A DRYROT CANKER OF SUGAR BEETS

By B. L. RICHARDS

Department of Plant Pathology, Utah Agricultural Experiment Station

What appears to be an undescribed rootrot of the sugar beet was first called to my attention on August 5, 1920, by Mr. A. H. Bateman. Specimens of the diseased beets collected at this date at Cornish, Utah, exhibited numerous brown, circular lesions that varied from $\frac{1}{16}$ inch to 1 inch in diameter (Pl. 4; 8, D; 9, A, B). The outer surface of the root covering these lesions, which in most cases remained entire, had so sunken as to give a definite undulating contour of alternating light and dark brown concentric areas or rings (Pl. 4). The removal of this outer layer of cells of the older lesions exposed deep cankers or pockets filled with hyaline-mycelium embedded in the dry remains of partially decayed host cells. This accompanying mycelium, when exposed to the atmosphere through the cracking open of the outer covering, appeared dark brown in color and immediately suggested the typical mycelium of the sterile, or "Rhizoctonia," stage of *Corticium vagum* B. and C. The general prevalence of black sclerotial bodies on the outside of the diseased beets (Pl. 7, A), together with the microscopic examinations made at this time, confirmed this initial suggestion.

An examination of the field from which these first diseased specimens were taken revealed the trouble to be of considerable economic importance; at least 20 per cent of the beets in this field of 40 acres were diseased. The disease appeared to be confined to definite areas wherein every beet might be found infected. These diseased spots varied considerably in size and appeared to be widening most rapidly in the direction parallel with rows. Three adjacent fields were found at this time to be seriously diseased, but none to the same degree as the field first visited.

The progress of the disease in these fields appeared of such ominous character as to require immediate investigation. However, as the season was well advanced, little more than preliminary experiments were undertaken. The results to date, while definite, are not sufficiently extensive to warrant final conclusions, and many of the important relations of the disease remain obscure; nevertheless it is felt that the apparent economic importance of the trouble justifies a preliminary description at this time.

The disease is first detected in the field by abnormal wilting of the leaves in the daytime with partial or complete recovery at night. Later

Journal of Agricultural Research, Washington, D. C.
zm
54817°—21——4

Vol. XXII, No. 1
Oct. 1, 1921
Key No. Utah-14

the older leaves fail to recover, turn brown, and die. This dying of the outer or older leaves continues with the progress of the disease in the root until all the leaves on the affected beets may succumb. Localized browning frequently occurs in the blade and petiole, but to date no suggestion of a parasitic relation has been found. Neither the petiole decay reported by Duggar (*2*)[1] nor the "western crownrot" described by Edson (*3*) have been found associated with the dryrot canker in the field. A peculiar type of crownrot, however, is found late in the season, usually well toward harvest time (Pl. 6; 8, B). A study of a number of these crownrot specimens indicates definitely that the causal organism enters the beet below the surface of the soil and works upward in the tissues, eventually destroying the crown. The fungus has not been observed to attack the beet above the soil line.

It is evident that the fungus is unable to destroy the outer corky cells of the beet root, but gains entrance to the inner tissue at a definite point and works tangentially just beneath this outer layer. As the fungus eats its way from the point of entrance the outer tissues, due to killing and subsequent drying out of the cells beneath, sink in such a manner as to produce the circular lesion with its very definite undulating contour of alternating raised and sunken concentric "rings" (Pl. 4). The lesions appear first as a small, brown, sunken spot with a minute perforation in the center (Pl. 8, D). The first definite concentric "ring" which is considerably sunken below the central area and usually dark brown in color is noted before the lesion reaches a diameter of $\frac{1}{8}$ inch. With continued enlargement a second and somewhat broader "ring," less sunken and much lighter in color, results. Similar concentric areas are developed alternately until the fungus reaches its limit of lateral spread. Individual lesions resulting from a single point of infection may obtain a size of from $\frac{3}{4}$ to 1 inch in diameter and develop as many as eight alternate "rings" (Pl. 4). When, however, adjacent lesions become confluent, as they frequently do (Pl. 4; 8, D; larger lesions result which may in severe cases cover a large part of the root surface. In such cases large concentric rings are produced, which become common to a number of centers of original infection (Pl. 4; 8, D; 9, A, B).

Another characteristic feature of the disease results in cases where infection occurs at or near the apex of the root. The root in such an event is usually severed at the point of infection and the fungus advances upward, producing the typical dryrot with resultant concentric rings which may encircle the entire root (Pl. 7, B). Again, cankers may occur with such frequency as to girdle completely the root (Pl. 8, A).

The distinctive feature of the contour, as shown in Plates 4 and 8, D, is obtained usually before the fungus penetrates deeply into the tissue of the beet and before a serious rupture of the outer layer occurs. With

[1] Reference is made by number (italic) to "Literature cited," p. 52.

the drying out and final cracking of this outer covering the fungus, possibly because of a better oxygen relation, eats radially into the beet, producing deep cankers (Pl. 5 and 6). The decaying tissues rapidly dry out as the fungus advances inward, leaving the cavity partially filled with a dry, pithy residue. Frequently the content of the canker appears as a definite plug, which, upon wetting, may be removed intact from the cavity of the canker (Pl. 9, D).

Except for slight cracking, the outer layer of dead cells remains entire and furnishes a definite covering until the lesion has reached approximately its limit of tangential spread. As the cells of this outer covering finally dry out the central perforation enlarges and ultimately gives rise to a definite crack which may extend the entire diameter of the lesion (Pl. 4; 8, D; 5). Frequently adjacent cracks become confluent, resulting in large characteristic fissures, which in severe cases of the disease may obtain from 2½ to 3 inches in length and from ½ to 1½ inches in depth (Pl. 5, 6). With numerous points of attack the beet by harvest time is converted into a dry, brittle shell filled with a pithy mass of host and fungous débris (Pl. 6).

During the season careful study was made of a large number of the beets taken from each of the different fields in which the dryrot had been found. In all cases the characteristic cankers exhibited the presence of the sterile stage of *Corticium vagum.* This fungus, it was found, may be obtained regularly in a pure form from any part of the typical canker, provided the outer covering of the lesion is not previously destroyed. The brown layer separating the normal from the diseased tissue (Pl. 8, A–C) has never failed to yield the fungus free from other organisms, and even from the open lesions cultures have been obtained with remarkable ease and regularity. The degree to which other organisms are found to be excluded is phenomenal.

To determine the etiological relation of the fungus, inoculations were made September 3 on partially grown beets. In the process of inoculation the soil was removed to a depth of approximately 4 inches from 21 beets in each of five rows. Each of the 21 beets in the first row was punctured a number of times with a sterile needle, and the inoculum, consisting of the beet fungus, grown for several days on potato agar, was then scattered throughout the soil as the latter was replaced about the beet. Row 2 was inoculated exactly as row 1 except that in place of needle punctures slight incisions were made in the beet by the use of a sterile scalpel. The beets in rows 3 and 4 were wounded as in row 1, and the soil was inoculated with two different "strains" of *Corticium vagum.*[1] Row 5 was left uninoculated, and the beets after wounding as in rows 1 and 2 were covered and grown as controls. All the wounded

[1] These "strains" were obtained from the surface of a potato tuber in 1918 and have proved virulent on potato stems in both sterilized and unsterilized soil.

beets in the control row healed normally. Infection occurred on but one beet in rows 3 and 4. The other beets in these two rows healed as perfectly as in row 5. The results of inoculation with the sugar-beet strain of the fungus in rows 1 and 2 are given in Table I. The types of lesions produced as a result of atrificial inoculation are shown in Plate 9, A. D.

TABLE I.—*Number of lesions on sugar beets inoculated with the sterile stage of Corticium vagum*

Beet No.	Row 1, needle puncture.	Row 2, incision.
1	23	8
2	7	0
3	11	3
4	13	13
5	8	8
6	8	2
7	17	6
8	14	2
9	11	6
10	17	4
11	6	4
12	17	6
13	13	7
14	17	8
15	20	3
16	19	2
17	10	6
18	15	4
19	11	4
20	12	0
21	16	0
Total	285	96
Average	13.5	4.5

Instructions for the inoculating of sugar beets with the beet fungus without puncture or incisions were not followed. As a result the question as to the ability of *Corticium vagum* to attack the sugar beet independently of other agents remains unsettled. It is quite conceivable that sugar-beet root aphis (*Pemphigus betae* Sloane) and other insects so prevalent in the soil may serve an important function in the initial entrance of the fungus. Having once gained access to the lower tissue, however, it appears evident from the results that this particular "strain" of *C. vagum* is capable of producing the type of canker and dryrot with which it is so constantly associated in the field.

The peculiar method of decay, together with the sharp line of demarcation between the diseased and the normal tissue (Pl. 8, A-C; 9, C–F), provide the most distinctive characteristics of the disease. A dark brown, watery layer invariably separates the dry, decayed mass occupying the cavity of the canker from the normal host tissue beneath. This layer

when examined under the microscope is found to be composed of masses of hyaline, vigorously growing young hyphae ramifying through and between the rapidly decaying host cells. It is in this advance layer that the major portion of the tissue destruction occurs. The brown layer advances uniformly inward by additions from the normal host tissue, while the outer surface of the layer rapidly dries out and constantly contributes its substance to the pithy mass occupying the resulting cavity of the canker. The thickness of the layer is dependent largely upon the rate at which the moisture is lost from it outer surface as the fungus eats its way radially into the normal tissue. No evidence of direct penetration of the normal cells by the fungus has been found. On the other hand, it appears that dissolving enzyms precede considerably the advancing mass of young hyphæ (Pl. 8, C).

This method of tissue destruction resembles in a very definite way that described by Ramsey (*5*), by which *Rhizoctonia solani* Kühn attacks and produces a definite pitting of the mature potato tuber. A similar process of decay is described by Atkinson (*1*) for the "sore shin" of cotton. He states that—

> the fungus (*Rhizoctonia solani*) never seems to penetrate far into the living tissues, but kills as it goes, and the tissues become brown, depressed, and present the appearance of a plant having a deep and ugly ulcer at the surface of the ground.

A type of decay most accurately resembling this particular beet rot is described by Richards (*7*) for the potato stem-canker caused by *Corticium vagum.*

The early production of definite cankers by a slow corroding of the normal tissue, finally resulting in a complete dryrot of the beet, suggests a possible name "dryrot canker" for the disease here described.

Various American workers. (*2*; *4*, *p. 243–54; 3*) have reported rootrots of the sugar beet which they attribute to the work of *Rhizoctonia solani* Kühn. It appears difficult at this time, however, to determine the possible relation of these to the particular type of dryrot described in this article. The indefiniteness of the literature on the subject in fact does not justify any general statement as to the possible distribution of the disease.

During September and October of 1920 a preliminary survey [1] was made of the beet-growing districts in four counties of Utah—Cache, Davis, Utah, and Salt Lake. The disease was found in 18 fields of the 51 visited in Cache County and in 3 fields of the 20 surveyed in Davis County. Very serious damage occurred in a number of these fields. No indication of the trouble was found in either Utah or Salt Lake Counties.

The limited survey does not permit of an estimate of the loss to the total sugar-beet crop of the State; nevertheless, the general prevalence

[1] This survey was conducted in cooperation with the Office of Plant Disease Survey, United States Department of Agriculture. The author wishes to express his indebtedness to Dr. G. R. Lyman for this support.

of the trouble would indicate that under more favorable conditions the disease may become a serious factor in beet culture. It is not improbable that a thorough survey may discover the "dryrot canker" in every beet-growing district in this and surrounding States.

Since the appearance of the author's abstract (*6*), Dr. George L. Peltier reports in a letter to the author that he noted during 1920 in Nebraska what appears to be the same trouble. Preserved specimens in the plant-disease herbarium of the Utah Agricultural College show that the disease was collected in Utah as early as 1915.

LITERATURE CITED

(1) ATKINSON, Leo. F.
1895. DAMPING OFF. N. Y. Cornell Agr. Exp. Sta. Bul. 94, p. 231–272, fig. 55, 6 pl.

(2) DUGGAR, B. M.
1899. THREE IMPORTANT DISEASES OF THE SUGAR-BEET. N. Y. Cornell Agr. Exp. Sta. Bul. 163, p. 335–363, fig. 49–63. Some references to the literature of beet diseases, p. 361–363.

(3) EDSON, H. A.
1915. SEEDLING DISEASES OF SUGAR BEETS AND THEIR RELATION TO ROOT-ROT AND CROWN-ROT. *In* Jour. Agr. Research, v. 4, no. 2, p. 135–168, pl. 16–26. Literature cited, p. 165–168.

(4) PAMMEL, L. H.
1891. FUNGUS DISEASES OF SUGAR BEET. Iowa Agr. Exp. Sta. Bul. 15, p. 234–254, 6 pl.

(5) RAMSEY, Glen B.
1917. A FORM OF POTATO DISEASE PRODUCED BY RHIZOCTONIA. *In* Jour. Agr. Research, v. 9, no. 12, p. 421–426, pl. 27–30.

(6) RICHARDS, B. L.
1921. A DRY ROT OF THE SUGAR-BEET CAUSED BY CORTICIUM VAGUM. (Abstract.) *In* Phytopathology, v. 11, no. 1, p. 48.

(7) ———
1921. THE POTATO STEM CANKER. Utah Agr. Exp. Sta. Bul. 178.

PLATE 4

Sugar beet showing typical lesions as a result of natural field infection. Lesions as shown may become confluent and develop common concentric rings. Initial stages in fissure formation are also evident. Photographed August 8, 1920.

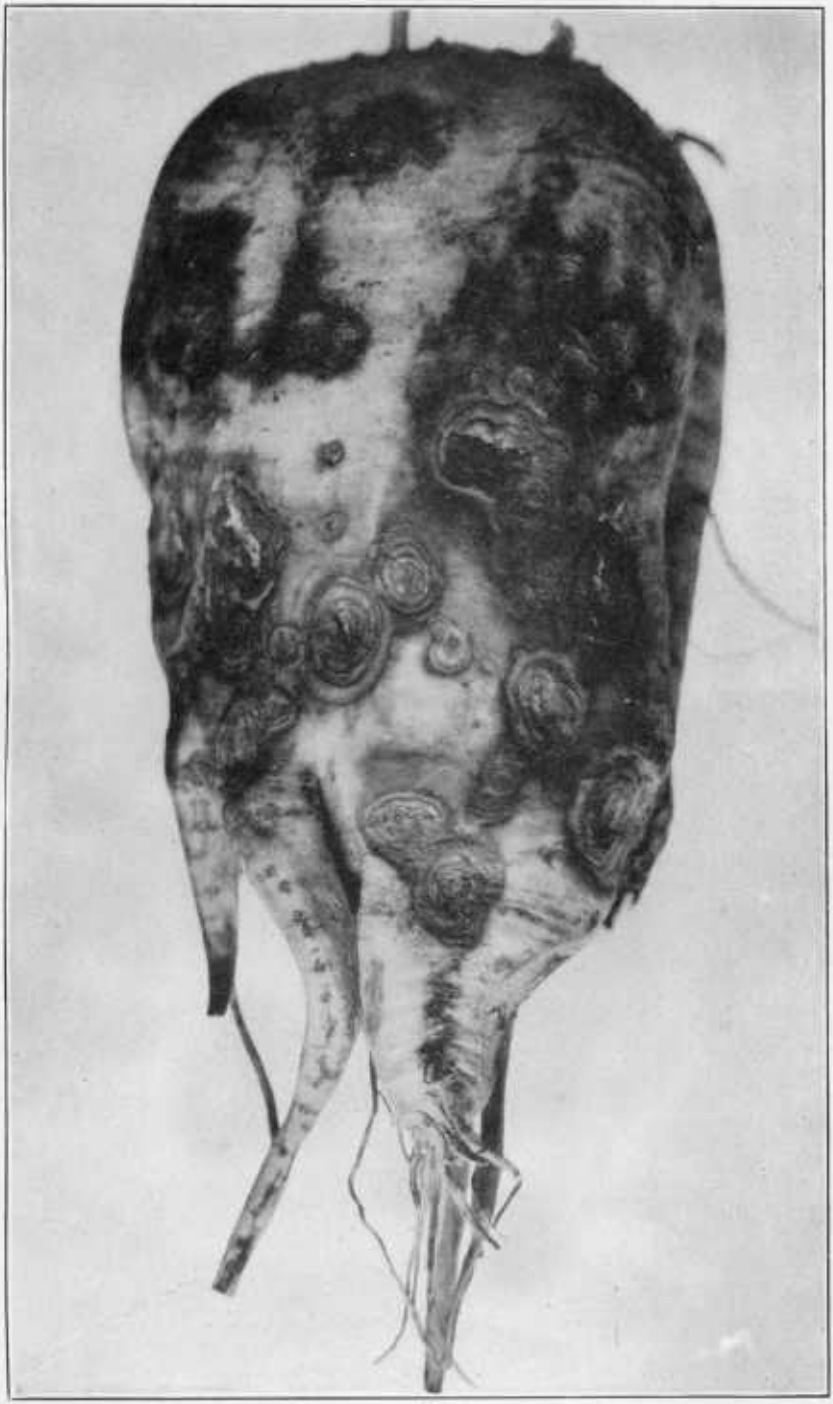
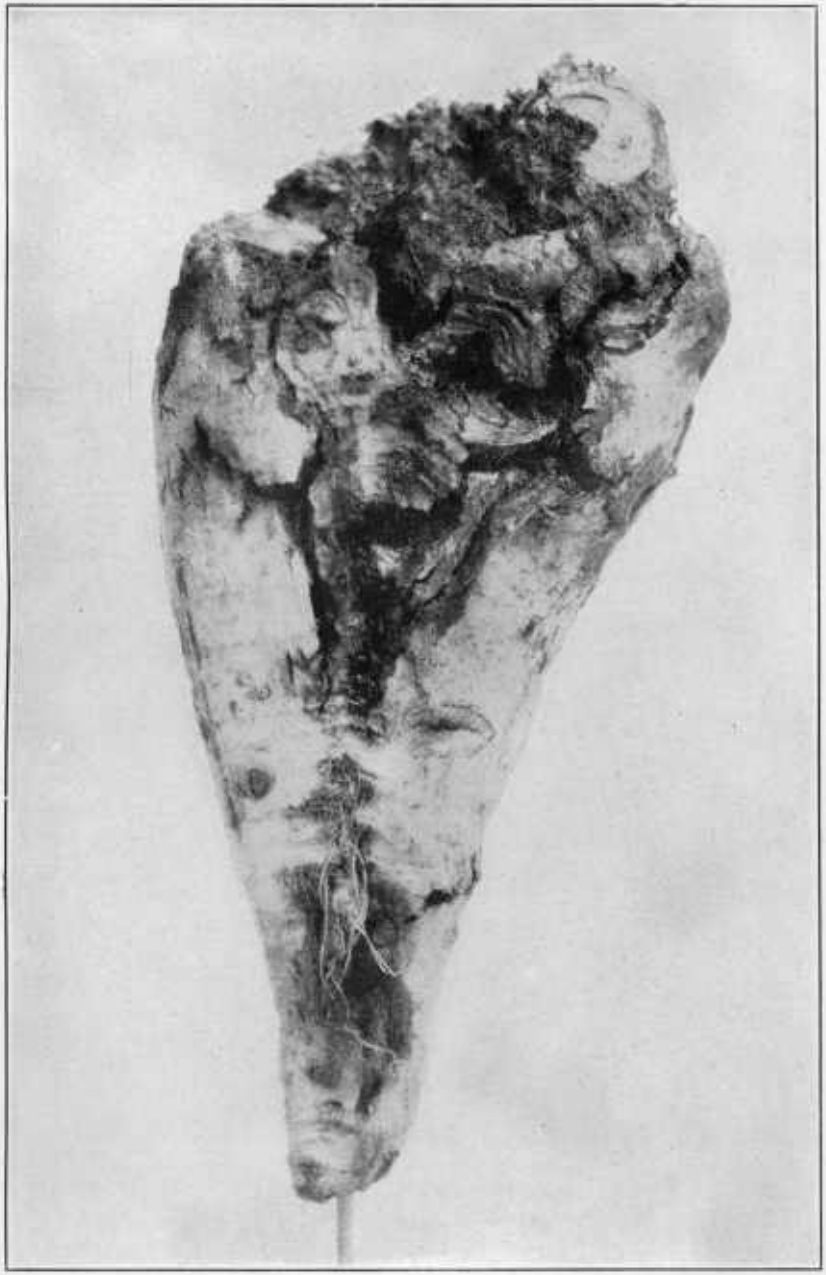


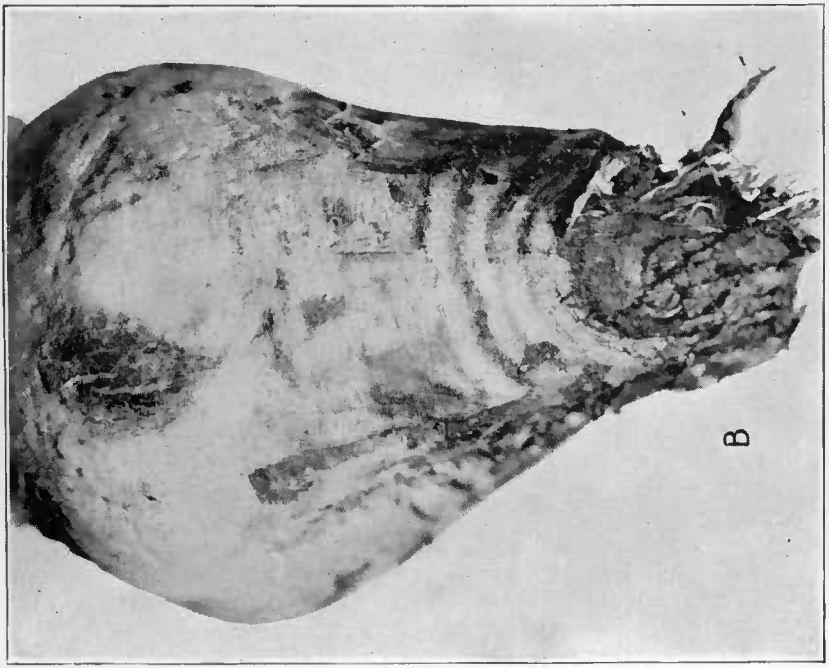
PLATE 5

Sugar beet showing various stages in the rupture of the outer covering of the lesion resulting in the formation of deep fissures. The lesions shown on this particular beet have not reached the size normally attained before rupture occurs.

PLATE 6

Late stage in the development of the disease, showing the beet as a dry shell partially filled with a pithy residue composed of mycelium and dead host tissue. The decay of the crown of this beet is a result of the fungus working upward from the point of infection below the surface of the soil. Remnants of the concentric rings of typical lesions are clearly visible. The cracking of the outer surface of the beet at this stage is shown to extend beyond the lesions.




B

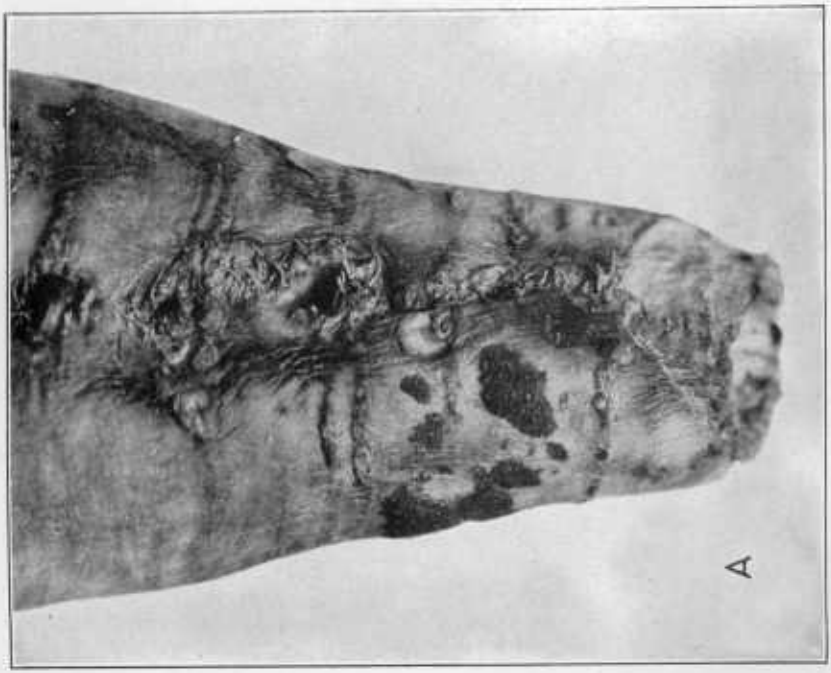

A

PLATE 7

A.—Portion of a sugar beet showing the typical sclerotial masses commonly found adhering to the beets in the infested areas.

B.—Sugar beet showing the result of natural infection near the apex of the beet, at which point the root has been completely severed. The dryrot advancing upward from the initial point of attack has produced the typical undulating contour so characteristic of the small lateral lesions.

PLATE 8

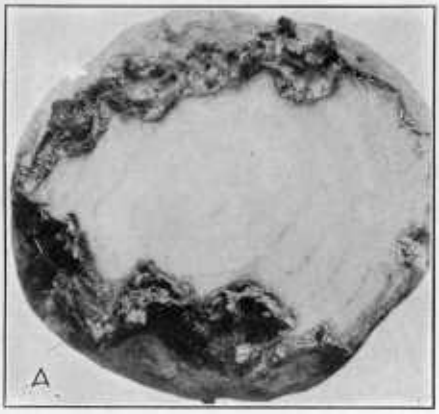
Sections of diseased sugar beets, showing the abrupt drying out between the diseased and healthy tissue. The prominent "feeding surfaces" composed of recently killed cells and the young hypha is clearly evident.

A.—Cross section, showing complete girdling of the beet by cankers resulting from separate points of infection. In such cases the continued penetration of the fungus may completely sever the root at the line of greatest infection.

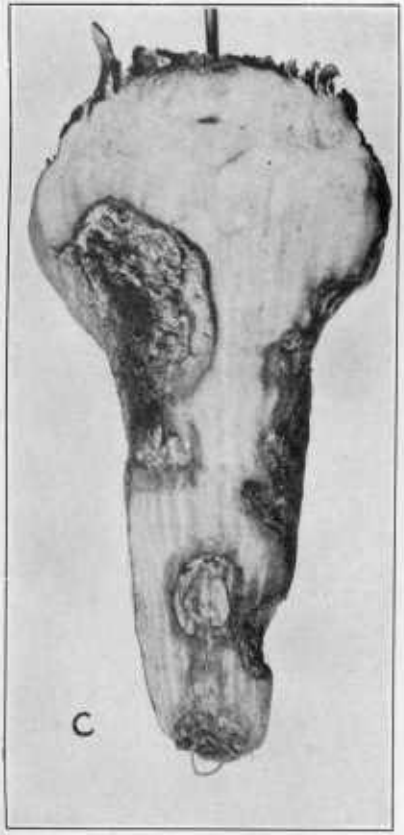
B.—Longtitudinal section of diseased beet, showing various stages of decay and the pulpy material partially filling the cankers.

C.—Sugar-beet crown, showing the definite type of crownrot caused by the fungus worked upward from a point of infection below the soil surface (Pl. 6). A small region of healthy tissue is shown to which a few sickly leaves were attached.

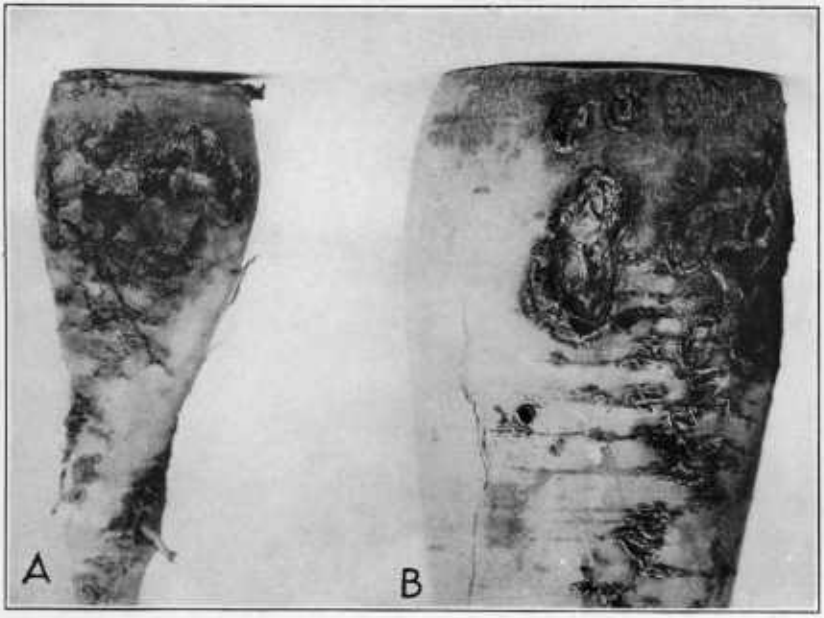
D.—Section of beet surface, showing progressive stages in the development of the lesions resulting from natural infection. The earliest visible stage is shown to exhibit a slight perforation of the outer surface at the center of the lesion. This small opening, present in all lesions, gradually enlarges with age and finally results in the large fissures (Pl. 5). Various stages in the coalescence of lesions are especially evident.

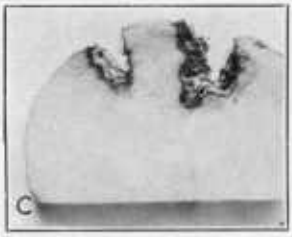

A

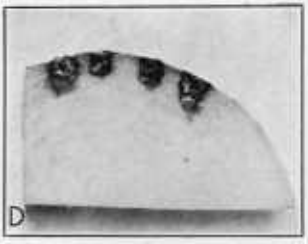

B


C


D


A
B


C


D


E

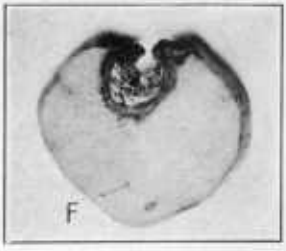

F

PLATE 9

A, B.—Beets showing typical lesions produced by artificial inoculation. Needle punctures through which the fungus entered permitted of rapid drying out of the diseased tissue and of an early rupture of the outer layer of cells at the margin of the lesions. A number of the lesions, however, show the concentric rings so characteristic of the disease produced by natural infection. Cross sections of these lesions are shown in C and D.

C, D.—Cross sections of the lesions in A and B. The lesions in C disclose the more advanced stage of the disease wherein the outer layers of cells are broken down. In D the outer layers of the lesions are more or less entire.

E, F.—Cross sections of cankers resulting from natural infection in the field. A more advanced stage is shown than in C and D; otherwise the lesion produced by the natural and artificial method of inoculation appeared identical.

ADDITIONAL COPIES

OF THIS PUBLICATION MAY BE PROCURED FROM
THE SUPERINTENDENT OF DOCUMENTS
GOVERNMENT PRINTING OFFICE
WASHINGTON, D. C.
AT

15 CENTS PER COPY

SUBSCRIPTION PRICE, $3 PER YEAR

▽