

Analysis of Recent Oat Stem Rust Epidemics

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ABSTRACT

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An oat stem rust epidemic occurred in the eastern Dakotas and western Minnesota in 1977. Because the pathogen races generally have been virulent on the commercial cultivars during the past 38 yr, this epidemic was chosen as a model disease to investigate for the purpose of making long-range disease forecasts. Temperatures and rainfall frequency and amount were above normal throughout the 1977 epidemic. Thus, these factors were examined to see if they were associated with other epidemics during a 38-yr

period for Minnesota, South Dakota, and North Dakota. However, little relationship was found between estimated percentage of yield loss and the minimum or maximum temperatures for May, the period May through July, or the mean frequency or amount of precipitation for the same periods. Date of onset of rust in an area was related to the estimated yield losses, and explained about 20-40% of the variation in estimated loss.

Additional key words: yield loss, *Puccinia graminis* f. sp. *avenae*, temperature, precipitation, *Avena* sp.

Oat stem rust (caused by *Puccinia graminis* Pers. f. sp. *avenae*) has been an important disease of oats (*Avena sativa* L.) in the United States. Serious epidemics in the north central states of Iowa, Minnesota, North Dakota, South Dakota, and Wisconsin occurred in 1943, 1944, 1953, 1954, and 1955 (9). This is the major oat-producing area in the United States. The lack of regional epidemics in this region since 1955, even though the predominant race was virulent on the commercial cultivars, resulted in speculation that the virulent race lacked the aggressiveness of earlier races, or the reduction of acreage of oats grown in the other portions of the United States had resulted in insufficient inoculum to initiate an epidemic.

The regional epidemic of oat stem rust in 1977 provided an opportunity to study the components of the epidemic (ie, host, pathogen, disease, and environment) on a regional basis. Because

the host and pathogen apparently had changed little in recent years (10), and the environmental conditions seemed especially favorable for stem rust, we chose to study disease records over a period of years to evaluate these variables. The weather variables associated with stem rust development on susceptible hosts have been studied individually and collectively in the laboratory, but have not been related to the initiation of a regional epidemic. This must be done to develop a long range (greater than 30-day) disease forecast for a wind disseminated pathogen on a relatively low value crop grown on millions of hectares. Thus, practical forecasts will require regional inputs for the epidemic components. This paper compares the 1977 epidemic to epidemics of previous years.

MATERIALS AND METHODS

Annual surveys of disease prevalence and severity have been conducted. The geographical route varied, but was preplanned to pass through the primary cereal-growing area. Commercial production fields were examined each 32.2 km (20 miles), based on the car odometer. Additional stops were made en route at

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TABLE 1. Weekly oat stem rust severity and prevalence in the most severely rusted fields observed during the 1977 rust epidemic in the north central states

Week of	South Dakota		North Dakota					
	Northeast		Southeast		East central		Northeast	
	Severity ^a	Prevalence ^a	Severity	Prevalence	Severity	Prevalence	Severity	Prevalence
June 27	Trace	Trace	... ^b
July 4	10	100
July 11	25	100	5	100	Trace	Trace	Trace	Trace
July 18	60	100	35	100	20	100	Trace	10
July 25	Harvested		Harvested		60	100	30	100

^aSeverity = based on modified Cobb scale (8), prevalence = percentage of culms effected.

^b... Data for this area are missing.

experimental plots.

The annual physiological race survey was made in spite of the lack of detected disease losses in recent years. Collections were obtained from the annual disease survey, one collection per diseased commercial field visited, plus one or two collections from rusted cultivars at the experimental plots visited (ie, the most susceptible and the most common local cultivar). Additional collections were made from rusted wild oats (*Avena fatua* L.). Our collections were supplemented by collections submitted by cooperators.

Weather data were obtained from North Dakota and South Dakota weather stations east of 99° W longitude and from Minnesota west of 95.5° W longitude for the period 1940-1977. The average of data from approximately 30 stations per state for May and the period May through July were compared by linear regression with the annual estimated yield losses (9) for the state. Temperatures studied were both the maximum and minimum for each period. Amount of precipitation was handled in the same manner. To examine the effect of precipitation frequency, the data were analyzed on the basis of the number of days of precipitation during the same two periods omitting days with less than 2.5 mm of precipitation.

Date of disease onset was obtained from the disease surveys for each state during the 38-yr period. This was defined as the day on which stem rust was first reported annually. In a few cases, the data are somewhat biased in that the data were from plot observations and not from commercial fields; in other cases, the rust may have been present for several weeks before it was observed. Julian days were compared by linear regression with the yield losses estimated for the state (9).

To evaluate the relationship of the date of onset in the northern growing areas with disease further south, the date of onset of the disease in the northern states was compared by linear regression with the date of onset in Iowa (central) and Texas (central). The latter is a winter oat-growing area where stem rust can occasionally overwinter. Lack of data currently limits comparisons with other locations.

RESULTS AND DISCUSSION

Disease. Oat stem rust was first observed during 1977 in mid February at Beeville, Texas. Our field surveys showed rust had overwintered in trace amounts as far north as southern Oklahoma. A very cold winter and removal of infected tissue by grazing had reduced the host tissue, and thus the amount of stem rust overwintering. However, the observed lack of crown rust provided sufficient host material for stem rust to increase rapidly without competition in the spring, when conditions became favorable. Stem rust spread into the Texas panhandle for the first time in recent years. By the end of the first week in July, oat stem rust was widespread in the north central states, but most of the crop was in the dough stage due to an early season. During the second week in July, on a routine cereal rust disease survey, a field of oats in western Minnesota was observed with 30% stem rust severity. This was found to be the eastern edge of an area of severely rusted oats that stretched 80 km either side of a line from Brookings to Milbank,



Fig. 1. Area affected by the 1977 oat stem rust epidemic; A, approximate area of initial infection; B, area with losses of 10-20% in late fields; and C, limits of 100% prevalence.

South Dakota. Approximately 10% of the oats in this area were in the milk stage. The initial infections in this area had been observed during the period 14-24 June. Over the last 2 wk of July, the disease increased rapidly (Table 1). Late oats were severely rusted at maturity in a fan-shaped area from Brookings, South Dakota northward. The most severely rusted area was along the North and South Dakota borders with Minnesota (Fig. 1). Farmers reported test weights for these late fields ranged from 23 to 31 kg/hl (18-24 lb/bu) with estimated yield losses of up to 30% (using the methods of Roelfs [9]).

Host. There have been three principal hosts of oat stem rust in the Great Plains. *Berberis vulgaris* (L.), the alternate host, has been eradicated from the immediate vicinity of oat fields in this area. The alternate host has not been a direct factor in oat stem rust epidemics for more than 40 yr in the Great Plains (12).

Wild oat is a common weed in commercial small grain cereal fields in northern Texas and southern Oklahoma and also in the Dakotas and Montana. No race-specific genes have been reported for this species. It is frequently severely rusted; so, if nonrace-specific resistance exists, it is apparently of a low degree.

Cultivated oat, *Avena sativa* L., is the principal host, based on area occupied, even though in some years most of the stem rust occurred on the wild oats. Oat acreage has decreased during the past 30 yr from over 15×10^6 to near 5×10^6 hectares. Changes have occurred in the cultivars grown during the past 30 yr (14), but changes since 1967 have been gradual. The current cultivars have genes Pg-1, -2, or -4, or the combination of Pg-1 and -4 or Pg-2 and -4. These genes have been ineffective in conferring resistance to the most prevalent race of the rust pathogen since 1965 (10). Although some nonrace-specific resistance may exist in some of the cultivars grown, no major differences in cultivar susceptibility have been reported.

Pathogen. The oat stem rust race most commonly identified during the 1943 and 1944 epidemics was virulent on oat cultivars

TABLE 2. Summary of environmental variables for the area between 95.5 and 100° longitude in the north central states for the years 1940-1975 with and without epidemics of oat stem rust

Location and year(s)	Mean monthly temperatures (C)				Mean monthly precipitation			
	Minimum		Maximum		Frequency ^a		Amount (cm)	
	May	May-July	May	May-July	May	May-July	May	May-July
Minnesota								
1940-1977	6.1	10.8	20.1	24.5	6.2	6.5	3.17	3.42
Epidemic mean (10) ^b	6.1	10.9	20.5	24.0	6.9	6.8	3.49	3.62
Non-epidemic mean	6.0	10.8	20.3	24.6	6.0	6.4	3.10	3.37
Highest	11.8	13.1	25.5	26.8	11.3	9.2	6.56	5.52
Lowest	2.4	8.4	16.3	16.7	1.8	4.2	0.49	1.74
South Dakota								
1940-1977	6.6	11.9	20.6	26.2	6.0	6.2	3.02	3.26
Epidemic mean (14)	7.0	11.7	20.1	25.7	6.6	6.6	3.08	3.50
Non-epidemic mean	6.3	11.9	20.3	26.4	5.8	6.0	3.00	3.15
Highest	11.8	13.9	25.9	27.9	10.8	9.4	6.71	5.81
Lowest	4.3	9.9	18.9	23.0	1.5	2.7	0.55	1.45
North Dakota								
1940-1977	5.3	7.7	19.7	24.3	5.8	6.0	2.58	3.02
Epidemic mean (9)	5.2	10.1	19.0	24.0	6.8	6.4	3.26	3.18
Non-epidemic mean	5.2	9.9	19.9	24.5	5.4	5.9	2.39	2.96
Highest	11.0	13.6	26.2	27.0	10.6	7.5	6.97	4.86
Lowest	3.0	8.2	15.2	21.4	1.8	4.0	0.67	1.61

^a Number of days with 2.5 mm precipitation or more per month.

^b Number of years with losses estimated to be 1% or more.

TABLE 3. Reported time periods of oat stem rust epidemic onset in selected years

Year(s)	Reported date of disease onset (Julian date)				
	Central Texas	Iowa	Eastern S. Dakota	Eastern N. Dakota	Minnesota
1940-1977 mean	107	176	190	202	183
Epidemic^a					
1943	141	174	179	202	175
1944	132	171	203	...	179
1945	104	183	198	188	183
1947	142	184	197	...	189
1950	... ^b	158	178	209	173
1951	130	179	200
1952	82	175	190
1953	100	155	182	173	161
1954	124	157	168	173	167
1955	79	161	165	164	161
1956	38	167	167
1957	45	171	171
1964	...	168
1970	147	175	...	195	...
1971	76	176	201
1977	118	158	178	193	165
Epidemic mean	104	170	185	187	172
Non-epidemic mean	108	180	193	206	187
Earliest ever	12	155	165	164	161
Latest ever	152	200	217	232	208

^a Statewide loss >1% in Minnesota, North Dakota, or South Dakota.

^b ...Data for Texas are missing, in other cases no epidemic occurred in the state.

carrying the *Pg-3* gene, and the second most common was virulent on cultivars having the *Pg-2* and *-3* genes. These races occurred in the epidemics of 1953, 1954, and 1955 as a small part of the population. The prevalent race in these epidemics was virulent on cultivars having *Pg-1* and *-3*. These races gradually decreased in frequency and by 1965 had been replaced by a race virulent on hosts with resistance genes *Pg-1*, *-2*, *-3*, *-4*, and *-8*. This race, variously designated (11) as race NA 27, 31, C10, and 6AF, has been the most commonly identified race for more than 12 yr (10). Thus, although races have changed, in general, there has been an increase in virulence that was more than sufficient to overcome the resistance of commercial cultivars. In a separate study (*unpublished*, J.B.

Rowell and A.P. Roelfs) no difference in aggressiveness was found between three random cultures obtained from 1977 and a culture from the 1960's.

Environment. The mean daily temperature in 1977, in the area of the oat stem rust epidemic, was higher than normal by 4.4, 3.9, and 3.3 C in May; 5.0, 6.1, and 6.7 C in June for northeast, South Dakota; southeast, North Dakota, and eastcentral-northeast, North Dakota, respectively. July temperatures were 1.1 C above normal for the entire area (15). Based on the studies of wheat stem rust by Lambert (5), Levine (6), Stakman and Lambert (13), and Peltier (7), this temperature would be anticipated to be favorable for disease development, but Wallin (16) thought that this temperature could be unfavorable for epidemics. The effect of maximum and minimum temperatures evaluated separately was helpful in short-range forecasts (3). From a comparison of epidemic and non-epidemic years (Table 2), appreciable differences do not exist between the two groups of years in the means for either maximum or minimum temperatures for May or the means for the period May through July. When the various temperature variables were regressed against the estimated yield losses, there was little or no association. The years with severe (losses >10%) epidemics tended to be warmer than average and years with no losses were those with lower-than-normal temperatures; however, moderate losses (1-9%) may occur in years with temperatures lower or higher than normal. This observation agrees with the data of Levine (6).

Lambert (5) found no association between frequency of rainfall and wheat stem rust severities, but Eversmeyer et al (3) found some association when making 30-day forecasts. Peltier (7) reported that lack of even distribution of rainfall was a limiting factor in wheat stem rust epidemics in Nebraska. Atkins (1) stated that frequent dews, that probably were correlated with higher frequency of rains, were a factor in the 1935 epidemic. Rainfall (>2.5 mm) frequency was 0.8-1.4 more than normal in May in epidemic years. However, a slight relationship existed when estimated yield losses were regressed against frequency of precipitation. This resulted from a large number of years that had nearly average rainfall frequency and a wide range of estimated losses.

Atkins (1) and Levine (6) reported that amounts of precipitation greater than normal enhanced epidemics, but Wallin (17), Lambert (5), Eversmeyer et al (3), and Stakman and Lambert (13) found little association between amounts of precipitation and disease epidemics. Analysis of the data in Table 2 for the upper midwest oat stem rust epidemics gave a very low coefficient of determination.

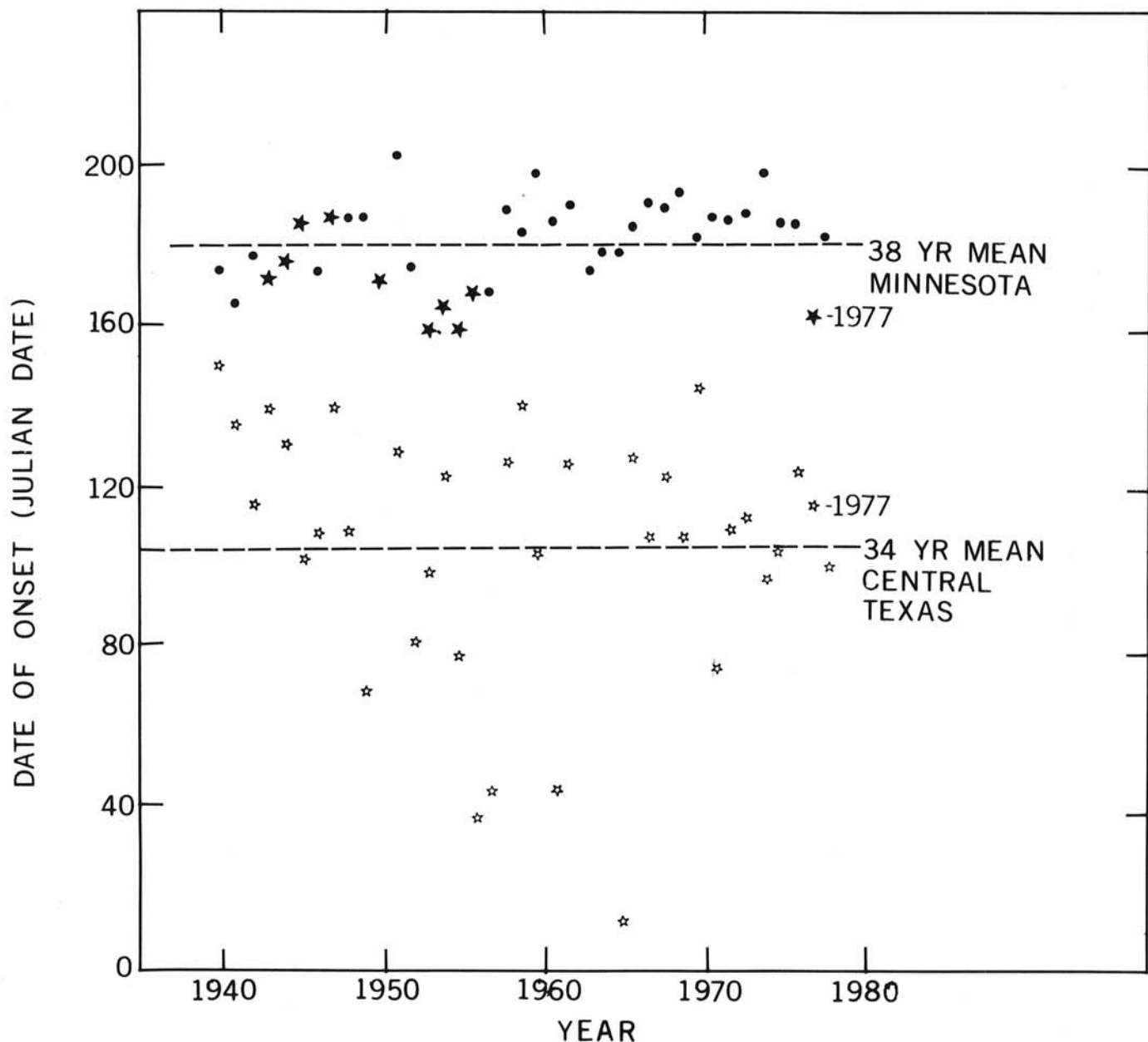


Fig. 2. Date of oat stem rust appearance (in days after 1 January) in Minnesota and central Texas, during the period 1940 through 1977. • = onset date for Minnesota; ★ = years with estimated losses greater than 1% in Minnesota; ☆ = onset date for central Texas.

However, the severe epidemics generally did not occur in years with much less than normal rainfall. Moderate losses (1–9%) occurred in years in which amounts of rainfall varied from much above to much below normal.

Disease. Hamilton and Stakman (4) reported that epidemics of wheat stem rust in the western Mississippi basin were related to the time of rust appearance. Similar data for oat stem rust were collected but not compiled. Linear regression of date of disease onset and estimated yield losses gave coefficient of determination (r^2) values of .25395, .42449, and .18906 for Minnesota, North Dakota, and South Dakota, respectively. These values were fourfold greater than those of any other factor studied. The mean date of disease onset (Table 3) for each state for epidemic and non-epidemic years showed that epidemics occurred in years when the disease onset was 7–19 days earlier than the mean of the non-epidemic years. An example of the relationship between date of onset for Minnesota and years with estimated losses greater than 1% is shown in Fig. 2. In a few years, particularly in the Dakotas, stem rust had already been present for at least one generation before it was reported. Thus, the date of disease onset could probably be even more useful if it were based on careful surveys and

if trap plots were observed frequently and early in the season.

The pathogen in the north central states was wind-borne from a southerly location, so a relationship exists between date of onset in the north and disease occurrence in the south. Currently compiled data are limited to date of onset without disease severity and prevalence on that date. Unfortunately, the only southern locations for which data are available for most of the 38-yr period are in central Texas and Iowa. The date of onset in Iowa averaged 1 wk earlier than in Minnesota; however, at that time of year the disease has an incubation period of 8–12 days, therefore it probably does not result from a cause and effect relationship; ie, uredospores produced in Iowa do not start Minnesota epidemics. In Fig. 2, the possible relationship between date of onset in Minnesota and central Texas is shown.

Another factor that can affect losses and that is not included in this analysis would be plant growth stage at date of disease onset. Unfortunately, such data are either not available or not collated.

For long-range forecasts of a disease such as oat stem rust, the actual temperature and precipitation data seem to be of little value except in overwintering areas (16). Although these variables are known to be important for disease initiation once inoculum is

present, they must not often be limiting in this area. Apparently Chester (2) was correct in his statement that "---the weather for the remainder of the growing season is poorly correlated with rust development, since even with marked departures from normal, it is still within the range favoring rust multiplication and usually the latter months also have adequate rainfall for rust even when the rainfall is below normal." Thus, long range forecasts (30-90 days) of cereal rust epidemics in the upper mid-western states apparently will have to be based on host resistance, pathogen virulence, date of disease onset, historical record of past epidemics, and perhaps host growth stage at disease onset.

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