

Research on potato wart disease in the U.S.S.R. - a literature review (1955-1977)

Michael C. Hampson¹

Potato wart disease, recorded in the Soviet Union in 1935, is now found in all six Western Soviet Socialist Republics. It is particularly evident in the Trans-Carpathian mountainous zone of the Ukrainian S.S.R. Since the establishment of a Potato Wart Disease Research Station in Trans-Carpathia in 1944, extensive work has been carried out on the pathology, biology and physiology of the disease and its causal agent. Particular emphasis has been paid to the elucidation of the mechanism of resistance. Since biotypes were discovered in 1962, work has been devoted to immune cultivar production and selection for resistance. A well systematized method is used for crop inspection, quarantine and disease management. Chemical controls have also been developed.

Can. Plant Dis. Surv. 59:1, 7-14. 1979

La galle verruqueuse de la pomme de terre, signalée en 1935 en Union Soviétique, touche maintenant les six républiques soviétiques socialistes de l'ouest. Elle sevit principalement dans la zone montagneuse transcarpatique de la république ukrainienne. Depuis la fondation en 1944 d'une Station de recherche sur la galle verruqueuse dans cette région, de vastes travaux ont porté sur la pathologie, la biologie et la physiologie de la maladie et de son agent pathogène. On s'est particulièrement intéressé à la compréhension des mécanismes de résistance. Depuis la découverte de biotypes en 1962, les recherches ont été consacrées à la production de cultivars immuns et à la sélection de variétés résistantes. On a élaboré une méthode bien structurée pour l'inspection, la surveillance sanitaire des cultures et l'éradication de la maladie. On a également mis au point des techniques de lutte chimique.

Introduction

Potato wart disease caused by *Synchytrium endobioticum* (Schilb.) Perc. was first described, from Hungary, in 1896. It has been present in Newfoundland, Canada, since the 1900's (16), and is no longer present in the U.S.A. (2,3).

It was extensively reported from 1898 onwards in Western Europe, but not observed in U.S.S.R. until 1935. It is generally conceded (22) that most infection foci in the Ukrainian S.S.R. - the most extensively infested area in Russia - originated from tubers imported from Western Europe during the German territorial occupation of the 1940's.

There is probably no other area in the world where the potato wart disease problem has been tackled with such vigor and vision as in the U.S.S.R. This article reviews work published in the Soviet Union from 1955-1977.

The information for this article was made available from translations of work available to Western observers supplied by the Multilingual Division, Translation Bureau, Secretary of State Department. As far as possible, the phraseology of the translators is used. For example, the uses of the terms "pathogenicity/aggressivity/virulence" are not clearly defined. Therefore, when pathological terms appear in the text and appear to run counter to accepted usage, or are ambiguous,

they have been marked with an asterisk, and the phraseology has been transferred verbatim from the translated copy.

Research centers

The main research center was located at Boyany, near Chernovtsy, in 1944, as most foci of potato wart disease were found in the so-called Carpathian arc (Fig. 1). At that time, the station was given a mandate to pursue research into determining immunity, resistance and tolerance of potato varieties* to wart disease with special emphasis on the mechanism of resistance. The station was staffed by approximately twenty research scientists grouped into five sections, and equipped extensively with much modern apparatus (17).

Laboratory, greenhouse and field work is pursued on biological, biochemical and biophysical questions at Boyany. Biochemical work on the mechanism of resistance is also carried out at other institutes, such as the Potato Research Institute, Moscow, the Ukraine Scientific Institute for Plant Physiology, and the Institute of Plant Pathology, Leningrad.

Field testing, for wart resistance, at Chernovtsy is done at high altitudes, where most infested areas are located. Substations at Leningrad, Russian S.F.S.R., Minsk, Byelorussian S.S.R.; and Vilnius, Lithuanian S.S.R. are also concerned with screening varieties for wart reactions.

Hosts affected

Soviet workers have examined the reactions of tomato cultivars and a large number of wild Solanaceous species

¹ Contribution No. 57, Research Station, Agriculture Canada, P.O. Box 7098, St. John's, Newfoundland, A1E 3Y3

Accepted for publication September 20, 1978

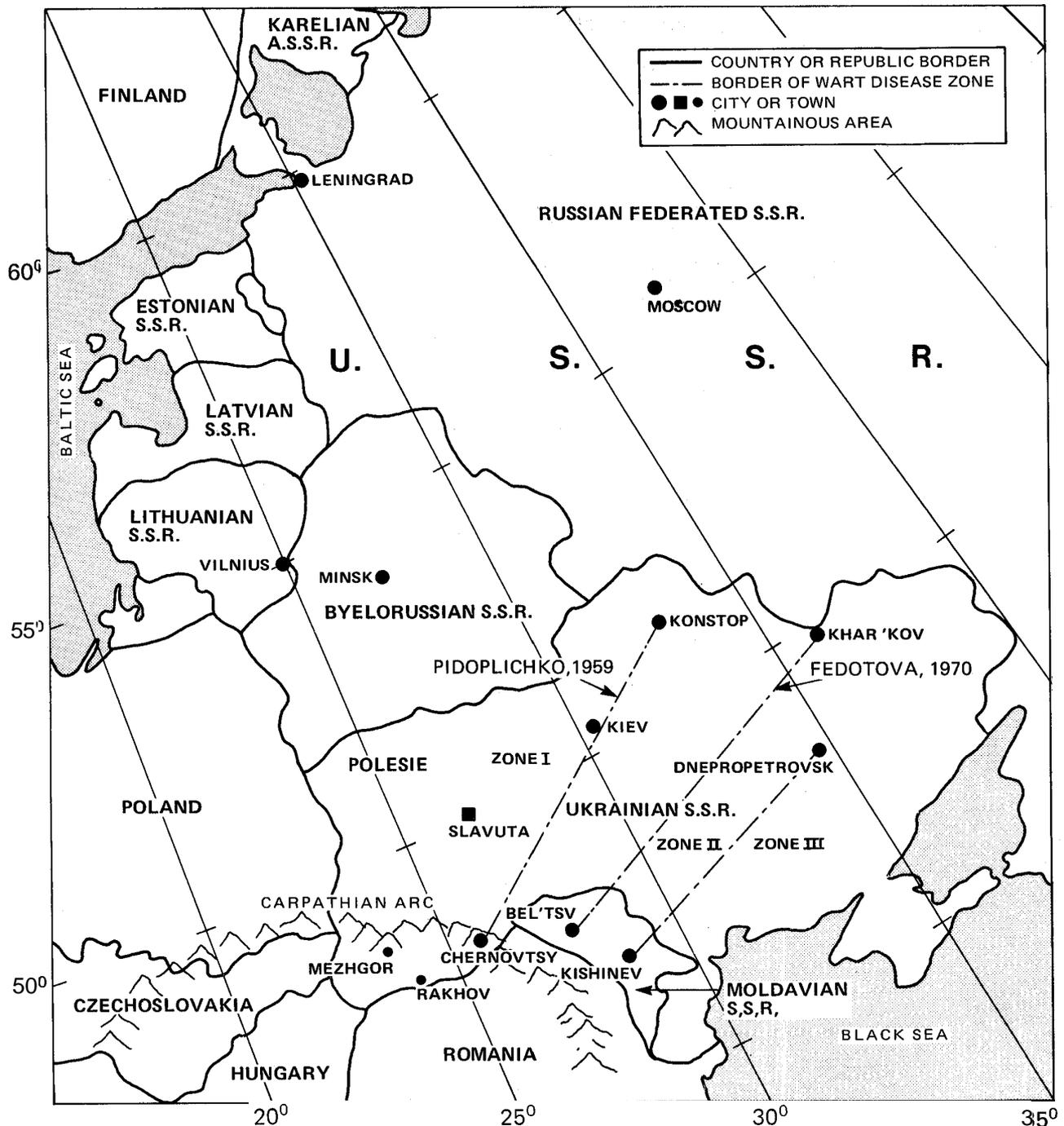


Figure 1. Outline map of Western Russia and adjoining countries, showing locations of major research centers. The sites of original discovery of the common biotype and two other biotypes are shown. Lines connecting cities in Ukrainian and Moldavian S.S.R. delineate zones favourable and unfavourable to establishment of potato wart disease.

to infection by *S. endobioticum* (47). On some species the life cycle of the fungus may not be completed following infection, and visible symptoms may not develop. Potato plants were found to be more affected

by sporangia obtained from other Solanaceous plants than by those obtained from potato plants (22). Several South American potato species were found to be susceptible to the Soviet Union biotypes (19).

Economic importance

The disease was first reported from Slavuta, in West-Central Ukraine, in 1935 (22). Potato wart disease is now known in the Russian Federated, Estonian, Latvian, Lithuanian, Byelorussian and Ukrainian Soviet Socialist Republics. (Fig. 1).

As of January, 1968, more than 16,000 ha of land in Russia were infested. The numbers of infested sites reported were: farms - 274; household plots - 118,472. In the Ukraine, disease foci are found in the Carpathian arc, the Polesie and Lesostep areas. Koretsky (24) estimates that 70% of the potato crops are concentrated in these areas. Household plots comprise nearly 90% of the infested area which represents about 56% of the area under potato crops.

In the Western Ukraine, wart disease was discovered in 84% of the populated areas, and in 98% of the districts. Only in isolated cases was the disease discovered on collective and state farms (40). In Trans-Carpathia, the disease is concentrated mainly in the highlands. In the valley areas the disease is minimal even when infected tubers imported from the infested highland areas are planted.

Pidoplichko (40) reported that all the concentrations of the disease were found northwest of a boundary connecting Chernovtsy, Kiev and Konstop (Fig. 1). Fedotova (9) challenged this distribution which was based on actual field findings. She used soils from different zones of the country, in bio-climatic chambers. She showed that the southern range could be extended much further south with the boundary passing through Bel'tsv and Khar'kov (Fig. 1).

In the Ukrainian foothills and mountainous areas, the losses are extensive in certain years. In areas east of Carpathia, the losses are minimal (40). Although tuber yield is not markedly reduced, in some cases infected tubers rot quickly and thereby infest the soil. In loss situations, 13-84% yield reduction occurs. In the Leningrad region in 1967, the crop loss was said to be as high as 96%. Yield reduction is at times found to be in tuber size rather than in number.

Regulations require the destruction of all plants within a 3-m radius of an infected plant. This increases the losses suffered by the householder in garden plots. Commercially, crops from an area placed under quarantine must be processed. Hence, an immediate reduction in the value of the crop occurs when the disease is discovered in the seed production areas (24).

Etiology

Sporangial studies: Miloslavova et al. (33) and Golik (14) developed an extraction system for isolating zoosporangia* from plant tissue uncontaminated with organic and mineral residues. The wart tissue was crumbled, dried and screened. Sub-samples were collected on 40-60 μm mesh and dried. Sporangia were floated off from the powder on a mixture of CCl_4

and sulfuric ether. Varying the mixture ratio yielded sporangia of different degrees of physiological maturity.

The sporangia were found to undergo maturation during the resting period. Germination followed if the resting period was accompanied by sufficient warmth and moisture (22). Vladimirskaia (54) reported on observations made over 5 years on the dormant character of the fungus. She found that about 3/4 of a sporangial population germinated in the year of production, 1/5 the following year, and the remainder over a long time base. These groups were designated: annual, biennial and perennial. Under laboratory conditions 60-75% germination occurred in the summer. This coincided with the host's tuberization phase. Under natural conditions, wintering retarded germination of the two dormant groups. Among the "annuals", the complete life-cycle took place in 21 days. Prolonged drought retarded germination, rainy summers accelerated germination.

Sporangial viability was determined by plasmolysis in NaCl , Na_2SO_4 , NH_4Cl or NH_4NO_3 , and confirmed by deplasmolysis (22). Mirzabekian (36) attempted to culture tumour tissue *in vitro*. He seeded disinfected tumour pieces (3-5 mm) on various nutrient media at pH 5.2-6, in the dark, at 24-25°C. Best gall stimulation occurred on galls harvested in September and cultured on a medium supplemented with coconut milk, next best on a gall extract or potato shoot extract supplemented medium. Growth ceased after 70-80 days. Summer sporangia were seen but no winter sporangia were found. In a later report, Vinkler and Lipsits (53) cultured wart tissue on White's medium and observed sporangia formation..

Biotype studies: The first biotypes of *S. endobioticum* were discovered in Czechoslovakia and Germany in 1941/2. Cultivars in the U.S.S.R. were tested against the new biotypes from 1957 on (23). Tests were also made against the pathogen obtained from four different Russian climatic zones. While none of the resistant cultivars broke down, degrees of susceptibility were found among control cultivars. Further testing of these differences revealed greatest pathogen activity was displayed from zones in the order Chernovtsy/Minsk > Leningrad/Vilnius. The pathogen from zones of greatest occurrence of the disease were said to be most active.' (10). Biotypes were later discovered in the Carpathian mountains in 1961-62 (55). They were different from the German biotypes discovered in 1941, '50, '51 and '52. Three biotypes were discovered, named according to the sites of discovery: Mezhgorsky, Rakhovskiy and Bukovskiy (22, 55). Naturally, the discovery of biotypes, in Trans-Carpathia, stimulated extensive studies on biotypes. In East and West Germany, between 1942 and 1960, 9 biotypes had become established, in East Germany alone 44 such foci had been determined. There was concern that a similar set of events would unfold in the Ukraine.

Yakovleva (56) reported on a study of factors responsible for the origin of new biotypes. Known factors to date were severe soil infestation, continuous (potato) crop production, and the wide distribution of resistant varieties. The hypothesis was developed that one of the responsible factors is an adaptive change following nutrition of the parasite upon cultivars of varying resistance. In support of this hypothesis it was found that necrosis, deformation, proliferation and the presence of sori and sporangia were observable on some resistant cultivars. On others, sporangia were observed but symptom expression did not occur even at 40 days after inoculation. The pathogen's activity was found to be intensified* after passaging through resistant cultivars and transferring to further resistant and susceptible cultivars.

Morphological differences between Mezhgorsky and Rakhovsky biotypes were noted (55). The summer sori, respectively, contained 2-3 sporangia and 5 sporangia. The common biotype displayed sori containing 5-9 zoosporangia.

The biotypes were found to not extend their ranges beyond the Carpathian zone (58). The Bukovsky biotype was found to be the least aggressive* of the three (57). Detailed work reported by Yakovleva, V.I. (58) indicated that the Carpathian biotypes may consist of "smaller specialized biotypes". An isolate from Leningrad, however, showed evidence of increased pathogenicity" (57). It was noted from the literature that there were indications of the affection of wart resistant potatoes on the Karelian isthmus (44). Work carried out in Byelorussia failed to detect the presence of aggressive* biotypes there (45).

Fedotova and Yakovleva (12) made the first mention in a Russian article of potato wart disease work in Newfoundland, citing Newfoundland at that time as one of the five geographic areas in the world where racial deviation had occurred (the others were: German Federal Republic, German Democratic Republic, Czechoslovakia and U.S.S.R.). Yakovleva (57, 58) dwelt at some length on the Newfoundland findings and concluded that Newfoundland biotypes differed from the Carpathian biotypes.

Epidemiology

Climate: Soviet researchers maintain that the main environmental factors that influence the establishment of wart disease are soil, temperature and precipitation (22). In the U.S.S.R. the fungus is best adapted to a moderate climate, cool and humid summer, and podzol-type soil.

Soil: Gedz (13) examined the influences of mineral salts on infection, in laboratory and field experiments. Mn, Cu and Mo reduced infection. Tarasova (48) observed that B, Zn and Cu stimulated sporangial germination. Tarasova (48) also noted that soil temperature and soil moisture played a large role in decay of wart tumours, and subsequent release of sporangia and germination.

The pH level did not appear to influence germination. The rate of decay in soil types was sandy 7 sandy-loam 7 loam, and pH 7.0 > 3.85.

Fedotova (9) noted that disease suppression occurred in certain soil types. The degree of disease development was felt to be related to soil type (22). Thus, dry and warm region soils fostered a slow development, piedmont and montane region soils fostered intensive development. The most serious cases of the disease were observed on light sandy and less frequently on clay and muddy soils. Acidic heavy textured structureless podzol enhanced the disease, calcareous chestnut chernozem did not.

Temperature: Pidoplichko (40) extensively surveyed soil temperatures across the Ukrainian S.S.R. He found that, based on soil temperatures, boundaries could be established between areas where the disease would and would not be a threat. He could not correlate the disease occurrence with the duration of the frost-free period, as Hartman had done in Pennsylvania. Disease-free areas had soil temperatures which reached 24-25°C at 10 cm soil depth. He found that sporangial viability was lost at summer temperatures > 30°C. Pidoplichko found 'disease-free area temperatures' in Trans-Carpathian valleys which had remained disease free. Using growth chambers, Fedotova (9) simulated meteorological combinations for different areas of the country. Thus, three zones of disease establishment were created (Fig. 1). Parallel field tests in all regions yielded the following results: in the North (Leningrad), 60-70% infection occurred annually; in the South (Zone III), small excrescences were noted in the first year, then in subsequent years no further disease nor viable sporangia were found. Temperature was suggested as the limiting factor, but since soil moisture and certain soil properties were also involved, its role was not clear.

Precipitation: Where soil temperatures were borderline, precipitation was found to be a limiting factor. The effect of soil type here was thought to be very important since it would influence the amount of retained moisture.

The southern boundary for infection was found to coincide with a July average of 75 mm at 20°C. As the majority of cultivars which were cultivated in the Ukraine form tubers in July, the ecological conditions during this period appeared to play decisive roles in disease development (22, 40). Thus season is also an important factor in disease development. Vladimirskaia (54) pointed out that massive germination of sporangia was observed in July. Tarasova (48) found that moisture levels above 90% and below 40% suppressed sporangial germination. Fedotova (9) observed that no disease developed at 50% or less of soil moisture content.

Control

Legislative: Potato wart disease control in the Soviet Union was originally based on the understanding that the disease was dangerous to potato crops wherever

such crops were cultivated. Thus, the fungus became the object of quarantine and restrictions on shipping potatoes were introduced. Pidoplichko (40) concluded that this notion was invalid, whereupon, based on his findings, the Ukraine was divided into three zones: Zone I - a wide distribution of the disease, resistant cultivars allowed only; Zone II - a moderate disease distribution, locally produced resistant cultivars only; Zone III - unfavorable to the disease.

Resistant cultivars could be grown on scheduled plots 3 years after prohibition of tomatoes, root and tuber crops.

Quick detection and elimination of disease foci are the current aims of control measures (22). This is bound up with a system of crop inspection. All farms, seed producing stations, research stations and household plots are subject to inspection (24). Inspections are made at time of flowering of potato crops. Fields are sampled and farm implements, etc., are inspected. Suspected samples are sent via an agronomist to the quarantining agents. All inspections, and actions taken are recorded. If infection is suspected the plot in question is quarantined. If confirmed, all produce from the plot is cooked for cattle food or destroyed in a cesspool. Infected areas are fenced off and posted. A lengthy, detailed form is completed for each confirmation thus describing the circumstances surrounding the infection.

Agrotechnical control, A complex of measures is instituted following positive confirmation. These include: planting resistant cultivars, bare-fallowing, organic fertilization of the soil, and summer planting of potato. In Byelorussia, it has been shown that disease suppression takes place over a five year period when resistant potato cultivars, cabbage, corn, lupin and bare-fallow are used on infested plots. Bare-fallowing for 3-5 years brings about 97% germination of zoosporangia. Organic fertilizers such as hog, cow or poultry manure cause 75% sporangia to germinate.

Chemical control' Following work in Czechoslovakia, it was suggested that dinitro-orthocresol be tried as a control agent (40, 46). Chloropicrin was tried but rejected as too expensive. Over 200 organic and inorganic compounds were tried (22). Nitrphen - a complex mixture of sodium salts, nitration products of alkylphenols, water and a wetting agent - was selected (20). Nitrphen testing was begun in the Leningrad oblast in 1957. It induces granularity and coagulation of sporangial contents. The substance is applied as a solution in May-June to fallowed ground (21).

Calcium cyanamide was introduced as a control chemical since it, too, was found to be effective when harrowed in at the rate of 150 g/m². It is widely available, inexpensive and useful for both commercial and home-garden plots. Carbamide is also used. At the rate of 1.5 kg/m², after 3 years the fungus could no longer be found (49).

Biological control: Small farming allotments and household plots are the most common sources of the disease.

Since the dangers and expense of chemical treatment of these sources are high, work had been initiated on finding a biological control (24, 37, 40). Mirzabekian et al. (37) cultured actinomycetes on potato slices. When diluted with sand, the cultures were incorporated into infested pot mixes. It was found that such treatment reduced disease incidence from 97% to 25%. Treatment of soil and tubers produced the best result. A 3-year treatment program, it was suggested might eliminate infection from infested soils.

Selection for resistance: Selection for resistance is complicated by two factors. On the one hand, Fedotova (8) found that host reactions varied from highly resistant, weakly resistant to susceptible. Thus resistant cultivars used to clean soil may perpetuate the disease since the pathogen can penetrate some resistant varieties and complete its developmental cycle. On the other hand, there is more than one biotype (19). The presence of the Carpathian biotypes - though not a threat to the potato industry - complicates selection work. South American potato species are used as sources of resistance. Interspecific hybridization is used in the breeding work at Chernovtsy (43). Numerous promising cultivars have thus been produced. As a result of a wide study of wart-resistance material at the breeding establishments, Bondarenko (1) concluded there were four levels of resistance to potato wart disease: viz 'high resistant', 'resistant', 'low-resistant', and 'susceptible'.

The study of wart resistance was initiated in 1957, and intensified in 1965 following the discovery of the Carpathian biotypes. Under a more closely developed screening process, cultivars that had appeared resistant under field conditions appeared susceptible under laboratory conditions. Saltykova (43) noted that the percentage of Soviet selections susceptible to the Rakhovsky and Mezhgorsky biotypes was much higher than that percentage susceptible to the common biotype.

Biochemical studies

The aim of the studies into the mechanism of resistance was to place the selection of wart-resistant cultivars on a scientific foundation through the biochemical determination of the nature of resistance (25). Through biochemistry, the mandate was to develop a method of rapid determination of resistance, thus shortening the time taken for testing breeders' selections for resistance.

In the forefront of this research stood D.V. Lipsits who authored or co-authored much of the work on resistance mechanisms, until his death in the early seventies. The three areas of study that were developed were: differences in physiology between resistant and susceptible plants; differences in protein between resistant and susceptible plants, and suppression of tumour development.

Lipsits believed that susceptible cultivars must be able to ensure the vigour of the physiological and biochemical

processes necessary to ensure pathogen development and tumour production. Although all classes of cultivars can be attacked, only in susceptible tissue does the fungus proceed to develop secondary infections (28). Therefore, the cause of resistance, it is stated, is not hypersensitivity but an absence of energetic cellular processes. In Lipsits' opinion, the zoospore affects large protein molecules, in their turn bringing about accelerative changes in the entire cellular metabolism. On this hypothesis, potato wart disease could be diagnosed by estimation of SH-groups, dye absorption, methionine-S³⁵ levels, protease activity, fermentative susceptibility of protein, protein immuno-chemistry, amino acid and polypeptide composition, protoplasmic and cell membrane permeability (27, 28).

Respiratory levels: Lipsits and Eisinger (30) measured respiratory levels in tissues taken from resistant, susceptible and infected plants. They found that infection was accompanied by an elevated respiration which remained high in susceptible sprouts but fell in leaf and root tissues. Lipsits (28) did not find any differences in the oxidation systems of resistant and susceptible cultivars, and no qualitative changes were found in fermentative respiration. Lipsits also examined differences in polyphenol levels in these plants. He noted that polyphenols accumulated only in susceptible cultivar tissue. He concluded that the polyphenols contributed to disease development.

Auxins: Reingard and Pashkar' (41) examined the differences between resistant and susceptible plants in respect of their auxin content. Increases in auxins were principally noted in the periphery of tumour tissue. More auxin-type substances were found in the free than in the bound fraction. Pashkar' (39) later demonstrated that specific physiologically active substances produced by roots were found essential to tumour development.

Sulfhydryl groups: Lipsits (26) analyzed the differences in number and localization of SH-groups in resistant and susceptible sprout tissue. The quantity of SH-groups in infected sprouts increased before the first visible signs of infection. Also, differences were noted in quantity of SH-groups in leaf and tuber tissue between resistant and susceptible plants. Young tumour tissue was most active in labelled P and S uptake. The proteins of the susceptible cultivars had an enhanced ability to incorporate labelled methionine. This was felt to be an excellent indicator of the degree of immunity.

Peroxidase activity: Kadyrmatov (18) studied the changes brought about in the peroxidase complex of resistant and susceptible sprouts at 2, 3 and 6 days after inoculation with *S. endobioticum*. Negligible changes were noted in the complex from susceptible plants. This finding was held to confirm Lipsits' thesis that an intensive metabolism of the host plant is necessary for attack by *S. endobioticum*.

Free radicals: Oxidation process levels are reflected in the changes of free radical levels (31). Healthy, infected,

resistant and susceptible potato sprouts were analyzed for free radicals. Free radical levels were observed in the order: infected susceptible sprouts > susceptible sprouts > resistant sprouts. Dolyagin et al. (4, 6) reported a much higher level of free radical states in the epidermis of resistant tubers than in that of susceptible ones.

Proteins: Okaneneko and Bershtein (38) studied the differences in hydrolyzed alkali-water-soluble and 'residual' protein in tubers and tumours. In susceptible tubers, alkali-soluble protein fraction displayed quantitative increases in amino-acid components, and in peripheral tumour tissue quantitative and qualitative increases in 'residual' protein components were found. The new protein material was regarded as a plant reaction to *S. endobioticum*. Lipsits (26) regarded the new or modified proteins in tumour tissue as active factors influencing the course of infection.

Fedotova et al. (11) extracted the proteins of the fungus, noting characteristic differences between specimens taken from different geographic zones, and between different biotypes. Golik et al. (15) then compared the fungal proteins with host plant proteins. A high level of protein similarity existed with susceptible plants, and as the degree of resistance increased there was a decrease in protein fractions common to incitant and host.

Timchuk and Lipsits (50) demonstrated differences in nucleoproteins between resistant and susceptible shoot tissue. Nucleoproteins from the latter could be enzymatically cleaved more rapidly than those from the former tissue. Timchuk and Lipsits (51) suggested that the stronger protein reaction in susceptible tissue could be accounted for in terms of both accumulation of proteins as well as changes in molecular structure, and could both explain the nature of resistance and aid in developing an accelerated method for resistance diagnosis.

Dolyagin et al. (5) extracted DNA from normal and tumorous potato tissue. They found a significantly higher DNA content in the tumorous tissue, with an absorbance at 320 mμ not present in the normal tissue extract. Reshetova et al. (42) also examined nucleoproteins. They established that DNA and RNA were more easily extracted from wart-susceptible cultivar shoots, but a greater amount was extracted from resistant shoots. They suggested that there existed a greater rigidity of the nucleic acid-protein bond in resistant than in susceptible tissues, and that this rigidity could explain the passive background activity of resistant tissues. Thus, activation of metabolism (Lipsits' thesis) after infection of resistant tissue may be made more difficult.

Chemotherapy: Work on disease physiology logically lead to the use of tumour suppressants. Lipsits (26) blocked the balance of SH-groups in susceptible tissue in the laboratory, and prevented tumorigenesis. Field tests, however, failed since the suppressants became phytotoxic. Propylgallate was shown to be an effective tumour suppressant. Lipsits et al. (32) also demonstrated the effectiveness of ionol (2,6-di-ter-butyl-

4-methyl-phenol) dispersed in Tween-60. Later, it was found that Tween-60 alone would suppress tumour formation. Miloslavova et al. (34) examined the effects of gossypol, ionol, Tween-60 and polyethyleneglycols of different molecular weights. These substances did not prevent pathogen penetration nor its development, but manifested anti-tumoural properties only. It was thought that the presence of degenerate tumour cells sensitized the tissue to the anti-tumoural agents.

Present research needs

The present needs of research into potato wart disease by Soviet researchers are summarized by Ephremenko (7)

1. Collaboration of specialists from other countries having the disease, to solve problems of quarantine and plant protection.
2. Standardization of evaluation of resistance of selection material, since the main method of control is breeding for resistance.
3. Development of effective methods of soil disinfection.
4. Improvement of soil samplings methods, assessment of levels of sporangia in soil, and methods of viability determination.
5. Development of trustworthy, indirect methods of diagnosing wart disease through the study of the nature of immunity and host/pathogen relationships.

Conclusion

The potato wart disease story in U.S.S.R., to date, is a fascinating unfolding of a wide-ranging but concentrated effort to come to terms with the world's number one potato disease. The story obviously is far from finished and we look forward to learning more about the attempts to control the disease, and the discoveries yet to be made in its etiology and epidemiology, and in the biology of the causal agent.

Aside from the concerted and organized effort to manage the disease, two points are particularly striking since they appear to be so disparate from our experience with the (disease and its) causal agent. The first is that Soviet workers can apparently clean their land within five years, whereas we find the organism capable of remaining dormant for more than 30 years. The second is that in the Soviet Union, it is observed that three-quarters of a population of the agent germinate in the first year of production, but we calculate that the half-life of the propagule under Newfoundland conditions cannot be less than 14 months, and 10% of a population is still present after four years. Thus, under Soviet soil conditions, the rate of sporangial loss appears to be about twice that for Newfoundland.

Acknowledgements

I wish to gratefully acknowledge the helpful suggestions made by Kenneth G. Proudfoot, Agriculture Canada Research Station, St. John's, Newfoundland, and Norman F. Haard, Department of Bio-

chemistry, Memorial University of Newfoundland, St. John's, Newfoundland, in development of the text.

Literature cited

1. Bondareko, E.E. 1973. Results of a study of the degree of potato resistance to the canker agent *Synchytrium endobioticum* (Schilb.) Perc. (in Russian, English summary). Trudy VN11 Zashchity rastenii 36, 95-97.
2. Brooks, J.L., J.B. Given, J.F. Baniecki and R.J. Young. 1974. Eradication of potato wart in West Virginia. Plant Dis. Rept. 58: 291-292.
3. Craig, F.W. and M.E. Gallegly. 1964. Potato wart persists in West Virginia. Plant Dis. Rept. 48: 468-469.
4. Dolyagin, A.B., M.S. Zshedek, K.W. Zschedek and D.V. Lipsits. 1970. Free radicals in shoots of potato under normal conditions and potato wart (*Synchytrium endobioticum*) (Schilb.) Perc. and action of substances suppressing tumour development (in Russian). Mikol. i Fitopatol. 4: 229-234.
5. Dolyagin, A.B., K.E. Krugliakova and D.V. Lipsits. 1974. DNA isolated from healthy potato tissues and from tumours caused by *Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 8(4): 345-347.
6. Dolyagin, A.B., D.V. Lipsits and K.E. Krugliakova. 1975. Investigation of the level of free radical states in the rind of potato sorts differing as to cancer resistance (in Russian, English summary). Izv. Akad. Nauk S.S.S.R. Ser. Biol. 6, 921-923.
7. Ephremenko, T. 1977. Wart disease (*Synchytrium endobioticum* (Schilb.) Perc.) and its control in the Soviet Union. pp 73-74 in 1st. Rep. Working Party on Potato Wart Disease. EPPO Publications, Series Cn. 50, Paris, France.
8. Fedotova, T.I. 1959. On the biology of *Synchytrium endobioticum* (Schilb.) Perc. and the immunity of potato varieties to wart disease (in Russian, English summary). Sbornik CSAZV Ros-tlinna Vyroba 32(6): 165-174.
9. Fedotova, T.I. 1970. Zones of potential distribution of the potato wart pathogen (in Russian, English summary). Byul. VN11 Zashchity rastenii 15, 15-17.
10. Fedotova, T.I., E.F. Karaseva and M.I. Rakovich. 1957. Differences in the activity of the causative agent of potato canker (in Russian). Doklady Vses. Akad. Sel'skok. Nauk im. V.I. Lenina 22(9): 31-33.
11. Fedotova, T.I., B.B.-O. Gromova and I.V. Golik. 1972. The electrophoretic and immunochemical properties of the proteins in *Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 6(1): 68-70.
12. Fedotova, T.I. and V.I. Yakovleva. 1964. Characteristic of the biotypes of the potato wart fungus in the Carpathian mountainous zone of U.S.S.R. (in Russian). pp. 78-97 In T.I. Fedotova, ed. Rak kartofelya i mery bor'by s nim. Kolos Publishers, Leningrad, U.S.S.R.
13. Gedz, S.M. 1957. Effects of Manganese, Boron, Molybdenum and Copper micro-elements on the rise of canker immunity in potatoes (in Russian, English summary), Rept. Acad. Sci. Ukraine 6, 605-608.
14. Golik, I.V. 1973. Method of isolation and fractionation of zoosporangia of the potato wart causative agent, *Synchytrium endobioticum* (Schilb.) Perc. (in Russian, English summary). Trudy VN11 Zashchity rastenii. 36, 112-116.
15. Golik, I.V., B.B.-O. Gromova and T.I. Fedotova. 1973. The immuno-chemical similarity of the proteins of *Synchytrium endobioticum* (Schilb.) Perc. and the host plant (in Russian, English summary). Trudy VN11 Zashchity rastenii. 36, 107-111.
16. Hampson, M.C. and K.G. Proudfoot. 1974. Potato wart disease, its introduction to North America, distribution and control problems in Newfoundland. FAO Plant Prot. Bull 22(3): 53-64.
17. Hulea, Ana. 1967. A visit to the station for potato canker in the Ukrainian Soviet Socialist Republic (in Rumanian). Prob. Agr. 19(10): 88-90.
18. Kadyrmatov, I.N. 1973. The change of the peroxidase complex in the sprouts of potato tubers after their inoculation with the

- pathogen of *Synchytrium endobioticum* (Schilb.) Perc. (in Russian, English summary). Trudy VN11 Zashchity rastenii. 36, 102-106.
19. Kameraz, A.Ya. 1968. Development of potato varieties which are resistant to cancer, p. 20-22. In, The World Potato Collection in the service of socialist agricultural production (in Russian). Trudy Po Prikladnoi Botanike, Genetike 1 seleksii 39(1): 241-260.
 20. Kharitonova, Z.M., A.S. Volovik and V.P. Tarasova. 1964. Commercial testing of nitrphen in the fight against the causal agent of potato canker (in Russian). pp. 152-159 In T.I. Fedotova, ed. Rak kartofelya i mery bor'by s nim. Kolos Publishers, Leningrad, U.S.S.R.
 21. Kharitonova, M., V.P. Tarasova and E.P. Larina. 1969. Nitrphen against potato canker (in Russian). Zashchita rastenii 7: 19.
 22. Kharitonova, Z.M. and V.P. Tarasova. 1971. Potato Wart Disease (in Russian). Kolos Publishers, Leningrad, USSR. 46 p.
 23. Khizhnyak, P.A. 1957. The biology of the pathogen of potato canker (in Russian). Zashchity rastenii ot vreditel'ei i boleznei 2(4): 41-42.
 24. Koretsky, P.M. 1970. Losses from potato canker on household plots in regions of frequent occurrence of this disease in the Ukraine (in Russian). Mikol. i Fitopatol. 4(4): 366-369.
 25. Lipsits, D.V. 1955. Biochemistry and potato canker (in Russian). Priroda (2): 62-63.
 26. Lipsits, D.V. 1962. Studies on the biochemistry of the immunity of potatoes to canker (in Russian). Biokhimiya Plodov i ovoshchei. Akad. nauk S.S.S.R., 7: 60-84.
 27. Lipsits, D.V. 1963. Incorporation of S³⁵ methionine into the proteins of varieties of potatoes susceptible or immune to wart disease. Doklady Biol. Sci. Sec. 146(1): 945-948.
 28. Lipsits, D.V. 1965. Die Biochemie der Kartoffel resistenz gegen den Krebs erregger *Synchytrium endobioticum* (Schilb.) Perc. (English summary), pp. 265-281. In Biochemische Probleme der Kranken Pflanze. Symposium Deut. Akad. Landwirt. Berlin. 1964.
 29. Lipsits, D.V. 1972. The possibilities of biochemical diagnosis of disease resistance in potato plants. (in Russian, English summary). Sel'skokhoziaistvennaya biologiya 7(6): 886-894.
 30. Lipsits, D.V. and F.Z. Eisinger. 1958. Respiration of potato plant tissues in connection with resistance of the plant to tumour-producing agents. Soviet Plant Physiol. 5(2): 172-174.
 31. Lipsits, D.V., E.K. Kruglyakova, V.M. Chibrikov, and A.B. Dolyagin. 1971. The concentration level of free radicals in the sprouts of various varieties of potatoes differing in their resistance to cancer (in Russian). Akad. Nauk S.S.S.R. Doklady 196(4): 975-976.
 32. Lipsits, D.V., K.E. Kruglyakova and A.B. Dolyagin. 1971. Action of ionol and Tween-60 on potato wart. Doklady Biochem. Sec. 198(1-6): 221-223.
 33. Miloslavova, T.A., E.K. Reshetova, A.S. Volovik and A.B. Dolyagin. 1973. A method isolating zoosporengia of *Synchytrium endobioticum* (Schilb.) Perc. from potato warts (in Russian). Mikol. i Fitopatol. 7(5): 459-461.
 34. Miloslavova, T.A., M.S. Zhedek, A.S. Volovik, K.Ye. Kruglyakova and A.B. Dolyagin. 1975. The effect of wart-inhibiting substances on the susceptibility of potatoes to the fungus (*Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 9(2): 113-116.
 35. Mirzabekian, R.O. 1959. Antibiotic substances of actinomycetes origin affecting phytopathogenic micro-organisms (in Russian, English summary). Akad. Nauk S.S.S.R. Izv. Ser. Biol. 24(1): 103-110.
 36. Mirzabekian, R.O. 1969. Tissue culture from the wart callus of the potato (in Russian). Mikol. i Fitopatol. 3(4): 337-342.
 37. Mirzabekian, R.O., N.V. Sinitina and O.G. Belyakova. 1961. Elaboration of a biological method for the control of potato wart (in Russian). Agrobiologiya 4(130): 566-572.
 38. Okanenko, A.S. and B. Bershtein. 1960. Proteins of cancerous growth in the use of potato infected with *Synchytrium endobioticum*. Akad. Nauk S.S.S.R. Doklady 134(3): 180-182.
 39. Pashkar', S.I. 1975. On the role of the root system in the growth of wart calluses on potato. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 403-404.
 40. Pidoplichko, N.M. 1959. Control of potato canker in the Ukrainian S.S.R. (in Russian, English summary). Rostlinna Vyroba 32(6): 47-58.
 41. Reingard, T.A. and S.I. Pashkar. 1958. Participation of growth substances of the auxin type information of tumours on potato plants. Soviet Plant Physiol. 5: 512-517.
 42. Reshetova, E.K., A.S. Volovik and A.B. Dolyagin. 1975. Nucleo-proteinases isolated from the sprouts of both *Synchytrium endobioticum* (Schilb.) Perc. resistant and susceptible potato cultivars (in Russian). Mikol. i Fitopatol. 9(5): 438-440.
 43. Saltykova, L.P. 1973. Varietal differences in potato resistance to ordinary and aggressive agents causing potato wart disease (in Russian, English summary). Trudy VN11 Zashchity rastenii. 36, 63-71.
 44. Saltykova, L.P. and V.I. Yakovleva. 1976. Soviet and Czechoslovak races of a causal agent of potato cancer, *Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 10: 503-507.
 45. Samersova, V.A., M.I. Danchenko. 1975. Detection of aggressive biotypes of potato wart in Byelorussia. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 399-401.
 46. Sedivy, J. 1975. In memory of Javoslav Zakopal/*Synchytrium endobioticum* Phytopathologist (in Russian). Ochr. Rostl. 11(3): 169-172.
 47. Sharikov, K.E. 1975. On canker infection of different representatives of the Solanaceae. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 398-399.
 48. Tarasova, V.P. 1969. Role of the environment in the decay of warts and germination of zoosporengia of *S. endobioticum* on Potato. (in Russian, English summary). Byull. VN11 Zashchity rastenii. 1(13): 42-44.
 49. Tarasova, V.P. and V.K. Beskorovainy. 1973. A complex method for controlling potato wart (in Russian). Zashchita rastenii 11: 45.
 50. Timchuk, K.S. and D.V. Lipsits. 1970. Histo- and cytochemical studies of inter-relations between the causal organism of potato wart disease *Synchytrium endobioticum* (Schilb.) Perc. and host plant. I. (in Russian). Mikol. i Fitopatol. 4(1): 34-43.
 51. Timchuk, K.S. and D.V. Lipsits. 1973. Cyto- and histo-chemical characteristics of the protein-nuclein complex of healthy potatoes and those infected by the cancer-inducing *Synchytrium endobioticum* (Schilb.) Perc. sprouts of varieties of potato contrasting in resistance (in Russian, English summary). Trudy VN11 Zashchity rastenii. 36, 117-122.
 52. Trochinskii, N., G. Karytko. Agricultural measures as means for eradication of potato wart. (in Russian). Zashch. rastenii ot Vred. i Boleznei 6: 49-50.
 53. Vinkler, G.N., D.V. Lipsits. 1975. Culture of pieces of wart callus detached from potato on nutrient media. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 401-403.
 54. Vladimirskaia, N.N. 1960. Physiological processes in *Synchytrium endobioticum* (Schilb.) Perc. in the dormant zoosporengia state (in Russian). Botan. Zhur. 45(1-6): 97-104.
 55. Yakovleva, N.N. 1970. Some morphological peculiarities of aggressive biotypes of potato wart pathogens (in Russian). Mikol. i Fitopatol. 4: 267-268.
 56. Yakovleva, V.I. 1961. Variability of the potato wart pathogen (in Russian). Vestnik Sel'skokhoziaistvenii Nauki. 12: 72-75.
 57. Yakovleva, V.I. 1973. Racial composition of the agent causing potato wart disease in the U.S.S.R. (in Russian, English summary). Trudy VN11 Zashchity rastenii. 36, 78-86.
 58. Yakovleva, V.I. 1975. Races of the causal agent of potato cancer and their virulence (in Russian). Mikol. i Fitopatol. 9(5): 421-452.