DRY ROT OF GLADIOLUS CORMS

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The term ''dry rot'' was first used by Wallace (9), who recognized four diseases of gladiolus corms, namely, hard rot, dry rot, soft rot, and scab. Although Wallace states (9, p. 69) that he was probably including two distinct diseases under the name ''dry rot,'' his brief description of one of the two fungi, isolated from corms from Germany and New York State, leaves no doubt as to the identity of the disease with the one here described. With the termination of the study by Wallace, Fitzpatrick (3) gave some attention to gladiolus diseases and throughout his study restricted the term dry rot to the disease as it is now known. The writer (4) adopted the terminology of the above-mentioned workers, as did Drayton (2) in presenting the most extensive account of the disease thus far published.

Although mention of gladiolus diseases is to be found in several publications antedating the articles cited here, the meager descriptions of both disease and pathogene and the absence of infection experiments create doubt as to what diseases were involved. Wallace (9, p. 14) states that ''the disease reported by Robinson (8, p. 139) is probably identical with that which we are calling dry rot, specimens of which were received from Germany and also from New York State. At least the presence of a fungus with Rhizoctonia-like mycelia in the tissue suggests this.'' Aside from this doubtful instance the dry rot disease can not be identified in literature prior to 1909.

Dry rot probably occurs generally wherever gladioli are grown. Hundreds of specimens have been received from growers in the United States and Canada and the disease has been found on corms from England, France, and Holland. Dry rot, hard rot (5), and neck rot (7) are the most common and prevalent diseases of the gladiolus, a plant which has greatly increased in popular favor and economic importance during the past two decades. The losses from these three diseases are large, especially in years of high precipitation and when there is unfavorable weather at the time of harvesting the corms.

SYMPTOMATOLOGY

All parts of the plant below ground may be infected, and a stem-rot at the surface of the soil involving the lower inch or two of the stem may

often be observed. This rotting of the stem frequently results in a pre-
mature yellowing of the tops, followed by drying and death. The plant
may or may not bend at the rotted area, depending on the extent of decay,
which in turn is dependent on the amount of moisture present. Usually in
cases of stem-rot the corm will be found to be affected, but instances may be
observed in which the corm appears quite free from lesions. It is possible
that in these cases the husks or sheathing leaf bases of the parent corm are
diseased. In stem-rot the outer sheathing leaf bases are characteristically
attacked first. Minute, black sclerotia of the pathogene are usually present
on diseased stems.

As in the case of hard rot (5, p. 157), but less frequently, yellowing of
the foliage and death of the plant may result from the decay of the parent
corm affected with dry rot when planted. This usually happens about mid-
season when temperatures are high and the soil dry. Saprophytic fungi are
frequently involved in the final destruction of the old corms, although the
dry rot advances more rapidly under the favorable environment of the soil
and may of itself cause the death of the plants.

The dry sheathing leaf bases or ''husks'' of stored corms affected with
dry rot may show evidences of disease by being abnormally dark in color
and brittle. These darkened areas are usually irregular in outline, rarely
being approximately circular as are the lesions on the corms. There is a
tendency for the affected husks to split longitudinally, and pieces may be
broken away easily, exposing the corm beneath (Plate X, B). The husk
lesions may or may not be immediately over lesions on the corms. In most
cases it is necessary to remove the husks to determine whether or not a corm
is diseased.

On removing the husks it will be found that diseased corms charac-
teristically bear many small lesions which range in size from mere dots to
areas about one centimeter in diameter (Plate X). Larger individual areas
are relatively uncommon, but are to be found and may involve half or more
of the corm. Frequently, numerous lesions coalesce to form a large lesion,
which in advanced cases may involve the entire corm and reduce it to a dry
and shriveled mummy. When this condition exists it is usually still pos-
sible to trace the outlines of individual lesions. Areas more or less healthy
may be left insulated in large areas of diseased tissue. Not uncommonly
the lesions occur along the juncture of the husk and the corm, forming rings
of diseased tissue. This would seem to indicate that the husk was first af-
fected, the rot spreading from the husk to the corm. On the other hand,
diseased corms with apparently healthy husks are commonly found.

The individual lesions are approximately circular in outline and appear
first as minute, reddish brown spots, usually on the side and lower half of
the corm but not infrequently on the upper half as well. The line of

demarcation between healthy and diseased tissue is rather sharp. As the lesions increase in size, the centers become sunken, the color usually deepens to black, and the margins become more definite. The sunken centers of the lesions probably result from the drying of the tissue. The margins of the lesions, especially noticeable in the larger and older diseased areas, are slightly elevated. The consistency of diseased tissue is characteristically corky.

As a rule, the lesions do not extend deeply into the corm, the range being from 1 to 5 or 6 millimeters and the average less than 3 millimeters. Usually the diseased tissue can be separated readily from the apparently healthy tissue beneath. This symptom is not restricted to the dry rot disease but is at least equally true of hard rot, Fusarium rot, and scab. Evidently, in this stage of the advancement of the disease, the corm has the advantage in being able to callous over the diseased area and, for a time at least, prevent further advance of the invading hyphae.

A more detailed study of the larger and older lesions will sometimes disclose the presence of minute black bodies buried in the diseased tissues. These are the sclerotia of the pathogene. They are sometimes present on the husks (Plate X, E) but are not found in a sufficiently high percentage of instances to be of much importance as an aid in diagnosis.

Cormels produced by diseased plants are commonly affected. At the time of harvesting the corms it is frequently possible to distinguish diseased cormels by their darker color, but this difference in color is less marked on drying, if at all evident. The lesions to be found by removing the hard outer coats are similar to those found on the corms, and mummification during storage results in a high percentage of cases.

<center>ETIOLOGY</center>

The dry rot of gladiolus is caused by the fungus **Sclerotium gladioli** n. sp.

The pathogene is readily obtained in culture by the usual methods of making isolations from infected tissue. It is a relatively rapid grower and will tolerate a wide range of media and cultural conditions. As stated below, growth varies somewhat with different media, but the variations are not great enough to render difficult the identification of the fungus.

Sclerotia similar to those produced on stems and husks, and sometimes within the infected tissues of the corms, are readily formed in culture in from 10 to 14 days (Plate XI). Measurements of 100 sclerotia produced on potato agar[1] in a petri dish after three weeks at about 20° C. gave a diameter

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[1] In preparing the potato agar 200 grams of potato were used per litre, to which were added 20 grams of agar. For cornmeal agar, 50 grams of cornmeal were used per litre, with 2 per cent agar. Media did not contain sugar unless indicated.

range of $90–300 \times 90–240$ μ, with an average of 191×164 μ; on potato agar in a petri dish, 27 days old, at about 20° C., a range of $125–261 \times 125–208$ μ, with an average of 188×161 μ; on cornmeal agar[1] in a petri dish, 27 days old, at about 20° C., a range of $115–229 \times 105–208$ μ, with an average of 182×159 μ; and for 100 sclerotia formed on the stem of a diseased plant (natural infection) growing in the greenhouse, measurements showed a range of $94–156 \times 73–135$ μ, with an average of 119×106 μ. Drayton (2, p. 206) gives the range as 187×156 to 93×93 with an average of 158×118 μ, the source of the sclerotia not being given. Evidently they vary somewhat, depending on conditions under which they are formed.

The sclerotium is black. It consists of a definite cortex of thick-walled cells, from one to two cells in width, surrounding thin-walled parenchymous cells (Fig. 1, A). On crushing a sclerotium, small globules of an oily substance are exuded. Although growths are obtained from planting sclerotia on artificial media in petri dishes, there is no evidence that the sclerotia, and not attached or adhering hyphae, initiate the culture.
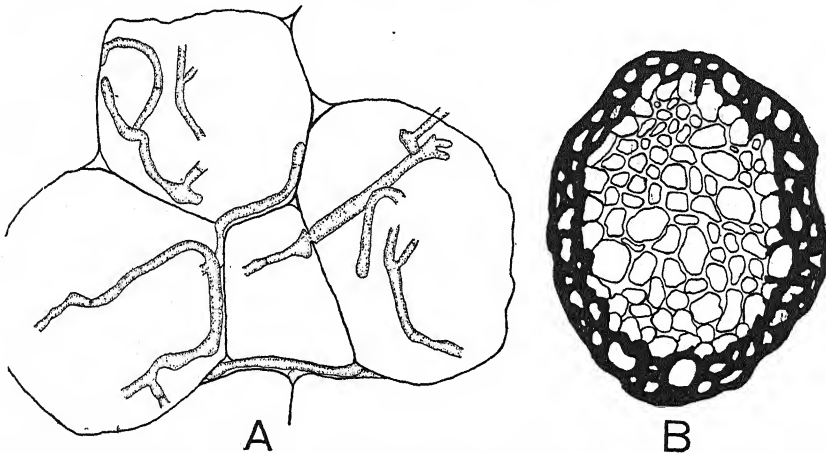

FIG. 1. A, Mycelium is inter- and intra-cellular. ×350. B. Section of sclerotium, from culture on potato agar, showing definite cortex and parenchymous medulla. ×300.

The only spores produced appear to be microconidia. These have been observed only in culture. These microconidia were first found in a culture of the fungus on a synthetic medium,[2] in test tubes, and later on potato agar, especially when adjusted to a pH of about 5 by the addition of hydrochloric acid. The cultures in which microconidia have been found

[2] Water 1000 c.c., agar 15 grams, glucose 20 grams, peptone 10 grams, dipotassium phosphate 0.25 gram, magnesium sulfate 0.25 gram, as given by Cook and Taubenhaus (1).

were from 25 to 40 days old. Small white granules, less than a millimeter in diameter (Plate XI, D), appear buried in the medium mostly at the top and back of the slant, adjacent to the wall of the tube. On examination under the microscope these granules are found to consist of numerous spores, borne on verticillately branching conidiophores (Fig. 2, A). The microconidia are approximately spherical, 1.7–3.7 μ in diameter, with an average of 2.5 μ. Attempts to germinate them have failed, and, since they have not been found under natural conditions, the role they play, if any, in disseminating the fungus and in infection is unknown.

The mycelium is septate, and both inter- and intra-cellular. On media used by the writer it is white in color, with the aerial mycelium tipped with clay[3] in older cultures.
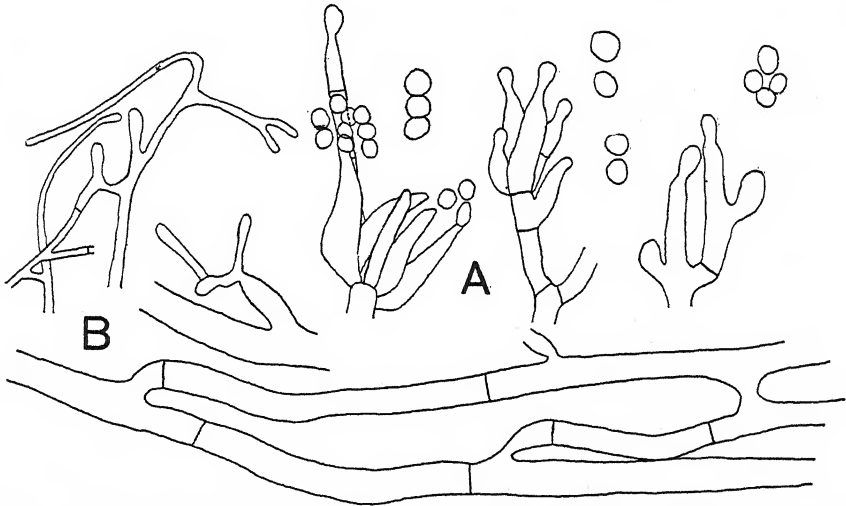

FIG. 2. A, Microconidia and portions of verticillately branching conidiophores. From a 40-day-old culture on potato agar, pH 5.04. ×1500. B, Mycelium from a 7-day-old culture on potato agar to show manner of branching. ×500.

Since no spore stage other than microconidia has been found, it seems necessary to continue the fungus in the form genus *Sclerotium* as was suggested by the writer in 1918 (4, p. 72). Because of the structure of the sclerotia the form genus *Sclerotium* is preferred to *Rhizoctonia*, even though the method of branching of the mycelium suggests to a certain extent that of the latter genus (Fig. 2, B). The species name **gladioli** is here proposed. The formation of sclerotia and microconidia, of the type figured (Fig. 2, A), strongly suggests that the fungus may be a species of *Sclerotinia*, but at-

[3] Designations of color are according to Ridgway, Robert. Color standards and color nomenclature. 44 pp., 53 col. pl. Washington, D. C. 1912.

tempts to bring about further development of the sclerotia have been unsuccessful.

<div align="center">PATHOGENICITY</div>

The ability of *Sclerotium gladioli* to infect the gladiolus and produce symptoms identical with those found under natural conditions has been established through numerous experiments both in the field and under greenhouse conditions. Wallace (9, p. 72), Fitzpatrick [according to Whetzel (10)] and Drayton (2, p. 205) report successful infection experiments. Infection takes place when corms are planted in infested soil, and when the progeny of healthy corms planted in clean soil are inoculated by placing mycelium in contact with them. Sound corms placed in sterilized moist sand in moist chambers and inoculated by pressing bits of medium carrying the fungus against the uninjured surface, when held at a temperature of 25° C., develop lesions in about ten days.

A phellogen layer is formed at the juncture of the diseased and healthy tissue, similar to that formed in hard rot (5, p. 168). Diseased tissue, especially in advanced cases, can be severed from the apparently healthy tissue beneath, the rupture occurring at this phellogen layer. Sections through lesions show from one to many layers of these thin-walled cells indicating progressive stages in the advancement of the fungus into the healthy tissues.

<div align="center">CULTURAL CHARACTERS</div>

Sclerotium gladioli has been grown on a large number of media, both standard and special, the latter being used largely in an attempt to induce sporulation. The fungus is tolerant of a wide range of conditions, and growth is fairly uniform. The mycelium is mostly white, at first appressed and somewhat silky, later becoming more distinctly aerial (in petri dishes) in the outer one-third or so of the growth. The surface of the medium in a standard petri dish is covered in about 7 days, with appressed mycelium in the inner two-thirds of the diameter and aerial mycelium forming a ring covering the outer one-third or so of the surface. Later the area of appressed mycelium is increased somewhat, but the ring of aerial mycelium near and on the wall of the dish persists indefinitely. With age the color of the aerial mycelium is gradually changed from white to clay.[3] Sclerotia appear in from 5 to 20 days, usually in the older parts of the culture following a darkening of the mycelium, although in some instances, *e.g.*, on cornmeal agar without sugar, they appear simultaneously throughout the culture and with no darkening of the hyphae (Plate XI, F).

The growth on oat-mush agar is more rank than on decoctions of potato and cornmeal, growth on the latter being scanty and relatively inconspicu-

ous. Sugars, 2 and 5 per cent, added to plant decoctions increase the growth rate slightly and hasten the formation of sclerotia (Plate XI, E, F). Fructose was found to be slightly better than dextrose and saccharose for sclerotial formation, and 2 per cent as effective as 5 per cent although the growth rate was somewhat increased with the larger amounts. With sugars added to decoctions of potato, cornmeal, and oats, a sclerotial crust is formed, while without sugars the sclerotia stand out as individuals. Zonation is sometimes present in cultures in petri dishes, but is not very conspicuous. Lactic acid, two drops to 15 c.c. of medium, slightly retards sclerotial formation, although the fungus is more tolerant toward a neutral or acid medium than toward one that has been made alkaline by the addition of sodium hydroxide. Saltations result frequently, especially where but a thin layer of medium in petri dishes is available to the fungus.

Cultures of *Sclerotium gladioli* have a very pronounced musty odor which may aid in the identification of the fungus. This odor is strongest in recently isolated cultures, becoming weaker as the growth ages. On several occasions recent isolations of the fungus have either failed completely or made irregular growths when cultures have been confined in closed containers of limited volume. Whether or not there is any relation between these failures and the presence of the substance responsible for the characteristic odor has not been established.

Reaction between cultures from different sources. Among other attempts to induce sporulation by *Sclerotium gladioli* two or more isolations of the fungus were grown at one time in the same petri dish. Although these attempts were failures so far as fruiting is concerned, it was found that certain isolations, when paired, resulted in the formation of a heavy dark line at the junction of the thalli, whereas other combinations did not react in this manner (Plate XI, A, B, C). It was thus possible to divide the cultures from numerous sources into groups, positive and negative in this respect, no two members of either group reacting with each other. The full significance of this characteristic, and whether or not anything other than the question of staling is involved, is not known.

Temperature relations. Cultures of *Sclerotium gladioli* were established by transferring bits of a solid medium carrying mycelium to the center of petri dishes of equal size containing equal amounts of hard potato agar (3 per cent) with 1 per cent dextrose. The medium was tested electrometrically and found to have a pH value of 7. After incubating all of the cultures for 24 hours at 20° C., they were divided into lots of four dishes each and each lot held for 10 hours at the particular temperature at which it was to be kept throughout the experiment. This permitted the fungus to establish itself in the medium in the petri dish and eliminated errors that might

arise from failure to allow sufficient time for temperature adjustments before making initial measurements. The diameters of the thalli were then recorded and the rate and amount of subsequent growth determined by measuring the increases over the original diameters. Four cultures were placed in each of a series of temperature chambers with the following temperatures: 0°, 5°, 10°, 15°, 20°, 25°, 27.5°, 30°, 32°, and 35° C. The diameters of the thalli at 72 hours were taken as a criterion for comparison, the results being represented graphically in figure 3. There is a gradual increase in rate of growth up to the optimum at 25° C., with a rapid drop beyond 27.5° C.
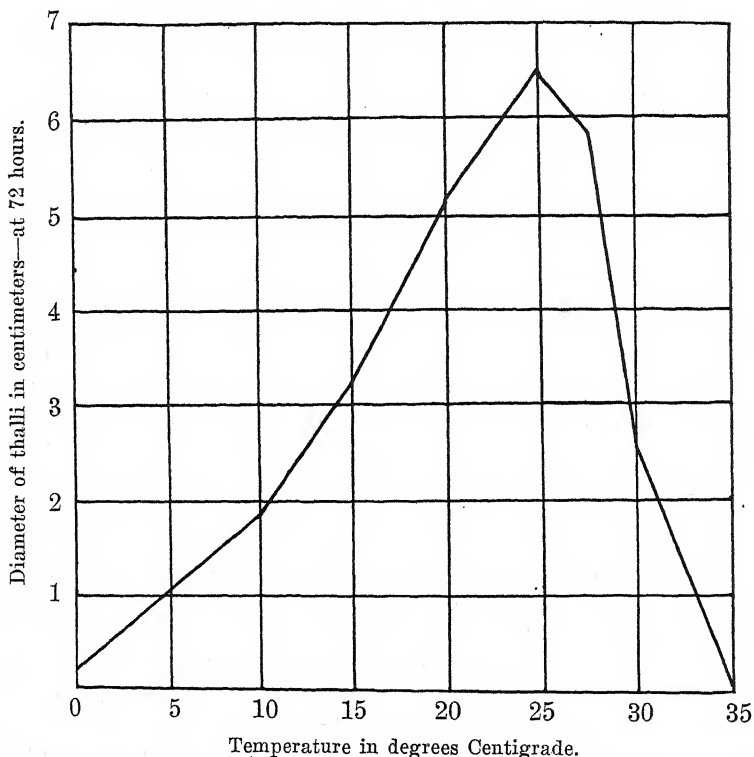

FIG. 3. Relation of temperature to growth of *Sclerotium gladioli* on hard potato agar with 1 per cent dextrose in petri dishes.

LIFE HISTORY

The dry rot fungus may be isolated from lesions on the corms at any time during winter or spring. This shows that the living organism is carried to the soil along with diseased corms at planting time. About 30 per cent of the offspring from diseased corms planted in clean soil are infected. The fungus does not grow directly from the old into the new corm. It either grows through the sheathing leaf bases or else cut into the soil from which

it attacks the newly developing corm.   In connection with inoculation experiments it was observed that the fungus freely enters the soil, and experiments previously reported (5, p. 162, 180) show that the fungus will live in soil in which no gladioli have been grown for at least four years.

The fungus, then, passes the winter in diseased corms, in infested soil in the field, and doubtless also to some extent in infected stems.   Just what rôle is played by the sclerotia, found especially on husks and the lower parts of the stems, is unknown.   Further, since the microconidia have not been germinated, the part they play, if any, in the life history of the fungus remains unsolved.   The fact that diseased corms almost always show numerous lesions suggests the possibility of infection by spores, but no evidence to pathogene, approximately 70 per cent of the progeny will escape infection. since sound corms planted in soil in which no gladioli have been grown have uniformly remained sound and produced disease-free offspring.

As previously reported (5, p. 173), hundreds of healthy corms have been housed over winter with many thousands of diseased corms without becoming infected.   This indicates that *Sclerotium gladioli* is not disseminated in storage.   It has been observed, however, that the percentage of infected corms is increased when considerable soil from the field is taken into storage on improperly harvested corms.   The excess soil holds moisture, delaying drying of the husks, and under such conditions the fungous mycelium may actually grow from corm to corm.   Lesions increase in size during storage especially where the temperature and humidity are high.

CONTROL

In connection with an investigation of the hard rot disease the writer included corms affected with dry rot in the several experiments on control measures (5, p. 172–180).   The treatments of diseased corms with (*1*) formalin at the rate of 1 pint of commercial formalin to 15 gallons of water for 18 hours, (*2*) corrosive sublimate, 1–1000 solution, for 18 hours, or (*3*) chemicals, including sulfur, air-slaked lime, acid phosphate, and soot, in which the corms were rolled and with which they were covered after placing in the row and before covering with soil, were found to be of no value. These treatments were made in the spring before planting.   Similar tests were made in the autumn immediately after digging, at which time the lesions are materially smaller than in the spring.   Failure again resulted. Recently dug corms have also been treated with formaldehyde gas (5, p. 177), with water at 50° C. for one-half hour, and with dry heat at 50° C. for one and one-half hours.   These treatments were unsuccessful in reducing the percentage of diseased corms, and the dry-rot fungus was isolated from numerous lesions, showing it to be viable still.   Drayton (2, p. 208) reports

failure in the use of formalin, mercuric chloride, Uspulun, and Bayer compound, both for the treatment of diseased corms and as a drench for soil in which diseased plants had grown. No satisfactory material for the disinfection of soil has been found.

When corms affected with dry rot are planted in soil free from the pathogene, approximately 70 per cent of the progeny will escape infection. It is evident, then, that soil infestation is one of the most important factors to be considered in connection with control measures.

In an article on Fusarium rot of gladiolus corms (6) the writer has proposed general recommendations for control, involving sorting, crop rotation, proper harvesting and storage, which are applicable to the several corm rots, including dry rot.

SUMMARY

Dry rot is an important disease of gladiolus corms in the United States and Canada, and is known to occur in England, France, and Holland. A description of the disease is given. Corms and cormels become infected in the field, and the rot advances in storage. Plants in the field may die during the season either from a stem rot or the decay of the corms. Soil becomes infested from planting diseased corms, and the pathogene will survive in the soil for at least five years.

The pathogene causing dry rot is placed in the genus *Sclerotium* because of the structure of the sclerotium and the absence of spores other than microconidia. A new species, *gladioli*, is made. The morphological and cultural characters of the fungus are recorded.

On hard potato agar with 1 per cent dextrose, the fungus grows over a range of from about 0° to 35° C. Optimum growth takes place at about 25° C.

Infection was readily obtained on uninjured corms in the laboratory and on those growing in the greenhouse and experimental gardens.

No satisfactory treatment of diseased corms to disinfect them has been found. Crop rotation, avoiding the use of the same soil oftener than once in four or five years, is believed to be of major importance in holding dry rot in check. This practice should be combined with sorting to eliminate diseased corms, together with the proper handling of corms at harvest and during storage.

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EXPLANATION OF PLATES

PLATE X

Corms affected with *Sclerotium gladioli*.   Corms at lower left and upper right show sclerotia on husks.   A, B, C, D, and F, approximately natural size; E, × 2.

PLATE XI

Cultures of *Sclerotium gladioli*.   A, B and C show results of growing paired isolations from different sources on potato agar in petri dishes.   Note reactions, indicated by dark-colored junctures, between A and C, and A and S.   Photographed after 10 days. D, Microconidia produced on potato agar, after 40 days.   Aggregations of spores and conidiophores appear as small, globose, granules, white by reflected light, buried in the medium, as indicated by arrow.   E, F, cornmeal agar with and without 2 per cent dextrose, respectively, after 40 days.   Note effect on formation of sclerotia.

PLATE X

PLATE XI