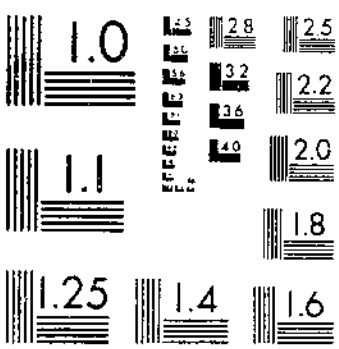
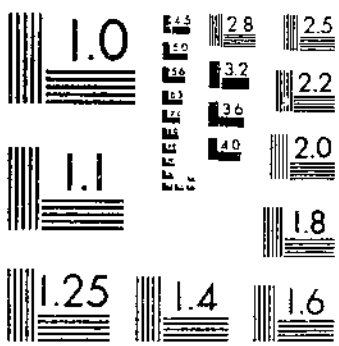
START



MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS-1963-A



MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS-1963-A

TECHNICAL BULLETIN No. 56 MARCH, 1928

UNITED STATES DEPARTMENT OF AGRICULTURE
WASHINGTON, D. C.

FACTORS OF SPREAD AND REPRESSION IN POTATO WART

By FREEMAN WEISS, *Associate Pathologist*, and PHILIP BRIERLEY, *Assistant Pathologist, Office of Vegetable and Forage Diseases, Bureau of Plant Industry*

CONTENTS

INTRODUCTION

Recent investigations in England, Germany, and the United States have considerably extended the knowledge of the appropriate conditions for infection by the organism, *Synchytrium endobioticum* (Schilb.) Perc., causing potato wart, and have indicated the possibility under certain conditions of destroying it in infested soil. The fact that this disease has been held well in check in the United States by the methods adopted against it in the several centers of infection has led to diminished interest in its further investigation. The undesirability of maintaining infectious material except within the areas covered by quarantines (and even there only within restricted experimental gardens, since extermination is sought as the ultimate goal) has induced the abandonment for the time being of the solution of several interesting but no longer urgent remaining problems. Certain data have been accumulated pertaining to questions which the writers need not further attempt to answer, but as some of these facts bear on the official policy (*4*, *8*)[1] adopted with respect to potato wart, it is thought best to publish such results as seem to warrant tentative conclusions.

[1] Reference is made by (*italic*) numbers in parentheses to "Literature cited," p. 13.

75069—28——1

MEANS OF DISSEMINATION OF POTATO WART

The predominant rôle of seed potatoes in the dissemination of this disease has been generally recognized, though usually no distinction has been made between tubers which are actually infected, but bear only small, dry, inconspicuous warts and tubers which have escaped infection but bear externally particles of infested soil. The distinction is not a practical one except in relation to the passive dissemination of spores on tubers of immune varieties, since both types of carriage may be presumed to occur on susceptible tubers. In this way quarantines can guard against the dissemination of soil that is known to be infested, but in the absence of the culture of susceptible varieties an index of contamination is lacking.

In the work herein described attempts were made to determine the actual importance of spore dissemination in the spread of the disease when immune tubers from an infested source were planted in clean soil either simultaneously with or followed by the planting of a susceptible tuber. The pot experiments were not sufficiently numerous to demonstrate this means of spread, and a field experiment of the same nature miscarried because of a destructive drought. In another experiment contaminated tubers were washed, and the wash water, in which sporangia of Synchytrium could be detected microscopically, was poured over pots of clean soil bearing susceptible plants, but this attempt also failed.

That viable spores may be carried on the surface of tubers is indicated by the following experiment: At harvest 20 tubers were selected which bore small, nearly indistinguishable warts and 20 others from the same hills in which no overgrowths could be detected even with a lens. As the soil was fairly dry at the time of digging, the tubers came out clean without conspicuously large soil particles adhering even in the eyes. These tubers later were planted in pots of steamed soil in the greenhouse, and observations were made on the development of wart infections. The results are shown in Table 1.

TABLE 1.—*Experiment showing that viable spores of potato wart may be carried on the surface of tubers*

Seed tubers	Number of plants	Number of infections	
		Primary	Secondary
Visibly infected	20	11	4
Not visibly infected	20	9	1

Primary infections were those which developed at the eyes of the seed piece; secondary, those on stolons or young tubers. Most of the primary infections originated in the ring of buds at the base of a shoot; that is, the primary sprout was not directly infected, even though it came from an infected eye.

Other experiments showed that when infected tubers were planted in either soil or sphagnum they gave rise almost invariably to infected plants if the temperature averaged about 15° C. and the pots were watered frequently. If the temperature ranged from 22° to

30° C., or if water was given in only about the minimum quantity to permit sprouting, normal growth occurred and healthy shoots developed even from warty eyes. If the entire bud cluster of an eye was involved in the overgrowth, usually no further development occurred, but instances of renewal of growth over the entire surface of the wart were observed.

Other sources of dissemination of the disease are believed to be (1) manure derived from animals fed on warted potatoes or contaminated with infected plant débris and (2) soil which may be variously carried. Actual spread of wart by both these means has been observed in the infested area around Freeland, Pa. (*4*). Carriage in manure was demonstrated by feeding a goose, a pigeon, a rabbit, and a cow on infected potatoes and collecting the manure with as little risk of contamination as possible. This was added to pots of clean soil planted to susceptible potatoes. Infection developed only from pigeon and goose manure, but failure in the other cases may have been due to unfavorable experimental conditions. The reluctance of these animals to feed upon warty tissue unmixed with bran or other vegetables greatly discounts the importance of this factor in spreading the disease. Malthouse (*7*) had already demonstrated the carriage of viable wart sporangia in the manure of swine.

A practical experiment on the importance of infected soil in establishing the disease may be cited. In 1922 need developed for an experimental plot more completely under control than the infested gardens which had previously been rented from the householders in the mining villages around Freeland, Pa. A site approximately 1¼ acres in size was selected in an old pasture which had been partly under cultivation but was not known to be infested. About 10 cubic yards of surface soil was taken from two gardens known to be heavily infested and spread over an area about 80 by 120 feet in extent as a covering equivalent to 0.34 of an inch if uniformly distributed. In the same spring about half the area was planted to susceptible potatoes. When these were harvested warted plants were found in most of the susceptible varieties, but only in the late-maturing ones was the proportion of infection as much as 5 to 10 per cent.

All infected material was left in the field, and the planting of a large proportion of susceptible varieties continued for three more years. Not until the harvest of 1925 was the soil sufficiently contaminated in an area about 40 by 100 feet to assure reliable variety tests, in which half or more of all susceptible plants become warted; that is about the proportion resulting from planting contaminated seed.

The slow distribution of the pathogene through soil doubtless explains the failure of wart to spread from the infested mining villages to the surrounding agricultural area, where the culture of susceptible varieties is carried on under permit and inspection. In the 15 years which have elapsed since wart was presumably introduced into this region it is unlikely that infested soil has not been transported on implements, shoes, vegetable containers, etc., to some of the surrounding farms. Up to the present time, however, no infections have been found in the fields of this farming area. On the other hand, the movement of potatoes, whether for table use or seed, is exclusively from the farms into the quarantined villages.

VIABILITY OF THE PATHOGENE IN THE DORMANT STATE

VERTICAL DISTRIBUTION OF THE PATHOGENE IN THE SOIL

General considerations lead to the view that resting spores of the pathogene should be found to the depth to which the soil is tilled. Samples of soil were taken from a heavily infested plot by 2-inch intervals down to 14 inches deep. Part of each sample was examined after it had been shaken up in water and centrifuged fractionally, by which means most of the sporangia were recovered in the successive top layers. The presence of wart sporangia was demonstrated at all depths to and including the 6 to 8 inch layer but not below. Simultaneous tests were made, soil samples being used to inoculate susceptible potatoes in plots of clean oil. Infection developed only from the 6 to 8 inch layer and at higher levels.

DURATION OF INFECTIOUSNESS OF SOIL

The observation has been repeatedly made in the writers' work that contaminated soil contained in pots and allowed to stand in practically air-dry conditions remains infectious at least 15 months. Several attempts have been made to determine the duration of infectiousness in field soil under natural conditions. Thus plots were laid off at Freeland to receive such surface treatments as bare fallow, turf, cultivation of nonsolanaceous crops, and constant culture of immunes. Subjected to unusual vicissitudes in a situation remote from the regular experiment station and with the added handicap of being cultivated by foreigners to whom the experimental work was incomprehensible, these plots suffered various disturbances and were abandoned. A subsequent experiment in which a series of barrels of infested soil was partly sunk in the ground in a fenced inclosure is still in progress. Half the series is turfed over and half is bare fallowed. Each year potatoes are planted in one barrel of each set. Thus far soil that was grassed over 10 months after it last bore a warty crop and has lain in turf for two years since has been found to be infectious, but a similar plot which was bare fallowed for two years bore a healthy crop in the third year. This, of course, is a far shorter period than that reported by Schaffnit (*9*), who found that an originally infested field grown to sod gave a warty crop in nine successive years when a portion of it was planted to potatoes.

In 1920 a large sample of infested soil was stored in a covered crock. In 1925 it was used to inoculate five pots of potatoes, all of which developed wart.

VIABILITY OF RESTING SPORANGIA IN ARTIFICIAL CONDITIONS

Although the resting sporangia are apparently incapable of germination as soon as morphological development is complete, there is considerable evidence that they may be induced to germinate in a period much less than the usual dormant interval in nature. Esmarch (*2*) considers a state of physiological ripeness a necessary antecedent to germination.[2] If the sporangia are in such a state, they germinate

[2] In a paper received after the manuscript for this bulletin was completed Esmarch (*2*) has shown that a small proportion of sporangia germinate in the year they are formed after a rest period of a few weeks, but others may remain dormant for at least three years.

more or less promptly when favorable conditions supervene, irrespective of the presence of an appropriate host, though certain substances present in loam and humus are said to stimulate germination. Such sporangia are quickly excluded from any rôle in long persistence of soil contamination. On the other hand, physiologically unripe sporangia do not respond to favorable conditions for germination within months of exposure. The factors involved in physiological ripening are unknown, but Köhler (*5*) states that neither intermittent nor continuous cold has an accelerating effect.

If fresh warty growths removed from a potato are allowed to dry for about two weeks and then used to inoculate growing plants, new infections develop in about 40 days, irrespective of whether the temperature during drying was cool or moderate or was periodically below freezing. Nor does continued storage beyond two weeks affect the incubation period. An example is shown in Table 2.

TABLE 2.—*Effect of storage conditions on length of the incubation period following inoculation with dry wart material*

Period of storage	Length of incubation period after storage under three indicated conditions		
	At room temperature	At 10° C.	Under periodic freezing and thawing
Days	*Days*	*Days*	*Days*
13	43	43	47
	47	47	47
24	35	35	57
	57	57	57
39	42	42	42
	42	----------	42
57	38	----------	----------
	38	----------	----------

When dry wart material is stored for much longer periods it eventually suffers complete loss of viability. In some cases viability is lost within two years; in others it is retained at least five years. For example, a considerable quantity of such material was preserved in stoppered vials in 1922, the intention being to use it in annual tests of viability as long as any infection resulted from inoculations with it. This material is still infectious, nearly six years after it was produced. According to Collins (*1*), Wilson of Aberdeen carried out a similar test and concluded that viability was lost within six years, as infection occurred after four years but not after six.

The difference in viability shown by different samples of preserved material can not be attributed to the storage conditions. Moist storage would of course be inimical to survival, as was determined with one batch of material that was kept wet for three months. On the other hand, in another test some sporangia were viable after being wet for a month and exposed to alternate freezing and thawing.

The writers' evidence up to the present time indicates that sporangia in dry wart material stored in artificial conditions survive about as long as those in their natural environment, the soil.

THE TOMATO AS A HOST FOR POTATO WART

Ever since the tomato was shown to be a host of *Synchytrium endobioticum* (*6*) the question of its importance in perpetuating wart infestation in areas under quarantine, despite the prescribed culture of immune potatoes, has been of official concern. In 1922, results of field tests carried on for four years in the infested area at Freeland, Pa., were published (*11*). At that time 28 varieties had been successfully infected with wart, but a number of others had escaped infection in one or more trials. It was noted that tomatoes generally were less readily infected than potatoes under similar environmental conditions, and that stimulating adventitious budding from the basal part of the stem by hilling up the soil greatly favored infection. Field tests were continued for two subsequent years, in both of which weather conditions were propitious for wart infection, and several varieties which had previously escaped developed wart. Only 12 of 65 accessible varieties remained apparently resistant. These 12 varieties were subjected to heavy inoculation under the conditions that had been found to be most favorable for infection of tomatoes in the greenhouse, with the result that all proved to be susceptible.

It has been shown that infection of the potato depends on conditions favorable to its growth (*10*), and that at a temperature of 15° C., for instance, a higher proportion of infection occurs than at 22° to 25°, in agreement with the effect of temperature on the host. With the tomato, however, the higher temperature promotes more rapid growth. Nevertheless, infection experiments with about 20 varieties known to be susceptible were all negative when the inoculations were made in the greenhouse at the end of May, and the temperature averaged 27° or above for the next two months. In these tests plants which had been rooted in 4-inch pots were set in the bottom of 8-inch pots filled with infested soil, thus exposing the basal three or four nodes to infection. When a similar test with the John Baer variety was carried out during the cooler part of the year the results were as shown in Table 3.

TABLE 3.—*Results of the exposure of John Baer tomato plants in 8-inch pots to potato-wart infection*

Average temperature at night	Treatment	Plants		Susceptible parts	
		Exposed	Infected	Exposed	Infected
22° C.	Hilled	5	2	19	3
	Not hilled	5	1	4	2
13° C.	Hilled	5	5	16	11
	Not hilled	5	2	5	2

It is evident that infection of tomatoes, as of potatoes, is favored by a temperature near 15° C. and is restricted by higher temperatures; also that hilling is favorable to infection but not indispensable. In a test in which tomato plants were distributed among the householders of one of the villages to grow after their own fashion in gardens having a previous history of wart, infection occurred several times.

TABLE 4.—*Susceptibility of tomato varieties to potato wart*

[The tests in 1925 were made in the greenhouse. Tests in other years were made in the field]

Variety	Number of plants										Average infection (per cent)
	1920		1922		1923		1924		1925		
	Exposed	Infected	Exposed	Infected	Exposed	Infected	Exposed	Infected	Exposed	Infected	
Acme	5	0	10	1	14	0	19	4			10
Atlantic Prize							18	5			28
Beauty	4	4	17	0							19
Bolgiano Extreme Early							20	2			10
Bolgiano Grand							21	6			29
Bonny Best	5	0	5	1							9
Brimmer	6	0					20	7			27
Buckeye State							17	5			29
Burbank											
Carter Sunrise	6	6									100
Chalk Early Jewel			12	0			19	7			22
Comet			18	1							6
Coreless							23	15			65
Crackerjack					16	5					31
Crimson Cushion			6	0			18	6			25
Duke of York	6	5			12	5					55
Dwarf Champion	6	1									17
Dwarf Stone	6	0					22	6			21
Earlinna	12	4	14	2							23
Early Detroit	4	4	14	2							33
Early Michigan	6	1			27	2					9
Enormous							18	2			11
Favorite			5	0			20	0	10	2	6
Freedom							19	10			53
Fregmore							21	17			81
Globe	6	2	26	0							6
Golden Queen			3	0			14	2			12
Greater Baltimore					14	4					29
Hummer							20	10			50
Ignotum							20	8			40
John Baer	4	4	13	2					10	7	48
June Pink			17	0			4	1			5
Long Keeper									12	7	58
Lorillard			7	2	15	0					9
Magnus	5	1			16	8					43
Mansfield Tree									10	1	10
Matchless	4	2			28	10					40
Maule New Imperial							18	1			6
Mikado (Turner Hybrid)					13	0	19	3			9
Mississippi Girl									10	5	50
New Century					14	2					14
Norton			7	0			19	9			35
Optimus							19	9			47
Paragon									10	5	50
Perfection	6	0					20	3			12
Ponderosa	6	2									33
Prize Taker							19	9	8	7	69
Red Cherry			14	1	6	1					10
Redfield Beauty									10	6	60
Red Head	6	4									67
Red Peach							22	13			59
Red Pear							9	3			33
Red Rock	5	0			14	6					32
Royal Red									10	0	00
San Jose Canner	3	3									100
Stone	6	6	6	0	14	7					50
Success	6	0	7	0							
Superb Salad			8	0					10	5	28
Ten Ton							22	7			32
Trophy			6	3	15	0					14
Trucker Favorite	5	4									80
Yellow Cherry			3	1							33
Yellow Peach							17	15			88
Yellow Pear							16	5			31
Yellow Plum							19	10			53

Although the overgrowths on tomatoes are usually small, they occasionally attain a size that might be significant in perpetuating soil contamination (pl. 1, D). As in the potato, all parts of the shoot

system are susceptible, the infection of buds, stem internodes, leaves, and flowers having been observed; but in the experience of the writers root infection does not occur (pl. 1, C). This conclusion is based not only on the negative results with soil-grown plants but on the absence of root infection when young roots in active growth are exposed in solution cultures to inoculation from bits of fresh potato warts, which resulted in infection of shoot buds (pl. 1, A, B).

A summary of the tomato-infection experiments by years is given in Table 4. Owing to varying weather conditions in the different years, the tests are not strictly comparable. Thus in 1921 a warm, dry year, only one among many susceptible varieties tested developed wart, so that no data are shown for that year. On the other hand, in 1924 infection was as general as it is likely to be in field-grown tomatoes.

In these tests no consistently resistant variety has been found, but this does not necessarily imply that none exists. However, the absence of even one immune variety among so large a collection of types makes it appear unlikely that wart-resistant tomatoes, comparable with immune potatoes, will be found. There is not even convincing evidence that differences exist in the degree of susceptibility, although the disparity in the proportion of infected plants among the different varieties, when all were grown in the same heavily infested garden (as in 1924), where the potato control plants were uniformly infected, makes it probable that not all tomatoes are susceptible to the same degree. No recommendation can be made as to what varieties might safely be permitted under a partial quarantine; but it would appear desirable to select those showing the least infection, such as Bonny Best, for further trial in the infested area.

SUSCEPTIBILITY OF RESTING SPORES TO HEAT AND DISINFECTANTS

During the time that the field experiments to exterminate wart infection within the quarantined area by soil sterilization were in progress, laboratory tests were begun to determine the lethal temperatures for the pathogene under various conditions and its susceptibility to some of the standard fungicides. Great difficulty was experienced in obtaining anything like uniform behavior from different lots of wart material used as the source of resting sporangia in heat and chemical treatments, and the experiments covered several years. In 1926 Glynne (*3*) published her researches along the same line. She devised a staining technic which greatly expedited the determination of the effect of any particular form of exposure on the sporangia. The validity of the method was confirmed by inoculating growing plants, hence the interpretation of the results of treatment was confidently based on the staining test. In the tests by the writers the criterion of survival or injury was, respectively, infection or failure when the treated material was used to inoculate growing plants. This method is admittedly open to the objection that infection depends partly on the condition of the inoculated plants and on environmental factors, but as an effort was made to have these as favorable as possible, the results are thought to indicate in the main the functional viability of the sporangia after treatment.

POTATO WART ON POTATO AND TOMATO PLANTS

A, Tomato and potato grown together in solution culture, infected by inoculating with pieces of fresh wart.
B, Portion of A, enlarged to show infection of an adventitious bud (indicated by arrow) on a tomato stem. The roots are free from infection.
C, Tomato shoot showing foliar infection.
D, Tomato stem showing large wart developing from an adventitious bud.

In a few particulars the results here given differ from those of Glynne, so it is considered desirable to present them, although in general her results are more consistent and complete.

EXPERIMENTAL METHODS IN TESTING RESISTANCE TO HEAT

The wart material used in these tests was collected for the most part in the field in Pennsylvania, and was dried, ground, and stored dry until required. In the earliest trials each inoculated plant received 1 c. c. of the test material; in later experiments, 2 to 5 c. c. In every case like quantities of untreated material were applied to two or more similar plants as a check on the infectiousness of the inoculum and the effectiveness of the test conditions. Susceptible varieties of potatoes were grown in pots of clean soil in the greenhouse until suitable young stolons and tubers developed. The soil was then washed away until these parts were exposed, the wart material was applied directly as a paste or dust, and the pot was refilled with steamed soil. Usually the soil was covered with sphagnum to keep the surface moist. Inoculations were made immediately after the exposure in the case of all treatments with moist heat, and as soon as the longest treatment was complete in the tests with dry heat.

The test material was exposed to the given conditions in open Petri dishes, test tubes, or flasks. Temperature control in the ovens was poor. The ranges recorded are based on continuous observations in the shorter tests and on hourly readings during the day for the longer periods. The thermometer was placed adjacent to the test material when it was exposed dry and was inserted in the liquid in the wet treatments. For the test in a saturated atmosphere at 60° C., spores were spread in a thin layer in Petri dishes supported over water and surrounded with wet filter paper within a closed glass vessel. In this and the flowing steam tests of up to five minutes' duration, the treated material appeared dry at the end of the test.

The results of the tests of the effect of heat on resting sporangia in dried wart material are shown in Table 5. Two general tendencies of importance are evident. The resting sporangia are very resistant to dry heat, withstanding it 8 to 12 hours at 100° C. and 4 to 7 days at 60°, with apparently undiminished viability. On the other hand, the dry sporangia are readily killed by moist heat. As short an exposure as 2½ minutes at 100° is uniformly fatal, and apparently a slightly longer exposure at 70° is likewise fatal. At 60° the spores withstood exposure to moist heat for 1½ hours in one test, but in other trials, with equally heavy infection of check plants, the resistance was less marked. Glynne (*3*) concluded that sporangia immersed in water are probably all dead after a 5-minute exposure at 90°, or an 8-hour exposure at 60°. In so far as they parallel hers, the writers' results indicate that the statements of Glynne are very conservative. For instance, the writers found no infection resulting from material treated for 2 or more hours at 60°, although in several tests the control plants became heavily infected. Glynne reported that sporangia exposed dry withstood a 15-minute exposure at 90°, a 1½-hour exposure at 80°, and a 20-hour exposure at 75°, the greatest interval tested in each case. The writers' data regarding exposures at 100° and 60° indicate that sporangia may survive exposures at considerably higher temperatures.

TABLE 5.—*Effect of dry heat and moist heat on the resting sporangia in dried wart material, as indicated by subsequent infectiousness to potato varieties*

[Data in number of plants expressed in fractional form: Denominator=plants inoculated; numerator=plants infected]

Temperature, potato variety, and date of inoculation	Date of harvest	Temperature range (°C.)	Duration of treatment and degree of subsequent infectiousness														Check (untreated)
			10 days	7 days	6 days	5 days	4 days	3 days	2 days	36 hours	24 hours	16 hours	12 hours	10 hours	8 hours	6 hours	4 hours	
DRY HEAT																		
Dry heat at 100° C.:																		
American Giant, Jan. 16, 1923	Mar. 14, 1923	97 -104	0/2	------	------	0/2	0/2	0/2	0/2	0/2	0/2	------	0/2	------	1/2	------	1/2	7/8
Australian Blue, Apr. 14, 1923	(?)	98 -103	------	------	------	------	------	------	------	------	0/2	------	0/2	------	0/2	------	1/2	0/3
American Giant, Dec. 5, 1923	Jan. 11, 1924	96 -99	------	------	------	------	------	------	------	------	------	------	0/5	0/5	0/5	0/5	------	1/4
Early Ohio, Feb. 7, 1924	Apr. 4, 1924	95 -105	------	------	------	------	------	------	------	------	------	------	3/5	2/5	3/5	4/5	------	5/5
Russet Rural, May 7, 1924	June 19, 1924	97 -106	------	------	------	------	------	------	------	0/5	0/5	------	------	------	------	------	------	0/10
Triumph, Dec. 3, 1924	Jan. 20, 1925	98 -100	------	------	------	------	------	------	------	------	0/5	------	0/5	------	------	------	------	4/5
Triumph, Jan. 28, 1925	Mar. 21, 1925	95 -100	------	------	------	------	------	------	------	------	------	0/5	0/5	1/5	------	------	------	5/5
Dry heat at 70° C.:																		
Russet Rural, May 8, 1924	June 19, 1924	64 -73	------	------	------	------	------	0/5	0/5	------	------	------	------	------	------	------	------	0/5
Dry heat at 60° C.:																		
Australian Blue, Apr. 23, 1923	(?)	57 -62	------	------	------	0/2	------	------	0/2	------	------	------	------	------	------	------	------	1/2
American Giant, Nov. 15, 1923	Jan. 10, 1924	57 -65	------	------	------	------	------	------	2/5	------	2/5	------	1/5	------	------	------	------	4/5
Early Ohio, Jan. 23, 1924	Mar. 25, 1924	60(?)	------	------	------	0/5	1/5	1/5	------	------	------	------	------	------	------	------	------	10/10
Triumph, Dec. 6, 1924	Jan. 20, 1925	52 -60	------	------	------	0/5	0/5	------	------	------	------	------	------	------	------	------	------	4/5
Triumph, Feb. 2, 1925	Mar. 21, 1925	58 -60	------	------	------	4/5	5/5	------	------	------	------	------	------	------	------	------	------	5/5
Triumph, Jan. 9, 1926	Feb. 26, 1926	58.5-63.5	------	3/3	3/3	3/3	3/3	------	------	------	------	------	------	------	------	------	------	6/6

Temperature, potato variety, and date of inoculation	Date of harvest	Temperature range °C	Duration of treatment and degree of subsequent infectiousness												Check (untreated)
			4 hours	3 hours	2 hours	90 min.	60 min.	45 min.	30 min.	20 min.	15 min.	10 min.	5 min.	2½ min.	1 min.	
MOIST HEAT																
Steam heat, 15 pounds pressure:																
American Giant, Jan. 6, 1923	Mar. 4, 1923										0/2	0/2	0/2	0/2		7/8
Australian Blue, Apr. 5, 1923	(?)												0/5	0/5		2/5
American Giant, Nov. 16, 1923	Jan. 10, 1924														[1] 1/5	4/5
Flowing steam at 100° C.:																
American Giant, Jan. 8, 1923	Mar. 14, 1923	100	0/2	0/2	0/2		0/2									7/8
Australian Blue, Apr. 5, 1923		100	0/2	0/2	0/5		0/5		0/2							2/5
American Giant, Oct. 23, 1923	Dec. 5, 1923	100					0/5		0/5	0/5						8/14
American Giant, Nov. 15, 1923	Jan. 10, 1924	100										0/5	0/5			8/14
Early Ohio, Jan. 29, 1924	Mar. 25, 1924	100								0/5		0/5	0/5	0/5		5/5
Triumph, Dec. 3, 1924	Jan. 20, 1925	100										0/5	0/5			4/5
Petoskey, Apr. 4, 1925	May 5, 1925	100											0/5	0/5		5/5
Immersed in water at 70° C.:																
Russet Rural, May 7, 1924	June 19, 1924	67–68.5				0/5	0/5		0/5							0/5
Russet Rural, Feb. 3, 1925	Apr. 4, 1925	65–70				0/5	0/5		0/5		0/5					5/5
Triumph, Jan. 8, 1926	Feb. 26, 1926	68–72									0/3	0/3	0/3			6/6
Immersed in water at 60° C.:																
American Giant, Jan. 15, 1923	Mar. 14, 1923	55–70	0/2		0/2											7/8
Australian Blue, Mar. 21, 1923	(?)	60–70	0/5		0/5											0/2
American Giant, Oct. 22, 1923	Dec. 5, 1923	55–69			0/5		0/5		3/5		1/5					8/14
Early Ohio, Jan. 18, 1924	Mar. 18, 1924	60(?)				3/5	4/5	5/5								5/5
Russet Rural, Apr. 26, 1924	June 19, 1924	55		0/5	0/5	0/5										0/5
Triumph, Jan. 12, 1925	Feb. 21, 1925	59–61			0/5	0/5										6/10
Triumph, Jan. 9, 1926	Feb. 26, 1926	57–62			0/3	0/3	0/3									6/6
Presoaked in cold water 24 hours, then immersed at 60° C.:																
American Giant, Apr. 3, 1925	May 20, 1925	51–61.5					0/5		0/5		0/5	0/5	0/5			3/4
Dry sporangia in saturated atmosphere at 60° C.:																
Early Ohio, Jan. 15, 1924	Mar. 18, 1924				5/5	0/5	5/5									5/5
Russet Rural, Apr. 26, 1924	June 19, 1924	55			0/5	0/5										0/5
Triumph, Jan. 12, 1925	Feb. 21, 1925	59–61			4/5	2/5	3/5									6/10

[1] Maximum 14 pounds; 4 minutes to reach 14 pounds, 1 minute at 14, 2 minutes to fall to normal pressure. Wart moist at top only, dry at bottom at end of test.

RESULTS OF TESTS WITH SEED DISINFECTANTS

Tests of the resistance of dry wart sporangia to mercuric chloride and formaldehyde are summarized in Table 6. The wart material was of the same character as that used in the heat tests. In earlier tests inoculation was effected by dipping whole tubers in thin glue, then rolling them in the dry inoculum. After the tubers had stood over night a fairly thick layer of sporangia still adhered to them. They were next carefully placed in the disinfectant solution for the desired interval and then planted. Much of the wart material soaked off in the solutions during treatment, but enough clung to the eyes to give a satisfactory percentage of infection. Wherever this method was used the check lots were treated in like manner but soaked only in tap water. It was thought that this treatment might afford some of the sporangia a protecting coat of glue and keep the fungicide from penetrating them. Hence in the last tests the dry sporangia were immersed directly in the test solutions and applied without being rinsed to susceptible stolon buds and young tubers. Infection still resulted in nearly all cases.

The data in Table 6 show that resting sporangia are very resistant to both the common-seed disinfectants. They withstood treatment in formaldehyde, 1 part commercial to 96 parts water, for 1 hour, the longest period of test. Infection was fully as severe as in the untreated check. The sporangia also survived immersion in 1:1,000 mercuric chloride for 2 and 3 hours, the longest periods tested. In the last test, April 3, 1925, 25 c. c. of dry sporangia was treated in 100 c. c. of 1:1,000 mercuric chloride for 3 hours, and the disinfectant solution was poured over the stolons at the time of inoculation. Infection resulted in four of five plants.

TABLE 6.—*Effect of mercuric chloride and formaldehyde on the resting sporangia in dried wart material, as indicated by subsequent infectiousness to potato varieties*

[In the test of Apr. 3, 1925, the sporangia were immersed directly in the test solutions; in other tests thin glue was used]

Disinfectant, potato variety, and date of inoculation	Date of harvest	Strength of disinfectant	Duration of treatment and subsequent infectiousness													
			3 hours		2 hours		1½ hours		1 hour		30 minutes		20 minutes		Check	
			Exposed	Infected	Exposed	Infected	Exposed	Infected	Exposed	Infected	Exposed	Infected	Exposed	Infected	Exposed	Infected
Mercuric chloride:																
Triumph, Nov. 8, 1923	Feb. 1, 1923	1:1,000	---	---	---	---	---	---	5	0	---	---	---	---	5	4
Sir Walter Raleigh, Jan. 18, 1923	Apr. 4, 1924	---do---	---	---	---	---	---	---	5	2	---	---	---	---	5	5
Russet Rural, Apr. 5, 1924	June 19, 1924	---do---	---	---	5	0	5	0	---	---	---	---	---	---	5	0
Triumph, Oct. 31, 1924	Jan. 20, 1925	---do---	---	---	5	2	5	1	---	---	---	---	---	---	5	4
American Giant, Apr. 3, 1925	May 20, 1925	---do---	5	4	5	4	5	5	---	---	---	---	---	---	10	9
Do	---do---	1:1,000	---	---	---	---	---	---	5	3	5	5	5	3	5	3
Formaldehyde:																
Triumph, Nov. 8, 1923	Feb. 1, 1924	1:128	---	---	---	---	---	---	5	4	---	---	---	---	5	4
Sir Walter Raleigh, Jan. 18, 1924	Apr. 4, 1924	1:96	---	---	---	---	---	---	5	5	---	---	---	---	5	5
Russet Rural, Mar. 8, 1924	May 29, 1924	1:128	---	---	---	---	---	---	5	0	---	---	---	---	5	0
Do	---do---	1:96	---	---	---	---	---	---	5	0	---	---	---	---	5	0

[1] Following presoaking for 24 hours in tap water.

SUMMARY

Viable sporangia of the potato-wart fungus (*Synchytrium endobioticum*) may be disseminated on soil adhering to tubers as ordinarily handled in commerce, as well as by actually infected tubers. The concomitant transfer of host and parasite is much more effective in distributing the disease than the dissemination of sporangia alone; hence the quarantine on movement of infected or contaminated seed potatoes in eastern Pennsylvania has effectively prevented the spread of potato wart into other agricultural areas.

Infection of the tomato is favored by the same conditions that make for copious infection of potatoes—temperature near 15° to 18° C., frequent wetting, and hilling soil about the stalks. No resistant variety has been found among the 65 varieties tested.

Resting sporangia were unable to infect susceptible potatoes after exposure to moist heat for 2½ minutes at 100° C., or for 2 hours at 60°. They resisted dry heat for 10 to 12 hours at 100°, and for 6 to 7 days at 60°.

Resting sporangia adherent to tubers were not destroyed by ordinary seed disinfection as applied to potatoes.

LITERATURE CITED

(1) COLLINS, E. J.
1921. THE PROBLEM OF THE INHERITANCE OF IMMUNITY TO WART DISEASE IN THE POTATO. Gard. Chron. (III) 70: 260, 271, 290, 314, 326.

(2) ESMARCH, F.
1926–27. UNTERSUCHUNGEN ZUR BIOLOGIE DES KARTOFFELKREBSES. I–II. Angew. Bot. 8: 102–135; 9: 88–124.

(3) GLYNNE, M. D.
1926. THE VIABILITY OF THE WINTER SPORANGIUM OF SYNCHYTRIUM ENDOBIOTICUM (SCHILB.) PERC., THE ORGANISM CAUSING WART DISEASE IN POTATO. Ann. Appl. Biol. 13: 19–36.

(4) HARTMAN, R. E., and MCCUBBIN, W. A.
1924. POTATO WART. Pa. Dept. Agr. Bul. 394, 28 p., illus.

(5) KÖHLER, E.
1924. BEITRÄGE ZUR KEIMUNGSPHYSIOLOGIE DER DAUERSPORANGIEN DES KARTOFFELKREBSERREGERS. Arb. Biol. Reichanst. Land.-u. Forstw. 13: 369–381.

(6) KUNKEL, L. O.
1920. A NEW HOST FOR THE POTATO WART DISEASE. U. S. Dept. Agr. Circ. 111: 17–19, illus.

(7) MALTHOUSE, G. T.
1910. WART DISEASE OF POTATOES. SYNCHYTRIUM ENDOBIOTICUM, PERC. 40 p., illus. Shrewsbury.

(8) MARLATT, C. L.
1924. POTATO WART—STATEMENT OF POLICY. FEBRUARY 7, 1924. Potato Assoc. Amer. Proc. (1923) 10: 38–39.

(9) SCHAFFNIT, E.
1920. VERSUCHE ZUR BEKÄMPFUNG DES KARTOFFELKREBSES IM JAHRE 1918–19. Ztschr. Pflanzenkrank. 30: 59–67.

(10) WEISS, F.
1925. THE CONDITIONS OF INFECTION IN POTATO WART. Amer. Jour. Bot. 12: 413–443, illus.

(11) ——— and ORTON, C. R.
1923. THE VARIETAL AND SPECIES HOSTS OF SYNCHYTRIUM ENDOBIOTICUM. U. S. Dept. Agr. Bul. 1156: 1–16, illus.

ORGANIZATION OF THE UNITED STATES DEPARTMENT OF AGRICULTURE

March 6, 1928

Secretary of Agriculture	W. M. JARDINE.
Assistant Secretary	R. W. DUNLAP.
Director of Scientific Work	A. F. WOODS.
Director of Regulatory Work	WALTER G. CAMPBELL.
Director of Extension	C. W. WARBURTON.
Director of Personnel and Business Administration	W. W. STOCKBERGER.
Director of Information	NELSON ANTRIM CRAWFORD.
Solicitor	R. W. WILLIAMS.
Weather Bureau	CHARLES F. MARVIN, *Chief.*
Bureau of Animal Industry	JOHN R. MOHLER, *Chief.*
Bureau of Dairy Industry	L. A. ROGERS, *Acting Chief.*
Bureau of Plant Industry	WILLIAM A. TAYLOR, *Chief.*
Forest Service	W. B. GREELEY, *Chief.*
Bureau of Chemistry and Soils	H. G. KNIGHT, *Chief.*
Bureau of Entomology	C. L. MARLATT, *Chief.*
Bureau of Biological Survey	PAUL G. REDINGTON, *Chief.*
Bureau of Public Roads	THOMAS H. MACDONALD, *Chief.*
Bureau of Agricultural Economics	LLOYD S. TENNY, *Chief.*
Bureau of Home Economics	LOUISE STANLEY, *Chief.*
Federal Horticultural Board	C. L. MARLATT, *Chairman.*
Grain Futures Administration	J. W. T. DUVEL, *Chief.*
Food, Drug, and Insecticide Administration	WALTER G. CAMPBELL, *Director of Regulatory Work, in Charge.*
Office of Experiment Stations	E. W. ALLEN, *Chief.*
Office of Cooperative Extension Work	C. B. SMITH, *Chief.*
Library	CLARIBEL R. BARNETT, *Librarian.*

This bulletin is a contribution from

Bureau of Plant Industry	WILLIAM A. TAYLOR, *Chief.*
Office of Vegetable and Forage Diseases	K. F. KELLERMAN, *Associate Chief of Bureau, in Charge.*

ADDITIONAL COPIES
OF THIS PUBLICATION MAY BE PROCURED FROM
THE SUPERINTENDENT OF DOCUMENTS
U.S.GOVERNMENT PRINTING OFFICE
WASHINGTON, D. C.
AT
5 CENTS PER COPY
▽

END