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Downy Mildew of Tobacco

P. J. ANDERSON

Connecticut
Agricultural Experiment Station
New Haven

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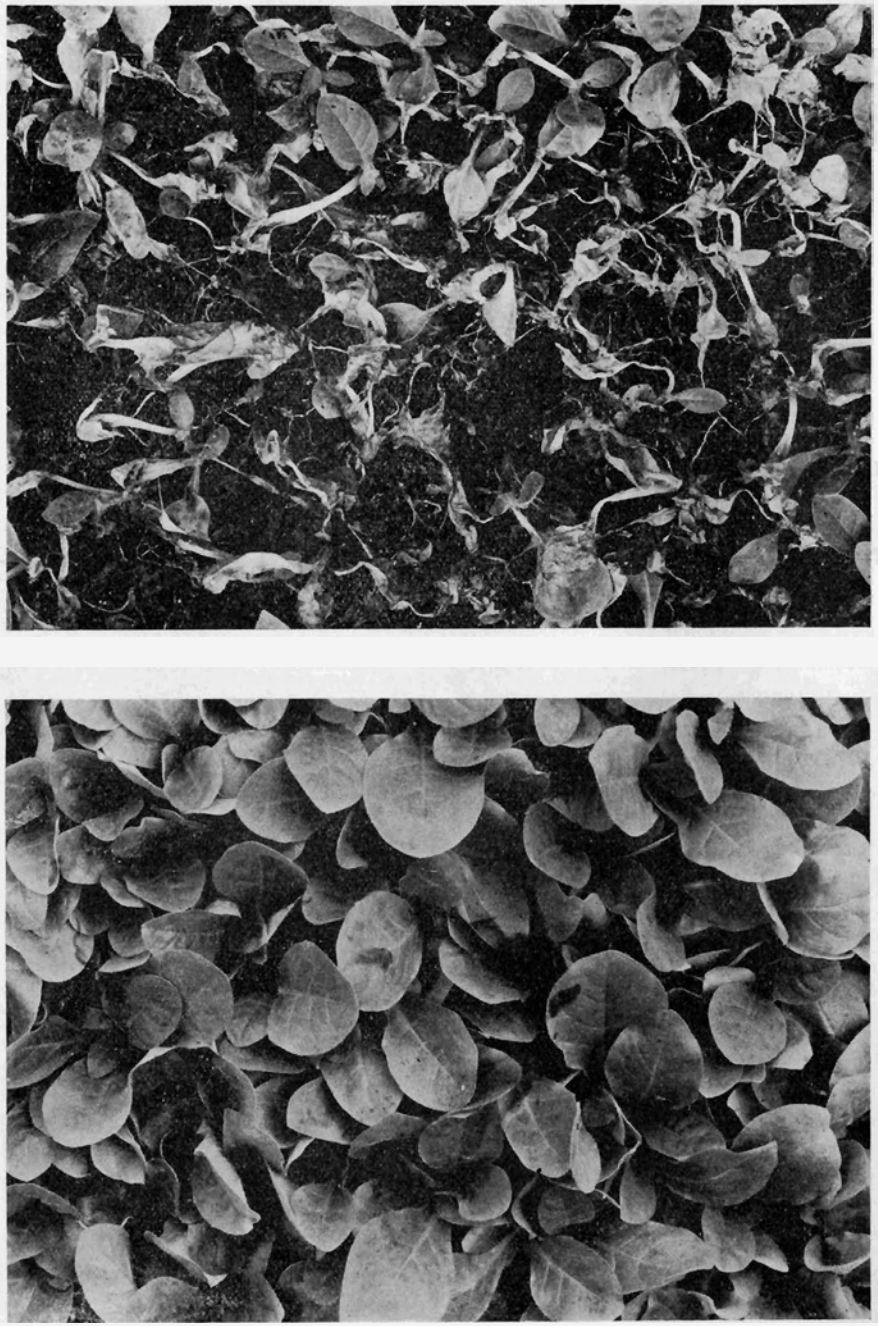

FIGURE 1. A healthy bed of plants contrasted with a diseased bed. Photographs taken from opposite ends of the same bed.

DOWNY MILDEW OF TOBACCO

P. J. ANDERSON

DOWNY MILDEW, a serious disease of tobacco plants, made its first appearance in Connecticut during the late seedbed season of 1937. After causing considerable damage in the beds, it spread to the fields during June, but disappeared with the coming of hot weather in July. However, it is primarily a disease of the seedbeds, and the greatest losses may be expected there. The erratic behavior of downy mildew in other tobacco sections makes it difficult to predict how serious it is destined to become in the Connecticut Valley. Since it is now thoroughly established and distributed here, we must assume that it may recur each year. Therefore, if growers are to insure against heavy losses they must apply the best known preventive or remedial measures to protect their seedbeds.

The purpose of this bulletin is to acquaint the growers with the essential facts about mildew so that they may be able to recognize the symptoms of the disease in every stage, to understand how it spreads and develops, and, most important, to have before them explicit directions for applying the best known methods of control.

Since downy mildew appeared so late in the seedbed season here, the time for conducting experiments on methods of controlling or preventing the disease has been too short to warrant conclusions. Therefore we are obliged to rely on results obtained in other states where the mildew has been prevalent for a longer time and, for the present at least, to use the methods found best in those states assuming that they will be practicable under our conditions in Connecticut[1].

HISTORY

The first published reference to a mildew disease of cultivated tobacco came from Queensland, Australia, in 1890 (59)[2], and a few months later from New South Wales (18). In succeeding years it was reported progressively from all the tobacco growing states of Australia, where it is considered the most serious and destructive of all tobacco diseases (7). Some tobacco growers in Australia said that it was present in the seedbeds of that continent as early as 1860, but since such opinions are usually expressed by some growers whenever any new disease occurs, this early date is questionable.

A similar disease on a wild species of the tobacco genus (*Nicotiana glauca*) was reported from southern California in 1885 (26), and one on another species (*N. longiflora*) from the Argentine in 1891 (56). Still a third was collected on *Nicotiana biglovii* in Nevada (60). There is no positive proof, however, that the disease on any of these wild species of the tobacco genus is the same as the disease of cultivated tobacco. The first record of a similar or identical disease on cultivated tobacco in the United States is furnished by a dried specimen in the fungous collection of the United

[1] The writer gratefully acknowledges the invaluable assistance rendered in correspondence and personal conference by Dr. E. E. Clayton of the Division of Tobacco and Plant Nutrition of the United States Department of Agriculture; by Dr. F. A. Wolf of the Department of Botany of Duke University, and by Mr. E. G. Moss and Mr. Thomas Smith of the Oxford Tobacco Station of the North Carolina Department of Agriculture.

[2] Numbers in parentheses refer to list of publications on p. 81

States Department of Agriculture, collected in Texas in 1906 (51). There is no indication that this caused any damage to the tobacco crop in that state as it is not mentioned in the literature of plant diseases.

The first outbreak of mildew in the United States was in the spring of 1921 when it became widespread in the seedbeds of northern Florida and southern Georgia. At that time there was great alarm lest the shade tobacco industry in those states should be wiped out (51, 52, 53). For some unknown reason, however, the disease did not appear the next year in the destructive proportions anticipated. In fact, it caused no trouble for the next 10 years and did not spread to neighboring states.

In the early spring of 1931, however, it was destructive and widespread in Florida and Georgia, and was reported from Louisiana. Before the end of the transplanting season it had also spread to South Carolina, North Carolina and Virginia. In 1932 it was prevalent in all the tobacco-growing Atlantic seaboard states as far north as Maryland. In 1933 it extended its range to Tennessee and Pennsylvania, and became more widespread in all the states where it was previously found. Since 1933 it has occurred every year in all these states with variable degrees of severity and has spread to Kentucky. The most extensive and destructive epidemic was in 1937.

It was anticipated that sooner or later mildew would reach Connecticut and therefore every suspicious disorder in the beds during recent years has been closely scrutinized but no mildew was found. It is practically certain that there was no tobacco mildew in this state before May, 1937.

The first case observed was on May 25 in one seedbed of a series covering an acre or more in Bloomfield. Since all the leaves on one whole end of this bed were dead and dry, the disease must have started at least a week before. Later it spread to all the beds of this plantation. Within less than a week after the first case was seen, similar spectacular and destructive infections were found in seedbeds on seven farms in Windsor, South Windsor, East Windsor, Manchester, Suffield and Glastonbury. It was widely distributed and appeared on all three types of tobacco. The simultaneous occurrence of the disease in such widely separated spots precludes the probability that it spread from one of these as a center to the others. It seems more likely that all the infections were primary and probably started from spores blown into this region from Pennsylvania or more southerly states where the disease was prevalent this year. Letters of warning with a full description of the mildew were sent to all Connecticut growers. A great deal of publicity was given to it in the newspapers and all suspicious cases were investigated. If there had been other infections besides the seven mentioned, this publicity would have brought them to light, but no others were found during that first week.

Beginning June 6, reports of additional cases came in rapidly and from various quarters indicating that the mildew was now spreading from the seven primary infections. By the twentieth of June it had been reported from about all the tobacco growing towns of Connecticut and the southern towns of Massachusetts, and additional cases were found every day until the seedbed period was over.

The first field infections were reported about the middle of June. Examination of a large number of such fields usually showed that the worst infections were in sections nearest to the seedbeds and there was unmistakable evidence that the spores were blowing from infected seedbeds into the fields. June was a rainy month and frequently the leaves had no opportunity to dry off for several days at a time, thus furnishing ideal conditions for the disease to spread. This weather continued until the first week in July when it became hot and dry stopping all further advance.

Mildew now appears to be established in the entire tobacco growing area of Connecticut and the southern part of Massachusetts.

NAME OF THE DISEASE

This same disease has been called by at least two common names to the confusion of growers. In Australia and in the southern states it is more often referred to as *blue mold* than as downy mildew. The use of this term seems unfortunate because it is confusing. In the first place, the blue color of the fungus is the most difficult of all symptoms to find and usually requires considerable imagination. In the second place, the same term is popularly used to refer to a truly blue colored mold of fruits and vegetables produced by fungi of the genus Penicillium, a genus far removed from the fungus causing tobacco mildew. The growers are also familiar with a blue mold (Penicillium) which sometimes runs over the soil of the seedbeds just after sowing, but which has no connection with mildew.

"Downy mildew" is much more descriptive of the appearance of the fungous growth on the back of the leaves, and, moreover, is the term in common use for diseases caused by this class of fungi (Peronosporales): for example, downy mildews of grape, onion, etc.

To avoid confusion it would seem best to use only the terms "downy mildew", or just "mildew", since there is no other kind of mildew on tobacco here.

SYMPTOMS [1]

The appearance of the infected plants or seedbeds shows great variation depending on the kind of weather prevailing during the development of the disease, on the age of the plants, the stage of the disease, and possibly other environmental factors. Downy mildew is essentially a disease of the seedlings in the seedbeds but this year has shown that it may also occur here sometimes in the field. Since the field symptoms are not just the same as those in the seedbed, it will be necessary to describe the two separately.

In the seedbed

A badly diseased bed, such as those first seen this year, looks as if it had been thoroughly burned by pouring scalding water or a toxic chemical, like formaldehyde, over it. All the leaves are dead, dry, and shrivelled to mere strings flattened out on the surface of the ground (Figure 1, page 64). Usually the plants are not affected equally in all parts of the bed. At one end they may be completely withered while they are progressively less affected as one approaches the other end. This gives the impression that the disease enters at one end and spreads toward the opposite.

Another symptom that is unmistakable after a little experience is the rank odor—especially if the beds have been closed—suggesting rapidly drying, decaying or steaming vegetable matter. It is not unlike the odor of potato mildew.

[1] This description was made by the writer with the diseased plants before him and is based only on observations of the season of 1937. Some symptoms described by persons in other sections were not observed here.

The smaller plants in these badly diseased areas are dead but the stronger ones still have green bud leaves although all the larger outer leaves are withered.

But if one wishes to see the beginnings of infection and observe the stages by which such destruction has come about, he must examine the opposite end of the bed or find beds where the infection is still new. In such places he will find the first indication of disease in small areas where the tips of the leaves, or indefinite spots on the leaves, are faded or rusty yellow. Such leaves are not flat, as they should normally be, but are irregularly puckered, humpy, contoured or cupped, or sometimes twisted until the lower surface faces upward.

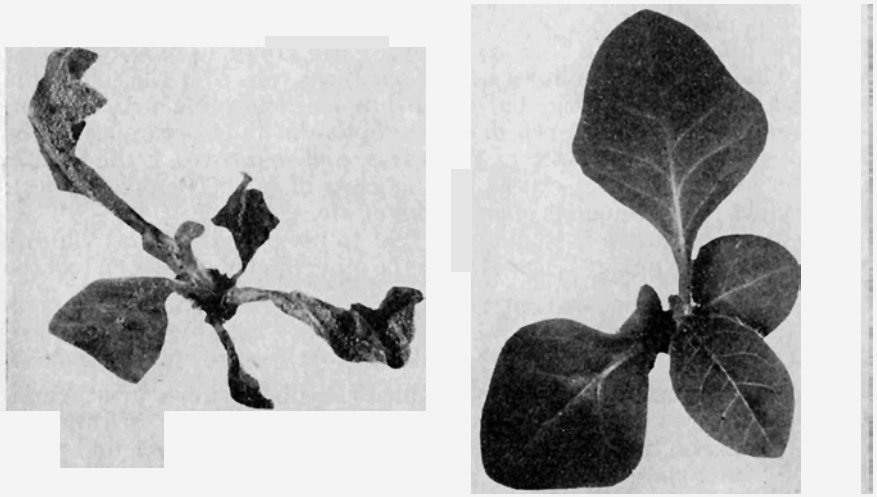

FIGURE 2. A healthy plant, right, contrasted with a badly mildewed plant, left.

If it is early in the morning or the weather is cloudy, the lower surface of some of these leaves will be covered with a downy felt of fungus (Figure 3), the symptom which gives this disease its name. The color of the down varies. Commonly it is white or gray, or, if older, rusty brown. Sometimes, however, especially if viewed obliquely, it has a distinct violet tint which accounts for the name, "blue mold". The presence of this felt-like growth on the lower surface of the leaves is the only infallible symptom of the disease visible to the naked eye. Later in the day and during dry weather, as well as during the later stages of the disease, this disappears and diagnosis becomes more difficult. The subsequent changes in appearance of the affected spots vary with the weather. When it is wet, the diseased tips take on a dark green to black, dead, water-soaked appearance as they wilt and wither progressively downward. In dry weather the affected spots become brown, dry and brittle. The colors which the dead tissue takes on

are so varied that they furnish no criterion for diagnosis. Neither is the shape of the spots regular or characteristic and it furnishes no proof of identity of the disease.

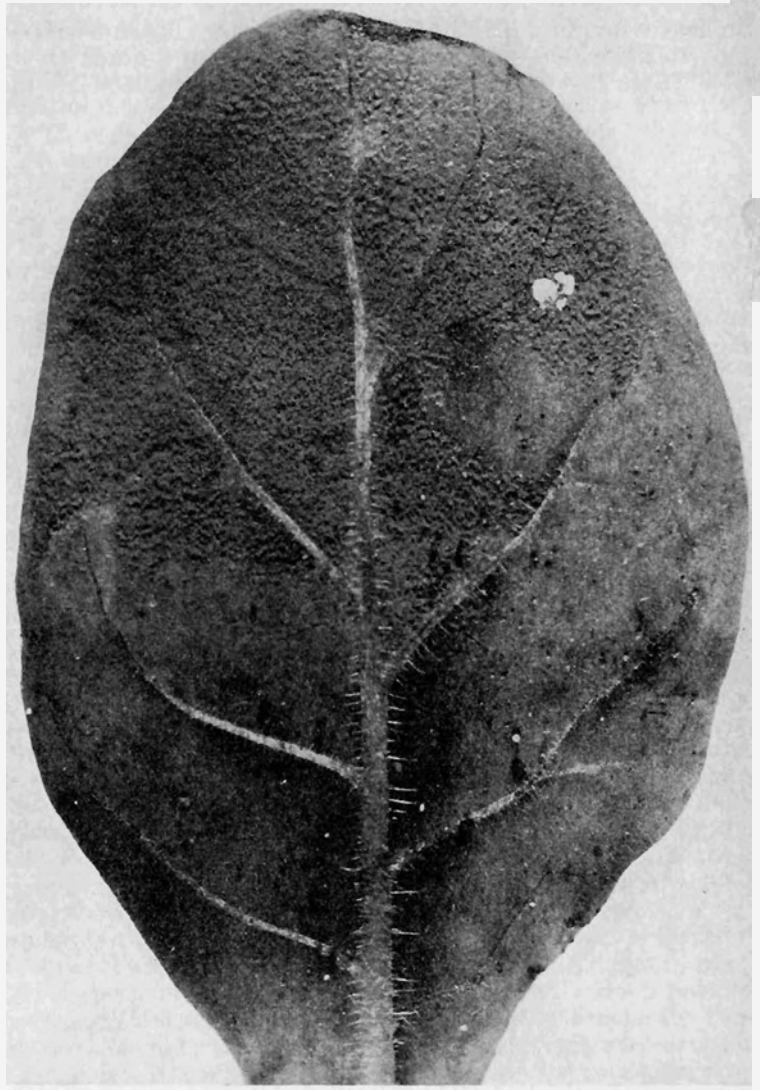

FIGURE 3. Leaves from the seedbed showing the fungus covering a part of the lower surface. Somewhat enlarged.

A remarkable characteristic of downy mildew is the capacity of badly diseased beds to recover. Except on the smallest plants, the bud and "chit"

leaves are not killed.[1] After the initial attack, the plant appears to acquire a certain degree of immunity and develops normally. Beds which seemed to be completely ruined when first observed this year were examined after 10 days and appeared perfectly normal, with no mildew on them. Plants from such beds showed no injurious effect when set in the field.

In the field

Entire leaves do not die in the field but the disease appears as spots of a half-inch to more than an inch in diameter, one to a dozen on a leaf (Figure 4). In the first stages one sees only a faint, indefinite yellow blotch

FIGURE 4. Mildew spots on shade leaf in the field. About one-third natural size.

on the upper side of the leaf. This blotch rapidly becomes more definite and more yellow, and as the leaf tissue dies, it turns to a light brown. The majority of the spots show no fungus on the lower surface at this time, but if the weather is damp one may find it, especially on leaves close to the

[1] Pathologists in other tobacco states report that often 80 or 90 percent of the plants are killed when infection occurs while the seedlings are quite small. This may well happen here if the mildew starts earlier in the season.

ground. On examining the young spots closely one notices numerous little brownish or blanched or sunken specks visible on both surfaces. Some persons have mistaken these for flea beetle injuries but examination under the glass shows that there has been no chewing of the tissue.

In wet weather the spots on the leaves enlarge to a certain extent but when dry they quickly cease to show any further development. When the tobacco is cured, the spots appear as blanched, dry areas which greatly reduce the value of the leaves as wrappers or binders.

Field infections were more common in shade tobacco than in the other types this year. The higher humidity under cloth may explain this, or possibly the fact that shade tobacco was set out earlier than the other types. There is no indication that any one type of our tobaccos is any more or less susceptible to mildew than the others.

CAUSAL PARASITE

The dead spots are due to the attack of a parasitic fungus which lives inside the tissues between the upper and lower epidermis of the leaves. This parasite forages its food from the leaf cells, causing them to die from starvation and poisoning, and thus producing a dead spot on the leaf.

The part of the fungus which lives in the interior of the leaf, the mycelium, consists of numerous, microscopically fine, branching threads, hyphae, running in every direction between the host cells. Specialized branches of these hyphae, haustoria, bore through the walls of the cells to reach the interior from which they absorb the food. A poisonous substance secreted by the mycelium also causes the death of cells not actually invaded. After fattening a few days on the food they have robbed from the leaf, the hyphae grow out to the lower surface, or occasionally the upper, making their exit through the numerous stomata, "breathing pores".

After passing through the stomata, each hyphal tip develops into a branched, tree-like structure (Figure 5A) called a sporophore. One or several sporophores may arise from each stoma. On the tips of the branches are borne egg-shaped, or lemon-shaped, colorless spores, variously called conidia, summer spores or sporangia (Figure 5 A and B). It is these sporophores and spores emerging in enormous numbers from the lower surface of the leaf that form the cottony or felt-like patches and furnish the most characteristic symptom of the disease. The development of these structures occurs early in the morning or on cloudy days, which accounts for the fact that the downy covering can be seen best at such times. The dust-like spores which are produced in enormous numbers are so light that they can be wafted about like the finest dust particles with the slightest air currents and can easily travel many miles through the air. The rapid spread of the disease and its wide distribution are thus accounted for by the quick development, enormous numbers and especially the easy aerial transportation of these summer spores. The spores may also be carried on the hands or clothes of workmen, by the splashing of water, and possibly by some insects. Later in the day, if it is clear, they blow away and the sporophores shrivel so that nothing can be seen on the surface of the leaf.

When the air is full of spores floating about, some of them are sure to fall on other tobacco plants in the same or other beds. Whether or not they infect the leaf on which they fall depends entirely on moisture conditions. If the leaf is dry and remains so, the spores die because they are short lived and most of them lose their power to germinate after a few

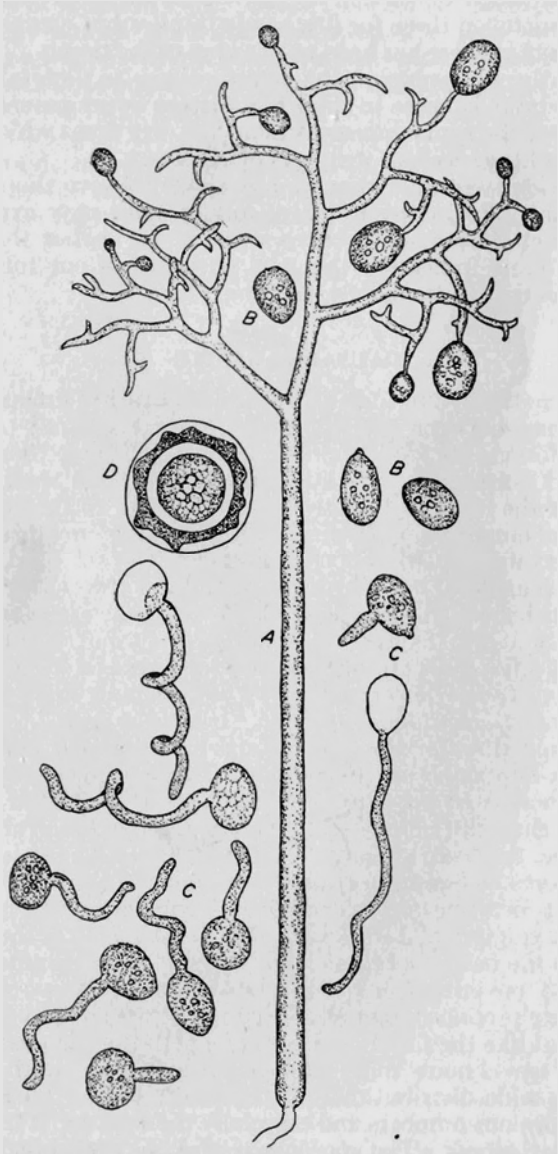

FIGURE 5. The causal fungus, *Peronospora tabacina*. A. A single spoorphore showing young summer spores in several stages of development. Most of the spores have already fallen off the tips of the branches. B. Mature summer spores (sporangia or conidia). C. Germination of the summer spores in various stages from one to four hours in a drop of water. D. An oospore (winter spore) from the interior of a dead leaf. (Magnified, 400 times natural size.)

hours or a day or two. They are also killed by exposure for an hour to direct sunlight or a temperature of 84° F. or more (61). If, however, there is moisture on the leaf, the spore germinates by pushing out a slender tube which elongates very rapidly (Figure 5C) and passes into the interior of the leaf through the stoma. Here it develops into a mycelium, as described above, and the life cycle is started again. In laboratory tests, the writer found it was only necessary for the spore to be in a drop of water two or three hours before it started to germinate, and the germ tube grows with unbelievable rapidity. According to Wolf, *et al.* (61) the life cycle, from inoculation to the production of a new crop of spores, requires four to seven days.

Oospores

A second type of spore, the "winter spore", is produced, not on the surface like the short-lived summer spores, but buried in the interior of the affected leaf. These occur in the collapsed dead leaves which are in contact with the soil. They have hard, thick, resistant shells (Figure 5D) and do not germinate at once when mature, but, after the leaf has decayed, remain in the soil until the following spring and then germinate at the right time to start new infections in the young beds. These oospores have been found rather frequently in the southern states where mildew is common, but rarely in Australia. The writer has found them in Connecticut and no doubt they occur here commonly, although not produced in such abundance as the summer spores.

NAME OF THE FUNGUS

The causal fungus belongs to the lowest or most simple of the three great classes of fungi, the Phycomycetes, in the genus Peronospora. The numerous species of this genus, Peronospora, are all parasitic on plants and produce diseases to which the name "downy mildews" has been given because of the plainly visible downy covering produced on the surface of the leaves during sporulation.

When Farlow (26) first found a Peronospora producing a mildew on a wild tobacco, *Nicotiana glauca*, he believed it was the same species as DeBary had described in Europe as *Peronospora Hyoscyami* on the black nightshade, *Hyoscyamus niger*, another plant of the same family as tobacco. Then when a similar disease was found on cultivated tobacco in Australia and later in Florida (51), the fungus was considered to be the same species and in the literature up to 1933 was referred to as *Peronospora Hyoscyami* de B. Inoculation experiments, however, by Angell and Hill (7) in Australia, and Wolf *et al.* (61) in North Carolina, in which spores from tobacco failed to produce any disease on the black nightshade, showed that the latter plant is immune to tobacco mildew although the two fungi are morphologically very similar. Because of these host differences it is now generally accepted that the tobacco mildew fungus should not be called *Peronospora Hyoscyami*.

In 1891 Spegazzini (56) described a mildew fungus on another wild tobacco species, *N. longiflora*, in the Argentine as *Peronospora nicotianae*. Since this fungus was morphologically very much like the tobacco mildew fungus, and since the latter was able to produce the disease on *N. longiflora* when inoculated, Wolf *et al.* (61) suggested that the pathogen on cultivated tobacco should be regarded as *P. nicotianae* Speg. Investigations by Adam (3) in Australia and later by Clayton and Stevenson (17) in America showed, however, that there were distinct morphological differences between the two species, particularly in the oospores. Also the latter point out that although *N. nicotianae* was shown by Spegazzini to infect various species of Nicotiana, still the tobacco growers in the Argentine are not troubled with any disease similar to our tobacco mildew.

Adam (3) in 1933 described the morphological differences between the three species and decided that the pathogen of cultivated tobacco is distinct from the other fungi and proposed a new name, *Peronospora tabacina*. This is the name now generally accepted by Australian and American pathologists working on tobacco mildew.

OTHER HOST PLANTS

In Australia, Angell and Hill (7) found the same mildew on about 20 other species of Nicotiana, the genus to which the cultivated tobacco belongs. They express the opinion that probably all species of this genus are susceptible. The genus Nicotiana comprises some 40 species, only two of which are of economic importance: *N. tabacum*, to which all of our cultivated types here belong, and *N. rustica*, used in some regions for smoking and grown in various places for the extraction of nicotine. All the others are wild weeds, none of which occur in New England. However, one species, *N. alata*, is sometimes cultivated in flower gardens here and is known popularly as "flowering tobacco".

Outside the genus Nicotiana, this fungus has been found to affect seedlings of tomato, eggplant and pepper (61, 10, 11). Albert and Sumner in South Carolina (11) report one case in which it did serious damage to pepper seedlings. Outside of this instance, there is no record of real damage to other plants and it seems unlikely that downy mildew will ever become a menace to other crops. Other host plants are of importance only because of their possible connection with the spread and overwintering of the disease.

Among the numerous varieties of cultivated tobacco, none has yet been found to be immune or highly resistant to mildew.

SOURCE OF INFECTION IN THE SPRING

Since the summer spores under ordinary conditions live only a few days at the most and the mycelium inside the cured leaf is no longer alive, how does the fungus live over the winter to start infection in the beds the next spring? The following possibilities suggest themselves: (1) Mycelium may remain alive over winter in the seed; (2) mycelium may remain alive on some perennial weed host and produce spores in the spring; (3) mycelium or summer spores may overwinter in the soil; (4) winter spores (oospores) may winter in the soil of the seedbeds; (5) summer spores may blow into New England from warmer southern states.

In the seed

In Australia, Angell (4) and Angell and Hill (7) found that the fungus sometimes occurred on the seed pods. By microtechnique they demonstrated the presence of mycelium in the seed of such infected pods but were not able to show that this mycelium lived until the next year, nor that such infected seed, when sowed, would produce diseased plants. American investigators have not reported the occurrence of mildew on pods or seeds. This possibility should be investigated further but at present there is no indication that infected seed is responsible for primary infection in the spring.

In perennial hosts

In Australia and in some of our southern states, the tobacco plants sometimes remain alive over winter and produce a new crop of suckers in the spring. Early spring infections sometimes found on these, and the fact that in Australia it has been shown that the mycelium is not always local in leaves but may become systemic and live in the tissues of the stalk, has led to the suggestion that such overwintering plants may furnish the

medium for starting spring outbreaks. Under Connecticut winter conditions, however, no part of the tobacco plants survive and therefore this possibility may be dismissed. Neither are there any other species of plants, known to be susceptible to this mildew, which survive the winter here.

Mycelium or summer spores in the soil

Although the summer spores and mycelium ordinarily are very short-lived, Angell and Hill (7) were able to cause spores to germinate after 117 days when kept in dry soil at a very low temperature, 3° to 5° C. Little is known about the longevity of the mycelium itself under various conditions. This possibility needs further investigation before it is completely dismissed.

Winter spores

By analogy with many other downy mildews, one would expect the oospores to be the most important if not the only source of spring infection. As stated on page 73, these spores occur in the decaying leaves and are known to be able to survive the winter in the soil and to germinate the following spring (62). Wolf *et al.* (61, 62) present evidence to show that this is the principal source of primary infection in the southern states.

Summer spores blown from states to the south

Reasons for believing that this was the source of primary infection in 1937 have been presented on a previous page. Repetition of this performance may be anticipated in the future if weather conditions are right. Investigations in the southern states have fully demonstrated that the spores blown for many miles in great numbers, remained capable of producing infection.

HOW THE WEATHER AFFECTS MILDEW

Mildew comes on suddenly and disappears as suddenly. Some years it is very destructive; other seasons it causes little or no damage. Sometimes it spreads with almost unbelievable rapidity, and again it remains stationary. Its erratic and puzzling behavior makes it impossible to predict what it will do at any one time. For the most part, these peculiarities of the disease can be explained by the effects of the weather on the development and distribution of the causal fungus.

Continued cool, moist weather is most favorable to the disease. Since, in general, our early growing season is cooler than that of the southern states, it may be anticipated that the disease will at least be as destructive here as it has been in the South.

Temperature

Dixon *et al.* (25), after exhaustive investigations on the effect of temperature, found that the spores in the beds are developed during the early hours of the morning at temperatures of 42° to 63° F. with the most abundant production at about 56°. Little, if any, production of spores occurs above 68° F., or below 36° F. Naturally the disease spreads little when no spores are produced. In the beginning of our seedbed period the nights are too cold for spore production; at the end of the season they may, at times, become too warm; but for most of the period, the night temperature range in seedbeds is quite favorable to sporulation.

High temperatures during the day inactivate or kill the mycelium. Thus in July of 1937, when the weather suddenly became very warm, spots on the leaves in the field made no further progress during the entire season and no more spores were found. That mildew has never caused damage in the field in the South is probably due mostly to the high temperatures that prevail during the summer. The critical temperature above which the mycelium does not develop is around 84° F. Except during unusual seasons, it is unlikely that this will be a serious field disease here.

No attempts have been made to determine the effect of winter temperatures on the fungus. Judging from analogy to other fungi of this group, we may anticipate that the severity of our winters will not kill the oospores and will give us no protection. The only advantage of cold weather is that it prevents the survival of suckers or any living part that might harbor the fungus until the following spring.

Moisture

Like most fungi, the mildew pathogen is favored by moisture. Since our beds are constantly watered and the sash prevent too much evaporation, the humidity is close to saturation most of the time. Even though the uppermost leaves may be dry, when the plants are crowded, there can be very little ventilation around the basal leaves and a high humidity near the ground is inevitable. Such conditions are ideal for the luxuriant development of the sporophores and spores that form the felt-like covering on the lower surface of the leaf.

Water plays a more important rôle, however, in the germination of the spores and infection of leaves. When a spore alights on a leaf it can germinate only when it is in water, i.e., it will not push out an infection tube on the dry surface of the leaf. It is naturally not possible to keep the leaf surfaces dry all the time. In a drop of water, as previously stated, the spore germinates within two hours or less and in another hour the germ tube could have entered the stomate of the leaf. Thus any condition under which drops of water remain on the leaf as long as three or four hours will permit infection. Naturally, the longer the leaves remain wet, the greater the chances for infection and the more severe the disease. In the field also, long periods of rain may easily result in spreading the disease.

Sunshine

Direct sunlight is lethal to the summer spores, killing them within a few hours. It also dries the leaf off more quickly and thus delays infection.

Wind

Since the spores are disseminated mostly by air currents, a windy day is most favorable for spreading the disease. On the other hand, however, wind may have a beneficial influence in evaporating the drops of water from the leaves.

PREVENTION AND CONTROL

Only recently have satisfactory methods of control been developed and even these have not been tested long enough. Certain methods developed in Australia and in our southern states appear promising but need further trial before we can be sure that they are effective under Connecticut

conditions. For convenience in discussion we may group the suggested methods of prevention and control under three heads: (1) Modification of cultural practices; (2) spraying the plants with fungicides, and (3) vapor treatment of the seedbeds.

Cultural Practices

1. In the southern states it has been recommended that the beds be located in a different place every year. This is based on the observation that the first infections found in the spring are usually in beds sowed where there were diseased seedbeds the previous year. Soil in diseased beds would naturally contain a larger number of overwintering oospores than new soil. Or, if there are other stages in which the fungus winters, the chances of infection would surely be greater on old bed sites. Continual yearly shifting would be practical on some Connecticut farms but on others, where there has been a considerable outlay for water systems, fencing, stationary steaming outfits, etc., such a plan would involve considerable expense which many growers would not wish to incur.

2. Steaming the soil is a common practice among the better growers in Connecticut and would kill the oospores or any of the other stages of the fungus that are present in or on the soil. Whether or not formaldehyde or acetic acid sterilization of soil would kill the oospores has not been determined. The spores probably do not remain over on the sideboards and sash so that there would seem to be no advantage in sterilizing them. Even when the soil is steamed there is always the chance of the fungus remaining alive in the walks between or about the beds. Nevertheless, steaming should not be neglected and may be an effective link in the chain of measures necessary to cope with the disease.

3. It has been recommended that beds be located on sites where good air drainage and proper exposure to sun would dry the water off the leaves quickly. Shaded, swampy, or poorly drained sites should be avoided. Any environment that will permit the water to stand for long periods on the leaves will furnish better opportunity for germination of the spores and thus favor infection.

4. Ventilation of the beds is of even more importance. The parasite requires high humidity of the air. When the sash are closed tightly the humidity is close to 100 percent all the time. By keeping one end of the sash raised, or leaving spaces between the sash, the humidity is quickly reduced. If the weather is warm, it is better to remove the sash completely. Plants grown with adequate ventilation are stronger and better regardless of mildew.

5. Just as soon as the setting season is over, all extra plants and debris should be removed from the beds to prevent them from harboring the oospores which would remain in the soil until the next spring.

6. In the South it is commonly recommended that growers increase the size of their customary seedbed area to provide a reserve of plants.

7. In Australia, the disease has been found on seed pods and, since it was suspected that infected seed could transmit mildew to the following crop, growers have been warned to avoid saving seed from plants known to be affected. In America, no one has reported the disease on pods, and it seems unlikely that this would be a source of danger.

8. In the southern states application of nitrate of soda to diseased plants is recommended to increase growth and recovery after infection. Since our plants in this section are raised on a pretty high level of fertility, it is questionable whether this practice would be of value.

9. The remarkable power of recovery of diseased beds has been mentioned on a previous page of this bulletin. It has been found that plants set after recovery live much better than those set during the early stages of the disease. Recovered plants appear to develop a partial immunity. If it is necessary to set from beds that have been attacked, it is best to wait until the plants have recovered. Naturally it is still better to set from beds which have no disease at all.

10. Although wind is probably the principal agent in spreading the spores, it is also certain that they may be carried from bed to bed on the hands or clothes of workmen. As far as is practicable, workmen should avoid handling diseased plants. Curious visitors who come to see affected beds may also carry the spores to healthy beds that they visit afterward.

11. Destruction of diseased suckers in the fall. In some sections, the disease has been found late in the fall on suckers growing from old stalks. Oospores would normally be developed as these leaves rotted and might start infection in the spring. As yet, the mildew has not been found in the fall here. If further investigation should show late infection, it would be best to plow the stubs under as soon as the crop is removed, or to remove all suckers from the field later.

Spraying the seedbeds with Fungicides

Since Bordeaux mixture has been extensively and successfully used in the control of various other downy mildew diseases, it was naturally the first to be tried against the tobacco downy mildew. The control obtained in experiments with this fungicide, however, has been disappointing. Angell and Hill (7), after one season of tests, succeeded only in delaying the appearance of mildew to a certain extent and stated, "Our experiments do not appear to offer much promise". Clayton and Gaines (14) in 1933, after reviewing the investigations in the South up to that time, state: "Bordeaux Mixture appears to be about as effective as any other spray or dust". None of them, however, had been found very satisfactory. Mandelson in Australia (38) conducted more extensive tests on Bordeaux in combination with various spreading agents. Also he included in his tests a number of other copper fungicides. Most of these gave some degree of control. He got poor control with all of the dusts. Bordeaux mixture alone was not as effective as when mixed with spreading agents such as soft soap or molasses. He used a weak Bordeaux of the formula 2-1-50. Most of the Bordeaux combinations also produced some injury to leaves.

Since Bordeaux mixture is in common use here on seedbeds for the control of wildfire and other diseases, and we can use it at the full 4-4-50 strength without injury, it appears worthy of further trial under our conditions. Connecticut growers who had been using Bordeaux on their seedbeds in 1937, however, did not escape the mildew, and in view of the rather poor results reported by pathologists in other sections, it would probably be unwise for growers to rely on Bordeaux mixture at present.

Mandelson (38) found two other copper fungicides to be more efficient than the Bordeaux, viz.: (1) Home-made colloidal copper with soft soap as a spreader, and (2) copper emulsion. He recommends particularly the former, which is prepared by adding molasses to a copper sulfate solution and neutralizing with caustic soda. This concentrated stock solution is stored throughout the season and diluted with water when needed for spraying. Armstrong and Sumner (11) in South Carolina also conducted spraying tests with colloidal copper, prepared as recommended by Mandelson, and obtained encouraging results. Although it did not prevent the disease it greatly reduced the severity of attack and gave somewhat better control than the other fungicides tried. It demands that the grower take considerable care in neutralizing to prevent injury, a point which may prevent the general use of this material.

Tests with a proprietary fungicide, Cal-Mo-Sul, reported by Armstrong and Sumner (11) also gave results not quite so good as the colloidal copper but much better than the unsprayed check.

In the same bulletin Armstrong and Sumner report favorable results with red copper oxide. This same fungicide, mixed with cottonseed oil and lethane spreader, has also been tested in Florida, Georgia, North Carolina, Virginia and Maryland. It is now recommended more than any other spray by the tobacco pathologists of these states. The cottonseed oil has a weak fungicidal value and when the two are combined they are more effective than either one used separately. The lethane spreader enables the copper to cover the leaf surface more thoroughly.

There are some minor variations in the quantities of materials and technique of mixing recommended by pathologists of the different states. The most generally accepted procedure is as follows:

1. Materials needed to make 50 gallons of spray mixture are: ½ pound of red copper oxide, 1 quart of lethane spreader, 2 quarts of cottonseed oil and 50 gallons of water.
2. Moisten the copper oxide with enough lethane spreader to make a dough. Then gradually add water, stirring all the time, to make a suspension.
3. Mix the quart of lethane spreader and 2 quarts of oil by stirring thoroughly.
4. Add 2 or 3 gallons of water to the above spreader-oil mixture.
5. Pump this through the spray pump (nozzle attached) into another container in order thoroughly to break up, emulsify, the oil into fine particles.
6. Add water and the copper oxide suspension (from 2 above) to bring the total volume up to 50 gallons.

This is now ready to spray on the plants. Make up just enough to spray all the beds each time. Do not try to store it for future applications. Use a fine nozzle with high pressure and apply enough to cover all leaves. Spray twice a week.

Experiments with the copper oxide oil treatment were started here late in the seedbed season. Results were not conclusive but showed some promise. Further investigations are in progress.

In view of results obtained in other sections, however, this seems to be the most promising treatment. It should be generally tried by the growers during the coming season.

Vapor Treatment

This treatment is accomplished by exposing volatile chemicals in the beds during the night, the fumes of which fill the confined air and are toxic

to the fungus. Various chemicals have been tested in Australia and in America (5, 8, 33, 34, 39, 42, 48). The one now commonly recommended is benzol, a distillate of coal tar. Other coal tar distillates such as toluol and xylol have also given control but not as complete as benzol[1].

The benzol is placed in shallow pans distributed throughout the beds. The total exposure surface of benzol in our glass-covered beds should be about .01 of the bed area to be protected. Starting when mildew first appears in a locality, the pans are set in the beds every evening about sundown and removed the following morning. It is also recommended that they be left in the beds during dark days, but not during bright days. Since benzol fumes are heavier than air, the treatment should be more effective if the evaporating pans are supported a few inches above the ground level. It requires about one-half to two-thirds of a gallon of benzol per night for 100 square yards of seedbed. The sash should be closed tightly during the night. Any unused benzol should be returned to the bottle and may be used for the next treatment. If just the required amount is used each time, there should be none left. This should be about one fluid ounce, (29.5 cc) per square yard of bed. (16 fluid ounces equal one pint.)

It is claimed by investigators who have worked with it in the South and in Australia that benzol completely prevents mildew.

If the benzol splashes on the leaves it will kill them or, at least, make dead spots on them. Water collecting on the underside of the sash may drop into the benzol during rainy weather and cause it to splash on to adjacent plants. This can be prevented by using some type of covers above the pans. There is also a possibility of an excess amount of the fumes causing some yellowing of the plants, particularly during warm nights. This injury may be prevented by mixing the benzol with lubricating oil, either fresh or waste (42). The mixture should consist of one part of benzol to five parts of oil. The oil may be used again and again. Benzol is inflammable and naturally should be kept away from lighted matches.

The benzol vapor treatment is a new method for controlling fungous diseases and has not been tried long enough to warrant a recommendation that it be universally adopted.

Further investigations are in progress here and recommendations must await further results, but it is worthy of some trial by tobacco growers.

Benzol vapor has the additional advantage that it kills flea beetles and other insects in the beds.

[1] Also called *benzene*, but not the same as *benzine* which is a commercial mixture and closely related to gasoline. Benzine should not be used.

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