia* spp.)

Von

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An Magnolien in der DDR und ČSSR, von denen zumindestens einige zu den Arten *Magnolia denudata* und *M. soulangiana* gehörten, konnten offensichtliche Virussymptome in Form von Linien-, Bogen-, Ring- und Eichenblattmustern beobachtet werden. Aus einer derartig befallenen Pflanze wurde wiederholt das Gurkenmosaik-Virus isoliert. Die mitgeteilten Befunde sind die ersten Feststellungen über Virusbefall an Magnoliaceen.

Die Magnolien gehören zu den attraktivsten Blühgehölzen in Parks und Gärten Mitteleuropas. Ihre Seltenheit und ihr Wert machen sie zu vielbeachteten Solitärpflanzen. Umso bemerkenswerter ist die Tatsache, daß die Magnoliaceen bisher zu der ständig kleiner werdenden Gruppe dikotyler Pflanzenfamilien gehörten, an deren Vertretern kein Virusbefall bekannt war. Erstmalig im September 1967 und auch im Frühjahr 1968 stellten wir jedoch Symptome fest, die unzweifelhaft viröser Natur sind.

Sie bestehen in chlorotischen bis weißlichen Linien-, Bogen-, Ring- bzw. Eichenblattmustern an den Blättern (Abb. 1). Die Gewebebezirke innerhalb der Zeichnungen können hellere Grüntöne als die übrige Blattfläche zeigen. Andere Krankheitsmerkmale, wie Deformationen, waren an den befallenen Magnolien nicht zu beobachten. Stets war nur ein Teil des Laubes symptomtragend. Erscheinungen dieser Art wurden bisher innerhalb der DDR im Park von Wörlitz bei Dessau und im Arboretum des Instituts für Spezielle Botanik der Humboldt-Universität Berlin-Baumschulenweg festgestellt. Innerhalb der ČSSR wurden kranke Magnolien im Waldsteingarten (Kleinseite von Prag) und auf dem Gelände der Internationalen Gartenbauausstellung in Olomouc (Olmütz) gefunden.

In Wörlitz und Prag konnte nicht genau ermittelt werden, welche Magnolienart jeweils vorlag. In Berlin-Baumschulenweg handelte es sich um *Magnolia soulangiana* Soul.-Bod. (hervorgegangen aus einer Kreuzung von *M. denudata* Desr. × *M. liliflora* Desr.), in Olomouc um *M. denudata* Desr. Beide sind sich sehr ähnlich und auch die Pflanzen der anderen Standorte gehören vermutlich in ihren Formenkreis. Wir sind davon überzeugt, daß die Erkrankung, für die wir den Namen »Linienmuster der Magnolie« (*Magnolia* line pattern) vorschlagen, auch an zahlreichen anderen Standorten des In- und Auslandes vorhanden ist. Dafür spricht unter anderem die Tatsache, daß die Magnolien vorwiegend vegetativ vermehrt werden.

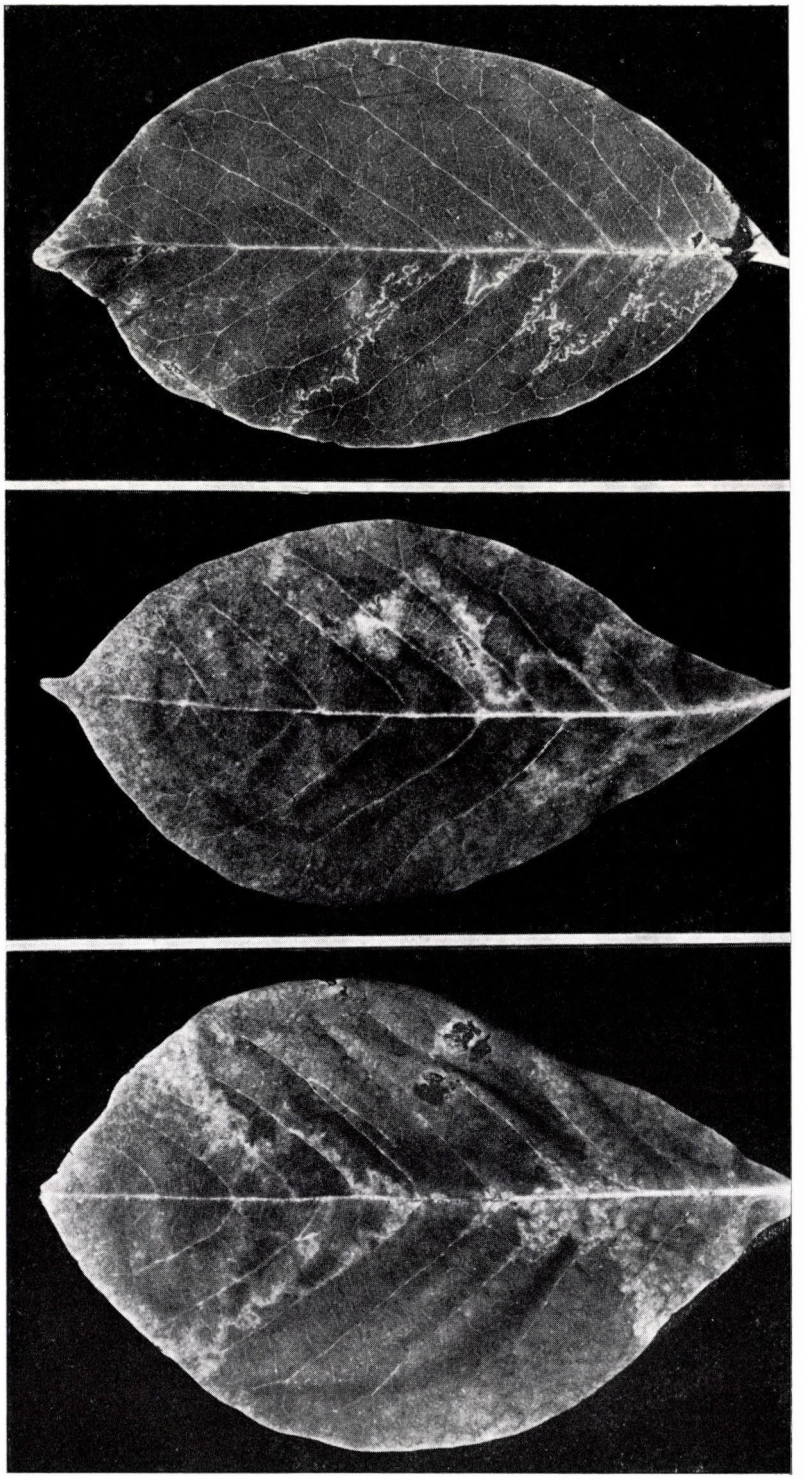


Abb. 1. Das Linienmuster der Magnolie (*Magnolia* line pattern)

Im Herbst 1967 angestellte Versuche zur mechanischen Übertragung eines Virus aus kranken Magnolienblättern auf krautige Testpflanzen verliefen negativ. Im Mai 1968 wurden dagegen in zwei von drei Versuchen mit Impfbrei aus symptomtragendem Blattmaterial einer Magnolie aus Wörlitz, der unter Zusatz von neutralem Phosphatpuffer und Aktivkohle hergestellt worden war, Erfolge erzielt. Es konnten *Nicotiana megalosiphon* Heurck et Muell., *N. glutinosa* L., *N. tabacum* L., Sorte ‚Samsun', *Chenopodium quinoa* Willd. und *C. murale* L. direkt infiziert werden. Von krautigen Pflanzen aus wurde das Virus zusätzlich auf die folgenden Pflanzenarten mechanisch übertragen: *Chenopodium amaranticolor* Coste et Reyn., *Cucumis sativus* L. und *Petunia hybrida* Vilm.

Alle Reaktionen der genannten krautigen Pflanzenarten deuteten darauf hin, daß ein Normalstamm des Gurkenmosaik-Virus (GMV) vorlag. Das Virus war auch durch *Myzus persicae* Sulz. und durch *Cuscuta californica* Choisy übertragbar. Prämunitätsversuche auf *Nicotiana megalosiphon*, in denen die Virusisolierung von der Magnolie Normalstämme des GMV sowohl abwehrte als auch von ihnen abgewehrt wurde, bewiesen die Richtigkeit der Zuordnung. Die Testpflanzenreaktionen, die Prämunitätsversuche sowie serologische Prüfungen auf verschiedene Stämme des Tomatenschwarzring-Virus, auf das *Arabis*-Mosaik-, latente Erdbeerringflecken-, Kirschenblattroll- und das Luzernemosaik-Virus ergaben keine Anhaltspunkte dafür, daß neben dem GMV noch ein anderes Virus auf krautige Pflanzen übertragen worden war. Zukünftige Untersuchungen müssen jedoch erst erweisen, ob das GMV das Linienmuster der Magnolie hervorzurufen vermag. Das ist umso notwendiger, als Versuche zur mechanischen Übertragung eines Virus auf krautige Pflanzen nicht nur bei symptomlosen sondern auch bei zwei anderen kranken Magnolien erfolglos blieben.

Summary

On magnolias in GDR and ČSSR, at least some of them belonging to *Magnolia denudata* and *M. soulangiana*, obviously virus-induced symptoms were visible, which consisted out of lines, arcs, rings and oak leaf patterns. From one of such plants cucumber mosaic virus was repeatedly isolated. This is the first communication about the fact that Magnoliaceae can be virus infected.

Triggering Mechanism in Rice Blast Disease

By

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Respiration, peroxidase and protein synthesis were activated within 15 min of inoculating rice (*Oryza sativa* L.) leaves with *Pyricularia oryzae* Cav. The fungal toxin, α-picolinic acid and spore germination fluid activated the respiration, however, to a lesser degree. The toxin inhibited peroxidase activity but had little effect on ascorbic acid oxidase. The spore germination fluid stimulated the peroxidase but markedly inhibited ascorbic acid oxidase activity of tissues. Both α-picolinic acid and spore germination fluid had no effect on protein synthesis. The symptoms are of host origin in response to the nature of inoculum; the maximum degree of response occurred in the presence of *P. oryzae* and the host cells react the moment they are in contact with the pathogen.

Introduction

ALLEN (1966) suggested that the visible host reactions to an invading organism are not caused by the pathogen produced metabolites but the result of a sequence of reactions in the host. SRIDHAR et al. (1968) showed that both the pathogen (*Fusarium oxysporum f. vasinfectum* (Atk.) Snyder and Hansen) and its metabolites are necessary to trigger off the physiological changes in cotton (*Gossypium herbaceum* L.) and such changes are influenced by the nature of the inoculum; the host response is maximum in the presence of the pathogen while the metabolites caused limited responses. In this paper, alterations in respiration of rice tissues (*Oryza sativa* L.) by inoculating them in Warburg vessels with a spore suspension of *Pyricularia oryzae* Cav., α-picolinic acid, a toxic metabolite of the fungus (TAMARI et al., 1963) and spore germination fluid are reported. The nature of enzyme changes, particularly oxidases and the amount of protein synthesized have also been investigated to understand the triggering mechanism in host-parasite interaction.

Materials and Methods

The variety Co 13, highly susceptible to blast was grown in pots for 15 days under field conditions. Leaves were used in all the experiments.

Respiration: Leaves were cut into 1–2 mm bits and suspended in 2 ml of 0.1 M sodium phosphate buffer at pH 6.5 in a 15 ml Warburg manometer

flask with 0.2 ml of 10% KOH solution and a folded filter paper in the central well. Four separate inocula were used to inoculate the leaves from the side arm of the manometer: (i) 0.5 ml of distilled water, (control), (ii) 0.5 ml of spore suspension (ca. 4,000 spores/ml) of *P. oryzae* prepared from 5 day old cultures grown on barley grain medium (GOTO et al. 1964) in the phosphate buffer, (iii) 0.5 ml of 2.5×10^{-4} M of α-picolinic acid (Fluka, Chemische Fabrik, Switzerland) dissolved in the phosphate and (iv) 0.5 ml of cell free spore germination fluid, which was prepared by incubating 25 ml of the spore suspension for 24 hrs at room temperature (28–30° C) and filtering through Seitz filter.

The manometers were equilibrated for 10 min with shaking at 30° C, the contents from the side arms were tilted into the main chamber and gas uptake was measured at 15 min interval up to one hr (UMBREIT et al., 1957). Duplicates were maintained in all the tests and the oxygen consumption was expressed on oven dry tissues. The gas uptake of flasks containing tissues + spore suspension was corrected by subtracting the gas uptake of spores.

Preparation of enzyme extracts: One g of leaf tissue cut into 1–2 mm bits was placed in sterile Petri dishes with 10 ml of the buffer and 2.5 ml of the inoculum. At 15 min interval, the tissues were removed, thoroughly washed in distilled water and crushed in 5 ml of chilled phosphate buffer, pH 6.5 in a chilled porcelain mortar with pestle. The extract was filtered, made up to 5 ml with the buffer, centrifuged at 2,100 × g. for 30 min and the supernatant was used as the enzyme source (SRIDHAR et al., 1968). Enzyme assays were performed in a Beckman DU-spectrophotometer. Cuvettes containing reaction mixture minus the enzyme source served as control. All the assays were conducted at 28–30° C and duplicate determinations were made.

Enzyme assays: (i) Polyphenol oxidase: To 0.5 ml of 0.2 M sodium phosphate buffer at pH 7.0, 0.5 ml of 0.1 M catechol, 1.5 ml of distilled water and 0.5 ml of the enzyme extract were added and enzyme activity was expressed as changes in absorbance at 30 sec interval up to 3 min at 495 mμ (MATTA and DIMOND, 1963).

(ii) Peroxidase: The reaction mixture contained 0.1 ml of enzyme source, 1.0 ml of 0.001 M pyrogallol in 0.05 M sodium phosphate buffer at pH 6.5, 1.8 ml of distilled water and 0.1 ml of 2 per cent hydrogen peroxide. Changes in absorbance at 470 mμ were measured at 30 sec interval up to 3 min (HAMPTON, 1963).

(iii) Ascorbic acid oxidase: To 0.1 ml of plant extract, 1.0 ml of 0.2 M sodium phosphate buffer at pH 6.2, 1.8 ml of distilled water and 0.1 ml of 0.001 M ascorbic acid were added and the absorbance changes at 265 mμ were followed up to 3 min at 30 sec interval (OBERBACHER and VINES, 1963).

Enzyme activities of healthy and inoculated plants were directly compared.

Protein estimation: 250 mg of the inoculated tissues were repeatedly washed in distilled water, moisture was removed by blotting and digested in a micro-Kjeldahl apparatus. Total nitrogen was estimated and proteins were calculated by multiplying the total nitrogen by 6.25 (A.O.A.C. 1960).

Results

Respiration: A rapid activation of oxygen consumption was noted in tissues within 15 min of tilting the spores into the main chamber of the Warburg flask (Fig. 1). α-Picolinic acid and spore germination fluid had little effect on respiration at this interval. Continued incubation showed an enhanced rate of

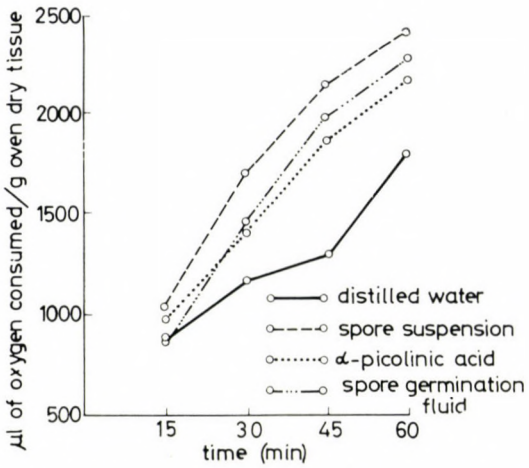


Fig. 1. Oxygen consumption (µl/g oven dry tissue) by the rice leaves in the various treatments

respiration when compared with the distilled water control. Respiration of the tissues strikingly increased in the presence of the pathogen.

Oxidase activity: Traces of polyphenol oxidase activity were noted in tissues suspended in the spore suspension at the end of 30 min. No enzyme activity was detected in the other treatments.

Peroxidase was markedly activated in tissues which were in contact with *P. oryzae* within 15 min (Fig. 2A). The activity gradually increased up to 30 min but decreased slightly at the end of 45 min, however, the activity was more than the control. α-Picolinic acid inhibited the peroxidase while spore germination fluid activated the enzyme at the end of 60 min.

Ascorbic acid oxidase activity of the tissues decreased in the initial stages of treatment with *P. oryzae* (Fig. 2B), however, at the end of 45 min, the enzyme activity was more than the distilled water control. The spore germination fluid distinctly inhibited the enzyme activity while α-picolinic acid exhibited little effect on ascorbic acid oxidase.

Changes in protein: In the leaves inoculated with *P. oryzae*, protein content increased while α-picolinic acid and the spore germination fluid had no effect (Fig. 3).

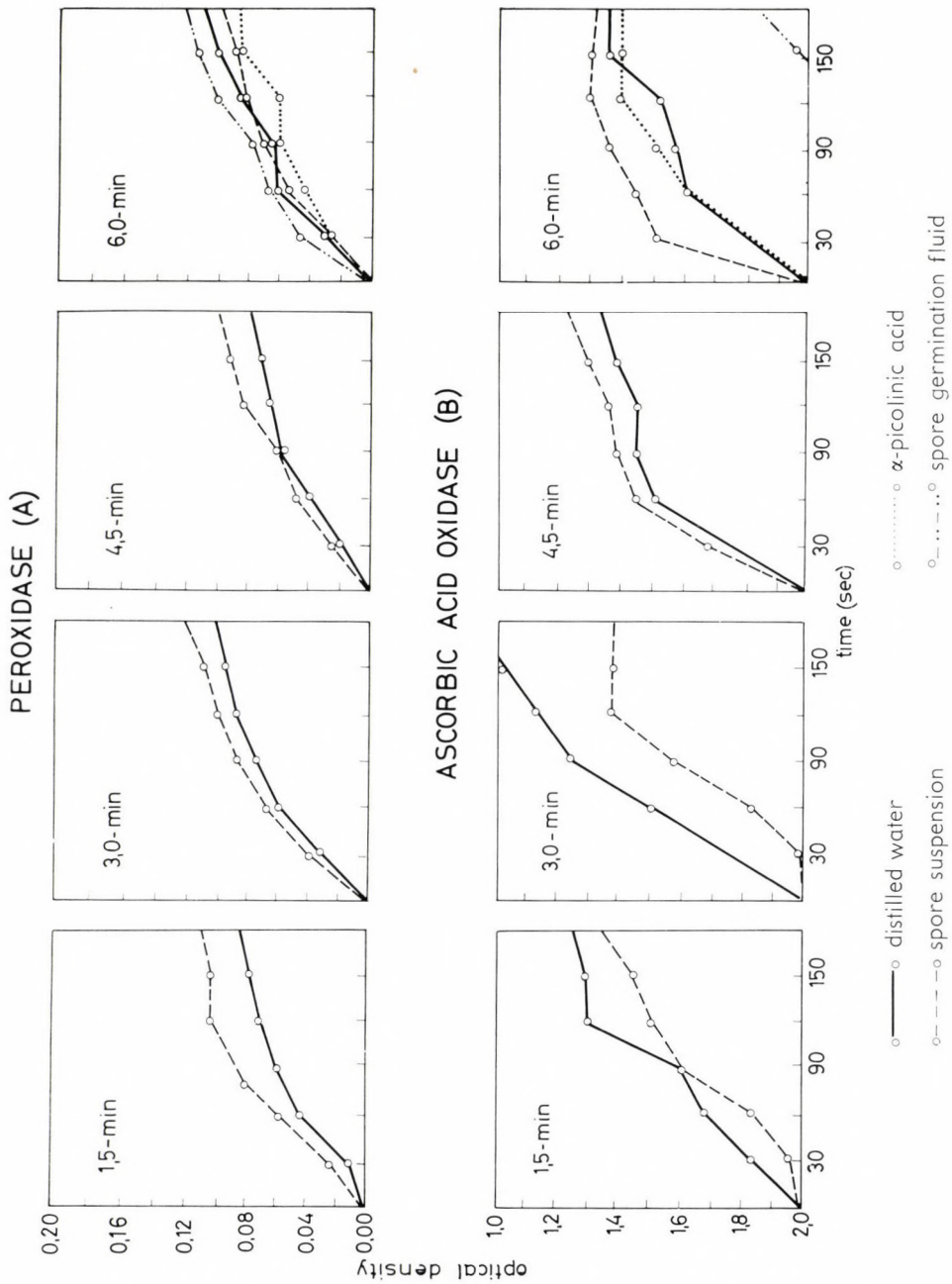


Fig. 2. Changes in optical density due to (A) peroxidase and (B) ascorbic acid oxidase

Discussion

The metabolism of the rice plant is altered by the pathogen, α-picolinic acid, the spore germination fluid and even injury. TOYODA and SUZUKI (1957) showed that the respiratory rate in the areas surrounding the necrotic spot of rice leaves increased 4 – 5 times over the healthy tissues. We found that activation

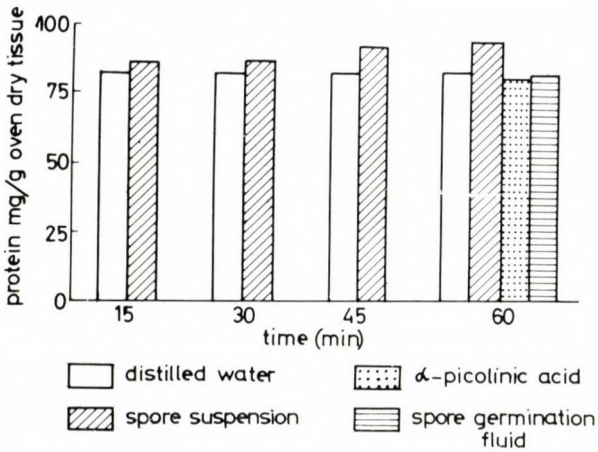


Fig. 3. Changes in protein content (mg/g oven dry tissue) of the treated tissues

of respiration is a general response exhibited by the leaves in response to *P. oryzae* and to its metabolites and the tissues react the moment they are in contact with any "intruding agent". Activation of respiration by α-picolinic acid and spore germination fluid which primarily contained α-picolinic acid is lower than that observed in the presence of the pathogen. Hence the pathogen alone causes maximum responses in the host while the metabolites are partially effective.

Rapid activation of terminal oxidases is characteristic of host parasite interactions (FARKAS and KIRÁLY, 1958) and such changes are reflections of "stress" in a host (KUĆ, 1967). The spore suspension activated the peroxidase up to 45 min and ascorbic acid oxidase 45 min after treatment. α-Picolinic acid inhibited the peroxidase but had little effect on ascorbic acid oxidase while the spore germination fluid enhanced peroxidase but distinctly inhibited ascorbic acid oxidase. Spore germination fluid may contain factors besides α-picolinic acid like piricularin which causes effects comparable to the pathogen (TAMARI et al. 1963).

Accumulation of protein soon after inoculation has been observed in many host parasite interactions (SHAW, 1963; URITANI et al. 1967). RAMAKRISHNAN (1966) found an increase in total nitrogen in rice plants at the end of 24 hrs of inoculation with *P. oryzae*. We found that protein synthesis is activated in the moment tissues come in contact with the parasite. Protein synthesis requires a

steady availability of adenosine triphosphate and is provided by the augmented breakdown of substrates, hence leading to rapid increase in respiration (CLICK and HACKETT, 1963; URITANI et al. 1967). These reactions are more regulated in a host-parasite combination while in other combinations, not regulated. Further the toxins and metabolites caused effects which are different from that of pathogen due to (i) limited concentration which may dissipate, (ii) not continuously available and (iii) inactivation by binding into cell walls or chemical transformation to non-toxic forms. Piricularin, a toxic metabolite of *P. oryzae* is detoxified by ferulic and chlorogenic acids (TAMARI et al. 1963).

The rapid onset of respiration and protein synthesis within 15 min of inoculation with *P. oryzae* suggests that the host cells react readily to the "trigger" given by the pathogen. The nature of "trigger" in host-parasite interaction has recently been studied by TOMIYAMA et al. (1967). They suggested that oxidized phenols act as a trigger in altering the metabolism of the cells. This type of alteration, we feel, is more limited and confined to the cells surrounding the invaded cells. KUĆ (1966) advanced an interesting proposal based on "operon theory"; in host-parasite interaction, the entrapment of repressor molecules leads to the synthesis of messenger ribonucleic acids (m-RNA) by the corresponding structural genes which direct the synthesis of the corresponding enzymes. So far no specific m-RNA has been isolated in host-parasite interaction. The sudden shifts in respiration and protein synthesis in the rice leaves inoculated with *P. oryzae* indirectly suggest the participation of KUĆ's (1966) mechanism.

The co-operation extended by Zoology and Geology departments to use Warburg respirometer and spectrophotometer, respectively, is gratefully acknowledged.

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A New Species of "Yeast-like" Achloric Algae, *Prototheca ubrizsyi* n. sp.

By

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A new species of *Prototheca*, *P. ubrizsyi* n. sp. was described. The new species is unable to assimilate KNO_3 and urea as a sole source of nitrogen contrary to the species of this genus described up to the present time.

During a study of the ecology of yeasts a strain of a microorganism with a macroscopic morphology resembling to yeasts was isolated from a flux of a mulberry tree (*Morus alba* L.) near to Szeged (Hungary) by the former author. Because of its interesting micromorphology it was studied further and finally it was identified as a new species of the genus *Prototheca* Krüger of algae.

Materials and Methods

The strain was isolated by direct inoculation of the fluid of a mulberry tree (*Morus alba* L.) onto malt-agar.

Subcultures were made on malt-agar. Identification was carried out by the method of Lodder and Kreger-van Rij (1952) partly modified by Zsolt and Novák (1961). Asparagine, glutamic acid, urea and peptone assimilation tests were performed by the auxanopraphic method too. Temperature relationship was studied on malt-agar slants. Sensitivity against antifungal antibiotics – cycloheximide (Actidione, Upjohn), Nystatin (Chinoin), Flavofungin* (Biogal) and Desertomycin** (Biogal) – was measured in Sabouraud glucose broth according a previously described turbidimetric method (Novák 1962). Bismuthsulphite agar was the standard (routine) bacteriologic type.

Results

Growth on malt agar 26°C, 3 days: Cells are globose or subglobose, 7.8–40.0 μ in diameter, single, containing one big of 2, 3, 6, 8 or more endospores with descending size parallel to their ascending number in one cell. Endospores measure 3.9–6.4–8.0 μ in diameter. (Figs 1–8.) Colony is cream-coloured, waxy, dull, margin entire, surface smooth.

* Flavofungin is a new antifungal antibiotic discovered by Uri and Békési (1958).

** Desertomycin is a new type of antifungal antibiotic discovered by Uri et al. (1958), recommended as an ingredient in selective media (Uri and Herpay 1961 a, b).



Fig. 1



Fig. 2

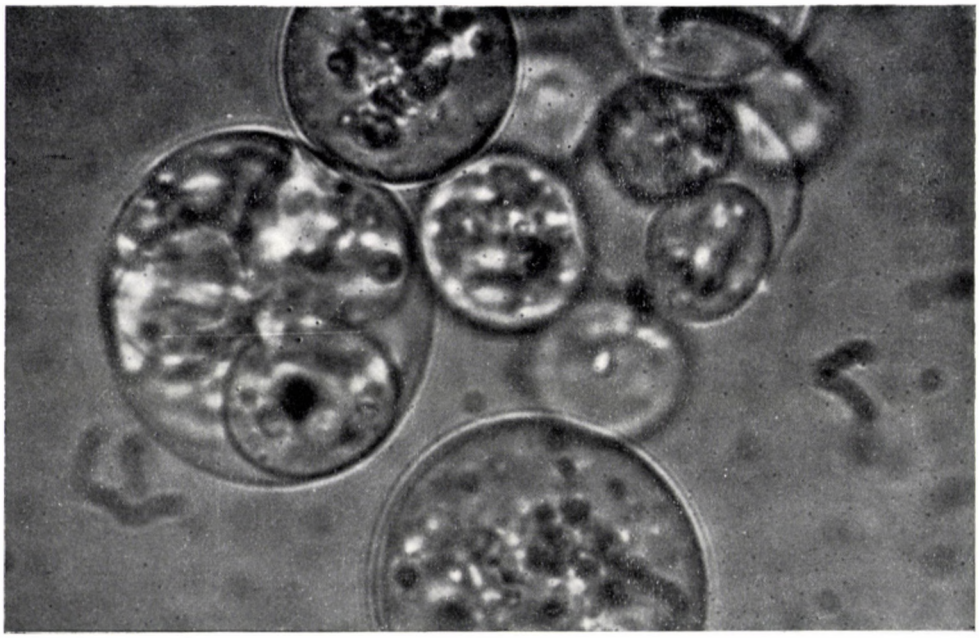


Fig. 3

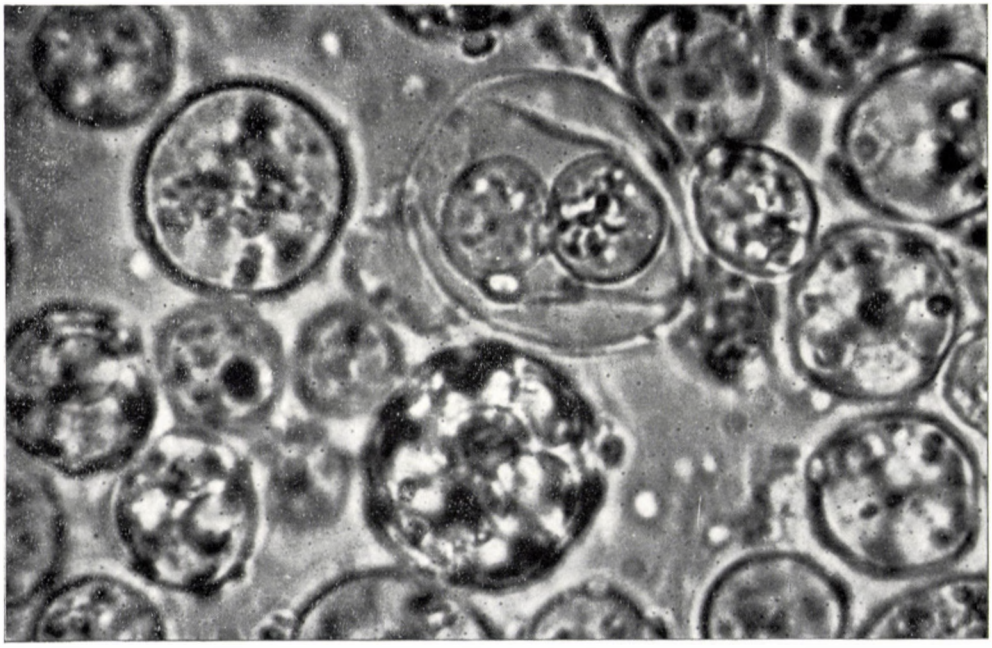


Fig. 4

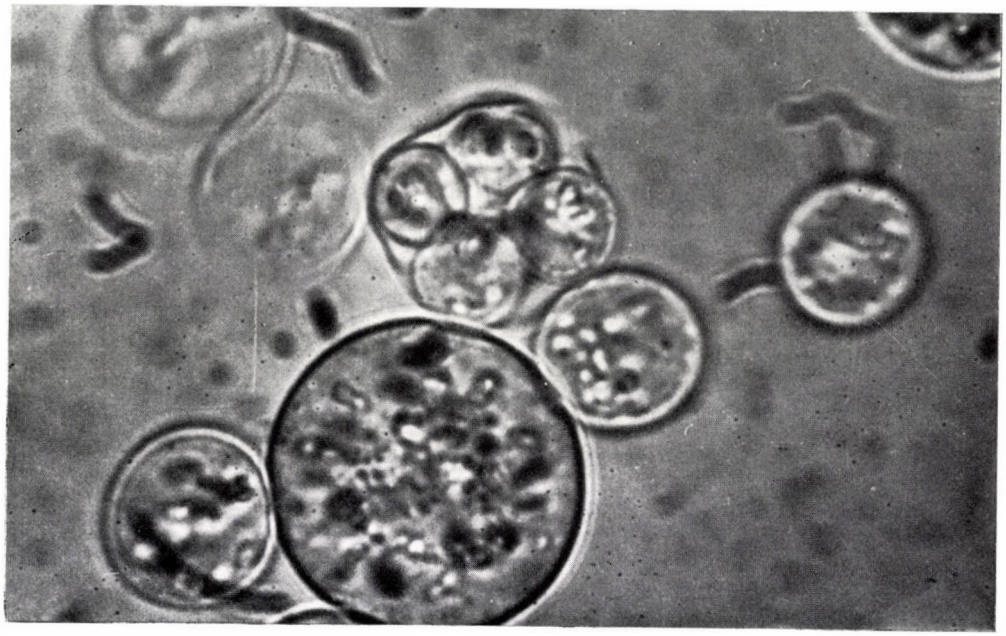


Fig. 5

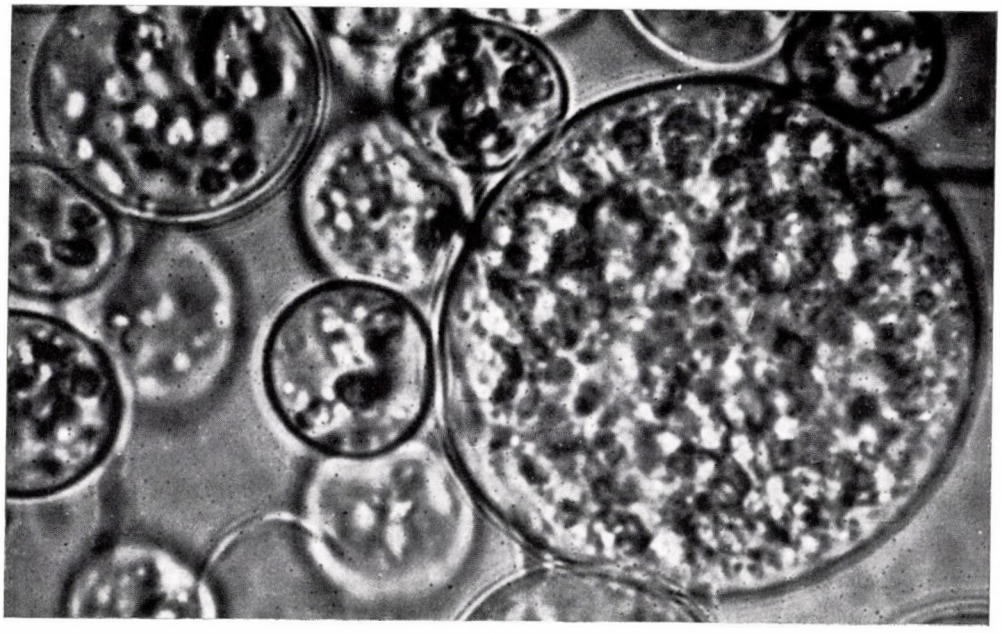


Fig. 6

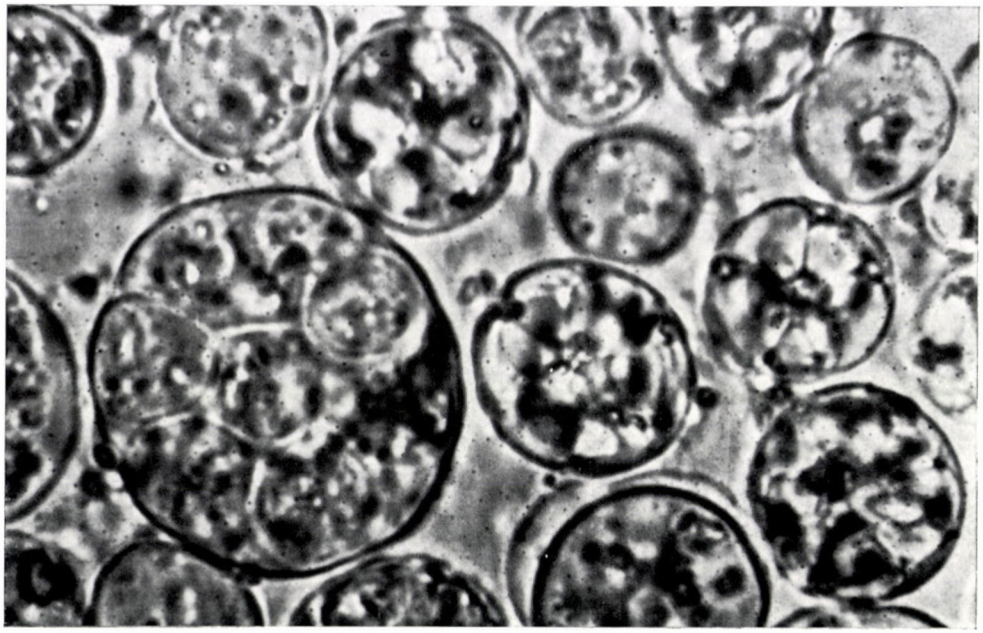
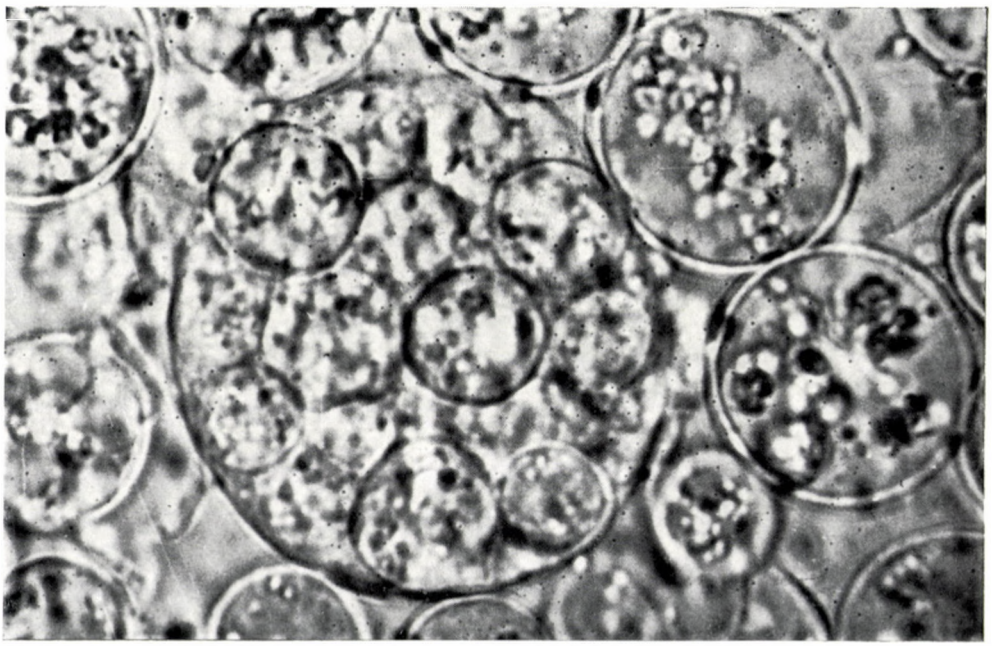


Fig. 7



Figs 1—8. The figures demonstrate the cells of *Prototheca ubrizsyi* n. sp. of various size and having various numbers of endospores. Note on Fig. 2. the big cell in the early phase of cytoplasma division forming two spores. (Magnif. 2500×.)

Growth on Pagano-Levin-Trejo TTC (1958) agar: Colony is pink, soft, surface smooth.

Growth in bi-sulphite agar: glistening, black, yeast like colonies.

Carbohydrate assimilation: DG/SMLR*

Carbohydrate fermentation: no fermentation of dgsmlr*, weak acidification with glucose and a delayed, slight reaction with galactose.

Nitrogen sources (after 10 days): $(NH_4)_2SO_4$, asparagine, glutamic acid and peptone positive, while KNO_3 and urea negative.

Growth on malt agar at various temperatures (4 days): 20° C and 26° C: good growth, 30° C: weak (moderate) growth, 37° C: no growth.

Assimilation of ethanol: positive

Splitting of arbutin: negative

Vitamine requirements: Thiamine gave a strong, and nicotinic amide a very weak stimulation of growth while riboflavine, pyridoxine, nicotinic acid, folic acid, calcium pantothenate and cyano-cobalamine (vitamin B_{12}) were indifferent in this respect.

Sensitivity to antifungal antibiotics: Flavofungin – in 25–200 μg per ml dilutions – was indifferent, Nystatin gave a moderate inhibition till 1–2 days (at 5th day in all dilutions – 2.5 to 80 μg per ml – reached the control values), Desertomycin showed a weak inhibition in 100–200 μg per ml and stimulation in 25–50 μg per ml concentrations, while Cycloheximide was strongly stimulative in 200 μg per ml, slightly stimulative in 50–100 μg per ml and indifferent in 25 μg per ml concentrations; as it is seen in Fig. 9.

The diagnosis of the species is as follows: The cells on malt-agar are globose to subglobose, single, producing 1–8-many endospores. The colonies are cream colored, flat, butyrous, dull, with entire margin and smooth surface. Obvious assimilation of glucose and galactose. Sucrose, maltose, lactose and raffinose are not assimilated. No fermentation of the former sugars, however, weak acidification with glucose and galactose (delayed). Ammonium sulfate, asparagine, glutamic acid and pepton served as nitrogen sources but not potassium nitrate and urea. Assimilation of ethanol, no splitting of arbutin. Good growth at 20–26° C, weak growth at 30° C, none at 37° C. Growth is stimulated by thiamin (strongly) and by nicotinic amide (very weakly), while riboflavine, pyridoxine, nicotinic acid, Ca-pantothenate, cyanocobalamine are indifferent.

Isolated from slime flux of mulberry tree (*Morus alba* L.).

Type strain: *Prototheca ubrizsyi* 322/1962 SIH. It is deposited in CBS. Yeast Division, Delft.

Latin diagnosis of Prototheca ubrizsyi n. sp.: In agaro maltato cellulae singulae, globosae vel subglobosae (7.8–40 μ). Multiplication cum 1–8-multis

* The data are expressed as a fraction in which the utilized compounds are in the numerator and the others in the denominator. D and d = glucose (dextrose), G and g = galactose, M and m = maltose, L and l = lactose, R and r = raffinose. Capital letters symbolize the respective sugar in assimilation, while the little ones in fermentation.

endosporis (3.9–8 μ). Colonia plana, flavoalbida, butyrosa, non-nitida, glabra, cum margine completa. Glucosum et galactosum at non saccharosum, maltosum lactosum et raffinosum assimilantur. Fermentatio nulla; paucum acidum e glucoso

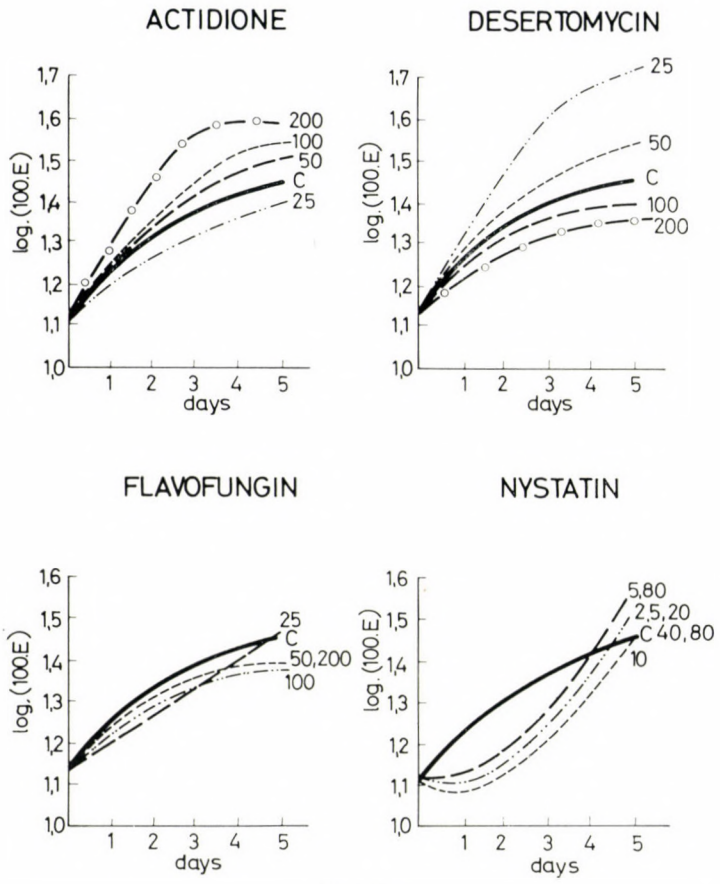


Fig. 9

et galactoso producetur. Sulfas ammonicus, asparaginum, acidum glutaminicum et peptonum at non nitras kalicus et urea assimilantur. Ethanolum assimilatur. Arbutinum non finditur. Optima temperatura crescentiae 20–26° C; super 37° C non crescit. Vitamini non sunt necessaria at thiaminum (valde) et amidum acidi nicotinici (multo lente) stimulant; riboflavinum, pyridoxinum, acidum nicotinicum, calcium pantothenicum et cyanocobalaminum indifferenti sunt. Isolabatur e exsudato *Mori albae* L.

Discussion

All *Prototheca* species described up to the present time assimilate KNO_3 and urea as a sole source of nitrogen (TUBAKI and SONEDA 1959), while our strain does not. For this reason we consider and describe it as a new species, under the name *Prototheca ubrizsyi* n. sp. in honorem Prof. Dr. GÁBOR UBRIZSY the excellent Hungarian phytopathologist and mycologist. The response to antifungal antibiotics by *P. ubrizsyi* n. sp. was very unusual, since no such stimulation was observed among algae investigated till now. *Bacillariophyceae*, *Chlorophyceae* and *Xantophyceae* showed sensitivity while *Cyanophyta* were resistant against cycloheximide (ZEHNDER and HUGHES 1958). By our strain, with cycloheximide a growth stimulation was observed in direct proportion with the increasing concentrations. Nystatin inhibited the growth of our strain only for two days and later the growth intensity raised over the control so that at the end of the investigation period (5 days) in all nystatin concentrations the cell-density was slightly higher than those measured in the control. Nystatin, however, inhibited all of the investigated algae, *Cyanophyta*, *Euglenophyta*, *Chlorophyta* and *Chrysophyta* out of *Bacillariophyceae* (LAMPEN and ARNOW 1961).

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Addition to the "Review of the Mycoflora of Hungary, Part I"

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Data relating to the *Myxomycotina: Plasmodiophorales* and *Phycomycotina: Phycomycetes* flora of Hungary were listed in an earlier publication, which appeared in *Acta Phytopathologica Hungarica*, Vol. 2., No. 2., in 1967. During the course of the further preparation of the literature on the subject as well as on the basis of the newer data, however, a completion becomes necessary.

The present paper contains all the data relating to the two fungal groups mentioned above. Literature on the subject was listed in the second part of this publication series *(Acta Phytopath. Hung.*, 1967, *2*(3), pp. 267–285). This article contains only the newer or complementary literature data.

Phylum. Mycota (Fungi)
Subphylum. Myxomycotina
Class. Myxomycetes
Subclass. Plasmodiophoromycetidae
(Archimycetes p. min. p.)
Ordo. Plasmodiophorales
Familia. Plasmodiophoraceae (incl. Woroninaceae)

Olpidiopsis fibrillosa DE WILDEMANN
Syn.: Pseudolpidiopsis fibrillosa DE WILD.
DOMJÁN 1. p. 16.; MOESZ 15. p. 72.
Olpidiopsis oedogoniarum (DE WILD.) SCHERFFEL
Syn.: Olpidium oedogoniarum (SOROKIN) DE WILD.
SCHERFFEL 1. p. 108.; SCHERFFEL 5. p. 103.; MOESZ 15. p. 69.; BÉRCZI 1. p. 80.; SCHERFFEL ap. CEJP 1. p. 372.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 41.
Olpidiopsis parasitica (FISCH.) A. FISCHER
Syn.: Olpidiopsis schenkiana ZOPF; Pseudolpidiopsis schenkiana (ZOPF) v. MINDEN
SCHERFFEL 1. p. 108.; SCHERFFEL 5. p. 15.; DOMJÁN 1. p. 32.; KRENNER 2. p. 201.; MOESZ 15. p. 69.; MOESZ 13. p. 43.; BÉRCZI 1. p. 80.; SCHERFFEL ap. CEJP 1. p. 374.; SÁNTHA–UBRIZSY in UBRIZSY 5. p. 41.
Olpidiopsis saprolegniae (A. BRAUN) CORNU

Syn.: Pseudolpidium saprolegniae (A. BRAUN) A. FISCHER
DOMJÁN 1. p. 32.; MOESZ 15. p. 69.

Plasmodiophora brassicae WORONIN
JABLONOWSKI 1. p. 93., 124.; SCHILBERSZKY 6. p. 346.; HEGYI 2. p. 41.; SCHILBERSZKY 7. p. 670.; BERNÁTSKY 2. p. 111.; MOESZ 40. p. 137.; LINHART, FÁY, LENGYEL, MÁGOCSY-DIETZ ap. MOESZ 15. p. 68.; UBRIZSY 1. p. 5.; KRENNER 1. p. 63.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 21.

Plasmodiophora solani BREHMER et BÄRNER
MOESZ 35.; BREHMER – BÄRNER ap. MOESZ 15. p. 68.; UBRIZSY 1. p. 5.

Pseudolpidiella sphaeritae (DANG.) CEJP
Syn.: Pseudolpidium sphaeritae (DANG.) FISCHER
ENTZ 1. p. 8.; MOESZ 15. p. 69.

Spongospora subterranea (WALLR.) JOHNSSON
UBRIZSY 1. p. 5.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 23.

Woronina glomerata (CORNU) A. FISCHER
SCHERFFEL 4. p. 16.; MOESZ 15. p. 69.; SCHERFFEL ap. CEJP 1. p. 336.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 41.

Woronina polycystis CORNU
BÄUMLER 1. II. No. 485.; MOESZ 15. p. 69.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 41.

Subphylum. Eumycotina
A. Phycomycotina
Class. Phycomycetes
Subclass. Chytridiomycetidae
Ordo. Chytridiales
Subgroup. Inoperculatae
Familia. Olpidiaceae

Olpidium brassicae (WOR.) DANGEARD
GULYÁS 1. p. 24.; MOESZ 15. p. 65.; UBRIZSY 1. p. 5.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 27.; UBRIZSY – VÖRÖS 2. p. 187.

Olpidium difflugiae SCHERFFEL
SCHERFFEL 7. p. 168.; MOESZ 15. p. 65.

Olpidium endogenum BRAUN
BÄUMLER 1. V. p. 26.; MOESZ 15. p. 65.

Olpidium entophytum BRAUN
SCHERFFEL 1. p. 108.; MOESZ 15. p. 65.

Olpidium gregarium (NOWAK.) SCHROETER
SCHERFFEL 4. p. 16.; MOESZ 15. p. 65.; BÉRCZI 1. p. 79.

Olpidium hyalothecae SCHERFFEL
SCHERFFEL 7. p. 200.; MOESZ 15. p. 65.

Olpidium leptophrydis SCHERFFEL
SCHERFFEL 7. p. 172.; MOESZ 15. p. 65.

Olpidium nicotianae PREISS
SÁNTHA – UBRIZSY in UBRIZSY 5. p. 29.
Olpidium pseudomorphum SCHERFFEL
SCHERFFEL 8. p. 510.; MOESZ 15. p. 65.
Olpidium pseudosporearum SCHERFFEL
SCHERFFEL 7. p. 170.; MOESZ 15. p. 65.
Olpidium saccatum SOROKIN
SCHERFFEL 3. p. 116.; SCHERFFEL 7. p. 210.; MOESZ 15. p. 65.
Olpidium trifolii (PERS.) SCHROETER
SÁNTHA – UBRIZSY in UBRIZSY 5. p. 29.
Olpidium utriculiforme SCHERFFEL
SCHERFFEL 7. p. 213.; MOESZ 15. p. 65.
Olpidium vampyrellae SCHERFFEL
SCHERFFEL 7. p. 168.; MOESZ 15. p. 65.
Pleotrachelus rotatorium SCHERFFEL
SCHERFFEL 10. p. 139.; MOESZ 15. p. 65.
Sphaerita endogena DANGEARD
ENDLICHER 1. p. 8.; SCHERFFEL 1. p. 108.; MOESZ 15. p. 66.

Familia. Synchytriaceae

Micromyces petersenii SCHERFFEL
Syn.: Mycromyces zygogonii AUCT. ap. SCHERFFEL
SCHERFFEL 3. p. 117.; SCHERFFEL 7. p. 209.; MOESZ 15. p. 66.
Micromycopsis cristata SCHERFFEL
SCHERFFEL 7. p. 202.; MOESZ 15. p. 66.
Micromycopsis fischerii SCHERFFEL
SCHERFFEL 7. p. 208.; MOESZ 15. p. 66.
Mitochytridium ramosum DANGEARD
BÉRCZI 1. p. 80.
Synchytrium anemones DE BARY et WORONIN
HAZSLINSZKY 1. p. 103.; FUSS 2. p. 428.; GRESCHIK 2. p. 117.; BRESADOLA 1. p. 112.; HOLLÓS 7. p. 107.; DOMJÁN 1. No. 140.; BÄUMLER, BOLLA, ENDLICHER, MOESZ, ZIMMERMANN ap. MOESZ 15. p. 66.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 34.; UBRIZSY – VÖRÖS 2. p. 189.
Synchytrium aureum SCHROETER
BÄUMLER 1. II. p. 67.; HOLLÓS 1. p. 92.; HOLLÓS 7. p 108.; KMET ap. MOESZ 15. p. 66.; LEHOCZKY 1. p. 235.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 33.; UBRIZSY – VÖRÖS 2. p. 189.
Synchytrium endobioticum (SCHILB.) PERCIVAL
Syn.: Chrysophlyctis endobiotica SCHILB.
SCHILBERSZKY 2. p. 36.; SCHILBERSZKY 4.; MOESZ 15. p. 66.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 30.; UBRIZSY – VÖRÖS 2. p. 189.
Synchytrium fulgens SCHROETER
BÄUMLER 1. V. p. 26.; MOESZ ap. MOESZ 15. p. 67.; MOESZ 13. p. 43.;

SÁNTHA – UBRIZSY in UBRIZSY 5. p. 34.; UBRIZSY – VÖRÖS 2. p. 189.

Synchytrium laetum SCHROETER

HOLLÓS 7. p. 108.; MOESZ 15. p. 67.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 33.; UBRIZSY – VÖRÖS 2. p. 189.

Synchytrium mercurialis (LIB.) FUCKEL

BRESADOLA 1. p. 112.; GRESCHIK, KMET ap. MOESZ 15. p. 68.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 34.; UBRIZSY – VÖRÖS 2. p. 189.

Synchytrium succisae DE BARY et WORONIN

HAZSLINSZKY 1. p. 103.; BÄUMLER 1. II. p. 67.; MOESZ ap. MOESZ 15. p. 68.; MOESZ 13. p. 44.; MOESZ 39. p. 175.; POLGÁR ap. MOESZ 1.; PICBAUER 1. p. 1.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 34.; UBRIZSY – VÖRÖS 2. p. 189.

Synchytrium taraxaci DE BARY et WORONIN

SCHERFFEL 1. p. 108.; HAZSLINSZKY 1. p. 14.; BÄUMLER, GRESCHIK, KMET, MOESZ, KALCHBRENNER, LINHART, GREINICH ap. MOESZ 15. p. 68.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 34.; UBRIZSY – VÖRÖS 2. p. 189.

Familia. Rhizidiaceae

Chytridium chaetophilum SCHERFFEL

SCHERFFEL 6. p. 45.; MOESZ 15. p. 70.

Chytridium (?) *characii* SCHERFFEL

SCHERFFEL 6. p. 13.; MOESZ 15. p. 70.

Chytridium confervae (WILLE) v. MINDEN

Syn.: Rhizidium confervae WILLE

SCHERFFEL 4. p. 17.; SCHERFFEL 6. p. 32.; MOESZ 15. p. 70.

Chytridium gibbosum SCHERFFEL

SCHERFFEL 1. p. 108.; SCHERFFEL 2. p. 105.; SCHERFFEL 7. p. 239.; v. MINDEN ap. MOESZ 15. p. 70.; MOESZ 13. p. 44.

Chytridium kolianum DOMJÁN

DOMJÁN 1. p. 26.; MOESZ 15. p. 70.

Chytridium lagenula (BRAUN) SCHERFFEL

Syn.: Rhizophydium lagenula (BRAUN) FISCHER

SCHERFFEL 7. p. 195.; MOESZ 15. p. 70.

Chytridium lateoperculatum SCHERFFEL

SCHERFFEL 7. p. 173.; MOESZ 15. p. 70.

Chytridium muricatum SCHERFFEL

SCHERFFEL 7. p. 216.; MOESZ 15. p. 70.

Chytridium olla BRAUN

SCHERFFEL 1. p. 108 ; SCHERFFEL 3 p. 117.; SCHERFFEL 4. p. 17.; SCHERFFEL 7. p. 225.; MOESZ 15. p. 70.

Chytridium pusillum SCHERFFEL

SCHERFFEL 10. p. 143.; MOESZ 15. p. 70.

Chytridium schenkii (DANG.) SCHERFFEL

Syn.: Rhizidium schenkii DANG.

SCHERFFEL 4. p. 16.; SCHERFFEL 7. p. 237.; DOMJÁN 1. p. 26.; MOESZ 15. p. 70.

Chytridium scherffelii BÉRCZI
BÉRCZI 1. p. 84.
Chytridium sphaerocarpum (ZOPF) DANGEARD
BÉRCZI 1. p. 84.
Chytridium spinulosum BLYTT
SCHERFFEL 3. p. 117.; SCHERFFEL 7. p. 216.; MOESZ 15. p. 71.
Chytridium spirotaeniae SCHERFFEL
SCHERFFEL 6. p. 14.; MOESZ 15. p. 71.
Chytridium versatile SCHERFFEL
SCHERFFEL 7. p. 177.; DOMJÁN 1. p. 26.; MOESZ 15. p. 71.
Coralliochytrium scherffelii DOMJÁN
DOMJÁN 1. p. 19.; MOESZ 15. p. 71.
Ectochytridium (Zygorhizidium) *willei* (LOEWENTHAL) SCHERFFEL
SCHERFFEL 6. p. 7.; DOMJÁN 1. p. 28.; MOESZ 15. p. 71.
Entophlyctis apiculata BRAUN
BÉRCZI 1. p. 86.
Entophlyctis aurantiaca SCHERFFEL
DOMJÁN 1. p. 25., 48.; MOESZ 15. p. 71.
Entophlyctis bulligera (ZOPF) A. FISCHER
SCHERFFEL 1. p. 108.; DOMJÁN 1. p. 23.; MOESZ 15. p. 71.; BÉRCZI 1. p. 86.
Entophlyctis pseudodistomum SCHERFFEL
DOMJÁN 1. p. 23.; MOESZ 15. p. 71.
Entophlyctis rhizina (SCHENK) v. MINDEN
Syn.: Entophlyctis vaucheriae FISCHER
SCHERFFEL 1. p. 108.; SCHERFFEL 9. p. 256.; DOMJÁN 1. p. 23.; MOESZ 15. p. 71.; BÉRCZI 1. p. 86.
Harpochytrium adpressum SCHERFFEL
SCHERFFEL 8. p. 521.; MOESZ 15. p. 71.
Harpochytrium hedinii WILLE
SCHERFFEL 4. p. 16.; SCHERFFEL 8. p. 520.; MOESZ 15. p. 71.
Harpochytrium hyalothecae LAGERHEIM
SCHERFFEL 4. p. 16.; SCHERFFEL 8. p. 521.; MOESZ 15. p. 71.
Harpochytrium intermedium ATKINSON
SCHERFFEL 8. p. 521.; MOESZ 15. p. 71.
Harpochytrium viride SCHERFFEL
SCHERFFEL 8. p. 519.; MOESZ 15. p. 71.
Nucleophaga sp.
SCHERFFEL 1. p. 109.; MOESZ 15. p. 71.
Olpidiomorpha pseudosporae SCHERFFEL
SCHERFFEL 8. p. 515.; MOESZ 15. p. 72.
Phlyctidium anatropum (BRAUN) SPARROW
BÉRCZI 1. p. 80.
Phlyctidium anomalum COUCH
BÉRCZI 1. p. 80.

Phlyctidium bumilleriae COUCH
BÉRCZI 1. p. 81.
Phlyctidium dentatum (ROSEN) SCHROETER
DOMJÁN 1. p. 19.; MOESZ 15. p. 72.
Phlyctidium eudorinae GIMESI
SCHERFFEL 1. p. 108.; GIMESI 1.; MOESZ 15. p. 72.; MOESZ 13. p. 44.
Phlyctidium haynaldii SCHARSCHMIDT
Syn.: Rhizophydium haynaldii A. FISCHER
SCHARSCHMIDT 1. p. 58.; SCHARSCHMIDT 2. p. 125.; FISCHER, v. MINDEN. SCHERFFEL ap. MOESZ 15. p. 72.; BÉRCZI 1. p. 81.
Phlyctochytrium dentatum (ROSEN) SCHROETER
BÉRCZI 1. p. 83.
Phlyctochytrium lagenaria (SCHENK) DOMJÁN
Syn.: Chytridium lagenaria SCHENK
DOMJÁN 1. p. 18.; MOESZ 15. p. 72.; BÉRCZI 1. p. 84.
Phlyctochytrium planicorne ATKINSON
BÉRCZI 1. p. 83.
Phlyctochytrium stellatum PETERSEN
SCHERFFEL 4. p. 16.; SCHERFFEL 7. p. 233.; MOESZ 15. p. 72.
Phlyctochytrium zygnematis (ROSEN) SCHROETER
DOMJÁN 1. p. 19.; MOESZ 15. p. 72.; BÉRCZI 1. p. 84.
Physorhizophydium pachydermum SCHERFFEL
SCHERFFEL 7. p. 181.; MOESZ 15. p. 72.
Podochytrium clavatum PFITZER
SCHERFFEL 3. p. 116.; SCHERFFEL 7. p. 174.; MOESZ 15. p. 72.
Polyphagus euglenae NOWAKOWSKI
Syn.: Chytridium (Rhizidium) euglenae BRAUN
ENTZ 1. p. 175.; SCHERFFEL 1. p. 108.; MOESZ 15. p. 72.
Polyphagus parasiticus NOWAKOWSKI
SCHERFFEL 1. p. 109.; SCHERFFEL 2. p. 106.; SCHERFFEL 3. p. 117.; SCHERFFEL 6. p. 2.; MOESZ 15. p. 72.
Rhizidiomyces apophysatus ZOPF
BÉRCZI 1. p. 84.
Rhizophydium ampullaceum (BRAUN) A. FISCHER
MOESZ ap. MOESZ 15. p. 72.
Rhyzophydium appendiculatum (ZOPF) A. FISCHER
SCHERFFEL 4. p. 16.; MOESZ 15. p. 73.
Rhizophydium asterosporum SCHERFFEL
SCHERFFEL 6. p. 17.; MOESZ 15. p. 73.
Rhizophydium braunii (DANG.) A. FISCHER
SCHERFFEL 1. p. 108.; MOESZ 15. p. 73.
Rhizophydium chrysopyxidis SCHERFFEL
SCHERFFEL 7. p. 174.; MOESZ 15. p. 73.
Rhizophydium clinopus SCHERFFEL

SCHERFFEL 10. p. 141.; MOESZ 15. p. 73.

Rhizophydium cyclotellae ZOPF
DOMJÁN 1. p. 17.; HORTOBÁGYI—DOMJÁN ap. MOESZ 15. p. 73.

Rhizophydium decipiens (BRAUN) FISCHER
SCHERFFEL 4. p. 16.; SCHERFFEL 7. p. 219.; MOESZ 15. p. 73.

Rhizophydium digitatum SCHERFFEL
SCHERFFEL 7. p. 223.; MOESZ 15. p. 73.

Rhizophydium eudorinae HOOD
ENTZ jun. 2. p. 12.; MOESZ 15. p. 73.

Rhizophydium fallax SCHERFFEL
SCHERFFEL 6. p. 30.; MOESZ 15. p. 73.

Rhizophydium fusus (ZOPF) A. FISCHER
SCHERFFEL 1. p. 108.; SCHERFFEL 2. p. 106.; MOESZ 15. p. 73.

Rhizophydium gibbosum (ZOPF) A. FISCHER
SCHERFFEL 3. p. 116.; MOESZ 15. p. 73.

Rhizophydium globosum (BRAUN) SCHROETER
SCHARSCHMIDT 1. p. 62.; SCHERFFEL 4. p. 16.; SCHERFFEL 7. p. 229.; ENTZ jun. 2. p. 12.; DOMJÁN 1. p. 16.; GYŐRFFY ap. MOESZ 15. p. 73.; BÉRCZI 1. p. 81.

Rhizophydium goniosporum SCHERFFEL
SCHERFFEL 6. p. 20.; MOESZ 15. p. 73.; BÉRCZI 1. p. 81.

Rhizophydium granulosporum SCHERFFEL
SCHERFFEL 6. p. 44.; MOESZ 15. p. 74.

Rhizophydium hyalothecae SCHERFFEL
SCHERFFEL 7. p. 201.; MOESZ 15. p. 74.

Rhizophydium irregulare DE WILDEMAN
SCHERFFEL 3. p. 116.; MOESZ 15. p. 74.

Rhizophydium leptophridis SCHERFFEL
SCHERFFEL 7. p. 172.; MOESZ 15. p. 74.

Rhizophydium mamillatum (BRAUN) FISCHER
BÉRCZI 1. p. 82.

Rhizophydium minutum ATKINSON
BÉRCZI 1. p. 82.

Rhizophydium mischococci SCHERFFEL
SCHERFFEL 7. p. 195.; MOESZ 15. p. 74.

Rhizophydium ovatum COUCH
BÉRCZI 1. p. 82.

Rhizophydium parasitans SCHERFFEL
SCHERFFEL 6. p. 26.; MOESZ 15. p. 74.

Rhizophydium persimilis SCHERFFEL
SCHERFFEL 7. p. 199.; MOESZ 15. p 74.

Rhizophydium pseudodistomum SCHERFFEL
SCHERFFEL 10. p. 140.; MOESZ 15. p. 74.

Rhizophydium quadricorne (DE BARY) SCHERFFEL
SCHERFFEL 4. p. 17.; SCHERFFEL 7. p. 224.; MOESZ 15. p. 74.

Rhizophydium sphaerocarpum (ZOPF) A. FISCHER
DOMJÁN 1. p. 17.; KRENNER 2. p. 201.; SCHERFFEL 4. p. 16.; MOESZ 15. p. 74.; MOESZ 13. p. 44.; BÉRCZI 1. p. 82.
Rhizophydium sporoctonum (BRAUN) A. FISCHER
SCHERFFEL 7. p. 222.; MOESZ 15. p. 74.
Rhizophydium subangulosum (BRAUN) RABENHORST
BÉRCZI 1. p. 83.
Rhizophydium utriculus SCHERFFEL
SCHERFFEL 7. p. 173.; MOESZ 15. p. 74.
Rhizophydium xylophilum (CORNU) A. FISCHER
SCHERFFEL 1. p. 108.; SCHERFFEL 2. p. 106.; SCHERFFEL 7. p. 247.; MOESZ 15. p. 74.
Rhizophydium sp.
SCHERFFEL 7. p. 241.; MOESZ 15. p. 74.
Rhizosiphon crassum SCHERFFEL
SCHERFFEL 7. p. 184.; MOESZ 15. p. 74.

Familia. Cladochytriaceae

Nowakowskiella endogena CONSTANTINESCU
DOMJÁN 1. p. 28.; MOESZ 15. p. 75.
Physoderma butomi SCHROETER
HOLLÓS 1. p. 92.; MOESZ 15. p. 75.; MOESZ 13. p. 44.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 37.; UBRIZSY – VÖRÖS 2. p. 192.
Physoderma gerhardti SCHROETER
KMET ap. MOESZ 15. p. 75.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 37.; UBRIZSY – VÖRÖS 2. p. 192.
Physoderma maculare WALLROTH
Syn.: Cladochytrium alismatis (WALLR.) BÜSGEN
HOLLÓS 1. p. 92.; HOLLÓS 7. p. 107.; MOESZ 15. p. 75.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 37.; UBRIZSY – VÖRÖS 1. p. 145.; UBRIZSY – VÖRÖS 2. p. 192.
Physoderma menyanthis DE BARY
Syn.: Cladochytrium menyanthis DE BARY
BÄUMLER 1. V. p. 26.; MOESZ 15. p. 75.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 37.; UBRIZSY – VÖRÖS 2. p. 192.
Physoderma pulposum WALLROTH
Syn.: Urophlyctis pulposa (WALLR.) SCHROETER
MOESZ 11. p. 95.; GÁYER, GREINICH, JÁVORKA, MOESZ, ZSÁK ap. MOESZ 15. p. 75.; MOESZ ap. MOESZ 13. p. 44.; UBRIZSY 1. p. 5.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 36.; UBRIZSY – VÖRÖS 2. p. 192.
Physoderma schroeteri KRIEGER
FILARSZKY, MOESZ ap. MOESZ 15. p. 75.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 37.; UBRIZSY – VÖRÖS 2. p. 192.
Physoderma zsákii (MOESZ) CIFERRI

Syn.: Entyloma zsákii MOESZ
MOESZ, ZSÁK ap. MOESZ 15. p. 75.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 37.; UBRIZSY – VÖRÖS 2. p. 192.

Ordo. Monoblepharidales
Familia. Monoblepharidaceae

Monoblepharis macrandra (LAGERH.) WORONIN
SCHERFFEL 10. p. 137.; DUDICH ap. MOESZ 15. p. 75.

Subclass. Siphomycetideae (Siphomycetes)
A. Coll. ordo. Oomycetes (Oomycetidae)
Ordo. Saprolegniales
Familia. Ectrogellaceae

Ectrogella bacillariacearum ZOPF
SCHERFFEL 1. p. 108.; SCHERFFEL 5. p. 6.; DOMJÁN 1. p. 31.; MOESZ 15. p. 77.; SCHERFFEL ap. CEJP 1. p. 68.
Ectrogella besseyi SPARROW et ELLISON
SCHERFFEL, DOMJÁN ap. CEJP 1. p. 67.
Ectrogella gomphonematis SCHERFFEL
SCHERFFEL 5. p. 9.; MOESZ 15. p. 77.; CEJP 1. p. 70.
Ectrogella monostoma SCHERFFEL
SCHERFFEL 5. p. 8.; MOESZ 15. p. 77.; SCHERFFEL ap. CEJP 1. p. 73.

Familia. Saprolegniaceae

Achlya conspicua COKER
CEJP 1. p. 142.
Achlya debaryana HUMPHREY
CEJP 1. p. 159.
Achlya flagellata COKER
CEJP 1. p. 144.
Achlya polyandra HILDEBRAND
KRENNER 2. p. 201.; MOESZ 15. p. 77.
Achlya prolifera (NEES) DE BARY
BÄUMLER 1. II. p. 72.; MOESZ 15. p. 77.; UBRIZSY 1. p. 5.
Achlya racemosa HILDEBRAND
MOESZ ap. MOESZ 15. p. 77.; CEJP 1. p. 182.
Aphanomycopsis bacillariacearum SCHERFFEL
SCHERFFEL 5. p. 14.; MOESZ 15. p. 77.; SCHERFFEL ap. CEJP 1. p. 75.
Aphanomyces cochlioides DRECHSLER
SÁNTHA – UBRIZSY in UBRIZSY 5. p. 39.
Aphanomyces gordejevi SKORTZOW
BÉRCZI 1. p. 87.

Aphanomyces laevis DE BARY
SÁNTHA–UBRIZSY in UBRIZSY 5. p. 39.
Aphanomyces phycophilus DE BARY
KRENNER 2. p. 201.; MOESZ 15. p. 77.; MOESZ 13. p. 44.
Saprolegnia ferax (GRUITH.) THURET
Syn.: Saprolegnia thureti DE BARY
BÄUMLER 1. V. p. 72.; HAZSLINSZKY ap. MOESZ 15. p. 77.
Saprolegnia litoralis COKER
BÉRCZI 1. p. 87.
Saprolegnia monoica PRINGSHEIM
BÄUMLER 1. II. p. 72.; MOESZ 15. p. 77.; UBRIZSY 1. p. 5.

Ordo. Leptomitales
Familia. Leptomitaceae

Leptomitus lacteus (ROTH) AGARDH
HAZSLINSZKY, KALCHBRENNER ap. MOESZ 15. p. 78.
Leptomitus lavandulae BIASOL.
KALCHBRENNER ap. MOESZ 15. p. 78.
Leptomitus rubi-idaei BIASOL.
KALCHBRENNER ap. MOESZ 15. p. 78.

Familia. Rhipidiaceae

Rhipidium interruptum CORNU
CEJP 1. p. 318.

Ordo. Lagenidiales
Familia. Lagenidiaceae

Ancylistes closterii PFITZER
SCHERFFEL 1. p. 109.; MOESZ 15. p. 76.; BÉRCZI 1. p. 86.
Lagenidium brachystomum SCHERFFEL
SCHERFFEL 5. p. 21.; SCHERFFEL 10. p. 256.; MOESZ 15. p. 76.; DOMJÁN ap. CEJP 1. p. 398.
Lagenidium cyclotellae SCHERFFEL
SCHERFFEL 5. p. 18.; MOESZ 15. p. 76.; SCHERFFEL ap. CEJP 1. p. 400.
Lagenidium enecans ZOPF
SCHERFFEL 1. p. 109.; SCHERFFEL 2. p. 106.; SCHERFFEL 5. p. 20.; MOESZ 15. p. 76.; SCHERFFEL ap. CEJP 1. p. 399.
Lagenidium entophytum (PRINGSH.) ZOPF
SCHERFFEL 3. p. 117.; MOESZ 15. p. 76.; BEIŠÁN ap. CEJP 1. p. 385.
Lagenidium nov. sp. SCHERFFEL
SCHERFFEL 5. p. 23.; MOESZ 15. p. 76.
Lagenidium oedogonii SCHERFFEL

DOMJÁN 1. p. 31.; SCHERFFEL 1. p. 109.; SCHERFFEL 2. p. 105.; SCHERFFEL 5. p. 109.; MOESZ 15. p. 76.; BÉRCZI 1. 86.; DOMJÁN, SCHERFFEL ap. CEJP 1. p. 396.

Lagenidium rabenhorstii ZOPF

SCHERFFEL 4. p. 17.; DOMJÁN 1. p. 31.; KRENNER 2. p. 201.; MOESZ 15. p. 76.; MOESZ 13. p. 44.; BÉRCZI 1. p. 86.; CEJP 1. p. 388.

Myzocytium irregulare PETERSEN

SCHERFFEL 4. p. 17.; MOESZ 15. p. 76.; SCHERFFEL ap. CEJP 1. p. 406.

Myzocytium megastomum DE WILDEMAN

SCHERFFEL 4. p. 17.; MOESZ 15. p. 76.

Myzocytium proliferum SCHENK

DOMJÁN 1. p. 31.; SCHERFFEL 1. p. 109.; MOESZ 15. p. 76.; BÉRCZI 1. p. 87.; SCHERFFEL ap. CEJP 1. p. 408.

Oovorus copepodorum ENTZ

ENTZ jun. 1. p. 175.; MOESZ 15. p. 77.

Ordo. Peronosporales
Familia. Pythiaceae

Phytophthora infestans (MONTAGNE) DE BARY

Syn.: Botrytis infestans MONT.; Botrytis devastatrix LIV.

KALCHBRENNER 1. No. 787.; SCHULZER 2. I. p. 153.; MIKA 1. p. 161.; FUSS 2. No. 72.; BÄUMLER 1. II. No. 502.; HAZSLINSZKY 4.; BARNA 1. p. 205.; HEGYI 2. p. 18.; RAPAICS 1. p. 214.; MOESZ 2.; MOESZ 35. p. 15.; TAKÁCS 1.; SCHILBERSZKY 3.; BEKE 1.; HOLLÓS 7. p. 111.; LINHART, MÁGOCSY-DIETZ ap. MOESZ 15. p. 78.; UBRIZSY 1. p. 5.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 47.; UBRIZSY—VÖRÖS 2. p. 203.

Phytophthora parasitica DASTUR.

Syn.: Phytophthora terrestris SCHERB.; Blepharospora terrestris (SCHERB.) PEYR. SÁNTHA—UBRIZSY in UBRIZSY 5. p. 50.; UBRIZSY—VÖRÖS 2. p. 203.

Pythium de baryanum HESSE

LINHART 3.; HEGYI 1., 3.; SCHILBERSZKY 5.; GULYÁS 1.; MOESZ 15. p. 79.; MOESZ 13. p. 48.; BÉRCZI 1. p. 88.; UBRIZSY 1. p. 5.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 43.; UBRIZSY—VÖRÖS 2. p. 200.

Pythium gracile SCHENK

SCHERFFEL 9. p. 256.; KRENNER 2. p. 201.; MOESZ 15. p. 79.; MOESZ 13. p. 48.; UBRIZSY—VÖRÖS 2. p. 200.

Pythium hydnosporum (MONT.) SCHROETER

Syn.: Pythium artrotrogus (MONT.) DE BARY; P. micranthum DE BARY

MIKA 1. p. 163.; MOESZ 15. p. 79.; BÉRCZI 1. p. 88.; UBRIZSY 1. p. 5.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 46.

Pythium reptans DE BARY emend. KRENNER

Syn.: Pythium gracile SCHENK

KRENNER 2. p. 201.; MOESZ 15. p. 79.; MOESZ 13. p. 48.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 45.

Pythium vexans DE BARY

MIKA 1. p. 162.; MOESZ 15. p. 79.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 45.; UBRIZSY – VÖRÖS 2. p. 200.

Familia. Peronosporaceae

Basidiophora entospora ROZE et CORNU

Syn.: Plasmopara entospora SCHROET.

BÄUMLER 1. II. No. 503.; GREINICH ap. MOESZ 15. p. 79.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 55.; UBRIZSY – VÖRÖS 2. p. 203.

Bremia REGEL

Bremia centaureae SYDOW

HOLLÓS 1. p. 93.; KMET ap. MOESZ 15. p. 79.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 85.; UBRIZSY – VÖRÖS 2. p. 205.

Bremia lactucae REGEL

Syn.: Peronospora gangliformis DE BARY

HOLLÓS 1. p. 93.; HOLLÓS 7. p. 108.; KMET ap. MOESZ 15. p. 79.; MOESZ 15. p. 80.; UBRIZSY 1. p. 5.; KRENNER 1. p. 63.; PICBAUER 1. p. 2.; PICBAUER 2. p. 178.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 84.; UBRIZSY – VÖRÖS 1. p. 145.; UBRIZSY – VÖRÖS 2. p. 205.

Bremia lapsanae GÄUMANN

HAZSLINSZKY ap. MOESZ 1.; UBRIZSY – VÖRÖS 1. p. 146.; UBRIZSY – VÖRÖS 2. p. 205.

Bremia sonchi SAWADA

KALCHBRENNER 1. No. 783.; BÄUMLER 1. II. No. 507.; RAPAICS 1. p. 218.; HOLLÓS 7. p. 108.; KMET, LINHART, MOESZ ap. MOESZ 15. p. 79.; MOESZ 13. p. 45.; UBRIZSY 1. p. 5.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 85.; UBRIZSY – VÖRÖS 1. p. 145.; UBRIZSY – VÖRÖS 2. p. 205.

Bremia spec. div. (sine nominibus)

BÄUMLER, GREINICH, HAZSLINSZKY, HOLLÓS, KMET, MOESZ ap. MOESZ 15. p. 80.; MOESZ 13. p. 45.; MOESZ 1.; UBRIZSY – VÖRÖS 1. p. 147.

Bremia spec. indet.

Syn.: Bremia lactucae REGEL ?

LEHOCZKY 1. p. 235.

Bremia tulasnei (HOFFM.) SYDOW

HOLLÓS 1. p. 93.; HOLLÓS 7. p. 108.; MOESZ 15. p. 80.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 86.; UBRIZSY – VÖRÖS 2. p. 205.

Bremia xanthii UBRIZSY

UBRIZSY – VÖRÖS 1. p. 146.; UBRIZSY – VÖRÖS 2. p. 205.

Peronospora CORDA

Peronospora achilleae SAVULESCU et RAYSS

UBRIZSY in litt.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora aestivalis SYDOW

SACCARDO, D. 1. p. 178.; RAPAICS 1. p. 215.; DEGEN, GREINICH, KMET, MOESZ ap. MOESZ 15. p. 86.; UBRIZSY 1. p. 5.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora affinis ROSSM.

HOLLÓS 1. p. 94.; HOLLÓS 7. p. 109.; HAZSLINSZKY ap. MOESZ 15. p. 84.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora agrestis GÄUMANN

BÄUMLER 1. II. No. 514.; MOESZ 15. p. 89.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora agrostemmatis GÄUMANN

Syn.: P. dianthi DE BARY

KALCHBRENNER 1. No. 750.; GREINICH, MOESZ ap. MOESZ 15. p. 82.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora alsinearum CASPARY

TIMÁR 1. p. 235.

Peronospora alta FUCKEL

BÄUMLER 1. V. No. 1665.; HAZSLINSZKY 4. p. 33.; HOLLÓS 1. p. 94.; MOESZ 21. p. 82.; LINHART, KMET ap. MOESZ 15. p. 90.; MOESZ 13. p. 45.; MOESZ 1.; UBRIZSY 1. p. 6 ; SZIGETI—TÓTH 1. p. 16.; UBRIZSY—VÖRÖS 1. p. 149.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora alyssi-calycini GÄUMANN

GREINICH ap. MOESZ 15. p. 84.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora aparines (DE BARY) GÄUMANN

HAZSLINSZKY 4. p. 31.; HOLLÓS 7. p. 109.; GREINICH, MOESZ ap. MOESZ 15. p. 90.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora arborescens (BERK.) DE BARY

HAZSLINSZKY 4. p. 32.; HOLLÓS 1. p. 94.; KRENNER, LINHART ap. MOESZ 15. p. 84.; MOESZ 1.; UBRIZSY 2. p. 152.; KRENNER 1. p. 63.; LEHOCZKY 1. p. 235.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 64.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora arenariae (BERK.) TULASNE

HAZSLINSZKY 4. p. 32.; MÁGOCSY-DIETZ ap. MOESZ 15. p. 82.; SZIGETI—TÓTH 1. p. 16.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora arvensis GÄUMANN

HAZSLINSZKY 4. p. 32.; LINHART, KMET, GREINICH, MOESZ ap. MOESZ 15. p. 89.; MOESZ 13. p. 45.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora astragalina SYDOW

UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora atriplicis-hastatae SAVULESCU et RAYSS

Syn.: Peronospora effusa GREV.; P. litoralis GÄUM.

BÄUMLER 1. II. No. 512.; MOESZ 23. p. 818.; HOLLÓS 1. p. 94.; MOESZ 15. p. 81.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 62.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora berteroae GÄUMANN

BÄUMLER 1. II. No. 517.; MOESZ 15. p. 84.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora borealis GÄUMANN

UBRIZSY—VÖRÖS 1. p. 149.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora brassicae GÄUMANN

HEGYI 2. p. 38., 68.; RAPAICS 1. p. 215.; AUGUSZTIN ap. MOESZ 15. p. 84.; UBRIZSY 1. p. 5.; VÖRÖS 9. p. 100.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 59.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora bulbocapni BECK

Syn.: Peronospora corydalis DE BARY f. corydalis cavae THUEM.

BÄUMLER 1. II. No. 511.; HOLLÓS 7. p. 110.; MOESZ 15. p. 84.; MOESZ 13. p. 45.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora buniadis GÄUMANN

MOESZ ap. MOESZ 15. p. 84.; MOESZ 13. p. 45.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora calaminthae FUCKEL

BUBÁK 1. p. 22.; MOESZ 15. p. 88.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora calotheca DE BARY

BÄUMLER 1. II. No. 509.; HOLLÓS 7. p. 109.; KMET, MOESZ ap. MOESZ 15. p. 90.; MOESZ 13. p. 45.; MOESZ 1.; SZIGETI—TÓTH 1. p. 17.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 64.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora camelinae GÄUMANN

KALCHBRENNER 1. No. 786.; HOLLÓS 1. p. 94.; GREINICH, LINHART, MOESZ ap. MOESZ 15. p. 84.; MOESZ 13. p. 45. UBRIZSY 1. p. 5.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora cardamines-laciniatae GÄUMANN

Syn.: Peronospora parasitica DE BARY ppe.

SACCARDO, D. 1. p. 178.; HOLLÓS 7. p. 111.; KMET, MOESZ, PÉNZES ap. MOESZ 15. p. 85.; MOESZ 13. p. 45.; MOESZ 1.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora cheiranthi GÄUMANN

Syn.: Peronospora parasitica DE BY ppe.

BÄUMLER 1. II. No. 517.; MOESZ 15. p. 85.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 59.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora chenopodii SCHLECHTENDAL

Syn.: Peronospora effusa RABENH.

FUSS 2. p. 429.; KALCHBRENNER 1. No. 781.; BÄUMLER 1. II. No. 512.; HOLLÓS 1. p. 94.; HOLLÓS 7. p. 110.; KMET, MÁGOCSY-DIETZ, MOESZ ap. MOESZ 15. p. 81.; MOESZ 13. p. 45.; UBRIZSY 2. p. 152.; VÖRÖS 9. p. 100.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora chenopodii-urbici SAVULESCU et RAYSS

Syn.: Peronospora effusa RABENH.

HOLLÓS 7. p. 110.; MOESZ 15. p. 81.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora chlorae DE BARY

BÄUMLER 1. III. No. 1403.; HOLLÓS 7. p. 109.; DEGEN, MOESZ ap. MOESZ 15. p. 88.; MOESZ 13. p. 45.; PICBAUER 1. p. 3.; PICBAUER 2. p. 179.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora chrysosplenii FUCKEL

HAZSLINSZKY 4. p. 32.; BÄUMLER 1. II. No. 510.; MOESZ 15. p. 86.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora conglomerata FUCKEL

HAZSLINSZKY 4. p. 33.; BÄUMLER 1. IV. No. 1485.; GREINICH ap. MOESZ 15. p. 87.; MOESZ 13. p. 45.; UBRIZSY – VÖRÖS 1. p. 149.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora coronillae GÄUMANN

HOLLÓS 1. p. 93.; MOESZ 15. p. 86.; MOESZ 1.; UBRIZSY – VÖRÖS 2. p. 215.

Peronospora corydalis DE BARY

HAZSLINSZKY 4. p. 32.; KMET, JÁVORKA, MOESZ ap. MOESZ 15. p. 84.; MOESZ 13. p. 45.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora cyparissiae DE BARY

MOESZ ap. MOESZ 15. p. 88.; MOESZ 13. p. 45.; SZIGETI – TÓTH 1. p. 17.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora destructor (BERK.) CASPARY

Syn.: Peronospora schleideni UNGER

RAPAICS 1. p. 215.; MOESZ ap. MOESZ 15. p. 81.; UBRIZSY 1. p. 5.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 61.; UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora digitalidis GÄUMANN

KMET, MOESZ ap. MOESZ 15. p. 89.; MOESZ 13. p. 45.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora diplotaxidis GÄUMANN

LEHOCZKY 1. p. 235.; UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora dipsaci (NEES v. ES.) TULASNE

BÄUMLER 1. V. No. 1666.; HOLLÓS 1. p. 93.; HOLLÓS 7. p. 110.; GREINICH, HAZSLINSZKY ap. MOESZ 15. p. 91.; UBRIZSY 1. p. 6.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora drabae GÄUMANN

MOESZ ap. MOESZ 15. p. 85.; MOESZ 13. p. 45.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora erophilae GÄUMANN

Syn.: Peronospora parasitica TUL. f. drabae FUCK.

BÄUMLER 1. II. No. 517.; HOLLÓS 1. p. 94.; BOROS, GREINICH, KMET, MOESZ ap. MOESZ 15. p. 85.; MOESZ 13. p. 45.; UBRIZSY 2. p. 152.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora erysimi GÄUMANN

MOESZ ap. MOESZ 15. p. 85.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora erythraeae (KÜHN.) GÄUMANN
HOLLÓS 7. p. 109.; GREINICH ap. MOESZ 15. p. 88.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora euphorbiae FUCKEL
BUDAI ap. MOESZ 15. p. 88.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora ficariae (NEES v. ES.) TULASNE
HAZSLINSZKY 4. p. 32.; BÄUMLER 1. II. No. 513.; HOLLÓS 1. p. 94.; HOLLÓS 7. p. 111.; GÁYER, LINHART, MOESZ, SZÉPLIGETI ap. MOESZ 15. p. 83.; MOESZ 13. p. 45.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora grisea UNGER
KMET, LINHART ap. MOESZ 15. p. 89.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora hiemalis GÄUMANN
KMET, MOESZ ap. MOESZ 15. p. 83.; MOESZ 13. p. 45.; SZIGETI – TÓTH 1. p. 17.; UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora holostei CASPARY
HAZSLINSZKY 4. p. 32.; BÄUMLER 1. IV. No. 1486.; GREINICH, LINHART, MOESZ ap. MOESZ 15. p. 82.; MOESZ 13. p. 46.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora hyoscyami DE BARY
KALCHBRENNER 1. No. 781/b; HAZSLINSZKY 4. p. 33.; HOLLÓS 1. p. 94.; AUGUSZTIN, PÁTER ap. MOESZ 15. p. 89.; MOESZ 39. p. 176.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 64.; UBRIZSY – VÖRÖS 1. p. 149.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora iberidis GÄUMANN
MOESZ 37. pp. 4 – 5.; MOESZ 13. p. 46.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora illyrica GÄUMANN
HOLLÓS 7. p. 111.; MOESZ ap. MOESZ 15. p. 83.; MOESZ 13. p. 46.; MOESZ 1.; UBRIZSY – VÖRÖS 2. p. 215.

Peronospora kochiae GÄUMANN
UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 211

Peronospora lamii BRAUN
KALCHBRENNER 1. No. 785.; BÄUMLER 1. II. No. 515.; HAZSLINSZKY 4. p. 32.; HOLLÓS 1. p. 94.; GREINICH, KMET, MOESZ ap. MOESZ 15. p. 89.; MOESZ 13. p. 46.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora lathyri-palustris GÄUMANN
HULJÁK ap. MOESZ 15. p. 86.; UBRIZSY – VÖRÖS 2. p. 215.

Peronospora lepidii-perfoliati SAVULESCU et RAYSS
UBRIZSY in litt.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora lepidii-sativi GÄUMANN
BÄUMLER 1. II. No. 517.; HOLLÓS 1. p. 94.; MOESZ 12. p. 817.; HOLLÓS 7. p. 111.; GREINICH ap. MOESZ 15. p. 85.; MOESZ 13. p. 46.; UBRIZSY 1. p. 5.; TIMÁR 1. p. 235.; UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora lepigoni FUCKEL
SACCARDO, D. 1. p. 179.; MOESZ, KMET ap. MOESZ 15. p. 82.; MOESZ 39. p. 176.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora leptosperma DE BARY
HOLLÓS 1. p. 93.; MOESZ 23. p. 818.; MOESZ 15. p. 91.; UBRIZSY – VÖRÖS 2. p. 213.
*Peronospora lithospermi*GÄUMANN
GREINICH ap. MOEZ 15. p. 88.; UBRIZSY – VÖRÖS 2. p. 213.
Peronospora lobulariae UBRIZSY 1966.
UBRIZSY – VÖRÖS 1. p. 147.; UBRIZSY – VÖRÖS 2. p. 209.
Peronospora lunariae GÄUMANN
Syn.: P. parasitica DE BY ppe.
BUBÁK 1. p. 22.; MOESZ 15. p. 85.; PICBAUER 1. p. 3.; ZELLER – TÓTH 1. p. 230.; TÓTH 10. p. 3.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY – VÖRÖS 2. p. 209.
Peronospora manshurica (NAOUMOFF) SYDOW
Syn.: Peronospora trifoliorum DE BARY var. manshurica NAOUM.; P. sojae LEHM. et WOLF
VÖRÖS – MOLNÁRNÉ 1. p. 371.; VÖRÖS 9. p. 100.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 66.; UBRIZSY – VÖRÖS 2. p. 213.
Peronospora matthiolae GÄUMANN
LEHOCZKY 2. p. 7.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY – VÖRÖS 2. p. 209.
Peronospora maublancii SAVULESCU et RAYSS
Syn.: Peronospora lepidii virginici GÄUM.
MOESZ 11. p. 95.; MOESZ 15. p. 85.; UBRIZSY – VÖRÖS 2. p. 209.
Peronospora mayorii GÄUMANN
MOESZ ap. MOESZ 15. p. 87.; MOESZ 13. p. 46.; UBRIZSY – VÖRÖS 2. p. 213.
Peronospora media GÄUMANN
Syn.: Peronospora alsinearum CASP.
HAZSLINSZKY 4. p. 31.; BÄUMLER 1. II. No. 508.; HOLLÓS 7. p. 109.; GREINICH, LINHART, MÁGOCSY-DIETZ ap. MOESZ 15. p. 82.; MOESZ 13. p. 46.; TIMÁR 1. p. 235.; UBRIZSY – VÖRÖS 2. p. 213.
Peronospora meliloti SYDOW
GREINICH, MOESZ ap. MOESZ 15. p. 86.; UBRIZSY 4. p. 54.; UBRIZSY 6. in SZÉKESSY p. 23.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 66.; TÓTHNÉ 1. p. 171.; UBRIZSY – VÖRÖS 2. p. 215.
Peronospora minor (CASP.) GÄUMANN
Syn.: Peronospora effusa v. minor CASP.
HAZSLINSZKY 4. p. 33.; BÄUMLER 1. II. No. 512.; MOESZ ap. MOESZ 15. p. 81.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY – VÖRÖS 2. p. 209.
Peronospora muralis GÄUMANN
MÁGOCSY-DIETZ ap. MOESZ 15. p. 81.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY – VÖRÖS 2. p. 209.
Peronospora myosotidis DE BARY
BÄUMLER 1. II. No. 516.; MOESZ 15. p. 88.; UBRIZSY – VÖRÖS 2. p. 213.
Peronospora niessleana BERLESE

Syn.: Peronospora parasitica DE BY ppe.
HOLLÓS 1. p. 94.; MOESZ 11. p. 95.; KMET, LINHART ap. MOESZ 15. p. 86.; MOESZ 13. p. 46.; UBRIZSY 2. p. 152.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora ochroleuca CESATI
MOESZ ap. MOESZ 15. p. 86.; MOESZ 13. p. 46.; MOESZ 39. p. 176.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora ononidis WILSON
KMET ap. MOESZ 15. p. 87.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora parasitica (PERS.) TULASNE
BÄUMLER 1. II. No. 517.; HOLLÓS 1. p. 94.; HOLLÓS 7. p. 111.; GREINICH, LINHART, MOESZ, SZABÓ ap. MOESZ 15. p. 86.; MOESZ 13. p. 46.; MOESZ 1.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 58.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora phyteumatis FUCKEL
KMET ap. MOESZ 15. p. 91.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora pisi SYDOW
RAPAICS 1. p. 215.; MOESZ 15. p. 87.; UBRIZSY 1. p. 5.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 65.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora polygoni (HALS.) FISCHER
UBRIZSY 4. p. 54.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora pratensis SYDOW
RAPAICS 1. p. 215.; MOESZ 15. p. 87.; UBRIZSY 1. p. 5.; UBRIZSY—VÖRÖS 2. p. 215.

Peronospora pulveracea FUCKEL
HOLLÓS 7. p. 111.; MOESZ ap. MOESZ 15. p. 83.; VASS—TÓTH 2. p. 46.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora ranunculi GÄUMANN
Syn.: Peronospora ficariae TUL. f. ranunculi SCHNEIDER
BRESADOLA 1. p. 112.; HAZSLINSZKY 4. p. 32.; BÄUMLER 1. II. No. 519.; HOLLÓS 1. p. 94.; HOLLÓS 7. p. 111.; GRESCHIK, LINHART, MOESZ ap. MOESZ 15. p. 83.; MOESZ 1.; UBRIZSY 1. p. 5.; UBRIZSY 2. p. 152.; SZIGETI—TÓTH 1. p. 17.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora rubi RABENHORST
LEHOCZKY 1. p. 235.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora rumicis CORDA
KMET, MÁGOCSY-DIETZ ap. MOESZ 15. p. 81.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora schachtii FUCKEL
HAZSLINSZKY 4. p. 33.; BÄUMLER 1. V. No. 1667.; MOESZ 15. p. 82.; VÖRÖS 9. p. 100.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 60.; UBRIZSY—VÖRÖS 1. p 148.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora scleranthi RABENHORST
KMET, MÁGOCSY-DIETZ, MOESZ, ZSÁK ap. MOESZ 15. p. 83.; MOESZ 13. p. 47.; MOESZ 1.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora senneniana FRAGOSO et SACCARDO
BÄUMLER 1. II. No. 520.; MOESZ ap. MOESZ 15. p. 87.; MOESZ 13. p. 47.; PICBAUER 2. p. 179.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora sepium GÄUMANN
HOLLÓS 7. p. 111.; MOESZ 15. p. 87.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora sherardiae FUCKEL
HAZSLINSZKY 4. p. 31.; MOESZ 15. p. 90.; MOESZ 13. p. 47.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora silvatica GÄUMANN
BÄUMLER 1. II. No. 509.; MOESZ ap. MOESZ 15. p. 90.; MOESZ 13. p. 47.; MOESZ 1.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora sisymbrii-officinalis GÄUMANN
MOESZ ap. MOESZ 15. p. 86.; MOESZ 13. p. 47.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora sisymbrii-orientalis GÄUMANN
MOESZ ap. MOESZ 15. p. 86.; UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora sordida BERKELEY
BÄUMLER 1. II. No. 518.; HAZSLINSZKY 4. p. 33.; KMET, LINHART, MOESZ ap. MESZ 15. p. 89.; MOESZ 13. p. 47.; MOESZ 1.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora spinaciae LAUBERT
Syn.: Peronospora effusa (GREV.) TUL.
HOLLÓS 7. p. 111.; LINHART, MOESZ ap. MOESZ 15. p. 82.; MOESZ 13. p. 47.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 62.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora stachydis SYDOW
MOESZ ap. MOESZ 15. p. 89.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora symphyti GÄUMANN
BÄUMLER ,GERSCHIK ap. MOESZ 15. p. 88.; UBRIZSY – VÖRÖS 2. p. 215.

Peronospora tabacina ADAM
TUBOLY 1. p. 55.; VÖRÖS 9. p. 101.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 66.; UBRIZSY – VÖRÖS 2. p. 211.

Peronospora tatarica SAVULESCU et RAYSS
UBRIZSY in litt.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora thlaspeos-arvensis GÄUMANN
KMET, LINHART ap. MOESZ 15. p. 86.; UBRIZSY – VÖRÖS 1. p. 148.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora thlaspeos-perfoliati GÄUMANN
Syn.: Peronospora parasitica DE BY ppe.
BÄUMLER 1. II. No. 517.; HOLLÓS 1. p. 94.; HOLLÓS 7. p. 111.; GREINICH, MOESZ ap. MOESZ 15. p. 86.; MOESZ 13. p. 47.; UBRIZSY – VÖRÖS 2. p. 209.

Peronospora tomentosa FUCKEL
KMET ap. MOESZ 15. p. 83.; UBRIZSY – VÖRÖS 2. p. 213.

Peronospora tribulina PASSERINI
HOLLÓS 1. p. 94.; MOESZ ap. MOESZ 15. p. 88.; MOESZ 13. p. 48.; UBRIZSY – VÖRÖS 2. p. 215.

Peronospora trifolii-alpestris GÄUMANN
MOESZ ap. MOESZ 15. p. 87.; MOESZ 13. p. 48.; MOESZ 1.; UBRIZSY 1. p. 6.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora trifolii-hybridi GÄUMANN
UBRIZSY—VÖRÖS 1. p. 149.; UBRIZSY—VÖRÖS 2. p. 215.

Peronospora trifolii-repentis SYDOW
HAZSLINSZKY 4. p. 32.; BÄUMLER 1. II. No. 519.; MOESZ 15. p. 87.; UBRIZSY—VÖRÖS 1. p. 149.; TÓTHNÉ 1. p. 171.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora trifoliorum DE BARY
SÁNTHA—UBRIZSY in UBRIZSY 5. p. 63.

Peronospora trivialis GÄUMANN
HAZSLINSZKY 4. p. 31.; BÄUMLER 1. II. No. 508.; HOLLÓS 7. p. 93.; GREINICH, KMET, PÉNZES ap. MOESZ 15. p. 83.; MOESZ 13. p. 48.; MOESZ 1.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora valerianae TRAIL.
UBRIZSY in litt.; UBRIZSY—VÖRÖS 2. p. 215.

Peronospora valerianellae FUCKEL
HAZSLINSZKY 4. p. 32.; GREINICH, LINHART ap. MOESZ 15. p. 90.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 65.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora valesiaca GÄUMANN
GREINICH ap. MOESZ 15. p. 88.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora variabilis GÄUMANN
Syn.: Peronospora effusa DE BY f. chenopodii albi THUEM.
HAZSLINSZKY 4. p. 33.; BÄUMLER 1. II. No. 512.; HOLLÓS 7. p. 111.; GRESCHIK, MOESZ ap. MOESZ 15. p. 82.; MOESZ 13. p. 48.; MOESZ 1.; UBRIZSY 4. p. 55.; UBRIZSY 6. in SZÉKESSY p. 23.; SZIGETI—TÓTH 1. p. 17.; VÖRÖS 9. p. 101.; UBRIZSY—VÖRÖS 1. p. 148.; UBRIZSY—VÖRÖS 2. p. 209.

Peronospora verbasci GÄUMANN
GREINICH ap. MOESZ 15. p. 90.; UBRIZSY—VÖRÖS 2. p. 215.

Peronospora verna GÄUMANN
KMET, MOESZ ap. MOESZ 15. p. 90.; MOESZ 13. p. 48.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora viciae (BERK.) GÄUMANN
KMET, HAZSLINSZKY, MOESZ ap. MOESZ 15. p. 87.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY—VÖRÖS 2. p. 213.

Peronospora viciae-sativae GÄUMANN
GREINICH ap. MOESZ 15. p. 87.; UBRIZSY—VÖRÖS 2. p. 215.

Peronospora violae DE BARY
BRESADOLA 1. No. 83.; BÄUMLER 1. V. No. 1668.; HAZSLINSZKY 4. p. 32.; GREINICH, GRESCHIK ap. MOESZ 15. p. 88.; MOESZ 13. p. 48.; UBRIZSY—VÖRÖS 2. p. 211.

Peronospora vistulensis WROBLEWSKI
GREINICH, MOESZ ap. MOESZ 15. p. 82.; MOESZ 13. p. 48.; UBRIZSY 1. p. 45.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 63.; UBRIZSY—VÖRÖS 2. p. 211.

Plasmopara SCHROETER

Plasmopara aegopodii (CASP.) TROTTER
Syn.: Pl. nivea (UNG.) SCHROET. ppe.
KALCHBRENNER 1.; BÄUMLER 1.; HOLLÓS 7. p. 112.; BUDAI, HOLUBY, KMET, MOESZ, HAZSLINSZKY, LINHART, MÁGOCSY-DIETZ, SIMONKAI ap. MOESZ 15. p. 91.; MOESZ 1.; UBRIZSY – VÖRÖS 1. p. 149.; UBRIZSY – VÖRÖS 2. p. 207.
Plasmopara conii (CASP.) TROTTER
Syn.: Pl. nivea (UNG.) SCHROET. ppe.
HOLLÓS 7. p. 112.; GREINICH, TAUSCHER ap. MOESZ 15. p. 91.; UBRIZSY – VÖRÖS 2. p. 207.
Plasmopara densa (RABENH.) SCHROETER
HAZSLINSZKY 4. p. 31.; BÄUMLER 1. IV. No. 1487.; HOLLÓS 7. p. 112.; GREINICH, LINHART, MOESZ ap. MOESZ 15. p. 91.; SÁNTHA – UBRIZSY in UBRIZSY 5. p. 83.; UBRIZSY – VÖRÖS 2. p. 205.
Plasmopara geranii-pratensis TR. et O. SAVULESCU
UBRIZSY – VÖRÖS 1. p. 149.; UBRIZSY – VÖRÖS 2. p. 205.
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Syn.: Peronospora urticae (LIB.) DE BARY

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Sclerospora graminicola (SACC.) SCHROETER

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Familia. Albuginaceae

Albugo (PERS.) GRAY

Albugo bliti (BIV.) KUNTZE

Syn.: Cystopus bliti (BIV. et BERN.) LÉV.

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Albugo candida (PERS.) KUNTZE

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KALCHBRENNER 1. No. 959.; SCHULZER 1.; FUSS 2. No. 77—79.; HAZSLINSZKY 2.; GRESCHIK 2.; BÄUMLER 1.; SACCARDO, D. 1.; HEGYI 3.; BUBÁK 1.; MOESZ 2. No. 3.; HOLLÓS 1. p. 93.; RAPAICS 1.; MOESZ 12.; MOESZ 21.; MOESZ 23.; MOESZ 11.; HOLLÓS 7. p. 108.; DOMJÁN 1. No. 74.; BORBÁS, DEGEN, BUDAI, GÁYER, GREINICH, GYŐRFFY, HOLUBY, JÁVORKA, KÁNITZ, KMET, KOVÁCS, KRENNER, LINHART, LOJKA, MÁGOCSY-DIETZ, SADLER, SZÉPLIGETI, TRAUTMANN, WAGNER, WIERZBICZKY ap. MOESZ 15. p. 95.; MOESZ 13. p. 44.; UBRIZSY 1. p. 6.; UBRIZSY 2. p. 152.; UBRIZSY 4. p. 55.; UBRIZSY 6. in SZÉKESSY p. 23.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 86.; VÖRÖS 9. p. 100.; UBRIZSY—VÖRÖS 1. p. 149.; TIMÁR 1. p. 235.; BÁNYAI, BOROS, DEGEN, GYŐRFFY ap. MOESZ 1.; O. SAVULESCU 1. pp. 12—58.; UBRIZSY—VÖRÖS 2. p. 220.

Albugo candida (PERS.) KUNTZE var. *globulosa* f. *microspora* O. SAVULESCU

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Albugo ipomoeae panduranae (SCHWEIN.) SCHWINGLE

SÁNTHA—UBRIZSY 5. p. 89.

Albugo lepigoni (DE BARY) KUNTZE

Syn.: Cystopus lepigoni DE BY.

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Albugo portulacae (DC.) KUNTZE

Syn.: Cystopus portulacae (DEK. et PASS.) LÉV.

HAZSLINSZKY 1. p. 105.; FUSS 2. p. 81.; HAZSLINSZKY 2. p. 12.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. III. p. 190.; HOLLÓS 1. p. 93.; MOESZ 21. p. 82.; HOLLÓS 7. p. 109.; KOVÁCS, LÁNYI, MÁGOCSY-DIETZ, PÉNZES ap. MOESZ 15. p. 96.; MOESZ 13. p. 44.; KRENNER ap. MOESZ 1.; UBRIZSY 1. p. 6.; VÖRÖS 9. p. 100.; SÁNTHA—UBRIZSY in UBRIZSY 5. p. 89.; UBRIZSY—VÖRÖS 1. p. 149.; HOLLÓS, MOESZ ap. O. SAVULESCU 1. pp. 59—61.; UBRIZSY—VÖRÖS 2. p. 220.

Albugo tragopogonis (PERS.) GRAY

Syn.: Cystopus tragopogi (PERS.) OUDEM.; C. tragopogonis SCHROET.; C. cubicus LÉV.

HAZSLINSZKY 1. p. 105.; HAZSLINSZKY 2. p. 12.; HAZSLINSZKY 3. p. 89.; BÄUMLER 1. II. p. 69.; SACCARDO, D. 1. p. 179.; BUBÁK 1. p. 22.; HOLLÓS 1. p. 93.; RAPAICS 1. p. 218.; MOESZ 21. p. 82.; MOESZ 11.; HOLLÓS 7. p.

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Albugo tragopogonis (PERS.) GRAY f. *tragopogi* O. SAVULESCU
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Ordo. Mucorales
Familia. Mucoraceae

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Absidia capillata VAN TIEGHEM
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Absidia corymbifera (COHN.) SACCARDO et TROTTER
ZELLER 2.

Absidia glauca HAGEM.
Syn.: Absidia septata VAN TIEGH.
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Absidia spinosa LENDNER
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Circinella simplex VAN TIEGHEM
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