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Blackleg Disease of Potatoes in Minnesota

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BLACKLEG DISEASE OF POTATOES IN MINNESOTA

J. G. LEACH

INTRODUCTION

Blackleg is one of the major diseases of the potato in Minnesota. It causes an average annual loss of approximately 2 per cent of the crop. In badly affected fields the loss may be as great as 50 per cent or more and it is not uncommon to find fields in which from 10 to 15 per cent of the plants have been killed by the disease. In the majority of the fields, however, the losses are seldom greater than 5 per cent of the crop. In addition to reduced yields, further losses may be caused by decay of the tubers in storage or transit. This is especially true where good storage conditions are not possible.

The disease may be transmitted from one crop to the next through infected seed tubers. In fact, some investigators have considered this to be the only means of transmission. The question of the possible sources of infection in the new crop has, however, never been satisfactorily answered. Most European workers, especially those in Germany, where the disease has been extensively investigated, are of the opinion that infection may take place from the soil, but the work of several American investigators has led to the opposite conclusion. With the development of the practice of certifying seed potatoes, the question becomes one of prime importance. If the disease were transmitted only by means of seed tubers, the planting of certified seed would offer a simple means of control. But this method is not always successful in practice. In the production and use of certified seed potatoes, observations indicated the probability of other sources of infection. Very severe outbreaks of the disease are frequently found in fields planted with tubers produced in fields known to be free from the disease. In other cases two fields planted with the same seed stock, known to have been produced in fields free from blackleg, varied so greatly in the incidence of the disease as to be inconsistent with the theory of seed transmission. To throw some light on this problem, an investigation of the disease was begun in 1923. Some of the results obtained have already been reported elsewhere, but sufficient information has been obtained to justify this general treatment of the disease.

Blackleg is a bacterial disease causing a soft rot of potato stems and tubers. It probably was first recognized as such in Germany, where it was commonly known as "'Schwarzbeinigkeit" or more rarely as "Stengelfäule." Frank (8), working in Germany in 1889, appears to have been the first to publish an accurate description of the disease. A few years later (1902) van Hall (43) reported it from Holland. In a

series of papers appearing from 1902 to 1906, Appel (1, 2, 3) published further descriptions of the disease as it occurred in Germany, together with the results of his extensive investigations of the cause. By that time it had been reported from most of the important potato growing countries of Europe. Jones (14), who had previously observed the disease in Europe, was the first to recognize and report it in the United States. He discovered it in Vermont in 1905 and in reporting it referred to it as "blackleg." This name has become practically universal in North America altho other names have been advocated i.e., "'Black-stem disease" (27), "Black-stalk rot," "Basal stem-rot," "Bacterial black rot" (40). Morse (26, 27, 28) was the first to study the disease extensively in this country. Harrison (11), in 1906, published a detailed account of the disease as it occurred in Canada. Since this time blackleg has been the subject of many investigations in all parts of the world, and it is known to occur to some extent almost wherever potatoes are grown. Jennison (12), in 1923, Kotila and Coons (17), in 1925, and Stapp (41), in 1928, published results of extensive investigations of the disease.

ECONOMIC IMPORTANCE OF BLACKLEG

The destructiveness of blackleg varies greatly in different regions and in different years. Still greater variations occur in the amount of the disease in different individual fields in the same year, and in the same region. Obviously, it is difficult to estimate accurately the amount of loss due to a disease that occurs in such a sporadic manner. Nevertheless, observations in a large number of localities over a period of years should give a fair estimate of the loss caused. The following table is compiled from the records of the Plant Disease Survey of the United States Department of Agriculture.

Table 1
Estimated Reduction in Yield of Potatoes in Minnesota and in the United States Due to Blackleg During the 10-Year Period 1918 to 1927, Inclusive

Year	Minnesota		United States	
	Per cent	Bushels (000 omitted)	Per cent	Bushels (000 omitted)
1918	3.0	1,013	0.40	2,001
1919	1.0	305	0.50	2,205
1920	1.5	486	0.40	2,380
1921	1.0	326	0.30	1,330
1922	2.0	1,023	0.60	3,603
1923	1.0	435	0.80	3,851
1924	2.0	1,152	1.01	5,705
1925	3.0	776	0.86	2,803
1926	3.0	1,161	0.70	2,999
1927	3.0	1,242	1.10	5,390
Average	2.05	792	0.67	3,226

An average annual loss of 0.67 per cent or even 2 per cent does not appear very great and, were the losses evenly distributed over the entire acreage, the disease doubtless would not be considered of great significance. But this is not the case. As mentioned above, the losses may range from 10 to 50 per cent or higher in individual cases. Such heavy losses, altho occurring more or less sporadically, can not easily be ignored.

The amount of loss in storage due to decay from blackleg is also difficult to estimate. In the first place it is not easy to determine the cause of the decay on account of the many saprophytic fungi and bacteria rapidly following the true pathogen. Moreover, as will be pointed out later, the decay of potato tubers by bacteria depends so greatly on storage conditions that there is no necessary relation between the amount of the disease in the field and the amount of decay of tubers in storage.

According to Paine and Haensaeler (32), in England, where potatoes are commonly stored in pits, or "clamps," the losses due to blackleg decay may be very great.

Geographic Distribution of Blackleg

Blackleg has been reported from time to time from practically all of the important potato growing countries of the world, and probably it would be safe to conclude that its occurrence is coexistant with potato culture. In the United States, blackleg appears consistently to be more prevalent in the northern part of the country. Figure 1 illustrates graphically the prevalence of blackleg in the United States, based on the reports of the Plant Disease Survey of the United States Department of Agriculture.

Symptoms of Blackleg

Blackleg is a bacterial soft rot. Any part of the potato plant may be affected, altho in nature it is generally the seed pieces, the stems, and the tubers that are rotted by bacteria. The destruction of these parts stunts the plant and eventually causes it to wilt and die (Figs. 2 and 3). Infection virtually always occurs through the seed pieces, which are usually decayed throughout before the bacteria spread upward into the stem or out into the stolons to the newly formed tubers (Fig. 5). Seed pieces destroyed by blackleg may vary in appearance. In the majority of cases the pathogenic bacteria are accompanied or closely followed by many saprophytic bacteria and fungi which reduce the seed piece to a slimy foul-smelling mass in which the larvae of insects, particularly the seed-corn maggot (*Hylemyia cilicrura*), may frequently be found (Fig. 4).

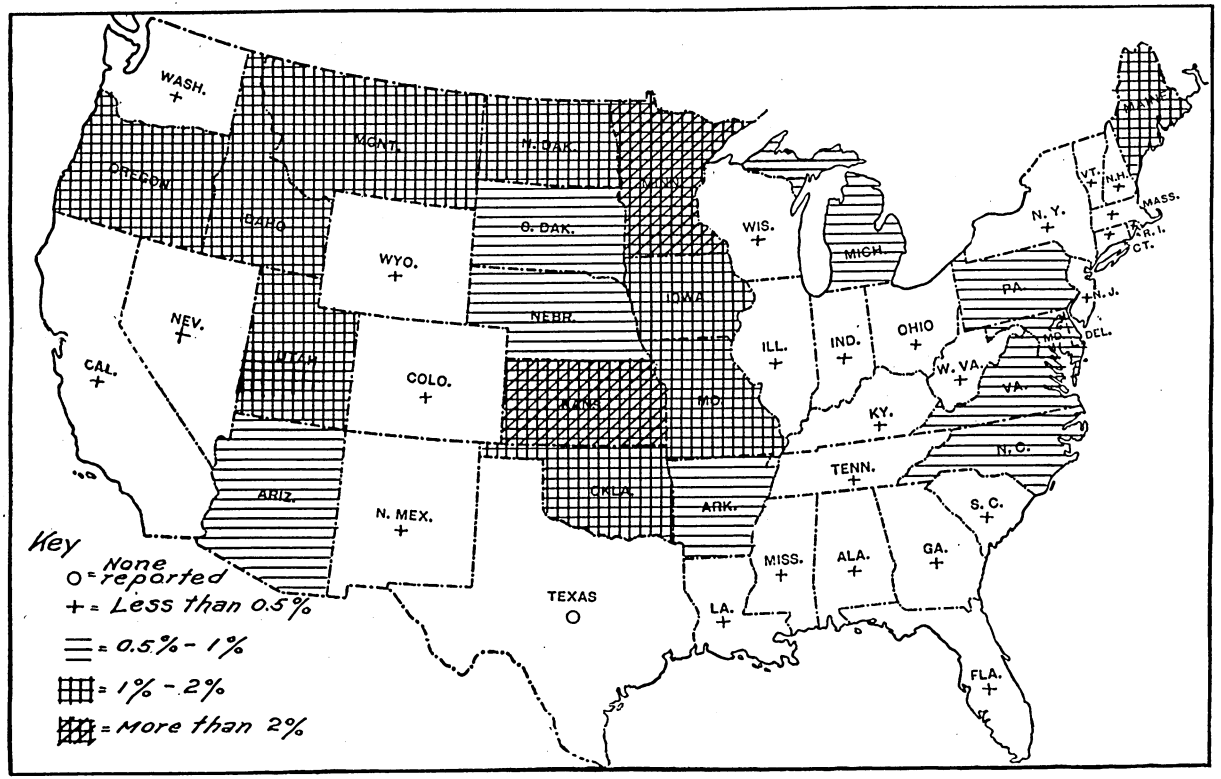


Fig. 1. Distribution and Prevalence of Potato Blackleg in the United States

Based on the records of the Plant Disease Survey of the United States Department of Agriculture. Average for the 10-year period, 1918 to 1927, inclusive.

On the other hand, especially late in the growing season, the seed piece may be destroyed by a watery translucent decay with no unpleasant odor. In such cases relatively few saprophytes are present.

The stems and leaves frequently show effects of the disease before the bacteria have advanced far into the stem. Apparently some toxic substance is carried up into the vascular system in advance of the bacteria. The importance of this toxic substance in the production of the disease has been pointed out by Kotila and Coons (17). The growth of the plant is checked, and the leaves turn yellow from the margins inward, curl upward along the margins, and lose their natural tugor. Branching is inhibited and the lateral stems tend to stand

Fig. 2. Potato Plant Affected with Blackleg (Left) Beside a Healthy Plant (Right) of the Same Age

Note the small size of the diseased plant, the lack of branching, and the upward rolling of the leaves.

more erect than those of normal plants (Fig. 2). The yellow color of the affected plants and their characteristic habit of growth make it relatively easy to detect them in a field among normal plants. As the decay progresses up the stem, the plant wilts and finally dies.

The decay, advancing into the stem, destroys the parenchyma and pith but has little effect on the lignified tissues. The bacteria may enter the vascular bundles, however, and in dry weather when the parenchyma decay is checked, they frequently advance some distance beyond the

affected portions of the parenchyma. In such cases the bundles are distinctly browned for several inches in advance of the limits of bacterial growth. In other cases the bacteria spread very rapidly in the pith and may extend through the major branches before attacking the parenchyma outside of the vascular ring.

The color of the decayed portions of the stems is usually dark brown or black. The intense black color that is sometimes present is not a characteristic symptom of the disease as is sometimes stated. In

Fig. 3. A Potato Plant Affected with Blackleg, Advanced Stage
Note the rolled and wilted leaves. The yellow color of such plants contrasts strongly with the normal green of healthy plants.

many cases the decay is a light brown or greenish brown. The latter type of decay is observed more frequently in wet weather because all types of decay on potato stems usually turn black on drying. It has also been observed in artificial inoculations that the color reaction differs somewhat with different varieties of potato. Different strains of the pathogen also differ in the amount of pigment produced under similar environmental conditions.

Plants affected with blackleg are easily pulled out of the soil because of the decay of the lower part of the stem (Fig. 5a). This enables one readily to distinguish blackleg from other diseases that may cause a wilt of the plant. Severe cases of stem injury caused by *Rhizoctonia solani* are sometimes mistaken for blackleg. The two can readily be distinguished by the more moist decay of blackleg, the reddish brown color of the Rhizoctania lesions, and the presence of Rhizoctonia mycelium, which often can be seen with the naked eye.

Frequently only one stem of a plant is affected while the remaining ones are healthy, but usually when one stem is infected the others eventually succumb.

Diseased plants usually appear at random over the field, showing no definite centers of infection. On the other hand, diseased plants are sometimes found in groups. This may or may not be the result of chance.

When a plant becomes diseased after tubers have been formed the decay frequently spreads out through the stolons and into the new tubers. In wet and heavy soils these may be completely destroyed before har-

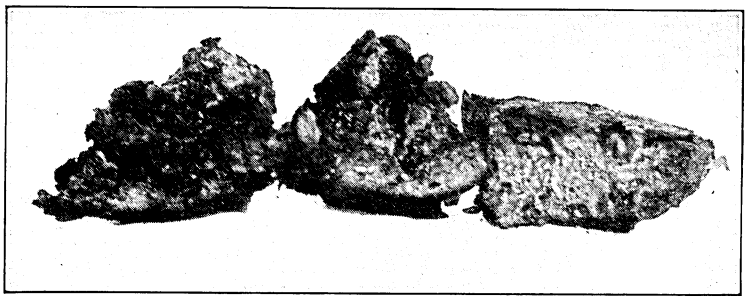


Fig. 4. Three Decayed Seed Pieces Taken from Plants Affected with Blackleg Note the tunnels made by the larvae of the seed-corn maggot.

vest. In light or very dry soils a small portion of the stem end of the tubers may be decayed (Fig. 6a) or only a browning of the vascular bundle results (Fig. 6b).

The symptoms manifested by tubers affected with blackleg also vary extensively. The decayed tissues may present a variety of colors varying through white, gray, brown, and black, depending chiefly upon the oxygen supply. The flesh of tubers rotted in the soil as a result of stolon infection may remain nearly as white as normal until cut open and exposed to the air, when it will rapidly turn brown or black, frequently passing through an intermediate stage of pink. These color changes are quite obviously due to oxidation.

The relationship of oxygen to the formation of pigment may readily be demonstrated by incubating artificially inoculated tubers in a tightly closed moist chamber. The decayed flesh will remain white until exposed to the air, when it rapidly changes to a brownish black. When tuber slices are inoculated and incubated in a moist chamber with a moderate supply of oxygen, the decayed area is usually whitish in the center, grading into a brown or black margin. Here, again, the degree

A B

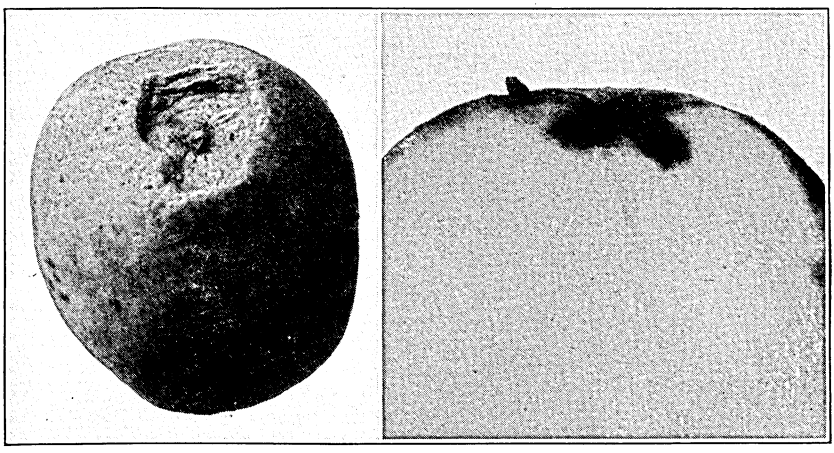
Fig. 5. Two Plants Affected with Blackleg

A. A plant pulled from the soil leaving the rotted seed piece behind.

B. A plant dug carefully so that the rotted seed piece is still attached to the blackened stem.

of discoloration varies with different strains of the pathogen, also with different varieties of potatoes.

The texture of the decayed flesh is influenced chiefly by the humidity of its immediate surroundings. When newly formed tubers decay in the soil the tissues are soft and somewhat mushy, altho they may be slimy, especially if accompanied by saprophytic species of bacteria.



A B

Fig. 6. Potato Tubers Affected with Blackleg at the Stem End as a Result of Infection Entering Through Decayed Stolons

A. Surface view.

B. A section through a tuber at the point of attachment of a decayed stolon.

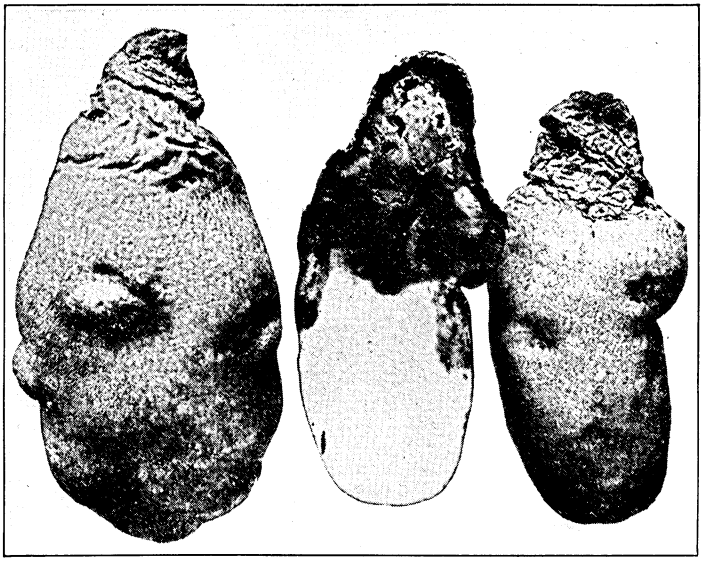


Fig. 7. Types of Blackleg Potato Tuber Rot on Netted Gem Potatoes Grown Under Irrigation in Idaho

Internal and external view one month after digging. (After Shapovalov and Edson).

Tubers decayed by a pure culture of the pathogen do not have an unpleasant odor, but when secondary organisms are present an extremely disagreeable odor may result.

Shapovalov and Edson (38) have described an atypical form of stem-end decay caused by the blackleg pathogen in irrigated sections of the West, especially on pointed-end Netted Gems. Under these conditions a dry and tough necrosis may be caused which is easily confused with the "jelly end-rot" caused by species of Fusarium (Fig. 7).

When tubers are stored under reasonably good conditions, very little blackleg decay results. If the tubers are so poorly ventilated that there is a deficiency of oxygen, cork formation is inhibited and much loss may result. Paine and Haenseler (32) have reported large losses in storage, in England. It is clear from their description that the disease was developing under at least semi-anaerobic conditions.

ETIOLOGY

Identity of the Blackleg Pathogen

The question of the identity and the correct name of the blackleg pathogen has been the subject of much discussion and the cause of considerable confusion. Frank (8), in 1899, was perhaps the first to publish a recognizable description of the disease and to attribute it to a definite bacterial pathogen. He described briefly a *Micrococcus* which he called *Micrococcus phytophthorus*. As no later workers have found a *Micrococcus* to be pathogenic to potatoes, and as abundant evidence has been presented to show that blackleg is due to a *Bacillus*, it is to be concluded that Frank's organism was not the true pathogen.

In 1902, van Hall (43) described a *Bacillus* as the pathogen and named it *Bacillus atrosepticus*. Shortly before this, Appel (1) reported the pathogen as *Bacillus phytophthorus*, but he did not publish a complete description until 1903 (2). Harrison (11), in 1907, published a description of the disease as it occurred in Canada. He described the pathogen as a *Bacillus* slightly different from those already reported and named it *Bacillus solanisaprus*. In 1911, Pethybridge and Murphy (35) attributed the disease in Ireland to *Bacillus melanogenes*, which they found to differ slightly from the previously described organisms. Morse (28), in 1917, published the results of comparative studies of *B. atrosepticus* van Hall, *B. solanisaprus* Harrison, *B. melanogenes* Pethybridge and Murphy, and three cultures isolated from diseased potatoes in Maine. Altho some slight differences were found, such as "slight variations in size, shown more particularly by *B. solanisaprus* and IIIA, and the production of a slight viscidity on different kinds of media shown by *B. solanisaprus* and *B. melanogenes*," he concluded that they "should be classed as one species or at the most, strains of the same

species." On the grounds of priority the name *B. atrosepticus* van Hall was held to be valid. Unfortunately, an authentic virulent culture of *B. phytophthorus* Appel was not included in the comparison.

Jennison (12), however, has repeated and extended this phase of Morse's work, comparing twelve strains of the pathogen, including a virulent culture of Appel's original strain of *B. phytophthorus* as well as cultures of the other three so-called "species." Jennison agreed with Morse that the slight differences were not sufficient to justify species characterization. He also agreed with Morse that the pathogen should bear the name *B. atrosepticus* van Hall. Smith (40), however, preferred to use the name *B. phytophthorus*.

Paine and Chaudhuri (33), in 1923, using a culture of *B. atroseepticus* isolated and identified by Paine in 1917 and one of *B. solanisaprus* obtained in the same year from the American Museum of Natural History, found the two to differ in several important respects such as resistance to heat, type of decay on potato tubers and stems, and gelatine liquefaction.

Brooks, Nain, and Rhodes (6), in 1925, compared strains of *B. carotovorus,* two strains of *B. Phytophthorus,* and one of *B. solanisaprus.* All strains except one of *B. carotovorus* produced identical reactions in sugar media and behaved alike on other media but were found to differ serologically.

In the course of work dealing with the relation of the seed-corn maggot and potato blackleg, the writer had occasion to isolate a large number of cultures of bacteria from the different stages of the insect, from the soil, and from potatoes affected with blackleg. Many of these cultures proved to be pathogenic to potato tubers and stems. As *B. carotovorus* Jones was known to be pathogenic to potatoes, and as the seed-corn maggot is known to breed in plants infected with *B. carotovorus,* it was expected that some of the cultures would prove to be this organism. An effort, therefore, was made to distinguish *B. carotovorus* from the blackleg pathogen, but it was found impossible to do so. Altho there were slight variations in minor cultural characteristics among the cultures isolated, they all proved to have the same primary characteristics as indicated by the index number[2] 5010-32120-0111. For closer comparison, a culture of *B. carotovorus* Jones was obtained from A. B. Massey. This was said to be a subculture of Jones' original type culture designated as 3a (23). This culture was found to be identical with an authentic culture of the blackleg pathogen (*B. phytophthorus* Appel) obtained by the writer from E. F. Smith, insofar as primary characteristics were concerned, altho there were a few differences in minor cultural characters. The most pronounced difference was the produc-

[2] The descriptive chart of the Society of American Bacteriologists.

tion of less black pigment by *B. carotovorus* Jones 3a on potato tubers and stems. When this character was used in an attempt to identify the unknown cultures, so many intermediate forms were found that it was impossible to do so. Massey (23) was able to distinguish *B. phytophthorus* and *B. carotovorus* on the basis of the production of acid by the latter on 5 per cent alcohol agar. This test was used by the writer also, but numerous other pathogenic cultures isolated from rotting carrots, cabbage, celery, onions, and iris, otherwise answering the description of *B carotovorus,* did not show this property. As some strains of *B. Carotovorus* did not possess this property, the test could not be relied upon for identifying the species.

Kotila and Coons (17) showed that *B. atrosepticus* produced a toxic substance that would diffuse through a colloidal membrane and cause potato leaves to wilt. They also showed that 5 cc. of a broth culture of *B. atrosepticus* added to a nutrient solution containing the uninjured roots of a potato plant did not infect the plant but produced a substance that was toxic to it. They conclude that "*B. atrosepticus* seems to possess the power of producing a toxic substance which affects potato cells so as to allow blackening, probably oxidation, to ensue, while *B. carotovorus* which softens potato tissues under some conditions without blackening, does not possess this power." These authors, however, did not report any experiments to show that *B. carotovorus* would not produce such a toxic substance. The writer, therefore, made experiments with both *B. carotovorus* and *B. phytophthorus.* It was found that both cultures produced a toxic substance that wilted cut leaves and caused the yellowing and ultimate death of plants grown in water cultures to which 5 cc. of broth cultures was added. This difference, therefore, could not be used in identifying the cultures.

The similarity between the blackleg pathogen and *B. carotovorus* Jones, which causes a soft rot of many succulent plants, has been recognized by many workers. After comparing several cultures of bacteria causing soft rots, Smith (40) stated that *B. phytophthorus* was "not sufficiently distinguished from *Bacillus carotovorus* Jones, which name is earlier."

Harding and Morse (10), working in collaboration with Jones, made a detailed physiological study of forty-three strains of bacteria causing soft rot of plants. They found that the strains could be separated into six groups on the basis of their action on glucose, lactose, and sucrose. They did not consider these differences sufficient to justify the recognition of six different species but considered the cultures as somewhat variant strains of a single species. Jones (15) also made a study of enzyme activity of the same strains studied by Harding and Morse. He found considerable variation in this respect and presented the re-

sults as "contributing further evidence to the general likeness of these strains and partly as emphasizing the minor variations which we believe must always be expected to occur with different bacterial strains, even of the same so-called species." These differences, however, are equally as great as the differences on which the various "species" of the blackleg pathogen have been distinguished from each other and from *B. carotovorus*.

A careful comparison of the data published by various workers dealing with the various soft-rotting bacteria reveals the following facts:

1. The differences between the various "blackleg pathogens" as described by different workers are as great as or greater than those which are supposed to distinguish them from *Bacillus carotovorus*.
2. The differences found by various workers using, presumably, the same organisms are as great as or greater than those used for distinguishing the blackleg pathogen from *Bacillus carotovorus*.
3. The differences between the six groups of soft-rotting bacteria decribed by Harding and Morse (10), of which *Bacillus carotovorus* is the type, are greater than those that are used for distinguishing the blackleg pathogen from them.
4. The revised description of the blackleg pathogen as given by Jennison (12) agrees in all essential details with the recognized characteristics of *Bacillus carotovorus*.

Many workers have observed the morphologic and physiologic similarity of *B. carotovorus* and the blackleg pathogen, but they usually have considered slight physiologic differences sufficient for differentiating two separate species. The recognition of these slight physiologic differences by different specific names has apparently carried with it an implication of parasitic differences. Jones (13), in his first paper on *B. carotovorus*, reported unsuccessful attempts to infect potato tubers, but in a footnote stated that later inoculations were successful. Smith (40) later showed that this organism was readily pathogenic to potato tubers and stems. However, it was not until 1926 (18) that a natural infection of potato tubers was proved to be due to *B. carotovorus*. Insofar as the present writer has been able to learn, no one has recorded in literature a natural primary infection of potato stems due to *B. carotovorus*. Is this due to the inability of *B. carotovorus* to infect the potato plant under natural conditions, or have we been deceived by the names applied to these organisms and assumed that all soft rot of carrots is due to *B. carotovorus* and that all soft rot of potato stems is due to *B. phytophthorus?*

If it is true that *B. carotovorus* is pathogenic to potato tubers and stems and that the blackleg pathogen is pathogenic to carrots and other

vegetables, it does not seem reasonable that they should be so closely specialized in their attack on these plants in nature. In studying this question several strains of virulent soft-rotting bacteria were isolated from carrots, onions, cabbage, celery, iris, and potato tubers and stems. Cross-inoculations were made and all of the cultures, without exception, were found to be strongly parasitic on all of the hosts. Some slight differences were observed in minor growth characters but all the cultures were identical insofar as the major physiologic tests were concerned; they all had the index number, 5010-32120-0111. Some slight differences in color of the decayed tissues were observed, but this character was very variable, being influenced by the variety of host plant used, the age of tissue, the humidity of the incubation chamber, and other factors. It was quite evident, however, that certain of the cultures differed in amount of pigment produced under the same conditions.

One of the most consistent differences between *B. carotovorus* and the blackleg pathogen as reported by various writers is the so-called "inky-black" discoloration of tissues affected with blackleg, which is absent in tissues affected with *B. carotovorus*. The culture used by the writer designated as *B. carotovorus* Jones 3a was definitely characterized by the production of less pigment than most of the cultures studied. On the other hand, several cultures obtained from carrots, onions, and cabbage produced more pigment than certain cultures isolated from typical blackleg plants. It should be stated, also, that the "inky black" discoloration so frequently mentioned in description of the blackleg disease is not always characteristic of the disease as it occurs in Minnesota. Potato tissue killed by almost any organism will turn black when thoroly dried and oxidized, but tissues in the process of decay by the blackleg pathogen are more frequently dull brown than "inky black."

Lacy (18) compared *B. carotovorus* with several other soft rotting organisms and concluded that "cultural, pathological, and serological tests all show that a close relationship exists between the three species, *B. carotovorus, B. solanisaprus,* and *B. phytophthorus,* but there are nevertheless sufficiently marked and constant differences to warrant continued separation into different species." Stapp (41), on the other hand, has compared many strains of the blackleg pathogen with *B. carotovorus* and other similar organisms. He classified them into five groups on the basis of serological and physiological differences but proposed to include all five groups in one uniform *"Bacillus phytophthorus* group." As our concept of what constitutes a species among the bacteria is extremely vague, it appears to be only a matter of opinion as to whether a distinct specific name should be given to each of these soft-rotting organisms. The writer is inclined to favor the idea, pro-

posed by Stapp, of including all in one species, but on the basis of priority it appears that *Bacillus carotovorus*[3] would be the correct name because *B. carotovorus* was described two years before *B. phytophthorus*.

At any rate it must be recognized that potato blackleg is a bacterial soft rot and may be caused by several closely related strains of bacteria and that the same bacteria may cause a soft rot of many other plants. If we separate the "soft rot" group of bacteria into separate species on the basis of minor physiological differences, we must recognize that potato blackleg can be caused by *B. carotovorus* as well as by *B. phytophthorus*. In other words, potato blackleg is considered to be nothing more than "soft rot" of the potato. The distinctive characters of the disease are due primarily to the nature of the host plant rather than to the causative agent. The potato, being propagated by tubers, is more subject to infection than most plants propagated by seed. The transmission of the organism through infected seed pieces and the tendency of decayed potato tissue to form a black pigment upon oxidation have been factors contributing to the distinctive character of the disease when affecting the potato.

Detailed Description of the Blackleg Pathogen

Erwinia carotovora (Jones) Comm. S. A. B., Jour. Bact. 5:191-229. 1920.

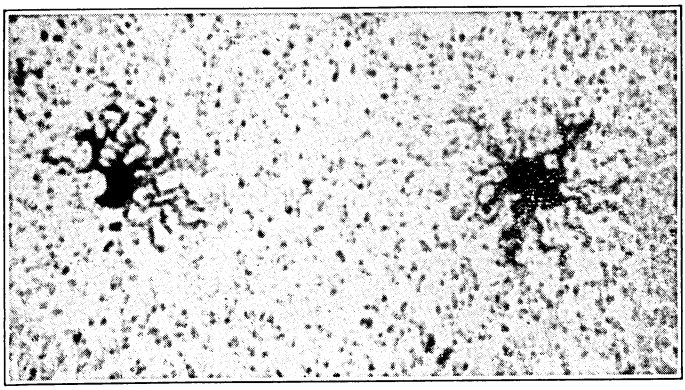


Fig. 8. Photomicrograph of the Blackleg Pathogen Showing the Flagella Approximately 5,000 X. Stained by Van Ermengem's silver nitrate method.

Morphology

Small rod, average dimensions, 0.6–0.8 μ x 1.5–2.5 μ, actively motile by means of peritrichic flagella (Fig. 8), non-sporiferous, non-capsulate, and Gram negative.

[3] Following the new system of classification of bacteria recommended by the Society of American Bacteriologists, the organism would bear the name, *Erwinia carotovora* (Jones) Comm. S. A. B.

Physiological Characteristics

Non-chromogenic on agar, gelatine, and beef extract broth. Growth on agar slants moderately abundant, surface smooth and glistening. Agar colonies round, appearing granular under the microscope. Gelatine is slowly liquefied, colonies on gelatine larger than those on agar. Beef extract is clouded and becomes slightly turbid after several days' growth. Surface growth varies from ring to slight pelicle.

Aerobe and facultative anaerobe. Nitrates reduced without formation of gas. Starch is not hydrolyzed. Acid and gas from d-glucose, lactose, and sucrose, no acid or gas from glycerine. Indol not produced. Sensitive to desiccation when dried rapidly on glass but very resistant when dried slowly in soil. Optimum temperature for growth 24°-27°C. Thermal death point 47°-50°C. Pathogenic to potato, carrot, cabbage, celery, onion, iris, and many other succulent plants. Index number 5010-32120-0111.

Synonymy

Bacillus carotovorus Jones, Centralb. f. Bakt. 7:12. 1901.
Bacillus atrosepticus van Hall, Inaug. Diss. Univ. Amsterdam. 1902.
Erwinia atroseptica (van Hall) Comm. S. A. B., Man. Determinative Bact. 1923.
Bacillus phytophthorus Appel, Ber. d. deut. bot. Ges. 20:128. 1902.
Erwinia phytophthora (Appel) Comm. S. A. B., Jour. Bact. 5:191. 1920.
Bacillus solanisanprus Harrison, Centralb. f. Bakt. II 17:34. 1907.
Erwinia solanisapra (Harrison) Comm. S. A. B., Jour. Bact. 5:191. 1920.
Bacillus melanogenes Pethybridge and Murphy, Proc. Roy. Irish Acad. 29B:1. 1911.
Bacillus oleraceae Harrison, Science (n. s.) 16:152. 1902.
Bacillus omnivorus van Hall, Inaug. Diss. Univ. Amsterdam. 1902.
Bacillus apivorus Wormald, Jour. Agr. Sci. 6:203-219. 1914.
Erwina oleraceae (Harrison) Comm. S. A. B., Man. Determinative Bact. 1923.

Sources of Infection

Blackleg as it occurs in nature starts first as a decay of the seed piece. The decay spreads from the seed piece into the stem, from which it may spread to all parts of the plant if conditions are favorable. It is generally agreed by those who have studied the disease that infection does not take place through the roots or from the parts above ground (12) (28). Altho Appel (2), in 1903, presented some evidence to show that infection could arise from the soil, Morse, in 1909

(26) and in 1917 (28), as a result of observations and experiments in Maine, expressed the opinion that seed tubers, already infected at planting time, were responsible for the propagation of the disease. The inability of Rosenbaum and Ramsey (27) and Ramsey (36) and others to find evidence of the survival of the pathogen in the soil added support to the views of Morse. Their views were accepted generally by pathologists and growers and for many years it was customary to attribute all blackleg to the use of infected seed. With the advent of potato seed certification and the resulting inspections of fields and records of seed stock, it became quite evident that there was much blackleg which could not be explained on the basis of seed tubers from diseased plants. Orton (31), Murphy (29), and others have expressed the opinion that contamination of the freshly cut surface of seed pieces with bacteria from the cutting knives was sufficient to produce infection of the resulting plants. On the other hand, the experiments of the writer reported in 1926 (19) and in 1930 (21) indicate that this is not an important source of infection if the seed pieces are planted under reasonably good growing conditions. In these experiments seed pieces partly decayed by artificial inoculation with the pathogen were planted under a variety of soil conditions. More than a thousand such seed pieces have been planted and less than one per cent have produced blackleg plants. Careful examination of such seed pieces show that the failure of the disease to develop is due to the formation of wound cork, which effectually walls off the bacteria and prevents further decay (Fig. 9). Knowing that infection must take place through the seed piece and that wound cork, which is so readily formed by potato tubers, is an effective barrier to infection by the blackleg pathogen, it is obvious that the development of the disease must depend largely upon factors that enable the bacteria to penetrate this protective layer of cork. Investigations extending over several years have shown that there are at least three ways in which the pathogen may succeed in overcoming the resistance offered by the tuber's ability to form wound cork (19, 20, 21). These are described in the following pages.

Systemic Infection of Seed Tubers

It has been know for many years that seed tubers taken from fields in which blackleg is present sometimes produce more diseased plants than those from fields in which no blackleg occurred. This has been attributed to natural infection of the seed tubers. Altho natural infection of seed tubers has been recognized for a long time, the exact nature of such infection has not been demonstrated clearly

until recently. In 1927 (20) the writer reported experiments in which naturally infected tubers from diseased plants were planted in comparison with artificially infected seed pieces. No disease developed from the artificially infected seed pieces, while about 10 per cent of the naturally infected tubers resulted in blackleg plants. As there was no doubt as to the thoroness of the artificial infection or the virulence of the culture used, these results led to the conclusion that there was some fundamental difference between such natural infection and artificial infection of the tubers. A histological study of naturally infected tubers before planting and during the early stages of the development of the disease verified this conclusion. The results of this study may be summarized briefly as follows: When a stem-end infected tuber is cut in half longitudinally through the point of at-

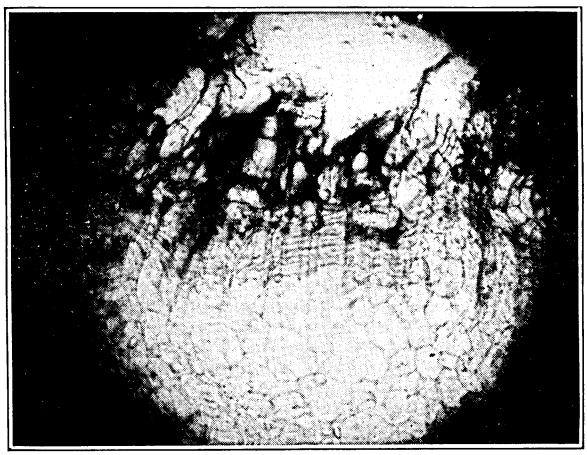


Fig. 9. A Photomicrograph of a Microtome Section Showing the Layer of Wound Cork Formed by the Parenchyma Tissue of a Potato Tuber at the Point Where Blackleg Decay Has Been Checked

tachment of the stolon, it readily may be seen that the decay extends farthest along the vascular bundles (Fig. 6). The presence of the pathogen in such lesions has been demonstrated by isolations in which it has been obtained in a fair percentage of trials. Whenever any considerable amount of the parenchyma has been destroyed, careful examination shows that the decay has been checked and cut off from the rest of the parenchyma by a layer of cork (Fig. 9). It appears that it would have been entirely cut off except for its ability to invade the vascular bundles. The cells of the vascular bundles, being unable to form cork, could not stop the advance of the bacteria. As soon as the bacteria advance beyond the cork elements, formed by the parenchyma

cells, they again break out of the vascular elements into the surrounding parenchyma but are soon checked by a layer of cork which quickly forms around the vascular bundle. Figure 10 shows a photomicrograph of a longitudinal section of an infected vascular bundle around which has been laid down a thick wall of cork cells. Thus the bacteria slowly advance along the vascular elements where they obtain sufficient moisture to remain alive, but from which they are unable to escape on account of the effective cork wall laid down by the parenchyma cells surrounding the bundles.

When such tubers are planted, the bacteria are unable to escape from the vascular bundles until the starch has been removed by the sprout. When this stage is reached, the seed piece is no longer able to form cork, so the bacteria develop rapidly throughout the seed piece and spread into the stem, producing the disease.

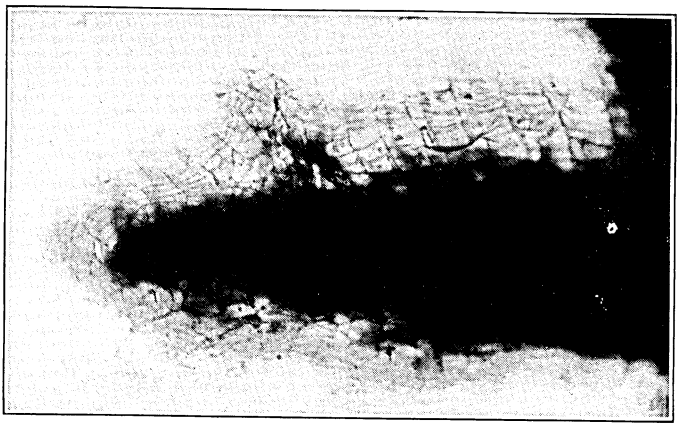


Fig. 10. A Photomicrograph of a Section Through a Vascular Bundle from a Stem End Infected Tuber Such as the One Shown in Figure 6

The elements of the vascular bundle have been destroyed along with some of the surrounding parenchyma cells, but a cylinder of cork cells has been formed around the old bundle, thus checking the spread of the bacteria. As soon as the starch has been removed from the seed piece by the sprout, cork can no longer be formed and the bacteria spread unhindered throughout the seed piece and into the growing sprout.

In order to prove the validity of the assumption that the seed piece is unable to cork off the decay after the starch has been removed, a number of sound seed pieces were removed from growing plants and inoculated with the blackleg pathogen. Normal tubers were inoculated at the same time, and all were placed over water in a closed chamber through which a stream of air saturated with moisture was drawn by means of a suction pump. The seed pieces from which the starch had been removed were completely decayed within three days; the inoculated normal tubers remained sound and completely corked-off the decay.

This study leads to the conclusion that blackleg is sometimes systemic and can be perpetuated by tubers naturally infected through the vascular bundles, which enter the tuber from decaying stolons. Artificial inoculation through the parenchyma tissues appears to be relatively ineffective in producing the disease unless aided by some agency which inhibits cork formation. Altho systemic infection of tubers does occur, it is unlikely that such infection would ever be responsible for a high percentage of blackleg under ordinary field conditions. As pointed out above, only 10 per cent of the plants grown from selected tubers known to be so infected, became diseased. It is, then, hardly reasonable to suppose that the general field run of tubers would result in a very high percentage of diseased plants. In all probability very high percentages of blackleg in commercial fields result from some other source of infection.

Infection of Seed Tubers by Bacteria from the Soil

The erroneous conclusion that the blackleg pathogen could not survive in the soil has led to a general assumption that infection could not arise from bacteria in the soil. The conclusion that the pathogen could not live long in the soil was based chiefly on the negative evidence presented by Rosenbaum and Ramsey (37) and Ramsey (36). Those investigators planted tubers in soil that had been heavily inoculated with bacteria the previous fall. The failure of such tubers to become infected led them to conclude that the bacteria were no longer present in the soil. However, the experiments of the writer, reported in 1926 (19) and in 1930 (21) show that this conclusion is not justified. In these experiments it was shown that seed tubers partly decayed as the result of artificial inoculation would shut out the decay by the formation of wound cork and produce healthy plants when planted in a soil favorable for growth. When we consider the results of these experiments it is obvious that altho the pathogen may have been extremely abundant in the soil, it would be illogical to expect infection to occur as long as conditions are suitable for the formation of wound cork by the seed tubers.

These authors attempted to isolate the pathogen from the infected soil by pouring dilution plates and picking out favorable looking colonies but were unable to do so. When one considers the great number of bacteria that inhabit most soils, and the similarity of their appearance on agar, it is not surprising that they did not succeed in reisolating the pathogen. Realizing this difficulty, the writer proceeded in the following manner: Several quart jars were filled with soil, inoculated with a broth culture of the pathogen, and buried in the soil, where they remained throughout the winter of 1928-29. About one gram of the soil to be

tested was thoroly mixed with about 10 cc. of sterile beef-extract broth in May of 1929. This suspension was used for inoculating several surface-sterilized tubers by the "hole and plug" method previously described (19). After incubation in a moist chamber for four or five days, a considerable amount of decay had developed. This decay was quite unlike decay resulting from a pure culture of the blackleg pathogen and advanced much more slowly. It was usually accompanied by a foul odor and frequently gas production was evident. Most of the decayed tissue was next removed with a scapel, and a small portion of the decaying tissue at the margin nearest the sound tissue was removed with a sterile needle and used for inoculating a second lot of tubers or tuber slices. When these had started to decay, a third lot was inoculated in the same manner. Usually the virulence of the inoculum increased with each successive inoculation, and after three or four transfers the decay was quite typical of blackleg. When a small amount of freshly decayed tissue from the fourth or fifth inoculation was used for pouring separation plates, little difficulty was experienced in isolating a vigorously pathogenic culture similar in all respects to the culture used for inoculating the soil.

Patel (34), by using crystal violet-bile-agar as a selective medium, has demonstrated that the organism may survive the winter in Iowa in sterilized soil.

These results, together with observations extending over several years, force one to the conclusion that the blackleg pathogen can and does commonly survive in the soil in Minnesota. If this is correct it is only reasonable to assume that the bacteria in the soil can infect seed tubers if for any reason wound cork formation is inhibited. There appears to be plently of evidence to show that this is true. It is a matter of common observation that blackleg is more destructive in heavy moist soils than in soils with an optimum moisture supply.

Morse (28) states that "more blackleg is observed in wet than in dry seasons" and that "all other things being equal, the disease is more likely to occur in wet than in dry soil, and is more prevalent when the early part of the growing season is characterized by abundant rainfall." He also described one example in which the disease was first noted "near the center of the affected area from which it gradually spread outward. The season had been excessively wet, and this area coincided with a low, undrained pocket or depression in the field, where water would stand for a few hours after each heavy rainfall."

Murphy (29) says "more diseased plants are found in low-lying parts of a field than in higher." One example is given in which "it was found in extensive counts that there were three blackleg plants in the low-lying area to one in the higher."

The writer has made numerous observations of similar nature, and any one who has studied the disease to any extent will agree that outbreaks of the disease very frequently occur in water-logged soils. Where attempts have been made to explain this relationship, emphasis has been put on the growth requirements of the pathogen, and very little attention has been paid to the influence of such conditions on the seed tubers. Without doubt such conditions are favorable to the growth of the pathogen, which is a facultative aenaerobe, but, at the same time, one should not overlook the influence of water-logged soils on the formation of wound cork by the seed pieces. That abundant oxygen is necessary for the development of cork in the potato tuber has been shown by Kny (16), Appel (2), Olufsen (30), and others. It is obvious that excessive moisture in the soil inhibits cork formation, but apparently there are no records of a careful study of the question.

In order to obtain a more accurate measure of this influence, freshly cut Early Ohio tubers were planted in a series of jars or cylinders of quartz sand, each of which was held at a different moisture content. In the jars with a higher moisture content, water was supplied from the bottom, and the differences in water content were obtained by varying the water level. In the dryer jars, the water was supplied through Livingston atmometers buried in the center of the jar, sealed, and connected to a bottle of water by a rubber tube. The differences in moisture content were obtained by varying the height of the jar above the bottle of water. On the 1st, 2nd, 4th, 6th, and 8th days after planting, a tuber was removed from each jar and the cut surface was examined for the presence of wound cork. Sections were cut on a sliding microtome, stained in Sudan III, and mounted in glycerine for microscopic examinations. In this way a very good idea of the effect of excessive moisture in the soil on the formation of cork was obtained.

It is realized that the conditions of the experiment were not comparable in all respects to those in nature, for the influence of a given moisture content would vary with the type of soil. The results obtained show only the relative effect of different degrees of moisture, not the absolute effect of a given moisture content.

The experiment was made during the month of March when the tubers were no longer dormant. The results, as indicated by the amount of cork formed on the 8th day, are given in Table 2.

Table 2

Influence of the Amount of Moisture in the Soil on the Amount of Wound Cork Formed by Cut Potato Tubers in Eight Days

Pot No.	Moisture content, per cent	Outer cells	New cross walls	
			No.	No. suberized
1	Less than 1	Suberized	2-4	2
2	2.39	do	5-8	3
3	5.91	do	5-8	3
4	7.88	do	5-8	3
5	9.38	do	4-6	2
6	12.03	Lightly suberized	1-4	None
7	12.59	do	1-4	do
8	14.75	Not suberized	None	Outer cells autolyzed

The progress of healing as observed in the experiment agreed closely with the process described by previous investigators. During the first 48 hours the walls of the outer layer of cells became infiltrated with a dark brown substance which apparently makes them impervious to water. These cells do not readily stain with suberin stains. The process, sometimes termed "blocking," is considered as a form of suberization and is inhibited by exclusion of oxygen. Beginning about the third day, under favorable conditions, the starch disappears from the second or third layer of cells beneath the wounded surface and cross walls are formed in it. These walls all lie parallel to the wounded surface. It usually requires from 8 to 10 days for the formation and suberization of these new cells that make up the new periderm. It will be noted that in this experiment an excessive amount of moisture resulted in a decided retardation or complete inhibition of these processes. In the more moist cylinders, not only was there no suberization or cell division, but the cells became soft and mushy, evidently because of asphyxiation and resulting autolysis.

Since the blackleg pathogen is a facultative anaerobe and may survive in the soil, it seems that under such conditions infections could easily arise from the soil. It is probable that such infection is largely responsible for the greater abundance of blackleg in excessively wet soils and in unusually rainy seasons.

The marked influence of oxygen on wound-cork formation and on infection by the blackleg pathogen was further demonstrated by the following experiment: Several potato tubers were inoculated with a pure culture of the blackleg pathogen by the "hole and plug" method. The inoculated tubers were divided into three lots and placed in three separate moist chambers. The first container was a 3-gallon earthenware crock with a fairly loosely-fitting top. The second was a large glass "desiccator" with a closely-fitting top. The lower section was

filled with water, and the inoculated tubers were suspended on a wire platform above the water. The third container was similar to the second but differed in that a stream of fresh air was drawn through the "desiccator" by means of two glass tubes sealed in the top of the vessel. The air-inlet tube extended to a point just below the tubers so that the air was drawn about the tubers and then removed from the top. The air current was supplied by a suction pump and the air was bubbled through two flasks of water before it was drawn into the moist chamber.

A saturated humidity in the chamber was insured by keeping the water in these flasks a few degrees warmer than the temperature in the chamber. In this way the warm, moist air was slightly cooled on entering the chamber and precipitated moisture was visible on the sides of the vessel throughout the duration of the experiment. The moist chambers were placed side by side on a table at room temperature.

In the first container the loosely-fitted top permitted sufficient interchange of air to cause some fluctuation and variation in relative humidity, altho it probably closely approached saturation at all times. The oxygen supply in the crock was no doubt soon used up by the respiration of the tubers and was not entirely replaced by diffusion through the leak in the top.

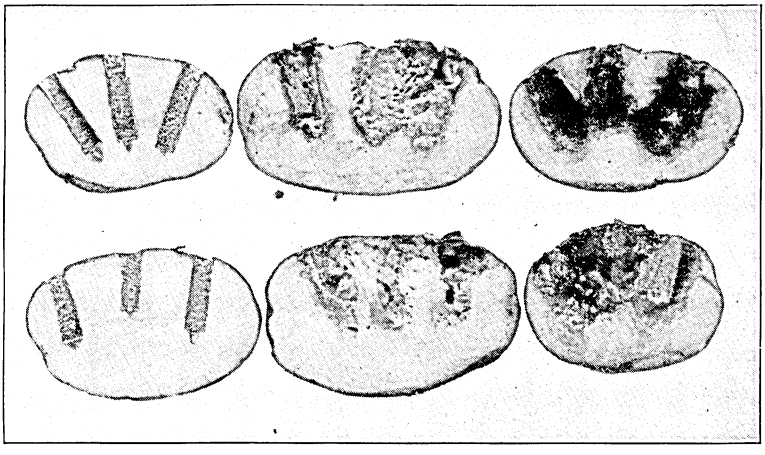
In the second container the air was saturated with moisture at all times, but the oxygen consumed in respiration could not be replaced.

In the third container a saturated relative humidity combined with abundant oxygen supply prevailed.

After four days' incubation two tubers were removed from each container and cut in half to determine the amount of decay. These tubers are shown in Figure 11. It will be seen that the tubers in the first two containers were badly decayed; those in the container receiving a constant supply of fresh air had not decayed at all. The decayed tissue of the tubers incubated in the closed "desiccator" was almost pure white, showing only traces of browning along the margins. The influence of the limited supply of oxygen available in the crock was evident in the darker color of the decayed tissue of the tubers incubated in the crock.

Microtome sections were cut through the inoculated portions of the tubers shown in Figures 11a and 11b and were examined for wound cork. Photomicrographs of these sections are shown in Figure 12. It was found that an abundance of wound cork had been formed by the tubers incubated in a constant supply of oxygen but that none had formed in the other tubers.

This experiment was repeated several times and it was found to be extremely difficult to rot tubers with the blackleg pathogen if they are given a constant supply of fresh air even when incubated at the optimum temperature and in a saturated atmosphere.



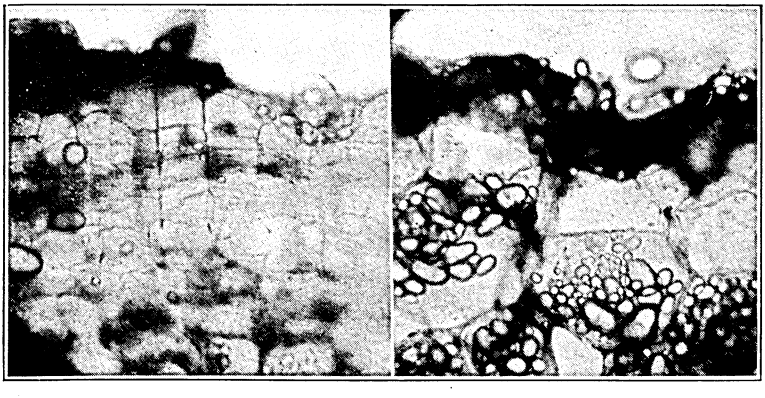
A B C

Fig. 11. Influence of Oxygen Supply on Infection and Decay of Potato Tubers by the Blackleg Pathogen

A. Tubers incubated in a moist chamber in saturated atmosphere, but with a constant supply of fresh air.

B. Tubers incubated in a moist chamber with a close-fitting top.

C. Tubers incubated in a moist chamber with a loose-fitting top.



A B

Fig. 12. Influence of Oxygen Supply on Wound-Cork Formation by Tubers Inoculated with the Blackleg Pathogen and Incubated in Moist Chambers

A. Photomicrograph of a section through the infection court of the tubers shown in Figure 11A.

B. The same for Figure 11C.

The Seed-Corn Maggot as an Agent of Dissemination and Inoculation of the Pathogen

During the spring and summer of 1923 and 1924 the writer investigated several outbreaks of blackleg which could not be explained on the basis of infected seed stock. This investigation led to the discovery that the seed piece of nearly every plant affected with blackleg was infested by larvae of the seed-corn maggot *(Hylemyia cilicrura* Rond.) Further studies led to the conclusion that this insect is an important agent of dissemination and inoculation of the blackleg pathogen. Since detailed accounts of these experiments have been published elsewhere (19, 22), only a summary of the salient facts will be presented here.

The seed-corn maggot (Fig. 13) is a common pest of the potato and many other plants. A survey of the literature revealed reports of

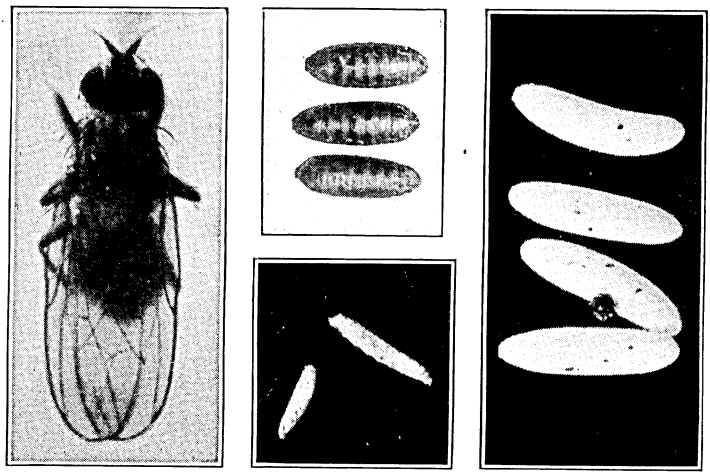


Fig. 13. The Seed-Corn Maggot *(Hylemyia cilicrura* Rond.)
A. Adult female fly, approx. 10x.
B. Four eggs, approx. 50x.
C. Two larvae, approx. 1½x.
D. Three puparia, approx. 3x.

injury on corn, peas, turnips, cabbage, radishes, onions, beets, potatoes, hedge mustard (9), tomatoes (42), wheat (39), and several other plants. The insect overwinters in the soil in the puparial stage. The adult flies emerge in early spring. They are usually present in Minnesota potato fields in great numbers during the planting season. They may be seen flitting about over the surface of the ground and appear to be attracted by freshly turned soil. They are about the size of a housefly, which they resemble in many respects (Fig. 14). Eggs are deposited on, or in, the soil about seed pieces and young potato plants (Figs. 15 and 16). The eggs are small, slightly less

.n one millimeter in length, but may be seen readily with the naked e,e. They are slightly curved, white, and distinctly reticulate (Fig. -3B). When freshly deposited they are covered with a sticky fluid, which causes them to adhere to anything with which they come in contact.

The eggs hatch after two or three days. The maggot, when it emerges, is nearly transparent and only slightly longer than the egg, but it is able to move about readily in search of food. It enlarges rapidly and reaches full size in about two weeks (Fig. 13C). The mouth of the maggot is equipped with two black, sharp, clawlike structures with which it is able to penetrate the tissues of the seed piece. Observations have shown that the maggots, during the first 24 hours, will crawl about over the cut surface of the seed piece, scraping with their mouth parts until the tissues begin to decay. During the next few days they penetrate deep into the tissues, thoroly inoculating the seed piece with bacteria. Experiments have shown that the eggs are

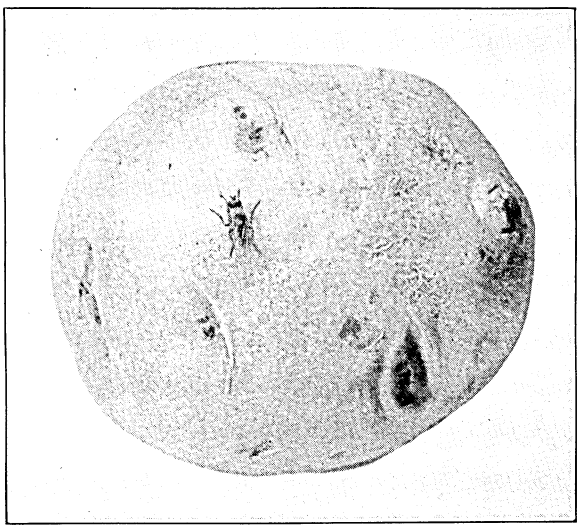


Fig. 14. Female Seed-Corn-Maggot Fly Resting upon a Potato Tuber. Approx. 1½x

sometimes surface-contaminated with the blackleg pathogen when deposited. It has also been shown that the pathogen can survive in the soil. The maggot, therefore, may pick up bacteria either from the egg shells, from the soil, or perhaps also from the surface of the contaminated seed pieces.

The maggot thus acts as a very effective agent of inoculation. By the continued burrowing of the maggot the bacteria are introduced deep into the seed piece and any tendency of the tissues to form wound cork is thoroly overcome.

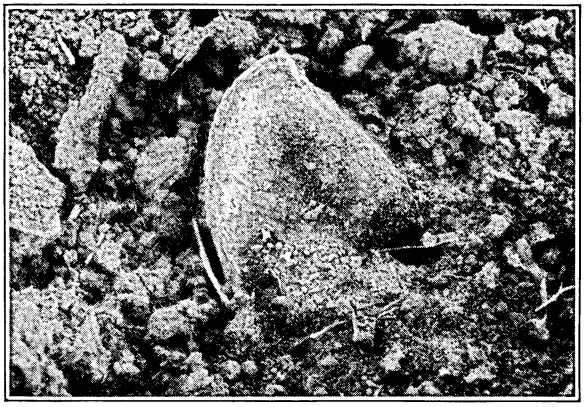


Fig. 15. Eggs of Seed-Corn Maggot on and in the Soil Near a Potato Seed Piece

Photographed three days after the seed pieeces were planted by machine in the usual manner. This seed piece was incompletely covered and when it was turned over the eggs were discovered underneath. Observations have shown that eggs are also frequently deposited in cracks near the stems of sprouting seed pieces.

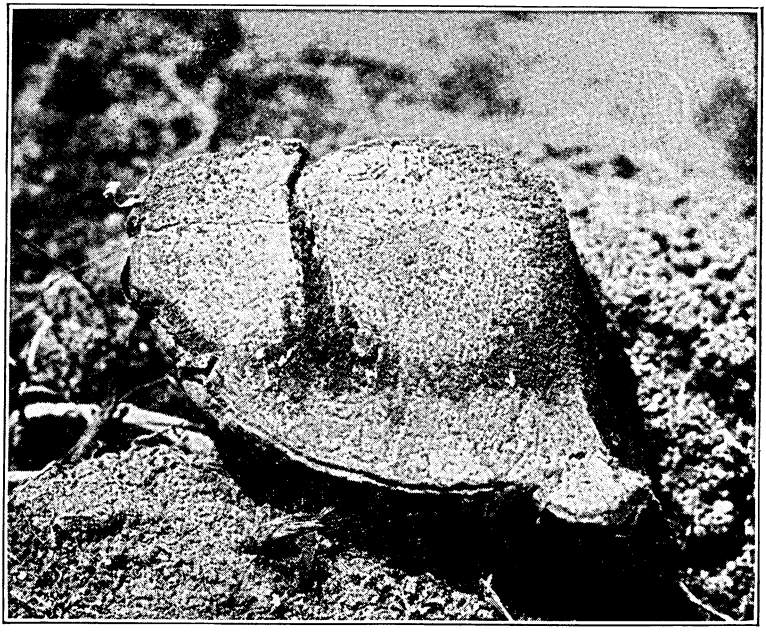


Fig. 16. Seed Piece Uncovered and Turned Over to Show the Eggs of the Seed-Corn Maggot Adhering to the Under Side

The larvae hatching from such eggs burrow into the seed piece, inhibiting wound-cork formation and inoculating it with the blackleg pathogen.

It has been demonstrated (19, 22) that bacteria are essential for the normal development of the larvae on potato tubers. The maggots are not able to grow on the normal tissues of the tubers, but, after the tissues have been acted upon by bacteria, they form a suitable medium for growth. The bacteria in all probability digest the tissues and transform them into materials readily available to the maggots.

After two or three weeks' development in the seed pieces, the maggots reach their maximum size, 7-8 mm. They then leave the seed-piece and go into the soil and pupate. In the meantime the seed piece is usually completely decayed and the bacteria are invading the stems of the plants producing typical symptoms of blackleg. Pupation is usually well under way by the time the first above-ground symptoms appear.

The puparia are 4-5 mm. in length and somewhat oval in shape (Fig. 13d). They vary from light brown to dark reddish brown according to age. The duration of the puparial stage varies from seven to fourteen days in summer.

There are usually two broods of the insect in Minnesota. The first brood is the more important in its relation to blackleg, as the eggs are deposited near seed pieces or young potato plants. The second brood usually deposits its eggs near or on the stems of plants already affected with blackleg. The maggots of the second brood may be found frequently in the stems of blackleg plants while the maggots of the first brood are confined chiefly to the seed pieces.

It has been shown (19) that the blackleg pathogen, along with certain saprophytic species, may pass through the intestinal tract of both larva and adult fly in a viable condition. It also has been shown that these bacteria may survive in the puparia and emerge with the adult fly. Thus the insect can serve as agent of both dissemination and inoculation of the blackleg pathogen.

No practical means of controlling this insect is known. It is obvious that if the insect could be controlled the losses from blackleg could be materially reduced.

PATHOLOGICAL ANATOMY

As previously mentioned, the blackleg pathogen infects chiefly through wounds. The organism at first is confined to the intercellular spaces. It secretes an enzyme (pectosinase), which dissolves the middle lamellae, thus loosening the parenchyma cells one from another. The protoplasmic membranes of the cells are destroyed, allowing the cell sap to flow into the intercellular spaces where it provides moisture and nourishment for the rapid multiplication of the bacteria. The tis-

sues lose their turgor and assume the soft mushy texture characteristic of bacterial soft rots.

Owing to the oxidation of the materials set free by the dead cells, a brownish or black pigment is formed which stains all infected tissues and sometimes adjacent living cells. The pigment is frequently taken up by the vascular bundles, which may be stained brown for several inches beyond the advance of the bacteria. In dry atmosphere, the affected tissues usually shrink as a result of the loss of water. Such drying is usually accompanied by an intensification of the brown or black pigment. In advanced stages the texture of decayed tissue may be greatly modified by the invasion of secondary organisms. Such saprophytic organisms are responsible for the slimy texture sometimes observed as well as the foul odor associated with mixed infections.

Artschwager (4) studied the pathological anatomy of blackleg plants as grown in the arid regions of western Colorado. He pointed out that plants affected with blackleg show an increase in strongly lignified vascular tissue and a transformation of part or most of the parenchyma cells of cortex and pith into sclerids. He also observed an abnormal appearance of protein crystals, especially in the cells of the leaves. Similar changes have been observed by the writer in blackleg plants in Minnesota, but such changes are not confined to plants affected by blackleg. Abnormal increase in lignefication of pith and cortex cells as well as an increase in protein granules has been observed frequently in plants injured by *Rhizoctonia solani* as well as in plants growing under conditions of extreme drought.

CONTROL MEASURES

It will be realized from the preceding discussion that a great many factors may influence the development of blackleg and that the control of the disease is not a simple matter. There are, however, a number of practices which, if properly carried out, should reduce the losses from the disease to a minimum. The most important of these are given below.

1. Use certified seed stock from fields known to have been free from blackleg. When growing tubers for seed purposes all blackleg plants should be removed and destroyed. When the disease appears late in the season, any tubers that have formed on blackleg plants should be dug and destroyed. If this precaution is carried out most of the systemically affected tubers should be eliminated.

2. All seed tubers should be disinfected before planting to kill any bacteria on the surface of the tubers that might cause decay of seed pieces when planted under unfavorable conditions. Bonde (5)

has recently shown that under some conditions small necrotic lesions may be formed on seed tubers soon after planting. These are attractive to the seed-corn maggot fly and also make the tubers more susceptible to maggot attack. Seed treatment methods that tend to prevent these lesions and preserve the seed pieces, should help in reducing the amount of blackleg.

3. Under Minnesota conditions it is advisable to plant the seed pieces as soon as possible after cutting them. The conditions obtaining in a good seedbed are excellent for the formation of wound cork over the cut surface. Seed stored for only a few days after cutting heal poorly and frequently develop some decay which, as stated above, is conducive to maggot attack.

4. On heavy soils, poorly drained soils, or peat soils that are likely to be wet, potatoes should be planted rather shallow. Deep planting in such soils usually results in poor healing of wounds and is conducive to infection from bacteria in the soil.

5. Crop rotation should be practiced because it is known that the organism may survive in the soil, altho little is known about the effect of continued cropping on its abundance in the soil. Another reason for rotating crops is that the first brood of seed-corn maggot flies emerging in the spring do not migrate very far during the first few weeks and are therefore to be found in greater abundance in and near old potato fields at planting time. There is also some evidence that flies arising from larvae that have developed in blackleg plants are more likely to be carriers of the pathogen than those that have developed in other decaying matter.

SUMMARY

1. Blackleg is one of the major diseases of the potato in Minnesota, causing an average annual loss of about 2 per cent of the crop. It is sporadic in its occurrence and may cause losses in excess of 50 per cent in some fields. When potatoes are stored under poor conditions the disease may cause heavy losses as the result of a soft rot.

2. Blackleg is world-wide in distribution, probably occurring wherever potatoes are grown.

3. Blackleg is a bacterial soft rot that may affect any part of the potato plant. In nature it practically always originates as a decay of the seed piece and then spreads to other parts of the plant. The characteristic symptoms of the disease are described in this bulletin.

4. Blackleg is caused by several closely related strains of bacteria that also cause a soft rot of many other vegetables. Various names have been applied to these strains of bacteria (*Bacillus phytophthorus* Appel,

B. atrosepticus van Hall, *B. solanisaprus* Harrison, *B. melanogenes* P. and M.), but a comparative study of the morphologic, physiologic, and parasitic properties of these strains lead to the conclusion that they should be considered as a single species, and on the basis of priority should be called *Erwinia caratovora* (Jones) Comm. S. A. B.

5. There are at least three distinct sources of infection: (a) Systemic infection of seed tubers in which the pathogen gains entrance into tubers produced on plants infected with blackleg. The bacteria enter through the stem end from decaying stolons. The bacteria overwinter in the vascular bundles and after the seed piece has sprouted it is destroyed by the bacteria, which spread into the stem of the plant and produce the disease. This is probably the least important of the three sources of infection. (b) Direct infection of the seed pieces by bacteria from the soil. This occurs when seed pieces are planted under conditions which are inhibitory to the formation of wound cork. (c) Infection following the attack of seed pieces by the seed-corn maggot. This insect is an effective agent of dissemination and inoculation of the blackleg pathogen. The maggots pick up the bacteria from several possible sources and introduce them into the wounds which they make in seed pieces, and by their continued burrowing into the seed piece, effectively inoculate the plant.

6. The action of the bacteria on the tissues of potato plants is essentially the same as that of the soft rot of other plants. The nature of this action is briefly described.

7. The control of blackleg is dependent upon a number of variable factors and is not a simple problem. Practices considered to be essential in avoiding losses from blackleg are outlined in this bulletin.

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