Wheat Stem Rust

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Black stem rust, caused by *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn., is potentially the most destructive disease of wheat crops in the United States. At least 12 severe epidemics have raged across the major wheat-producing areas of the Great Plains and caused serious economic losses since the late nineteenth century. More often, less extensive epidemics damaged the crops in smaller regional areas. The disease is under control today in the U.S. because of an effective program of integrated pest management: cultural practices, barberry eradication, quarantine, race identification, and host resistance. Much credit for this noteworthy accomplishment belongs to Dr. E. C. Stakman.

The Host

More than 20 million hectares (50 million acres) annually are planted to wheat in the U.S. The crop consists of five principal market types (19). The greatest proportion of our wheat crop consists of hard red winter wheat, planted mainly in the central and southern Great Plains. The northeast and west grow mostly white winter wheats. Hard red and durum spring wheats predominate in the northern Great Plains. In the eastern U.S., soft red winter wheats predominate. Among these market classes, about 270 wheat cultivars are in commercial use (20). In the south, much of the wheat acreage is grazed for winter forage, which often delays maturity and reduces grain yield.

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The winter wheats are planted in August through December and are harvested in April through August, depending on latitude. The spring wheats are planted in April and May and are harvested in August and September. Thus, living plants of wheat are available to the rust pathogen somewhere in the U.S. throughout the year.

The Pest

P. graminis in the natural environment is an obligate parasite; i.e., it grows and reproduces only on compatible hosts. This rust is long-cycled, having the full complement of five distinct spore forms. Also, it is heteroecious: it produces some of these spore forms on the gramineous host and others on the barberry host. Briefly, the life cycle is as follows: The resting phase of the fungus is the teliospore, a two-celled, black, thick-walled spore formed in sori on the maturing gramineous host. Only these spores are long-lived and resistant to the environmental extremes of winter in the north. With favorable conditions in the spring, teliospores undergo meiosis and germinate to produce haploid, short-lived basidiospores. The aerially transported basidiospores infect young leaves of susceptible species of the Berberidaceae by direct cuticular penetration. In these hosts, the parasitic mycelium develops into a pycnium, which consists of pycniospores and receptive hyphae. Insects attracted by a sugary secretion from the pycnia transport mononucleate pycniospores from one pycnium to another and thereby induce cross-fertilization to initiate the binucleate condition. When a pycnium is fertilized by pycniospores of the opposite compatible sex, the

mycelium develops into an aecium, which contains binucleate, single-celled aeciospores. This part of the life cycle on barberry results in hybridization of fungal characters and is the major source of the diversity of virulence in physiologic races.

Aeciospores infect only compatible gramineous hosts, in which the parasitic mycelium develops uredia and binucleate, single-celled uredospores. These rust-colored spores reinfect the gramineous host and this is the repeating stage of the fungus responsible for severe epidemics. These spores are resistant to desiccation and survive about one month at moderate temperatures but are killed by temperatures below 0 or above 40°C. They require only water at favorable temperatures to germinate. Successful infection of the host by uredospores requires a sequence of environmental conditions. The spore germinates best in the dark on the wet leaf surface at 18 C to form a germ tube and an appressorium over a host stomate. The appressorium develops a penetration peg, a substomatal vesicle and infection hyphae to initiate parasitism in the light at 29 C (25, 29). As the wheat ripens, uredia are transformed to telia, which contain binucleate, two-celled teliospores. With maturation, the two nuclei in each cell of the teliospore fuse to produce the diplophase, and the spore becomes dormant.

The Disease

The destructive phase of black stem rust is the numerous pustules produced during the repeating uredial phase on the leaves, stems, and inflorescence of the living wheat plant. When a uredospore germinates and penetrates a compatible host, the mycelium grows intercellularly about 1.1 mm/day (3). Hyphal branches penetrate the parenchyma cells and form specialized feeding structures, the haustoria. At favorable temperatures, the infection becomes visible, and the developing uredium erupts through the host epidermis about one week after penetration. The fungus continues to grow and sporulate, often forming concentric rings of secondary uredia, for two weeks or more if the lesion is surrounded by vigorous host tissue. In a greenhouse, a uredium produces about 5,000 to 25,000 spores/day (10). As the numbers of infections increase, competition for nutrients limits the size of each infection. The developing disease lesion is a metabolic sink. Respiration increases (3, 28) and photosynthate and nitrogenous compounds accumulate (3, 28, 30) in the infected area. The surface area of the blades and stem of an adult tiller of a typical wheat plant is about 150 cm^2 (35). Therefore, 1000 pustules uniformly distributed per tiller (or 6.7 infections/cm^2) essentially destroy the tiller's productivity. As rust approaches 100 percent severity, plant growth halts, the grain ceases to fill and shrivels, while the stem weakens and lodges, and the plant dies prematurely.

The Ecosystem

The most devastating epidemics of wheat stem rust occur in the central U.S. where most of our wheat is grown. Records of the estimated losses caused by stem rust since 1918 show that the disease damages the crop most often in the north-central states (Table 1). Rust affected wheat production on the average of one year in three in North and South Dakota and Minnesota, one year in five in Nebraska, and somewhat less than one year in ten in Kansas and Texas. These records reveal the frequency of rust damage for those years in which a significant part of the commercial crop was susceptible, inoculum of virulent races of rust was abundant, and the weather was favorable. However, these records underestimate the hazard, because rust is often present and weather favorable, but the crop consists predominantly of resistant varieties (22). In Minnesota, epidemics develop from natural infection on test plots of susceptible varieties in two out of three years. Thus, wheat would not be profitable in the north-central states without effective resistance to stem rust.

Years ago, barberries were an important source of primary inoculum of stem rust early in the growing season. A cooperative federal and state program for eradicating barberries

Table 1. Number of Years per Decade with Estimated Losses of 5 Percent or More in Wheat Yields Caused by Stem Rust in Some of the Major Wheat-Producing States of the USA, 1921 to 1970

State	Number of Years					Total Years
	1921-30	1931-40	1941-50	1951-60	1961-70	
North Dakota	5	3	1	5	2	16
South Dakota	4	3	1	4	3	15
Minnesota	7	3	1	4	0	15
Nebraska	0	3	1	3	3	10
Kansas	0	3	0	0	1	4
Oklahoma	0	0	0	0	0	0
Texas	1	1	1	0	0	3

was started in 1918. Since then, more than 5×10^8 bushes have been eliminated from more than one million square miles. No significant infection of wheat by stem rust has been traced to barberry in the north-central states in more than 20 years. Thus, the barberry phase of the life cycle of *P. graminis* is no longer important in the development of epidemics of stem rust in the central U.S.

However, eradication of the barberries was only a partly effective control, because uredospores are disseminated aerially between northern and southern wheat areas. The classic studies by E. C. Stakman and his associates (31) established the south-to-north movement of stem rust in the "*Puccinia* pathway" through observations of rust development, spore trapping, and occurrence of physiologic races. Similarly, inoculum from the north re-establishes stem rust in the southern overwintering areas during the fall when the rust fails to survive hot, dry summer weather in the south. Thus, rust develops cyclically each year: infection survives on wheat in the southern overwintering areas during the fall and winter, increases and spreads northward on the winter wheats of the central U.S. in the spring, develops on the spring wheats of the northern U.S. and prairie provinces of Canada in summer, and finally re-establishes and develops on fall-sown wheat in the south. Any prediction models for wheat stem rust must be designed on this framework of continental movement of inoculum.

Presently, the main barrier in the movement of stem rust between northern and southern wheat areas is the distribution of genes for resistance among the commercial cultivars and the genes for virulence in the prevailing biotypes of the rust fungus. The existence of physiologic races within *P. graminis* f. sp. *tritici* was discovered by Stakman and Piemeisel (34) in 1915. Later, Stakman and Levine (33) developed a system to identify the prevalent races in the U.S. Recognition of the differential reaction of wheat lines to different rust races provided the basis for developing varieties with specific resistance against stem rust. Varieties with resistance governed by a single specific gene have been impermanent, because new virulent races of the pathogen have soon appeared after the variety was planted widely. The gene-for-gene concept developed by Flor (7) with flax rust established that each gene for reaction in the host has a complementary gene for pathogenicity in the pathogen. Evidence from studies by Loegering and Powers (13) showed the gene-for-gene relationship in wheat stem rust. Thus, a single specific gene for resistance in the host is effective only as long as the corresponding gene for avirulence exists universally throughout the population of stem rust. Therefore, prediction models for wheat stem rust require knowledge of the specific genes for resistance present in the host cultivar and of the corresponding genes for virulence in the rust fungus.

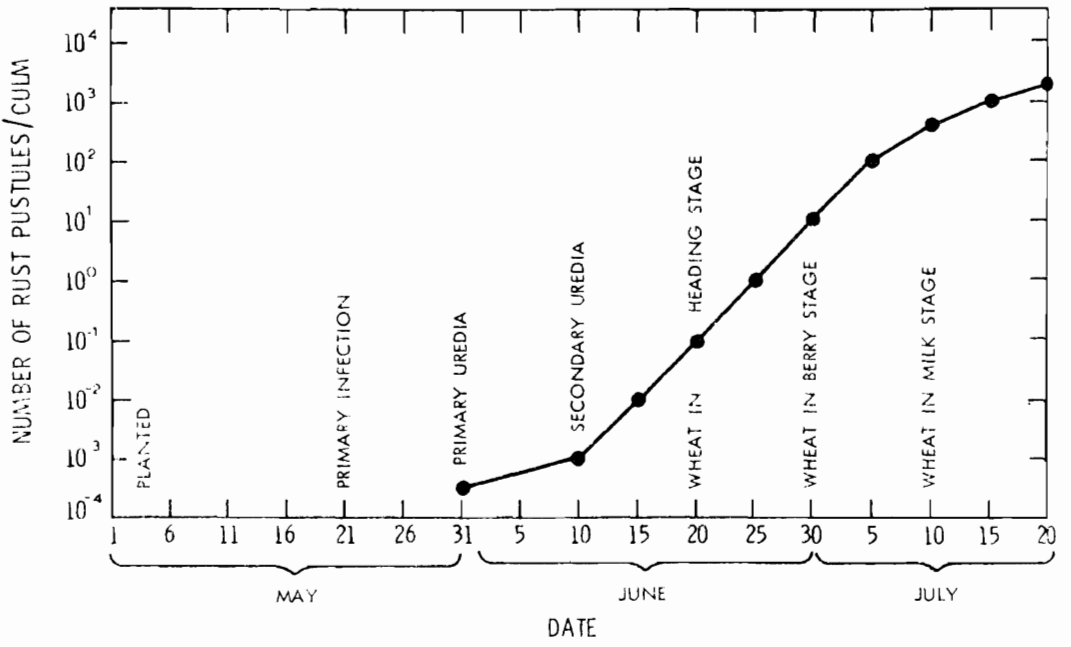

Figure 1. Relations between rust development, date, and stage of host development during a typical severe epidemic of stem rust on a susceptible variety of spring wheat in Minnesota.

Short-Term Prediction Models

Prediction of Epidemics

Development of a stem rust epidemic follows the typical sigmoid curve for population growth with the optimal combination of susceptible host, virulent pathogen, and favorable weather. Figure 1 shows a typical epidemic on spring wheat in the north-central states. There, wheat generally is planted about May 1, and primary inoculum may arrive any time during May. In Figure 1 primary inoculation occurred at mid-May, when the seedlings had only two fully expanded leaves. The frequency of primary infection at breakout about June 1 was 1 uredium/30 m of row and equivalent to 5 uredia/10,000 culms for a mature stand. Secondary infection appeared about June 10, and the amount of visible infection was doubled. Thereafter, rust increased exponentially. The figure shows a severe epidemic, in which uredial numbers increased tenfold every five days at a rate of 0.48 per unit per day as calculated by van der Plank (18). At this rate, the numbers of rust pustules increased one millionfold within one month and the crop was destroyed in the milk stage.

The effect of rust on crop productivity is negligible during the exponential phase of increase. As the amount of rust approaches 100 uredia/tiller (10 percent severity), it increasingly affects crop productivity, and the rate of rust increase slows as the proportion of uninfected tissue available for new infections declines. Thus, models that describe stem rust epidemics are essentially descriptors of population growth.

Van der Plank (18) has shown that stem rust epidemics conform to the model for "compound interest disease." In this model, the data fit a linear regression when transformed to logits by $\log_e (X/1\text{-}X)$, where X = the observed units of disease, and 1 = the total units at 100 percent disease. Either numbers of trapped spores (24) or uredia (2) may be used as measures of disease incidence. Van der Plank (18) showed that the rate of disease increase (r) is calculated by the formula:

$$r = \frac{1}{t_2 - t_1}\left(\log_e \frac{X_2}{1-X_2} - \log_e \frac{X_1}{1-X_1}\right)$$

$$\frac{\log_e 7X_2}{1-X_2} - \frac{\log_e 7X_1}{1-X_1}$$

where t_1 and t_2 = the times of the first and second disease observations, X_1 and X_2 = the numbers of disease units in the two disease observations, and 1 = the total number of units at 100 percent disease.

If one knows the amount of rust present in a field, the approximate rate of increase, and the stage of plant development, one can reasonably predict the course of the epidemic. Buchenau (1) has used this relationship to design a relatively simple model for growers to determine the economic feasibility of applying sprays for the control of rust. His model (Fig. 2) is based on eight years of observing rust development in tests of chemical control of rust. To predict rust development, the operator counts the number of uredia on 100 stems and plots the mean/stem on the logarithm axis at the days from heading on the time axis. The operator selects either a fast rate of increase, if the weather of the week before was wet with frequent dews, or a slow rate, if weather was dry with no dew. For a fast rate of rust increase, disease progress is extrapolated at a slope angle of 71°, and for a slow rate of rust increase, it is extrapolated at a slope angle of 41°. The point at which the plot intercepts the line for 40 days after heading gives the final severity of stem rust if 100 percent severity (1000 pustules/stem) is not reached earlier. Thus, at 0.1 pustules/tiller at heading, the model predicts that stem rust would be about 100 percent severe 17 days later when the wheat would be in the milk stage, for a fast rate of increase, or about 1 percent severe at 40 days, when the wheat is ripe, for a slow rate of increase. Thus, with this relatively simple model, the grower can quickly forecast the stem rust epidemic in his wheat field.

Precise short-term predictions would require not only a measure of the disease units, but also an accurate determination of the rate of rust increase. Eversmeyer et al. (6) used stepwise multiple-linear-regression computer programs to identify and quantify the biological and meteorological variables useful in explaining the variation in stem rust development observed for four years in test plots at six sites. Of 15 variables examined, they found

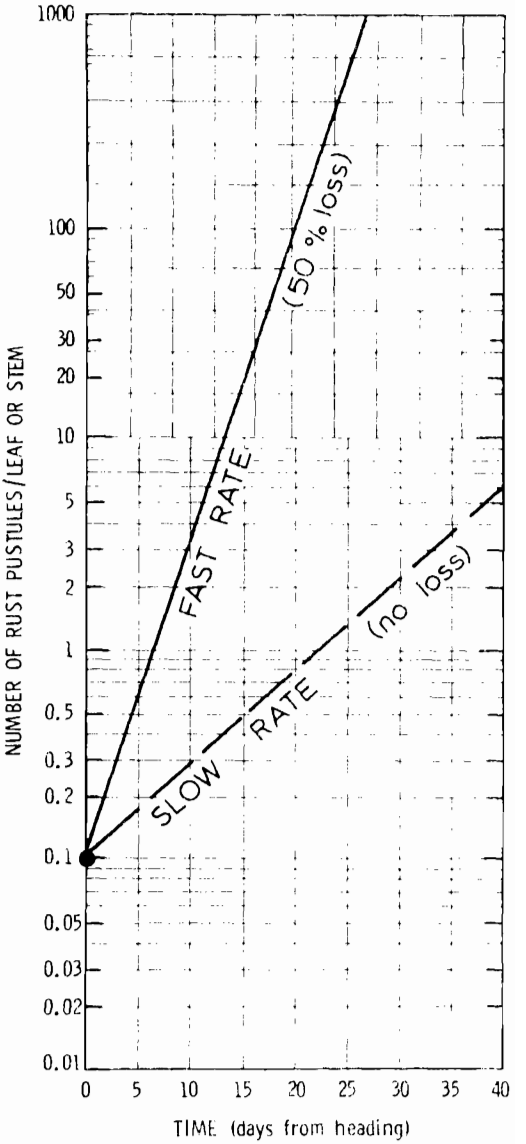

Figure 2. Short-term prediction model developed by Buchenau (1) to forecast the extent of rust development and losses in yield of wheat in South Dakota. Predictions are shown for stem rust epidemics at an incidence of 0.1 pustules/stem at heading for a fast rate of increase with a 50 percent loss in yield when weather was wet during the preceding week, and for a slow rate of increase with no loss when weather was dry during the preceding week.

that disease severity estimates, weekly and cumulative number of uredospores trapped/cm^2, cultivar, wheat growth stages, maximum temperature, minimum temperature, a fungal-temperature growth function, and a weekly

infection function were the most significant for the successful prediction of stem rust. Standard errors of the estimates obtained with their equations indicated that stem rust development could be predicted within 18 percent at 30 days from the prediction date.

The relative success of the two models for short-term prediction of stem rust epidemics depends on a disease observation at the prediction date and environmental observations for the seven previous days. Thus, the events of the immediate past during an epidemic largely govern the future course of disease development.

Prediction of Losses

The relationship between amount of stem rust and reduced grain production is evident to any farmer. The increase in weight of the developing kernels is linear with time between the heading and dough stages of plant development (16), and plant vigor is critical during this period. Thus, careful observers of wheat crops become proficient estimators of yield production. Such experience was used by Kirby and Archer (12) in the earliest model for predicting losses caused by stem rust. They composed a table for seven increasingly severe epidemics, with disease severity listed at six stages of crop development for each. All epidemics had the same constant rate of rust increase. The percentage of loss given for the epidemics was used to predict losses from observed rust severity and plant stage.

Greaney (8) and coworkers determined empirically the relation between stem rust and losses in yield by analyzing data from many experiments, in which stem rust was controlled to varying degrees with different schedules of sulfur dusting. Their model indicated that the percentage of loss in yield equalled half the percentage of terminal severity of rust. Kingsolver et al. (11) used different levels of inoculum applied to winter wheat at jointing to determine how severity of rust epidemics related to yields. Although cross contamination of inoculum between plots made all epidemics relatively severe, their data indicated a linear relation between terminal severity and yield. The data also showed a relation between yield and the stage of wheat development when rust became 1 percent severe.

Using the evidence that the loss in yield is a function of the amount and the duration of rust, van der Plank (18) formulated the hypothesis that the loss in yield is directly proportional to the area under the disease-progress curve. His analysis of the information for estimating losses given by Kirby and Archer (12), as well as the experimental data of Kingsolver et al. (11), supports the area hypothesis. Buchenau (1) uses the area hypothesis in his model to predict the course and destructiveness of a rust epidemic (Fig. 2). Thus, the top part of his graph for rust severities of 10 to 1000 uredia/tiller, or about 1 to 100 percent severity, is divided equally into a grid of 100 squares. Counts of the number of squares to the right of the extrapolated plot of the rust epidemic give the numerical percentage of loss in yield. His model predicts a 50 percent loss in the example with 0.1 pustules/tiller at heading for a fast rate of epidemic increase and no loss is predicted for the slow rate of epidemic increase.

Some doubt exists that the area hypothesis adequately describes the relations between rust epidemics and crop losses. Romig and Calpouzos (23) could not relate loss to the area under the disease-progress curve when they tested different stem rust epidemics after different fungicidal treatments on a single variety. In their data, the best estimate of loss was the $\log_e$ of disease severity at the stage when kernels had developed to three-fourths of their final size. Later, in a large scale test, Calpouzos et al.[2] examined 466 stem rust epidemics on six varieties at three sites for three years. Their data yielded three dimensional physical models of the percentage of loss in yield on a grid of the stage of plant development at the point of epidemic onset and the slope of the epidemic development. The percentage of variability in the raw loss data explained by fit to the proposed model

2. Calpouzos, L., M. Madison, F. B. Martin, A. P. Roelfs, and R. D. Wilcoxson. A model for describing the relationship between stem rust of wheat and wheat yields. Manuscript form.

was 69 percent. The model for percentage of yield loss (Y) as a function of slope and onset was as follows:

$$Y = f(X_1, X_2)$$

where X_1 = the value of onset coordinate
X_2 = the value of the slope coordinating axis
f = the functional relationship for the loss contour

Not surprisingly, researchers disagree about the relation of stem rust epidemics to yield. A recent review by James (9) summarized the development of methods to assess disease losses. Under most crop conditions, actual yields are appreciably short of potential production. Actual farm yields of 11,353 and 14,057 kg/ha have been recorded for wheat in Washington (19), where the 10-year average production is 2,825 kg/ha. Many environmental and biological factors affect tissue function and curtail the productivity of wheat. The effect of rust is measured against this background of other restraints on yield. When fungicides are used to alter the course of an epidemic or to determine the yield potential of the crop free of rust, the effect of other diseases or physiological factors on yield losses also may be altered. Furthermore, the least significant differences observed between treatments in experimental tests generally range from 134 to 268 kg/ha. This amount of imprecision limits determination of the differences in losses that are caused by small differences in rust epidemics.

Measurement and prediction of stem rust epidemics depend on the increase in number of units of the pathogen, whereas yields are reduced by the loss of functional host tissue. Correlating units of the pathogen to crop loss assumes a fixed relation between amount of disease and loss of tissue function. Stem rust infections do not develop with a random distribution, but rather first become abundant near the early infections on the lower plant parts and increase progressively to the top of the plant. When rust becomes too abundant for ready counting, the observer relies on estimations of disease severity, using reference scales such as that of Cobb (5) or the modified scales of Melchers and Parker (15). As the numbers of infections increase within a limited area on the host, nutritional competition markedly reduces the size of each pustule. Peterson et al. (17) expanded the Cobb scale from 5 to 48 diagrams, to represent more adequately the range in size and numbers of uredia that are found on different parts of the plant during an epidemic. Because rust is not uniformly distributed, its average severity value is estimated from observations on how rust frequency varies on and between plants in a plot. The precision of these estimates depends greatly on the skill of the observer. Such estimates can be only approximate measures of the loss of tissue function.

Accurate estimates of average rust severities may not measure the average loss of plant productivity. Many researchers have tried to determine how various plant parts contribute to the filling of the grain after heading. Results suggest that the flag leaf is important in the final yield production. Studies by Bushnell and Rowell (4) showed that the lowermost leaves and sheaths on the wheat plant sustain the growth and respiration of the adventitious root system. When these tissues are severely rusted or removed, root vigor declines and the plant dies prematurely. Thus, a concentration of rust in one area of the plant may have a greater effect on plant productivity than a more dispersed amount of rust on the same average rust severity.

Long-Term Predictive Models

No model exists for the long-term prediction of wheat stem rust in the U.S. We can only warn about a stem rust hazard when we know that a virulent race is prevalent in the stem rust population and that much of the planted acreage consists of one or more susceptible cultivars. The virulence characteristics of the stem rust population is determined annually by race identifications among the isolates from about 500 collections of wheat stem rust. The susceptibility of the 270 or so commercial varieties in use is determined from seedling tests with selected isolates of diverse

races and from observations on adult plants in nurseries. Field surveys in April by experienced observers determine the amount of infection present in the southern overwintering areas of south Texas and states along the coast of the Gulf of Mexico. Surveys in north Texas, Oklahoma, and Kansas during May; in Nebraska and South Dakota in June; and in Minnesota, North Dakota, and Montana in July follow the northward spread and development of stem rust. The information from these activities provides the basis for assessing the stem rust threat and issuing warnings when needed.

Developing long-term predictive models for stem rust in the major wheat-producing areas is hindered mainly by our inability to predict the time of initiation and amount of primary infection. With this information, plus reasonably accurate long-range meteorological forecasts for an area, we could predict stem rust epidemics and losses by the short-term predictive models. Thus, prediction of the time and frequency of primary infection on northern wheat requires a model based on the incidence of infection in the source area and on the efficiency of atmospheric conditions for launching, transporting, and depositing of inoculum.

The primary inoculum arriving in the northern wheat areas depends on the amount of inoculum in the southern overwintering areas. Stem rust often overwinters on wheat south of the 30° parallel in the U.S., where wheat is a minor crop planted mostly as winter forage for cattle. There, the fields are scattered widely and are difficult to find for a systematic survey. In southern Texas, the fall-sown wheats are semi-dwarf spring types of indeterminate day-length requirement, whereas soft red winter wheats are planted in the other states along the Gulf coast. Stem rust often is found readily in these areas during fall and winter. Its survival through the winter is limited by either extended droughts or extended periods of severe cold that kill the leaves back to the crowns and eliminate stem rust from the area. Remote monitoring of crop conditions by satellite in the Earth Resources Technology Satellite program (14) promises to help determine the potential for stem rust overwintering in the source areas.

Recent information suggests significant overwintering of stem rust in the winter wheat area of the Great Plains north of the 30° parallel (21). In race identifications of southern Texas collections from 1970 to 1973, the members of the race 15B group were not detected, but later appeared and predominated in the hard red winter and spring wheat areas. Researchers are studying test plantings inoculated with marker isolates of stem rust with distinctive virulence patterns in an attempt to identify sites and conditions of overwintering north of the 30° parallel.

Locating the overwintered sources of stem rust is a problem, because it is difficult to detect low levels of rust incidence. The lowest threshold at which our most proficient observers can reliably detect the incidence of stem rust is about 1 uredium/15 m of plant row. At this level, if they find one uredium, they can find other uredia. If more than 15 m of row are examined before one uredium is found, it is not likely that extensive search will reveal others. Experience with the various types of spore traps suggests that they detect primarily spores arising from infections near the trap. When an occasional spore is trapped, we generally can find a low incidence of natural infection on the wheat around the trap. Thus, spore traps are no better than direct search for the presence of stem rust, although they are useful for monitoring the increase of stem rust above the detection threshold level (27).

Establishing the source of inoculum was a problem when a widespread, natural primary infection of stem rust was observed on 100,000 km^2 in northern Oklahoma and eastern Kansas in May, 1970 (26). Conditions were particularly unfavorable for the survival of stem rust in south Texas that year. Race identifications of collections from the area with uniform primary infection in Kansas and Oklahoma yielded predominantly race 15B, whereas collections from the few infections found in Texas yielded predominantly races 11 and 151. Thus, the primary inoculum for the affected area in Oklahoma and Kansas

appeared to arise from some source other than that in Texas.

Only the broad general principles are known about the long-range dissemination of uredospores from the source areas. Many years ago, Stakman and coworkers (32) established the presence of uredospores in the atmosphere as high as 5 km. Rowell and Romig(27) showed that rains washed uredospores from the air and initiated primary infection on spring wheats early in the season. We lack information on the atmospheric processes required to launch the uredospores into the upper air masses favorable for long-range transport and on the trajectories of viable inoculum in the upper air.

Conclusions

In the development of short-term models of wheat stem rust, we have modestly succeeded in predicting disease development and yield losses within wheat fields after disease is present. This success has resulted mostly from how the prevailing weather during the week before the disease observation affected the course of the epidemic. We lack adequate knowledge to develop reliable long-term predictive models for regional occurrence of stem rust. Such models require methods to detect and estimate inoculum sources, information on the quantitative aspects of long-range aerial dissemination, and accurate long-range weather forecasts.