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CHARCOAL ROT OF SUGAR BEET

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INTRODUCTION

CHARCOAL ROT OF SUGAR BEET (*Beta vulgaris* L.), caused by *Macrophomina phaseoli* (Maubl.) Ashby, was found in August, 1932, in Sutter County in the Sacramento Valley and subsequently near Davis, Marysville, Walnut Grove, and on Victoria Island in the delta region west of Stockton. The incidence of infection in numerous fields ranged from 8 to 30 per cent (*5*).[4] Field observations indicated that the fungus attacks half-grown and mature sugar beets during the season of prevailing high temperatures and is probably confined to the hot, interior valleys. It is not known to cause damping-off of sugar-beet seedlings. Inspection of sugar-beet plantings in the cool, coastal valleys has shown them to be free from infection.

In studies of a seedling blight of beans (*Phaseolus vulgaris* L.) caused by this fungus, Kendrick (*3*) showed that the disease was favored by high temperatures. Later, under controlled conditions, Tompkins and Gardner (*6*) corroborated Kendrick's results and found that the fungus from charcoal rot of sugar beet grew throughout a temperature range of 12° to 37° C, with an optimum at 31° and was pathogenic to bean seedlings at high temperatures.

A brief discussion on symptoms of the disease, the causal organism, and pathogenicity of the fungus is presented in this paper.

SYMPTOMS OF THE DISEASE

The leaves of diseased plants show pronounced wilting and eventually turn brown and die. Dead leaves remain firmly attached to the crowns. When an infected plant is pulled from the soil, the symptoms of charcoal rot are distinctive enough to readily differentiate it from all other known root rots of sugar beet. Externally, infection is usually confined to the crown region as indicated by brownish-black[5] lesions of irregular size and shape (fig. 1) and with a silvery sheen. On old lesions the periderm is very thin, papery in texture, and loosely attached to the underlying

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[2] The writer is indebted to Dr. M. W. Gardner for advice and assistance.

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[4] Italic numbers in parentheses refer to "Literature Cited" at the end of this paper.

[5] In determination of root discoloration, Ridgway's (*4*) system of color nomenclature has been followed.

tissues. Under slight pressure it cracks and becomes detached from the root, exposing dry, black, carbonaceous masses of sclerotia (fig. 1).

When examined in cross section soon after infection, the outer or advancing part of a lesion is mustard yellow but later the inner or older

Fig. 1.—Natural infection of sugar beet by *Macrophomina phaseoli;* an advanced stage of infection, showing black lesions with a silvery sheen, which completely involve all of the crown and most of the taproot tissues. The thin, papery periderm has been partly ruptured on the crowns, exposing masses of black sclerotia directly beneath.

part changes to buffy citrine. These colors merge irregularly into each other, with no sharp line of separation (fig. 2). Occasionally the infected tissues may be a uniform buffy citrine. The advancing margin of a lesion, next to apparently healthy tissues, is undifferentiated in color from the tissues invaded earlier and has no distinctive dark band such as characterizes the root rot of sugar beet caused by *Phytophthora drechsleri* Tucker (*7*, fig. 2, *C*). After the entire root has become invaded, the tissues

turn in color from buffy citrine to old gold and finally to brownish black. In the late stages of decay, masses of black sclerotia largely displace the periderm and parenchymatous tissues, forming in pockets or cavities of

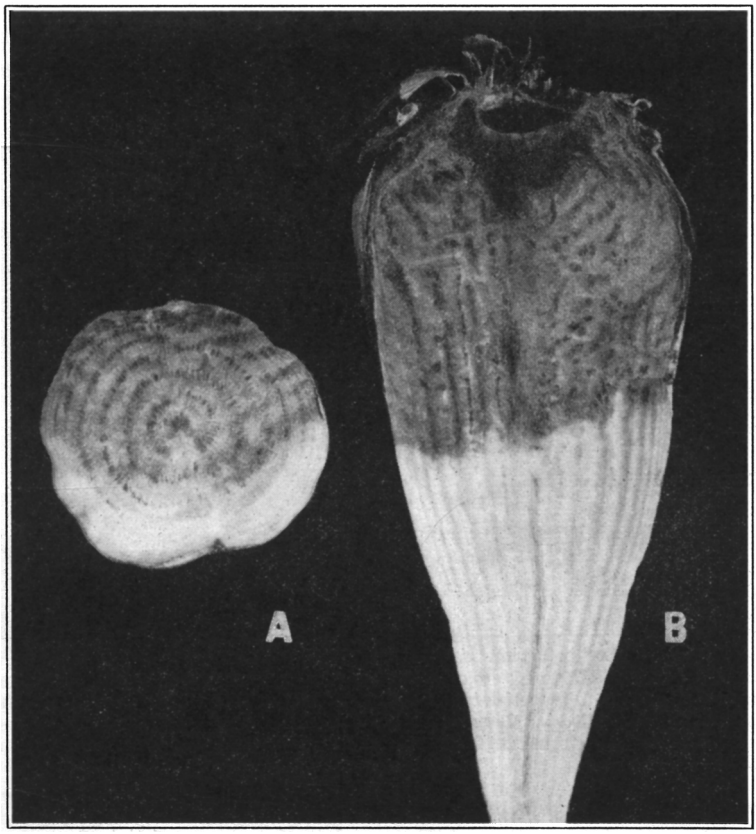

Fig. 2.—Natural infection of sugar beet by *Macrophomina phaseoli: A,* cross section of diseased taproot; *B,* longitudinal section of diseased taproot showing a large mass of black sclerotia at the crown.

irregular size and shape immediately beneath the periderm and extending inward for several centimeters; they may also be found scattered irregularly throughout the mustard-yellow tissues, in marked contrast. Eventually, sclerotial masses occupy the pith and only the vascular elements retain their identity (fig. 3). Completely invaded sugar beets shrink, tend to become mummified, and are of no value for extraction.

Microscopic examination of sections of the infected tissue from inoculated roots stained with magdala red and fast green showed that the mycelium of the fungus was confined to the intercellular spaces.

THE CAUSAL FUNGUS

Tissue fragments from the advancing edge of lesions on approximately 200 sugar beets from various localities were planted on prune agar in petri dishes which were then incubated at room temperature. Colonies containing colorless mycelium usually developed within 24 hours, with but scanty aerial growth. In 36 hours, the medium was darkened by the

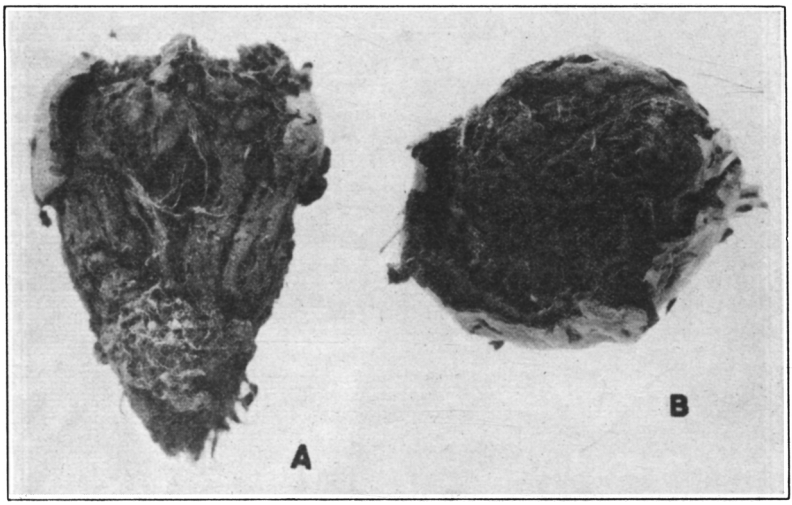

Fig. 3.—Natural infection of sugar beet by *Macrophomina phaseoli: A,* longitudinal view and, *B,* top view of crown, showing symptoms typical of the final stages of decay. The periderm has been completely ruptured and, with the parenchymatous tissues, largely destroyed, exposing vascular elements and masses of black sclerotia. Heavy shrinkage and mummification are not uncommon.

formation of sclerotia which in general were small, spherical, and black and rather evenly distributed. Pure cultures were established on prune agar slopes in test tubes by mass transfers of mycelium and sclerotia.

Six isolates of the fungus from diseased sugar beets collected near Stockton, Sutter Basin, and Walnut Grove were grown on potato dextrose agar, pH 5.6, and incubated at 28° for 14 days. A total of 100 sclerotia from each isolate were measured. The diameters of sclerotia ranged from 46.2 to 146.3 microns, with a mean diameter of 73.8 to 87.2 microns. These isolates, therefore, fall within the limits of Haigh's C group, in which the diameter of the sclerotia is 120 microns or less, and, according to the work of Ashby (*1*) and of Haigh (*2*), should be designated as *Macrophomina phaseoli* (Maubl.) Ashby, although no isolate of the fungus from sugar beets has produced any pycnidia.

PATHOGENICITY OF THE FUNGUS

Healthy sugar-beet roots were washed in tap water, rinsed in three changes of sterile distilled water, and allowed to dry. Two small areas on opposite sides from the root sutures were washed with 95 per cent alcohol, after which small cubes of tissue, averaging $\frac{3}{16}$ inch in size, were removed with a flamed scalpel. Small squares of prune agar containing

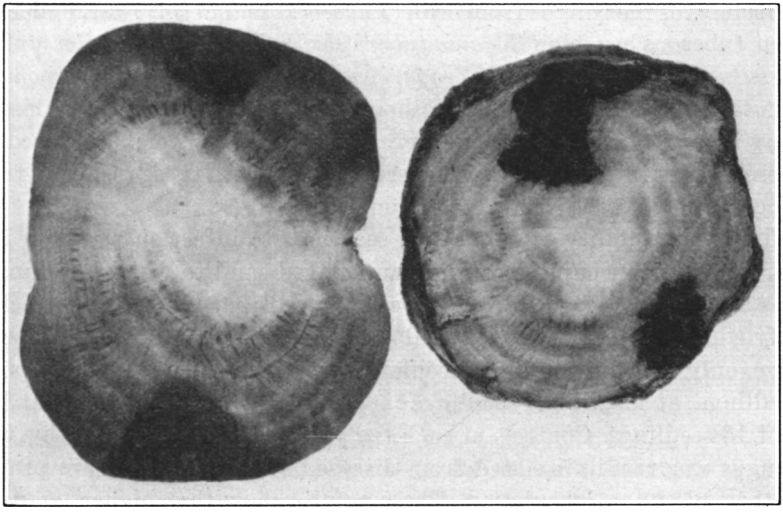

Fig. 4.—Artificial infection of sugar beet by *Macrophomina phaseoli;* cross sections of roots almost completely invaded by the fungus. The black areas on opposite sides represent the sites of inoculation wells and are packed with masses of black sclerotia. A small amount of healthy tissue remains in the centers of the cross sections.

new growth of mycelium and sclerotia were then inserted into these wells, after which the openings were covered with adhesive tape to prevent desiccation. Cultures from Walnut Grove, Sutter Basin, and Stockton, respectively, were tested for pathogenicity. Noninoculated controls received the same treatment except that sterile prune agar was substituted for the inoculum.

After inoculation, all sugar beets were placed in large moist chambers consisting of 5-gallon tin cans with pie tins for covers. Each can was provided with a wire-mesh platform to prevent contact of the roots with the water in the bottom of the can. The inoculated sugar beets were incubated at room temperature (20° to 23° C). After 42 days, 32 roots of 37 inoculated with the Walnut Grove culture, 8 roots of 9 inoculated with the Sutter Basin culture, and 3 roots of 4 inoculated with the Stockton cul-

ture, became infected (fig. 4). The noninoculated controls remained healthy. The fungus was reisolated in pure culture from all diseased sugar beets. The reisolated fungus proved pathogenic upon inoculation into healthy sugar beets.

Six sugar beets were inoculated by placing inoculum on the unwounded periderm under aseptic conditions and were held in moist chambers. After 15 days, 5 roots were infected and the fungus was reisolated. This suggests that the fungus may penetrate the unwounded periderm.

Isolates of the fungus from bean (*Phaseolus vulgaris* L.) var. Red Mexican, tuberous begonia (*Begonia tuberhybrida* Voss), cotton (*Gossypium hirsutum* L.), strawberry (*Fragaria* sp.), and sweet potato (*Ipomoea batatas* Poir.) proved highly pathogenic to sugar-beet roots within 15 days after wound inoculations were made. The symptoms produced by these isolates in sugar-beet roots were identical with those resulting from inoculation with the isolates from sugar beet.

The susceptibility of sugar-beet seedlings to infection was tested in paraffined paper cups containing autoclaved sand to which the fungus was added (*6*). After 7 days, it was observed that the fungus attacked cotyledons as well as the roots and stems of seedlings. At room temperature, only 3 of 56 seedlings inoculated were infected; at 25° C, 3 of 53 seedlings; at 28°, 9 of 31 seedlings; at 31°, 21 of 25 seedlings; and at 34°, 8 of 13 seedlings. Controls at each temperature continued healthy. The fungus was readily isolated from diseased seedlings and again proved pathogenic upon inoculation. These results show that higher temperatures are especially favorable to infection.

SUMMARY

A crown rot of sugar beet, caused by *Macrophomina phaseoli* (Maubl.) Ashby, is described.

The disease occurs only in the interior valleys of California and is apparently dependent upon high temperatures.

Infection of sugar-beet roots and seedlings was obtained in the laboratory with different isolates of the sclerotial form of the fungus from sugar beet.

Infection of sugar-beet roots was also obtained in the laboratory with isolates from other hosts.

The optimum temperature for growth of one of the isolates from sugar beet was shown to be approximately 31° C.

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