Effect of Long-Term Continuous Cropping of Spring Wheat on Disease-Causing Ability of *Cochliobolus sativus*, Agent of Wheat Root Rot

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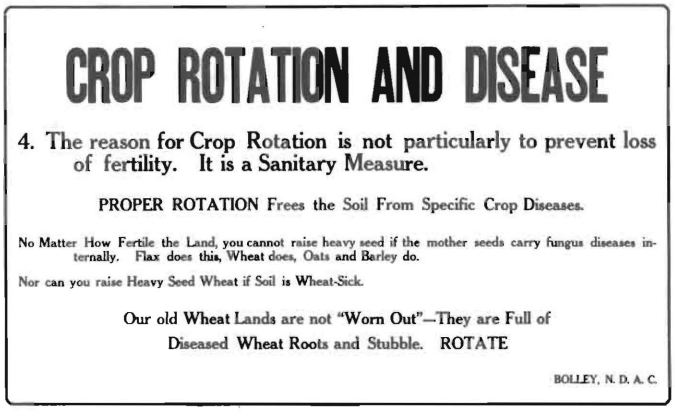
In the year of the Centennial of the North Dakota Agricultural Experiment Station, it is particularly appropriate to present research from an experimental plot which dates from the founding of the NDAC. Beginning in 1882, the land just north of Fargo--which was to become NDAC--was planted to wheat for the first time. With the founding of the North Dakota Agricultural Experiment Station and establishment of experimental land, several small plots (¼ acre) were kept in wheat production. One of those remains to this day. Professor H.L. Bolley, NDAC station botanist and the first North Dakota plant pathologist, maintained this plot continuously in wheat to serve as a "disease garden," to study the effects of continuous wheat culture on diseases.

In those early years, many people believed that a series of wheat crops wore out the land; they called it "wheat-sick soil." By comparing plots of continuous wheat to others where crop rotation was practiced, Bolley showed that the land wasn't worn out but rather that "wheat-sick soil" was due to build-up of disease (1). Bolley promoted crop rotation and clean seed as ways to reduce disease and make 'wheat-sick soil' productive again (Figure 1).

Bolley's continuous wheat plot is one of the oldest spring wheat disease gardens in the United States. This plot provides a unique and irreplaceable resource to NDSU scientists. This plot has been seeded to spring wheat every year since 1882 (1,13).

One of the most widespread diseases of dryland spring wheat and barley in North America is common root rot caused principally by the soil-inhabiting fungus *Cochliobolus sativus* (= *Helminthosporium sativum*). It was Bolley who first discovered that it was the extreme build-up of this fungus after repeated crops of wheat which was responsible for "wheat-sick soil." The level of common root rot in this plot continues to be high (8,9,10).

Common root rot caused by *C. sativus* is strongly influenced by cultural practices such as nitrogen fertilization or crop rotation and by environmental factors (2,6). Repeated cropping to wheat has been shown to increase inoculum density of *C. sativus* in the soil, resulting in an increased level of common root rot. In Saskatchewan, Chinn (1976) found the spore population of *C. sativus* in the soil was increased in fields seeded to wheat and declined in fields under summer fallow. Rotation with certain noncereal crops also led to a reduction in spore numbers compared to repeated cropping (2,3,4).


CROP ROTATION AND DISEASE

4. The reason for Crop Rotation is not particularly to prevent loss of fertility. It is a Sanitary Measure.

PROPER ROTATION Frees the Soil From Specific Crop Diseases.

No Matter How Fertile the Land, you cannot raise heavy seed if the mother seeds carry fungus diseases internally. Flax does this, Wheat does, Oats and Barley do.

Nor can you raise Heavy Seed Wheat if Soil is Wheat-Sick.

Our old Wheat Lands are not "Worn Out"—They are Full of Diseased Wheat Roots and Stubble. ROTATE

BOLLEY, N. D. A. C.

Figure 1. Wall poster promoting crop rotation used by Professor Bolley. The recommendations are still valid today!

In comparison to the effect of repeated cropping on spore populations in soil, little is known of the effect this practice has on the innate disease-causing ability of the pathogen population itself. Plant pathologists use the term "aggressiveness" to describe generalized disease-causing ability of a particular culture or population of infectious fungi. Individual cultures of *Cochliobolus sativus* show great variability in many biological characters including aggressiveness (5,6). The purpose of the experiments reported here was to see if the extreme condition of 100+ years of wheat culture without a break had influenced the aggressiveness of this root rot fungus.

MATERIALS AND METHODS

Isolation and maintenance of cultures. All cultures of *C. sativus* tested in these experiments were collected during the 1981 growing season from diseased spring wheat plants. Basal portions of plants collected from the field were washed thoroughly in running water. The crown and subcrown internode tissues were separated and cut into small pieces (3-5 mm), immersed in dilute bleach (0.25% NaOCl) for 10 min, rinsed in sterile distilled water, dried and planted on agar in petri dishes. Dishes were incubated at room temperature under fluorescent lights. Individual colonies of *C. sativus* were visible after 3 to 5 days (Figure 2) and were transferred to tubes and stored in a refrigerator.

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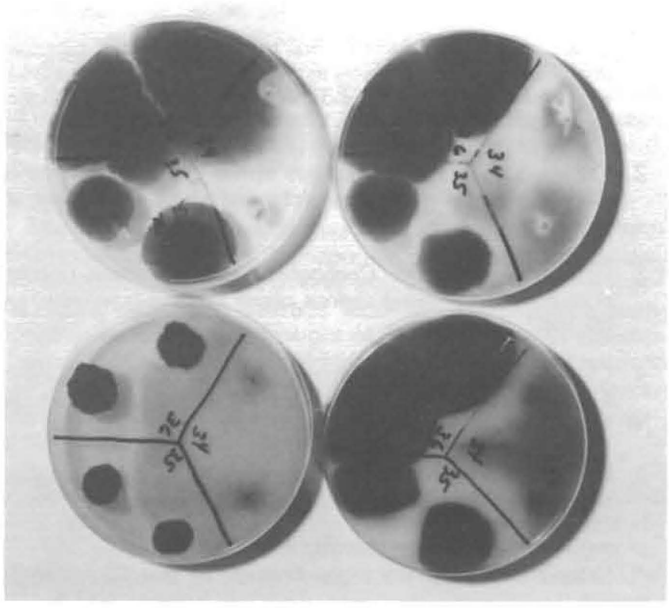

Figure 2. Black Colonies of *Cochliobolus sativus* growing from wheat root pieces placed on agar in petri dishes. Pure cultures like those tested are derived by such isolation methods.

Cultures of fungi obtained in this manner are individuals comparable to individual plants or animals in a population. There were 98 cultures from Bolley's continuous-cropped wheat disease garden (CW) and 38 cultures from conventionally rotated wheat fields (RF) from farms within a 15-mile radius of Fargo. Farm fields in this region are cropped to wheat every 2 to 4 years on average and are estimated to have grown 30 to 40 wheat crops in the last century.

Preparation of inoculum. *C. sativus* cultures were grown on petri plates for 10-15 days, then blended in sterile water and strained through layers of cheesecloth. This suspension was added to dry sterile sand to give a level of 400 spores per gram in this "mixed sand inoculum."

Test of pathogenicity. *C. sativus* cultures were tested for their ability to cause disease by experiments done under controlled conditions to minimize variability from weather, season, etc. Cultures were tested in three experiments extending over a five-month period. Experiment 1 and 2 each included 49 isolates from Bolley's plot and Experiment 3 included 38 isolates from farm fields in the region.

Wheat plants were grown in the greenhouse in clay pots containing the mixed sand inoculum. Each pot received a single culture. Wheat seeds were surface disinfected using dilute bleach and planted 4-5 cm deep.

In each of the three experiments there were 18 noninoculated pots (six per replicate) to serve as controls. To test the experimental conditions for disease development and to allow comparisons between experiments, three tester cultures of *C. sativus* known to cause disease were also included as disease standards in each replicate of each experiment.

After six weeks, plants were carefully uprooted and roots were thoroughly washed. From each pot 40 plants were scored for infection by *C. sativus*. Disease severity on each plant was visually rated according to the extent of lesions on the subcrown internode (12). Disease was expressed numerically on a scale of 1 to 4 where 1 = clean, 2 = slight symptoms, 3 = moderate symptoms, and 4 = severe symptoms and discoloration or rotting. A mean disease rating (DR) for each isolate was calculated from the individual plant values. Aggressiveness of an isolate was expressed as the amount of disease caused under these standard conditions.

RESULTS

In each experiment, the noninoculated control plants had very little disease with a mean DR of 1.19, 1.18, and 1.14 for the three experiments, respectively. Plants inoculated by the tester cultures caused moderate levels of disease; their mean DR in the three experiments was 2.33, 2.44, and 2.35, respectively. Neither the DR of controls nor of the tester isolates differed significantly between experiments.

Aggressiveness of isolates from the two sources was compared (Figure 3). Aggressiveness appeared as a normally distributed character with most cultures response in the middle and fewer at the extremes. There was a range of aggressiveness in cultures from both sources. The mean DR of the 98 cultures from Bolley's continuous-cropped wheat disease garden (CW) was 2.38 while that of the 38 cultures from the commercial fields (RF) was 2.16, a significant difference. When the frequency distributions by aggressiveness category for the two groups were compared, a large significant difference was also found. To check that *C. sativus* was

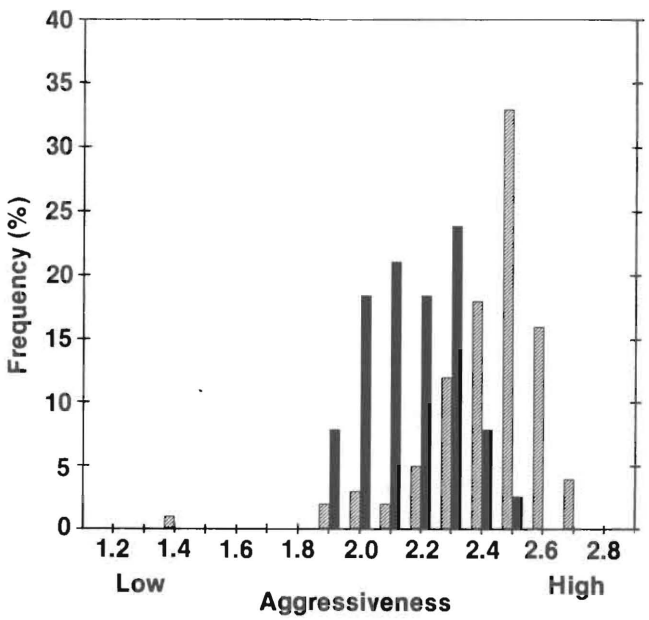

Figure 3. Aggressiveness of *C. sativus*. Position of bars (horizontal axis) indicates the amount of disease produced under standard test conditions. Height of bars indicates number of cultures at each level of disease. Cultures (98) collected from continuous-cropped wheat plot (CW) indicated by hatched bars; cultures (38) from wheat plants collected from commercial rotated fields (RF) indicated by solid bars. Relative aggressiveness scored on a scale of 1 = clean to 4 = severe disease on inoculated wheat plants.

causing the symptoms observed, 300 randomly selected symptomatic plants were re-isolated and yielded only *C. sativus*.

There were more highly aggressive cultures from CW than from RF. For example, 72 percent of CW cultures caused disease levels greater than or equal to DR 2.4, while only 10 percent of RF cultures did so (Figure 3). On the other hand, 26 percent of the RF cultures caused disease levels at or less than DR 2.0, whereas only 5 percent of CW cultures did so. The disease-causing ability of the 38 RF cultures tested here was approximately the same as for 384 other cultures from four widely separated North Dakota counties tested in another study (El-Nashaar, 1983).

DISCUSSION

This study was possible only because NDSU has retained valuable biological resources such as Bolley's continuous wheat plot. Recently, Shipton (7) reviewed the effects of monoculture on a range of crops and diseases caused by soilborne plant pathogens. He described several patterns in disease occurrence over time. Disease may increase to the point where the crop can no longer be grown, or may increase and then decrease as with take-all decline. In other situations disease may stabilize or undergo periodic fluctuations. He also stated that our understanding of such processes is limited by the small number of systems studied. The present work shows the wide variation in aggressiveness of field isolates of *C. sativus*. That its aggressiveness appears as a continuous variable suggests that the character is probably determined by several or many genes. This would correspond to the complex inheritance of root rot resistance in wheat and barley.

Innate disease-causing ability ("aggressiveness") is only one factor in disease. By testing these cultures under standardized environmental conditions, on fixed hosts, and at uniform inoculum densities, we believe that the contribution of other factors to disease has been minimized and the differences observed here are due to inherent characters of the cultures themselves. The change detected in this study took 100 years of extreme conditions to increase by 10 percent. Based on these results, it seems very likely that present resistance to root rot in small grains will continue to provide protection for many years to come.

All living organisms, including plant pathogenic fungi, possess the ability to vary over time in response to changes in their environment or in the case of parasites, changes in their hosts. It appears that long-term continuous cropping of wheat in Bolley's disease garden has shifted the population of *C. sativus* toward more aggressive types when compared to the population in surrounding commercial fields subject to crop rotation. The degree of shift, while significant, is relatively small considering the time span involved. This suggests that the aggressiveness of the soil population of *C. sativus* is relatively stable. Because of the high level of disease in Bolley's continuous wheat plot, it continues to be a valuable tool in screening wheat varieties for root rot resistance (8,9,10).

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