

STUDY OF PLANT DISEASES STARTED WITH COLLEGE

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The formal study of plant diseases in North Dakota is almost as old as the University. Some of the first publications from the then North Dakota Agricultural College reported on flax wilt. Bolley studied this malady in 1891-92. Later he recognized the soil-borne nature of crown and root rots of cereals.

Dr. H. H. Flor studied flax rust and the inheritance of resistance of plants and inheritance of virulence in pathogens, resulting in the world-recognized gene-for-gene hypothesis.

More recently, North Dakota State University achieved world recognition for research on tan spot of wheat under the leadership of Dr. R. M. Hosford. A workshop with scientists from around the world was held on the NDSU campus in 1981.

The uninformed might wonder why plant pathologists couldn't "wrap up" plant diseases like medical doctors took care of smallpox (they haven't done as well with the common cold). Much of the need for constant surveillance on plant diseases goes back to the constant variability of plant pathogens.

"It's something a lot of people don't understand. They understand that a plant has a genetic system. But what's often forgotten is that the pathogen has a genetic system, too. Just as in plants, variation in pathogens occur. They change. Because a variety was released with resistance to a disease doesn't mean it will retain resistance, especially if grown over a large acreage," says Dr. Richard Kiesling, head of the NDSU Department of Plant Pathology.

Many farmers assumed that with new crops such as dry edible beans and sunflower they would have no disease problems. It turns out that sunflower wasn't a new crop, having been grown considerably in the 1930s before their "discovery" 30 years later. The sunflower was also domesticated by the Indians right here in North Dakota. And while plant pathologists have been able to come up with resistance to handle downy mildew, sunflower rust and Verticillium wilt rather quickly, they have not found resistance to white mold.

Dr. Jim Venette, who conducts white mold research in edible beans, describes white mold as a low grade

pathogen which dissolves bean tissue ahead of the fungus. "So we don't have the really nice cellular mechanisms for resistance as we have for downy mildew and rusts," he points out. White mold has been studied longer than many other diseases and it still is very difficult to control. Researchers have published over 1,000 reports on its physiology, taxonomy, distribution and the like. But nobody has come up with inexpensive commercially acceptable controls.

Much of the problem with controlling white mold on beans is getting a fungicide into the plant canopy where it could be effective. Many of the bean varieties grown in the state have been chosen for their yield rather than their ability to resist the white mold organism. UI-114, Wyo-166 and UI-111 all are sprawling indeterminate plants that flower through the season. Fresh blossoms and mature beans are present on the vine at the same time. And blossoms are a susceptible part of the plant. The fungus needs dead or senescing plant parts to "get going."



Dr. Jim Venette examines beans for disease development in laboratory tests. Petri dishes contain cultures of pathogenic fungi.

A bush type bean might help reduce white mold because it would dry off sooner than present varieties, but not without risk to bean growers. Bush type beans

set flowers once or twice and would allow for timely spraying if needed. But because of their limited blooming, a hot dry spell during bloom could abort blooms and wipe out a crop. So far, bean producers have chosen to take their chances with white mold. But growers in the northeastern part of the state who have lost two or three successive bean crops to white mold might want to reconsider, says Venette.

He continues to screen fungicides that may be helpful in controlling white mold. Competition would bring the price down and make chemicals relatively inexpensive. An increase in the price of beans from their present low price of about 9 cents a pound to about 25 cents would also be helpful.

Another phase of white mold research involves the use of microbes that might break down fruiting bodies of the fungus. White mold is known to live in the soil for 10 years. It also can send up spores which can be airborne to infect a wide range of hosts: sunflower, potatoes, sugar beets, soybeans, rapeseed, mustard and many fleshy weeds.

Until there is plant resistance to white mold, good cultural practices provide the best hope to reduce — or live with — the pathogen: clean seed, rotations that allow several years away from susceptible crops, weed control, and avoiding solid seeding — especially of soybeans — that allows moisture to persist in the crop canopy.

Sclerotinia, the fungus responsible for white mold diseases, is common in and on the edges of the Red River Valley but is found in most counties in the state. *Sclerotinia* wilt of sunflower is caused by sclerotia (hard, black resting bodies) in the soil which germinate and infect the roots. Commercial hybrids are not resistant to *Sclerotinia* and chemical control is not feasible. Management practices, primarily rotation to non-susceptible crops such as small grains or corn are the only way to minimize damage from wilt.

One of the major differences between white mold of beans and *Sclerotinia* wilt of sunflower is that development of the bean disease depends on high soil moisture and on wet conditions in the crop canopy, while sunflower wilt develops in wet or dry soil. Wilt also occurs in a wide range of soil types. Therefore, any season sunflower is planted on *Sclerotinia* infested soil, there will be a wilt problem.

To understand how management practices affect the disease, Dr. B. D. Nelson each year is monitoring the populations of sclerotia in 30 commercial fields naturally infested with *Sclerotinia*. He is also determining the effect of different populations of sclerotia on the amount of disease. With information from these studies it will be possible to determine the rotation times needed to reduce sclerotia populations to acceptable levels.

Managing *Sclerotinia* may be the only way sunflower producers can live with white mold. The sclerotia of



In these greenhouse studies at North Dakota State University, Dr. Berlin Nelson studies the relationship between the amount of sunflower white mold (stalk rot) in the soil to the amount of the disease present on sunflower.

Sclerotinia also germinate to form mushroom-like apothecia which liberate spores called ascospores. These ascospores infect many susceptible crops. The origin of ascospores inoculum in North Dakota was determined by surveying infested crop fields for the presence of apothecia. They were found in many fields of susceptible crops and in non-susceptible crops such as small grains and corn. Ascospores have been trapped above a wheat field where apothecia were produced, indicating that *Sclerotinia* infested fields planted to non-host crops could be sources of ascospore inoculum for adjacent susceptible crops.

Head rot of sunflower is caused by spores blown to the head; middle stalk rot by spores blown to that part of the plant. Head rot usually is not a problem except in wet years when ascospores are produced. Sunflower fields with 30 percent head rot have been observed. Irrigation, which maintains high moisture, sets up ideal conditions for the formation of ascospores.

If small grain — and especially wheat — farmers suffer from the “40 bushel straw, 10 bushel wheat” syndrome, chances are their fields have been on the receiving end of *Helminthosporium sativum*. It is responsible for common root rot of spring wheat and barley, spot blotch of barley, wheat leaf spot, and head blight and black point on durum.

Surveys conducted in 21 eastern and central North Dakota counties during the last three years indicate that losses caused from common root rot averaged 6.3 percent for wheat and 6.9 percent for barley in 1980; in 1981, the loss in barley was 13.1 percent. The 1982 results are not yet complete at this writing but a preliminary count indicates that many barley fields showed root rot losses in the 8 to 10 percent range, according to Dr. R. W. Stack.

While root rot doesn't have the capability to wipe out a crop — it's sneaky rather than dramatic — the losses are considerable when penciled out. A 10 million acre crop with a statewide yield of 25 bushels per acre is 250 million bushels. And if only 5 percent was lost, that still is 12 million bushels per year that farmers don't harvest. Even at today's deflated wheat prices, that's a good piece of change.

H. sativum survives on crop debris and in the soil. It is a natural organism that was around before wheat because it is also a parasite of native prairie grass. It's not realistic to talk about eliminating the fungus. But root rots and other such plant diseases can be managed to a

certain extent. Stack's research is aimed at working closely with plant breeders at NDSU to identify lines and varieties that have levels of resistance to *H. sativum*. Some durum lines are quite resistant to the fungus and work is continuing to improve that level of resistance.

Because root rot is so devious, farmers may not recognize the disease unless it is severe. And it can be severe when the plant is under other stresses such as drouth or high temperature. The south-central part of the state suffered barley losses as high as 70 percent in 1980 when drouth and root rot combined forces.

Root rot is present in almost every field every year. Usually it weakens the plant, reduces the number of heads produced, causes smaller heads and grain doesn't fill as well or as heavily as healthy plants.

Farmers can reduce losses from root rots by good farming practices: especially crop rotation, using clean heavy seed and planting resistant or tolerant varieties.

Posters used by Prof. H. L. Bolley from 1909 onward to publicize the importance of common root rot. The recommendations are still valid today.

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BOLLEY, N. D. A. C.