Wheat Stem Rust: A Classic Disease of Cereals

Fig. 109.—Black Rust of Oats.
a, stem of oat with uredo-pustules; b and c, summer spores, one of which is germinating; d, stem of oat with puccinia-pustules; e and f, winter spores, one of which is germinating; g, barberry leaf with aecidia. (Orig.)

The story of stem rust caused by *Puccinia graminis* gained interest from the idea of "bridging hosts"—a concept developed in England in the late 1800s. According to this concept, varieties of *P. graminis* that, for example, infect rye, and to a limited extent barley but not wheat, could infect wheat after growing for a while on barley. The pathogen was thought to have adapted to wheat after growing on barley, which meant that barley was the "bridging host." If this theory were true, breeding for rust resistance would be futile. In 1915, E. C. Stakman discredited this bridging theory and went on to develop the concept of physiologic specialization. This involved the selection of a set of differential cultivars to identify races of rust. Of the four stages in the life cycle of rust, the pycnial and aecial stages are on barberry (*Berberis vulgaris*) and the uredial and telial stages are on wheat, oats, barley, rye, and many grasses. Specific knowledge of the different stages of the life cycle, especially the cytological aspects, is credited to J. H. Craigie, who discovered the sexual function of the pycnia in 1927.

Hybridization of rust, with formation of new races, occurs in pycnia on the barberry and elicited another reason for eradicating barberry bushes.

The damage is done by the uredospores, the "repeating stage" of the fungus; i.e., uredospores can go from wheat to wheat to infect new

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plants. They disrupt the epidermis of all aboveground parts of the plant, increase water loss by increasing transpiration, reduce photosynthetic area, and interfere with flow of nutrients from leaves and roots.

In 1916, a devastating epidemic of stem rust occurred in North America, generating in 1918 a campaign of barberry eradication as a cooperative state and federal program. Stakman was mainly responsible for organizing this campaign, which covered about one-third the area of the United States. By 1920, 4 million barberry bushes had been destroyed. Losses from stem rust in the barberry eradication area was estimated to be 184 million bushels of wheat in 1916, compared with 12 million bushels in 1925. However, the cumulative killing of barberry bushes failed to stop the great epidemics of 1935, 1953, and 1954. But barberry eradication had accomplished its goals by destroying bushes where outbreaks had occurred. It reduced stem rust prevalence, prevented early infection, and stopped the generation of new races from the barberry. The three epidemics resulted not from inoculum on barberry but from spores blown in from Mexico and the southern states northward into Canada, the so-called "Puccinia Pathway." Methodology of aerial dispersal of spores became integral in the investigation to understand spore dispersal up and down the Puccinia Pathway. The federal eradication program ended in 1975, after more than 500 million barberry bushes had been destroyed, with states accepting responsibility for further eradication. There has not been a significant wheat stem rust epidemic in the United States or Canada since 1974, although the pathogen is still present and potentially dangerous.

With support from the private sector and the U.S. Department of Agriculture, the Cereal Rust Laboratory (now the Cereal Disease Laboratory) was established at the University of Minnesota in 1915 and has become a center for national and international study of cereal rust diseases. Research is currently focused on genetics and molecular genetics on the host-parasite interaction as well as continuing investigations on the incidence and prevalence of cereal rusts. With consideration of Flor's gene-for-gene interaction, resistance genes that provide effective and durable resistance are identified. For example, the *Sr31* gene (developed in Mexico) provided stem rust resistance in wheat for more than 30 years. No virulence to this gene was found until recently, when a new rust collection was reported in Uganda (U99), for which no resistance is known, not even gene *Sr31.*

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