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Epidemiology in North America

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I. Introduction and History

The classical studies of epidemics of plant disease include many examples involving the cereal rusts. Among the first plant disease epidemics that were studied as they developed on a regional basis were the wheat stem rust epidemics that occurred in the United States in 1923 and 1925 (Stakman and Harrar, 1957). Earlier epidemics had been studied after the disease had become severe over a large area or, in some cases, years later. The studies by Stakman and his co-workers became the basis for the area of phytopathology that became known as epidemiology.

Chester (1946) in his monograph provides an insight of early botanists and their concern about the rusts. He also reevaluated previous opinions on epidemiology, to which he added his experience, and then laid the basis for the regional development of the leaf and stem rusts of wheat. The rust diseases are particularly apt to cause epidemics, because these fungi have a high capacity to produce spores, the urediospore is well adapted for wind transportation, and there is a vast area of cultivated host plants.

A. WHEAT STEM RUST

Records of the earliest epidemics in colonial North America are scant and most were lost. However, at least in localized areas, wheat stem rust (caused by *Puccinia graminis* Pers. f. sp. *tritici*) must have been a problem because barberry eradication laws were passed in Massachusetts, Rhode Island, and Connecticut in 1754, 1766, and 1776, respectively. The north central region of the United States suffered a severe wheat stem rust epidemic in 1878 (Hamilton, 1939). This epidemic affected southern Minnesota, northern Iowa, and Wisconsin. As more of the northern Great Plains was planted to wheat, the frequency of reported regional epidemics increased. Bolley (1891) raised the question concerning the source of inoculum that generated stem rust epidemics. He noted that epidemics could arise from local (aeciospores) or from general (urediospores) inoculum sources. Regional epidemics occurred in 1904 (Carleton, 1905), 1916, 1923, 1925, 1935, 1937, 1953, and 1954 (Stakman and Harrar, 1957). The epidemic of 1904 was more extensive than the one of 1878 and included Minnesota, North Dakota, South Dakota, and to a lesser degree extended northward into Canada and south and eastward through Iowa, Nebraska, Kansas, and Wisconsin. The next three epidemics of 1916, 1923, and 1925 were similar in size to the 1904 epidemic. The 1916 epidemic caused a series of actions that resulted in programs for barberry eradication, pathogen quarantine, breeding for disease resistance, determination of pathogen variation (Chapter 1), and disease epidemiology. Again in 1935, 1937, 1953, and 1954, epidemics affected approximately the same area as the 1904 epidemic did and occurred when a shift in the principal pathogen race resulted in the majority of the wheat cultivars being susceptible to that race. Large regional epidemics, such as those that extended as far south as Kansas, probably resulted from urediospore movement from a southern overwintering source. Winds in this area move generally from the south in the spring and summer. Infected barberry has seldom been found as far south as Kansas. The regional epidemic of 1878 could have been an exception, because it was limited to northern areas; however, the epidemics resulting from documented aeciospore spreads were always much more local in nature. The extent of the eradication of the barberry by 1930 makes it obvious that the epidemics of 1935, 1937, 1953, and 1954 were not the direct result of aeciospore spreads. The epidemics from aeciospore spreads generally occurred annually but resulted in serious disease losses in an area of less than 2.9 km^2 downwind of the infected bushes. Epidemics resulting from aeciospore spreads were studied by Beeson (1923), Melhus *et al.*, (1920), Stakman *et al.* (1927), Durrell and Lungren (1927), and Walker (1927).

Early stem rust epidemics in Canada occurred in 1904, 1909, 1916, 1919, 1923, 1925, and 1927 (Bailey, 1928). The epidemic of 1916 was the most severe, causing a loss of 200 million dollars. Although the epidemics of 1904, 1909, 1919, 1925, and 1927 were less severe than in 1916, Canada still lost an average of 25 million dollars per year from 1918 through 1927. During the next 30 years, epidemics were less frequent, occurring in 1935, 1937, and 1938 when race 56 became established in North America. The last series of epidemics occurred in 1953, 1954, and 1955 with the establishment of race 15-B (Peturson, 1958). Craigie (1945) thought that the epidemics of 1925, 1927, 1930, 1935, 1937, and 1938 were not directly related to aeciospore spreads from barberry.

The difference in years that epidemics were reported in the adjacent areas of the United States and Canada can be related to differences in (1) crop maturity at disease onset, (2) host resistance used, and (3) environmental conditions. In addition, epidemic years were designated by differences from the normal amount of disease that occurred in the area.

According to Borlaug (1954), severe epidemics of stem rust occurred in Mexico in 1938 and 1939. Another occurred in central Mexico in

1948 (Rupert, 1951). In most years, however, epidemics were avoided in spite of many susceptible host cultivars and a year-round source of inoculum. Borlaug (1954) stated that the Mexican farmer had "learned to manipulate dates of planting and to use irrigation water properly to minimize the losses from rust." Late-planted wheat maturing in June or wheat planted during the summer was often seriously damaged by stem rust, but this practice was seldom used on a regional basis.

B. WHEAT LEAF RUST

The severity of the wheat stem rust problem in North America overshadowed the effects of wheat leaf rust caused by *Puccinia recondita* Rob. ex. Desm. f. sp. *tritici* (Chester, 1946) in the early part of the twentieth century. However, a major regional epidemic in 1938 throughout much of central United States on winter wheat emphasized its importance. Leaf rust primarily affects the leaf blades, whereas stem rust also attacks the stem; thus there is less time for a leaf rust epidemic development during the grain-filling period. Yield losses are usually less from leaf rust than from stem and stripe rust. Growers have often overlooked leaf rust losses because the disease is usually more severe in warm years with adequate rainfall conditions, which also favor wheat growth. Thus, even with heavy leaf rust infections, yields often can be higher than the long-term average. However, when leaf rust is controlled by chemicals or other means in these years, yields are much higher than the average. Chester (1946) stated that losses due to leaf rust were often underestimated. The severe losses caused by leaf rust have generally been in winter wheat areas where the disease overwintered. Few cases of severe regional leaf rust epidemics on spring wheat have been observed. However, losses can be significant. Losses of 1–5% commonly occur on spring wheat (Roelfs, 1978). Epidemics of statewide importance in Georgia occurred in 1935, 1937, and 1972; in Illinois in 1922, 1932, 1935, 1938, 1939, 1940, 1945, 1946, 1948, 1949, and 1950; in Indiana in 1927, 1931, 1932, 1935, 1937, and 1945; in Iowa in 1927 and 1938; in Kansas in 1938, 1949, 1957, and 1974; in Missouri in 1938, 1945, and 1957; in Nebraska in 1938; in Oklahoma in 1921, 1938, 1945, 1973, 1974, and 1975; in Pennsylvania in 1929, 1931, 1932, 1934, 1935, 1936, 1937, 1938, 1939, 1945, 1946, 1949, and 1950; in South Carolina in 1972, 1973, and 1974; in South Dakota in 1965; and in Texas in 1949 and 1974 (Roelfs, 1978). The leaf rust epidemics that occurred in 1974 in California, Kansas, Oklahoma, Texas, and South Carolina, developed from three separate sources of inoculum. The frequent epidemics in Illinois, Indiana, and Pennsylvania were curbed from 1950 to 1981 by the use of resistant cultivars; however, in 1982 leaf rust was again epidemic in the eastern United States. The epidemics that occurred in Georgia and South Carolina resulted in part from the cultivation of the very susceptible but high-yielding cultivar Blueboy.

Severe leaf rust epidemics have occurred infrequently on a regional basis where spring wheat is the principal crop. However, losses from 1 to 10% occur frequently enough that leaf rust resistance is an important objective in wheat breeding in Canada (Chapter 15). Current cultivars in the rust liable area possess effective multigene resistance (Chapter 2).

Leaf rust in Mexico is often an important disease; however, records of epidemics are not readily available. The crop is planted late enough in the fall to avoid heavy seedling infections in the fall, but planted early enough to mature before the rainy season, and losses are generally light. Failure to do this results in an epidemic such as occurred in 1977 (Dubin and Torres, 1981). Evidence shows little direct relationship of leaf rust epidemics in Mexico to those in the rest of North America.

C. WHEAT STRIPE RUST

In the United States and Canada, the same *forma specialis* of *Puccinia striiformis* West. attacks wheat and barley. Stripe rust of wheat is adapted to cooler temperatures than are the other two rusts in the United States. It has been a major disease of fall-planted wheat only and to nearby spring wheat but only in the area west of the Rocky Mountains where summer temperatures are cool, as in parts of Idaho, Oregon, Washington, and western Montana, as well as in California during the winter. Most epidemics of stripe rust have resulted from early fall infections that survived the winter and then spread in the early spring. Although not totally clear, evidence indicates the source of inoculum for initial infection is from nearby sources of volunteer wheat, wheat planted for a cover crop in orchards, or from wild grasses (Shaner and Powelson, 1973; Tollenaar and Houston, 1967; Burleigh and Hendrix, 1970). A series of stripe rust epidemics occurred between 1915 and 1935 and again from 1959 through 1961. Otherwise the disease was present in minor amounts until 1976 (Roelfs, 1978). Since 1976, stripe rust has been an important disease in this area.

Stripe rust has not caused serious epidemics in the major wheat-growing areas east of the Rocky Mountains. It was first reported in central Texas in 1941 and again in 1953 and 1956 (Futrell, 1957). Then in 1957, it occurred from Texas to South Dakota and Wyoming on the north. This epidemic was followed by a wider distribution in 1958, when it was found throughout the Great Plains. Since then, stripe rust has frequently been found in the south central areas of the United States with little loss except to individual growers in Texas. Stripe rust was again present from Texas to North Dakota in 1981, but in only trace amounts.

Stripe rust has been relatively unimportant in Canada, because most of the crop is planted in the spring. Stripe rust in Canada is most commonly found in Alberta (Sanford and Broadfoot, 1932), which is adjacent to western Montana.

Although stripe rust is common in Mexico and can become very severe on highly susceptible cultivars, there seems to be little information available on serious epidemics. It has caused losses locally in the high valleys, but no epidemic years were reported by Campos (1960).

D. OTHER RUSTS

Oat stem rust (caused by *P. graminis* Pers. f. sp. *avenae*), like wheat stem rust, has been the most severe in the north central states and adjacent Canada (Chapter 4). Fewer epidemics of oat stem rust have occurred than of wheat stem rust (Roelfs, 1978), which in part could be due to a much smaller area planted to oats (approximately 20% that of wheat) and because oat foliage is more frequently winterkilled to the crown than wheat in southern Oklahoma and north central Texas. Currently, the most common race, NA 27, is virulent on most of the cultivars. Occurrences of epidemics in the north central states are highly correlated with the date of disease onset (Roelfs and Long, 1980). Oats is a minor crop in Mexico; however, oats and wild oats (*Avena fatua* L.) are commonly rusted.

Crown rust (caused by *Puccinia coronata* Cda.) is the most serious rust disease of oats in North America (Chapter 5). Epidemics are common in the southern states, where the disease overwinters, and again on late seeded oats in Minnesota, the eastern Dakotas, and Manitoba (Roelfs, 1978). Crown rust is common in Mexico. In the southern states of the United States and Mexico, the loss of oat forage is probably as important as or more important than losses in grain production.

Stem rust (caused by *P. graminis* Pers.) epidemics on barley develop more slowly than on wheat for a number of reasons that vary in importance with year and location. Barley as a host has a shorter vegetative period, grows at lower temperatures, is more likely to winter kill to the crown, and is grown on smaller areas in Mexico and the southern states than is wheat. In addition, barley has a level of resistance to *P. graminis* both as seedling and as adult plant (Steffenson *et al.*, 1982a,b). Epidemics of stem rust on barley have occurred most frequently in Iowa, Minnesota, North Dakota, Illinois, South Dakota, and Wisconsin (Roelfs, 1978).

Leaf rust of barley (caused by *Puccinia hordei* Otth.) has been even less important than stem rust, mainly due to the small area planted to winter barley, the only known host on which the disease can overwinter, and because of the earliness of the crop, which limits the time for disease development (Roelfs, 1978).

The rusts of rye (caused by *P. graminis* Pers. f. sp. *secalis* and *P. recondita* Rob. ex Desm. f. sp. *secalis*) are not a major problem in North America. The crop is very winter-hardy, and stem and leaf rust diseases can survive the winter into southern Canada (Johnson and Green, 1952). Rye is cross-pollinated, resulting in some diversity for resistance, and rye is grown on small widely scattered areas. Stem rust has never caused major losses in the United States, and leaf rust has caused only local losses (Roelfs, 1978). Rye leaves normally senesce at an earlier growth stage than in other cereals; therefore, leaf rust must be severe early in the season to have a major effect on yield. Most of the inoculum is endogenous to the area where the disease develops.

Rusts of corn have been minor diseases in the United States, regardless of the millions of hectares of corn grown (Chapter 7). This may be partly due to host resistance, but also to the lack of an overwintering host (Kingsland, 1975) such as occurs with the winter planted cereals. When present the disease has been the most common on late-planted fields (Wallin, 1951; Futrell, 1975) and on sweet corn.

Rust of sugarcane is relatively new to North America and is discussed in Chapter 8 with rusts of sugarcane.

II. Wheat Production and Rust Epidemics

The cereal rust epidemiology studies in North America have mainly involved the wheat rusts. Those at least nationwide in scope were of wheat stem rust in Canada (Craigie, 1945; Peturson, 1958), Mexico (Stakman *et al.*, 1940; Borlaug, 1954), and the United States (Stakman, 1934; Stakman and Harrar, 1957). Epidemics of leaf rust of wheat in the

United States were reviewed by Chester (1946). Stripe rust epidemics were studied in California (Tollenaar and Houston, 1967), Oregon (Shaner and Powelson, 1973), Washington (Burleigh and Hendrix, 1970), Montana (Sharp and Hehn, 1963), and Canada (Sanford and Broadfoot, 1932). A review article by Rajaram and Campos (1974) reviewed the epidemiology of the three rusts in the Western Hemisphere. Due to the abundance of information on the epidemiology of the wheat rusts, the rest of this chapter will be concentrated on them, but will draw on information from epidemiological studies of the other cereal rust diseases when it is advantageous to do so. Additional information on epidemiology of the other diseases will be found in the specific chapters on that disease in this volume.

Wheat is the principal cultivated crop in much of North America, especially in the drier areas (Fig. 1). The approximately 40 million hectares of wheat grown annually in North America is comprised of about 400 different cultivars; however, only about 20 of these are grown on over 400 thousand hectares each (Table I).

A. WHEAT AND RUST IN MEXICO

Wheat is intensively cultivated in isolated areas across Mexico where irrigation water is available. Wheat is generally seeded in November and early December; however, volunteer plants can exist in the summer crops. The period November through April is generally the dry season, which creates the need for irrigation. Wheat is generally harvested in April and May and is followed by a summer crop of corn. Rust is usually light through the growing season, but leaf and stem rust can become severe near maturity with the onset of the summer rains. The major wheat-producing areas are the El Bajio, mainly in the state of Guanajuato (Gj) (Fig. 1), and the Pacific coastal plain, mostly in the states of Sonora (So) and Sinaloa (Si). The cultivars grown are selections made and released by INIA (Instituto Nacional de Investigaciones Agricolas) from the wheat program of Centro Internacional de Mejoramiento de Maíz y Trigo (CIMMYT). Stripe, leaf, and stem rusts occur but the cultivars are generally resistant and epidemics have been few in the past 30 years. The most recent leaf epidemic in the state of Sonora resulted in part from a virulent race oversummering on volunteer wheat plants and then moving to early-planted wheat (Dubin and Torres, 1981). The data are meager, but it appears that the rusts normally survive throughout the year in the intermountain highlands and, with favorable conditions, can move northeast into the southern

Fig. 1. Distribution of wheat in North America. Values are in 1000 hectares using the period 1978–1981 as a guide. Figures for Mexico were obtained from various officials of Instituto Nacional Investigaciones Agricolas, for the United States from Briggle *et al.* (1982), and for Canada from the Canadian Cooperative Wheat Producers.

Table I

The Principal Wheat Cultivars in North America and Their Rust Resistance as Known[a]

Cultivar	Hectares	*Sr* gene(s)	*Lr* gene(s)	Market Class
Neepawa	6,884,160	*5,+*	*13*	HRS[b]
Centurk	1,813,296	*5,6,8,9a,17*	—[c]	HRW
Tam W101	1,527,973	—	—	HRW
Scout	1,414,769	*2,17*	—	HRW
Olaf	1,195,444	*8,9b,12,Wld-1*	*2a,10,+*	HRS
Eagle	1,133,600	*2*	—	HRW
Scout 66	993,941	*2,17*	—	HRW
Arthur 71	916,561	*5,36*	*9*	SRW
Sage	890,808	*17,24*	*24*	HRW
Butte	745,760	*6,8,9g,Wld-1*	*10*	HRS
Era	731,075	*5,6,12,17*	*10,13,+*	HRS
Arthur	724,075	*5,36*	—	SRW
Wakooma	704,160	?	?	Dur[b]
Sinton	663,880	?	*10,13,+*	HRS[b]
Waldron	657,446	*5,11,Wld-1,+*[d]	*2a,10,+*	HRS
Bento	655,920	?	*1,2a,12,13*	HRS[b]
Ward	605,619	*9e,+*	?	Dur
Abe	546,144	*5,36*	*9*	SRS
Triumph	529,664	*Tmp*	—	HRW
Larned	418,812	—	—	HRW
Triumph 64	418,004	*Tmp*	—	HRW
New cultivars with high potential use				
Newton	1,997,460[e]	—	*1,11,+*[f]	HRW
Tam W105		—	—	HRW
Columbus		?	*13,16*	HRS

[a] Data are 1982 for Canada and 1979 for the United States; no cultivar from Mexico is grown on 400,000 hectares.

[b] Canadian cultivars.

[c] Normally susceptible in the field.

[d] Segregates for *Sr5* and *Sr11*.

[e] 1980.

[f] Segregates for *Lr11*.

United States and northwestward into the major production areas in Sinaloa and Sonora. Barley production in Mexico is limited now, although earlier it was an important source of stem rust inoculum (Borlaug, 1954). Wheat leaf rust appears to survive throughout the year in at least trace amounts in all but the production areas of the northwest. There seems to be relatively little direct movement of rust from

Mexico into the main production areas of the United States in most years. The pathogen races in Mexico and south Texas are similar, but generally are different from those in the central and north central areas of the United States (Roelfs *et al.*, 1982). Although early evidence indicated an annual interchange of inoculum from north to south across North America in the fall (Stakman and Harrar, 1957), since 1970 with less rust in the United States and Canada this has not been true. Race 15 has predominated in areas north of Mexico but has not been found in Mexico. Leaf rust virulence for either *Lr9* or *Lr24* is common in south Texas and the very small wheat-producing area along the Texas border in Mexico, but neither of these virulences has been found in the central or northwest areas of Mexico. With wheat rusts oversummering at least as far south as Kansas in the summer (Burleigh *et al.*, 1969b), inoculum from the far north probably causes few if any infections in the southern United States and Mexico.

B. WHEAT AND RUST IN THE UNITED STATES

Wheat is grown in 42 of the states in the United States. Approximately 80% of the wheat is planted in the fall. The remaining 20% is planted in the spring to bread and durum wheats (Table I). Fall seeding starts in late August on the Canadian border and ends in mid December along the coastal area of the Gulf of Mexico, and southern Texas. Spring planting starts in April as soon as the ground can be worked and continues into early May. Harvesting of the crop starts in the south in May and continues northward until it reaches the Canadian border in early to mid August. About 370 different cultivars of wheat are grown annually (Briggle *et al.*, 1982), but generally a few cultivars make up most of the area. Because of the differences in grain characteristics of the five major classes of wheat, diversity is maintained on a national, if not a regional, basis (Table I). Cultivars are developed by both private and public breeding programs. The 17 most popular cultivars grown in the United States in 1979, were developed in eight different breeding programs. In the higher rainfall area (over 900 mm) in the eastern third of the United States, soft red winter wheat (SRW) is grown. In the southern two-thirds of the Great Plains (Texas, Oklahoma, Kansas, Nebraska, Colorado), hard red winter wheat (HRW) is grown with a narrow band extending through western South Dakota, southern North Dakota, into north central Montana. The hard red spring (HRS) and durum (Dur) wheats are grown in the Dakotas, Minnesota, and northeastern Montana. In the Pacific northwest, Michigan, and New York, white winter wheats (WW) are the most important. Most of the wheat in the United States (90%) is grown under dry land conditions, except in the drier western portions of Kansas, Oklahoma, Texas, and the eastern portions of Colorado and Wyoming, where a portion of the crop is irrigated. There are also important areas of irrigation in California and Washington (Reitz, 1976). There are many areas where planting practices vary from this outline, but they are small and too numerous to be considered on a continental basis.

Leaf rust is a major disease of wheat throughout most of the United States, with the greatest losses historically occurring along a band from Pennsylvania through Kansas and Oklahoma (Roelfs, 1978). Leaf rust can overwinter on wheat wherever the fall-infected leaves survive through the winter. Although it probably is rare, leaf rust has overwintered on wheat as far north as southern Wisconsin (Christman, 1904). Rarely does overwintering occur north of 41°N latitude but it usually survives as far north as 40°N latitude. Leaf rust can also overwinter throughout most of the Pacific northwest.

Stem rust is a major disease of spring and durum wheats in the north central states and extends southward into Nebraska (Roelfs, 1978). It originally survived the winter in the northern states as teliospores, and then by subsequent infection of the barberry in the spring it continued its cycle. However, an extensive barberry eradication program had eliminated most of the barberry growing near grain fields by the mid 1920s (Stakman and Harrar, 1957). Currently, inoculum for this area is principally from overwintering rust south of 34°N (Stakman, 1923), but rust can overwinter farther north (Paxton, 1921). In the southern half of the country, stem rust seldom causes any loss (Roelfs, 1978), because the winter wheat heads soon after the last freeze and leaves the pathogen little time to increase to epidemic levels. However, adequate inoculum is produced so that it can be blown northward in sufficient amounts to initiate epidemics. The date of the first detected infection in the northern areas is closely related to yield loss, with greater losses occurring in years when the disease arrives earlier (Hamilton and Stakman, 1967; Roelfs and Long, 1980) than usual.

Stripe rust has been the major rust of wheat west of the Rocky Mountains. Epidemics have occurred in cycles (Section I,C). These cycles may well be related to variation in temperatures between years (Coakley and Line, 1981a,b). Stripe rust also occurs in the central third of the United States. The inoculum may be from the highlands of central Mexico (Guanajuato, Gj; Fig. 1), arriving in December and January and infecting susceptible wheat in Texas, Louisiana, and Arkansas. No study of the epidemiology of this population has been attempted.

C. WHEAT AND RUST IN CANADA

Wheat is an important crop in Manitoba, Saskatchewan, and Alberta. Nearly two-thirds of the area is of a single cultivar, and due to quality standards, most cultivars are rather closely related. The major wheat crop is hard red spring (87%), with the remainder mostly durum wheat along with small amounts of winter wheat in Alberta, Ontario, and the Maritime provinces. Little if any rust overwinters on wheat in Canada, except possibly for stripe rust in Alberta or leaf rust in Ontario. Most of the inoculum comes from the United States.

III. Sources of Inoculum

Although the hosts are different, most of the cereal rust fungi have two basic sources of inoculum for the cereal host, urediospores and aeciospores. The exceptions are stripe rust, for which there is no known alternate host, and sugarcane rust, which is autoecious. However, the principal source of inoculum for all of the cereal rusts in North America currently is from urediospores.

In epidemiological studies, inoculum is often spoken of as exogenous, from outside the area under consideration, and as endogenous, from inside the area under consideration. These terms are not always clearly distinguishable, because what is an endogenous source of inoculum for field A can also be an exogenous inoculum source for a nearby field B. Thus, when it is said that the state of Texas serves as a source of exogenous inoculum for the northern states, it must be understood that some fields in Texas probably also have exogenous sources. Once the initial infection occurs in any field or on any plant, generally the endogenous inoculum produced there is the primary source of inoculum for further disease development.

The initial source of annual infection of wheat in North America is almost exclusively by urediospores. These spores may have been produced on volunteer plants nearby or on the wheat crop to the south. Other uredial hosts probably have a minor role, except perhaps *Hordeum jubatum* L. (for wheat and rye stem rust) in the north central states. However, *Avena fatua* L. may play an important role in oat stem and crown rust epidemics in some areas. Although urediospores can, if unweathered, survive on crop debris (Orr and Tippetts, 1971) for a short period, they probably have little potential for infecting the next crop.

A. AECIOSPORES

Aeciospores are released singly or in clumps, and only in approximately 100% humidity. However, the numbers of spores produced are very large. An average-sized barberry bush in southern Minnesota was the source of an estimated 64 billion spores in a few days (Stakman *et al.*, 1927).

The alternate hosts in North America were an important source of inoculum for *P. graminis* and *P. coronata.* Barberry, *Berberis vulgaris* L., once was a major inoculum source of wheat, oat, and rye stem rust in the north central states of the United States and Canada, and can still be a source of early inoculum for oat stem rust from Pennsylvania through southern Ontario, and of wheat stem rust in eastern Washington and northern Idaho. Most of the aeciospores currently isolated in the eastern United States are of *P. graminis* f. sp. *secalis,* which spreads to noncereal grasses. The barberry eradication programs of the United States and Canadian governments and their subdivisions have removed nearly all barberry from close proximity to small grain cereal fields. However, a few barberry bushes remain in most of the north central states of the United States and eastern Canada.

The importance of aeciospores as inoculum was in their genetic diversity (Roelfs and Groth, 1980; Groth and Roelfs, 1982). Aeciospores also served as a source of inoculum early in the season (Roelfs, 1982). Epidemics that were initiated by aeciospore infections were generally local in effect (Stakman *et al.*, 1927). Most aeciospores travel less than 100 m; however, a few spores can travel much longer distances. Lambert (1929) found effective spreads were limited to 3.5 km. Urediospores resulting from the initial aeciospore infection generally formed a fan-shaped infected area with a length of up to 15–30 km and a width of one-fourth the length. Hutton (1927) indicates a spread of 160 km was known. Aeciospore-initiated epidemics were usually north of the 40° parallel (Stakman, 1923). Viability of teliospores were generally reduced when they were produced in hot dry conditions further south. However, aeciospores were observed farther south at high elevations (2000 m) (Stakman, 1923), and epidemics resulting from aeciospores were documented in Colorado by Durrell and Lungren (1927) south of the 40° parallel. The Colorado locations varied from 1200 to 1500 m elevation compared with 300 m in eastern Nebraska at 40° at the southern edge of barberry-initiated epidemics. Local epidemics that were the result of initial inoculum from barberry were documented in Illinois and Indiana (Beeson, 1923), Iowa (Melhus *et al.*, 1920), Minnesota (Stakman *et al.*, 1927), North Dakota, South Dakota, and Wisconsin (Walker, 1927).

Rhamus cathartica L. was associated with epidemics of crown rust in the northern United States and eastern Canada. Inoculum from *Rhamus* has never resulted in as serious regional losses as has the inoculum from barberry. This difference is due in part to the oat crop having lesser value, the smaller area of oats grown in a region, and because crown rust attacks only the leaves and leaf sheaths. Thus, there is less tissue available that senesces earlier than true stem spike tissue, which also serves as a host for the stem rusts. Current evidence, including race stability, pathogen uniformity, and few local early epidemics, would indicate that the sexual cycle is essentially a local problem occurring principally in limited areas of Iowa, Minnesota, Wisconsin, and Ontario.

B. UREDIOSPORES

Urediospores are the principal source of inoculum of all the cereal rusts in the United States. A single uredium can produce over 1000 urediospores per day for a period of several weeks (Section VI). These spores are relatively long-lived (Bromfield, 1967; Peltier, 1925; Reed and Holmes, 1913; Thiel, 1938; Johnson and Green, 1952; Mohamed, 1960), survive in the field away from the host for periods of several weeks (Stakman *et al.*, 1923; Orr and Tippetts, 1971), and can be carried long distances (Christensen, 1942) before they are generally scrubbed from the air by rain (Rowell and Romig, 1966).

IV. Exogenous Inoculum

Exogenous urediospores seldom, if ever, arrive in such amounts as to directly cause an epidemic, but increase in inoculum from the resulting infections often can initiate an epidemic. Exogenous inoculum is mostly ineffective. The loss in numbers and viability of urediospores during transport is tremendous. Even upon deposition, most viable urediospores are deposited on nonvegetative material, nonhost plants, or resistant cultivars, or under environmental conditions that are unfavorable for germination, penetration, and establishment.

A. SPORE TRAPPING

Exogenous inoculum can be detected by spore trapping or by direct observation of disease occurrence. Spore trapping has been done in North America for over 60 years. Initially, spores were trapped by impaction on microscope slides that had been thinly coated with petroleum jelly. Although large numbers of spores are trapped, impaction traps are very sensitive to local inoculum (Roelfs *et al.*, 1968). Volumetric traps have also been used in North America, but on a more limited scale. Although spores are trapped, correlations are poor with initial arrival of inoculum (exogenous). This is probably due to several factors: trapping of nonviable urediospores, viable spores but conditions unfavorable for infection, failure to properly distinguish between urediospores of closely related species or *formae speciales,* resistance of host plants, and an inability to sample enough air volume. The most effective exogenous inoculum is deposited during rain storms, when volumetric samplers currently used are rather ineffective. Thus, in the United States, rain samples are the best device for the detection of exogenous inoculum (Rowell and Romig, 1966; Roelfs *et al.*, 1970). To be most effective, the inoculum source must be far enough away so that the spore clouds are relatively randomly dispersed. Urediospores of a similar morphology cannot already be present in the area of the trap, and the rainfall pattern needs to be such that sampler locations can be maintained with assurance that at least one will be in the area of each storm.

B. MONITORING DISEASE SPREAD

Because of the problem of recognizing urediospore morphological differences between some species, *formae speciales,* and races, it is necessary to do field surveys of the diseases to detect inoculum movement. These surveys have been conducted over most of North America for over 60 years.

The following techniques have been developed by the Cereal Rust Laboratory, partly because of cost and partly because they are effective in detecting rust, especially when it is present in trace amounts. Survey routes are chosen that cross the major small-grain cereal-producing areas when the majority of the crop is from late boot to early dough growth stages. The routes chosen are normally all-weather roads, and surveys are made on approximately the same date annually, adjusting only plus or minus 7 days for annual differences in the crop growth stage. Once in the area, the surveyor stops at the first small-grain cereal field after the odometer reading is divisible evenly by 10. Subsequently the surveyor stops every 20 miles (32 km) or at the first field after that distance. The surveyor normally spends approximately 15 min in each field or plot (or at a stop in case of several adjoining fields). Notes are taken on environmental conditions, host condition, host maturity, cultivar grown, rust severity and prevalence, loss potential, and other diseases. Collections of urediospores are made for physiological race determinations. If rust is common, the surveyor examines a strip approximately 1 m wide along a loop extending 100 m into the field and reports an average, except when the loop is obviously atypical to the field. When rust is scarce, the surveyor uses previous experience to determine the area of the field that is most likely to be rusted. However, data recorded is for the field mean. Experimental, demonstration, and trap plots are examined to ensure that the resistance of the commercial cultivars are not excluding rust development. For example, in the hard red spring wheat areas of the Northern Great Plains, in recent years stem rust is seldom found in commercial fields; however, susceptible cultivars in experimental plots are often severely rusted. Thus, the surveyor rapidly determines the potential for rust in an area and the virulence for the commonly occurring cultivars in the experimental plots. If no rust is found on susceptible cultivars, no extra effort is required in the commercial fields; however, if susceptible cultivars in research or demonstration plots are rusted, the surveyor usually makes a special effort to check in nearby fields or check additional fields to verify the extent of the disease occurrence. When fields are examined more frequently than every 32 km, the additional stops are recorded as special stops. Occasionally a change of route will be necessitated by local conditions affecting the host, disease, or road. This can only be done when the surveyor is well versed in both agriculture and roads in the area.

C. CHARACTERISTICS OF SPREAD FROM EXOGENOUS INOCULUM

Disease resulting from exogenous inoculum characteristically has the oldest infections at a standard plant height (Roelfs and Rowell, 1973). The initial infections from exogenous inoculum frequently are at the top of the canopy when the inoculum arrives. The secondary infections tend to be at the same height or just above or below older infections. The oldest infections are generally distributed at random through the area (Rowell and Roelfs, 1971), except when the environment is so marginal for rust infections that it occurs only in certain areas of the field or in certain fields. However, the distribution over a larger area will still be random. These infections are often still high in the canopy when sporulation occurs, which results in the spores spreading very rapidly horizontally, and often vertically, up from the field.

V. Endogenous Inoculum

In a regional approach to epidemiology, the term endogenous inoculum is normally used to refer to inoculum produced within a given field or local area, although in experimental epidemiology the term may be used for very much smaller units. Endogenous inoculum is the immediate cause of nearly all cereal rust epidemics, even though the initial infections may have been caused by exogenous inoculum.

A. SPORE TRAPPING

Spore traps can be used as an estimate of the disease severity. Our experience indicates that impaction-type traps are the most effective, and one may choose from the volumetric type (rotobar) (Asai, 1960) or wind-impaction types such as a rod or slide (Roelfs, 1970; Bromfield *et al.*, 1959). Volumetric samplers can be used, but they may be more likely to be influenced by exogenous inoculum than are impaction traps. Although it is not always possible or even desirable to do so, spores usually should be collected over areas with a radius of 10 m (Roelfs, 1972) if spore concentrations are to be representative of what happens in large fields. Traps exposed outside of the crop are affected by distance from the source and wind direction (Roelfs, 1972), causing erratic data and low spore numbers (Roelfs *et al.*, 1972; Roelfs, 1972). Neither sedimentation nor rain samples are recommended for studying endogenous inoculum. Spore trapping can be a very valuable tool in studying epidemiology in plots. However, until an electronic counter is developed that rapidly distinguishes particles by color, shape, and size, it is currently too expensive to adequately study epidemics over millions of hectares by spore trapping.

B. MONITORING DISEASE SPREAD

The frequency or intensity of disease caused by endogenous inoculum decreases as distance from the source increases. The best-documented natural spreads of this type in the United States are those of *P. graminis* from infected barberry bushes (Section III,A) and from urediospore spreads by Celik (1974), Elliot (1960), Emge and Shrum (1976), Kingsolver (1980), and Underwood *et al.* (1959). Spread from endogenous inoculum is found around volunteer plants in newly seeded winter wheats and when the disease spreads from a winter to a nearby spring wheat, and most commonly in the spring from an overwintering focus. The spread usually is fan-shaped with its origin at the upwind source. This spread pattern can be modified by areas of nonhost plants or local environmental conditions or both. The downwind spread follows the direction the wind is blowing at the time most of the spores are released and when conditions were favorable for infection after their dispersal. Thus, downwind may not be the mean direction of air flow during any specific time period.

Disease resulting from endogenous inoculum sources generally occurs relatively early in the season, often within a few weeks of emergence, or after a period of very unfavorable environmental conditions for the disease, such as winter for a fall-planted crop. Generally the host is growing rapidly so that infection at the top of the canopy may be under the canopy when sporulation occurs. Thus, many of the spores are trapped within the canopy, which results in a heavy infection low on the plant that can be overlooked by inexperienced observers. These foci of severely rusted plants grow gradually in size but seldom reach more than a meter in diameter before reaching the top of the canopy. Once the disease reaches the canopy top, it rapidly spreads horizontally across the field. These foci may have severities of 10–100%, and the ground beneath them may take on the color of urediospores before the disease has spread horizontally more than a meter. Because these foci generally result from a single infection that survives the unfavorable environmental conditions of winter, they seldom occur in a random manner, but occur in specific sites, e.g., sheltered, most fertile, or wetter parts of the field.

C. CHARACTERISTICS OF SPREAD FROM ENDOGENOUS INOCULUM

The most noticeable difference between infections from endogenous and exogenous inoculum is that the oldest infections from endogenous sources are generally low in the canopy. Foci are common, and horizontal spread is limited until the disease reaches the top of the canopy. Foci are nonrandom in distribution, and different foci are often the result of a different pathogen race. Foci are likely to occur in some predictable even though erratic pattern. For example, they may occur near old volunteer plants, on plants protected by snow cover along a tree row (Eversmeyer and Skidmore, 1974) or snow fence, etc. Normally on a single cultivar the most severe rust infection occurs in the earliest plantings.

VI. Urediospore Movement

Urediospores of the cereal rusts are passively released, transported by air currents, and deposited under the forces of gravity, impaction, and rain. Urediospores vary in size and shape depending on the fungus, but the size usually ranges from about 20 to 30 μm (Savile, Vol. 1). Urediospores have a terminal velocity of 0.97 to 1.24 cm/sec (Ukkelberg, 1933). Thus, in still air, a spore would fall 0.67 m/min, 40 m/hr, or approximately 0.9 km/day. At this rate of descent a spore at 1500 m (about half the normal maximum height of heavy spore concentrations) in a 13.3 m/sec (30 mph) wind would travel 1760 km before landing. Of course, with this wind speed the air is composed of many eddies, updrafts, etc., which tend to extend the time a spore is airborne. Urediospores generally move in greatest numbers when the plants are rapidly growing (warm to hot temperatures), foliage remains dry (low relative humidity), and near mid-day (usually the windiest and driest period of the day).

The shape of a urediospore probably is important to its response to transport and deposition, but much more information is required about this relationship. In studying different impaction traps under field conditions, Roelfs *et al.* (1968) found that urediospores of leaf and stem rust of wheat vary in their frequency of impaction on both vertically exposed microscope slides (2.54 × 7.62 cm) and 5-mm-diameter rods. On the rods, the urediospores of leaf rust tended to be on the mid arc facing the wind, while the majority of stem rust urediospores were more on the sides. These differences in impaction may relate to differences in the size and shape of urediospores.

Large numbers of urediospores are produced from a uredium, and this production continues for several weeks, resulting in the production of 100,000 urediospores for wheat stem rust (Katsuya and Green, 1967; Stakman, 1934) and over 28,000 for wheat leaf rust (Chester, 1946). The urediospores are released on a diurnal cycle peaking near mid-day (Asai, 1960). Most are trapped by the canopy and are deposited by sedimentation or impaction. Sedimentation is deposition due to the force of gravity while impaction results from urediospores being driven onto plant tissue by air movement within the canopy. Thus, of the millions of urediospores produced per acre, only a small fraction escape the canopy.

A. TRANSPORTATION

As the urediospore escapes the canopy, it is influenced by eddies of many types. Again, most of the urediospores are probably deposited within a 100 m of the source (Roelfs, 1972). Only 10% of the urediospores in a horizontal plane above the canopy are in the same plane 100 m downwind. However, because of the large numbers of spores produced, significant numbers of urediospores are transported vertically. These urediospores transported vertically generally decrease in number with elevation but are relatively numerous up to 3300 m above rusted fields (Stakman *et al.*, 1923). Claims of movements of viable urediospores have been reported for distances of over 8000 km. However, some of the distances are questionable because ground surveys were inadequate to establish that there were no spores produced between the stated source and the point of investigation. This is important because even 0.4 hectare of wheat with less than a 10% severity can produce a trillion urediospores (1.02×10^{12}) (Rowell and Roelfs, 1971). Furthermore, urediospores trapped of the various rusts are often difficult or impossible to distinguish microscopically with certainty on an individual spore basis. From most traps, it has also been impossible to determine if the urediospore was alive when it was trapped. Perhaps some of the new trapping methods will solve this last problem (Schwarzbach, 1979). The longest documented single movement by urediospores of *P. graminis* in North America was at least 680 km. Infections of wheat stem rust were found on volunteer wheat plants at two locations at Churchill, Manitoba, which is separated from the nearest wheat-producing area near Winnipeg by forest, lakes, and tundra (Newton, 1938). The world record may well be the transport of spores from Africa to Australia (see Chapter 10). However, Stakman (1934) found that few spores were in the air more than 400 km from a source area.

Vertical distribution of urediospores of wheat stem and wheat leaf rust was studied 1–6 m above the canopy by Eversmeyer *et al.* (1973). They found a decreasing number of urediospores of *P. recondita* as height increased, but an increasing number of *P. graminis.* This difference was attributed to differences in amounts of exogenous inoculum. Additional studies are needed on the vertical spore concentrations over isolated infected sources.

B. DEPOSITION

Urediospore deposition is caused by three different mechanisms; (1) sedimentation, (2) impaction, and (3) rain scrubbing. Transportation of spores within the crop canopy usually ends by sedimentation onto leaves, stems, or the ground by the force of gravity. The longer the spore remains airborne within the canopy, the more likely it is to be impacted by small eddies on a nearby stem or leaf. Those urediospores that escape the canopy by moving out vertically are carried by stronger wind eddies, updrafts, and currents. Although most are still deposited in the first 100 m from the source (Roelfs, 1972), the farther a spore travels, the less likely it is to be deposited by sedimentation or impaction. Therefore, the importance of deposition by rain scrubbing (Rowell and Romig, 1966) increases. Samples of rain water analyzed for urediospore content were successfully used to predict disease onset in the north central area of the United States (Roelfs *et al.*, 1970).

VII. Factors Affecting Epidemic Development

Epidemics of the cereal rusts are affected by four independent yet related factors: host, pathogen, environment, and time. The relationship between these factors and the disease is such that an independent discussion of each is not realistic, but is so complex that understanding and studying all the interactions together is difficult.

A. HOST RESISTANCE—PATHOGEN VIRULENCE

As pointed out in Chapters 5 and 6 in Volume 1 of this series, a host is susceptible if it does not possess a gene for resistance, or if the pathogen culture has virulence matching the host gene(s) for resistance. Besides this race-specific resistance, most cultivars have additional non-race-specific resistance [at least not known to be race-specific (Chapter 16)]. Both types of resistance exist in nearly all cultivars (Table I) grown in areas of North America where a rust disease has been historically important. In 1972, Blueboy, a soft red winter wheat cultivar that had the ineffective *Lr1* and *Lr10* resistance, was grown in Georgia, and losses of 50% were incurred (Roelfs, 1978), even though leaf rust does not usually cause serious losses in wheat in Georgia. In 1974, the soft red winter wheat cultivar McNair 701 was introduced in Florida, Georgia, and South Carolina, where stem rust seldom occurs; however, the pathogen rapidly increased and severe losses were incurred (Roelfs, 1978). In both of these cases, other susceptible cultivars (no detectable race-specific resistance to the pathogen genotypes present) suffered much less loss. Thus, in addition to knowing the virulence of the pathogen population (Vol. 1, Chapter 5) and resistance of the host, it becomes necessary to study nonspecific type of resistance of the host.

Additionally, in asexual-reproducing organisms like most of the cereal rusts, important differences may exist between cultures for characteristics other than virulence. Aggressiveness measured as reproduction potential was studied by Katsuya and Green (1967). Many other uncharacterized differences exist between cultures. However, if these differences are polygenic in nature, they may remain together long enough in asexually reproducing organisms to be important for detailed study.

In oat stem rust, the most common race is NA-27, which is virulent on the *Pg-2* and *Pg-4,* which are the only resistance genes widely used. In commercial oat fields, scattered plants of wild oats (*Avena fatua* L.) exist that have no known resistance. Races Na-27 and NA-16 both occur on the wild oats, with the frequency of NA-16 greater than on commercial oat cultivars, although the numbers of urediospores of race NA-27 deposited on the wild oats must be 15–1000 times greater than that of NA-16. The greater frequency of NA-16 on wild oats apparently indicates more aggressiveness in this race than in race NA-27 on wild oats.

B. DATE OF DISEASE ONSET

Hamilton and Stakman (1967) found that epidemics of wheat stem rusts in the central third of the United States were related to the date of rust appearance. Roelfs and Long (1980) found in the north central part of the United States that over a 38-year period the date of disease onset explained about 20–40% of the estimated yield losses of oats from stem rust. Date of onset explained a greater portion of the variation than any other factor studied, including temperature and rainfall. The most prevalent race NA-27 is virulent on nearly all the cultivars grown in North America; thus resistance–virulence was not a factor (Roelfs and Long, 1980). The pathogen in this case was windborne from a southerly location, so the relationship between date of disease onset and losses in the north should be expected. The relationship between disease onset and loss could be improved with better data. Disease severity on date of disease onset was not considered and it varied from a single uredium (less than 0.001 severity) to 20% (200 uredia per tiller). Another variable not considered in this study was the host growth stage at time of disease onset. An early infection generally results in a greater disease loss, all other factors being the same. In the north central area studies, the planting data within and between years may vary as much as 21 days. In this study, the yield loss was estimated for the entire state. Had actual yield losses been available for the fields in which the rust was reported, the date of disease onset in oat stem rust might have explained most of the variation in losses due to oat stem rust.

C. INITIAL DISEASE SEVERITY

In areas where the disease overwinters, the "critical month" principle (Chester, 1943) is useful in predicting losses. Essentially this theory is based on the premise that if rust is present before winter, some disease will survive the winter. However, in the spring when the host can grow at temperatures too cool for disease increase, or when the old leaves die before the rust spreads from them to the newer leaves, a critical period occurs for the survival of the disease. At the end of this critical period, Chester's critical month, the increase of disease is very constant between years, because environmental conditions are similar and the disease at the end of the critical period is closely correlated with final severity. Although the critical month theory appears to be crude and is quite simple, it has worked relatively well over the years. The environmental conditions that favor wheat during its reproductive process also are favorable for the obligate parasite of wheat. The environmental conditions after the "critical" month are those in the broad area of the fungal growth curve, one standard deviation either side of the optimum for the pathogen when a change of several degrees in temperature has little effect on the pathogen. During and before the critical month (just before the boot stage of the host in Oklahoma),

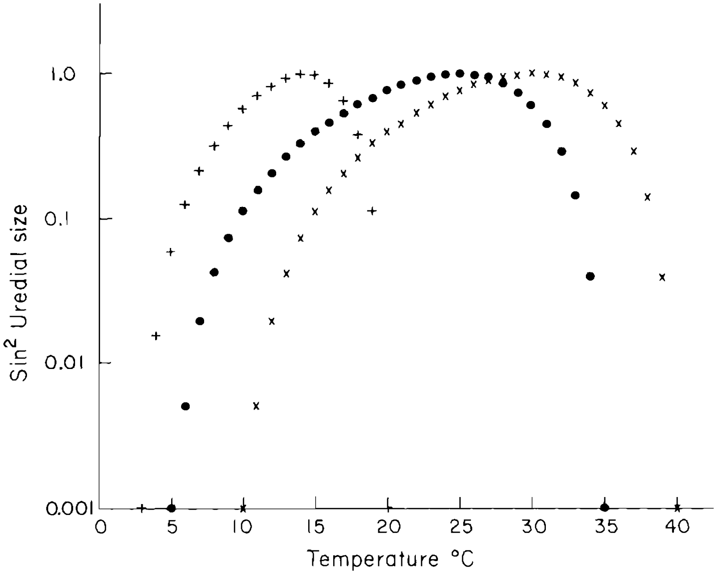

Fig. 2. The effect of temperature on fungal growth for stripe +, leaf ●, and stem X diseases of wheat using Schrodter $\sin^2$ transformation, as modified by Dirks and Romig (1970).

conditions can be in the tail of the growth curve, which is near the minimum for pathogen growth, where a single degree difference in temperature has a major effect on fungal growth (Fig. 2).

The frequency of initial infection and/or the frequency of infection on a given date are also useful for predicting losses due to disease. The common measurement of disease severity is based on the percentage of the maximum possible infection present. The scale most used worldwide is the modified Cobb Scale (Peterson *et al.*, 1948). Another sacle was designed by James (1971) and is based on percent of the leaf tissue diseased (percentage of the green tissue occupied). Either scale is satisfactory, but the scale used should be indicated. Prevalence is used by most cereal rust workers to indicate the percentage of tillers infected (plants are usually not possible to identify without pulling them). However, in studying the entire epidemic it may be desirable to use a single value for disease severity and prevalence. This value can be calculated determining the number of ureida per tiller that equal 1%. Kingsolver *et al.* (1959) found for stem rust that 10 uredia per tiller equals 1% severity on the modified Cobb Scale. Thus, 1 uredium/tiller = 0.1%, 1 uredium/10 tillers = 0.01%, 1 uredium/100 tillers = 0.001%, etc. For wheat leaf rust, Burleigh *et al.* (1969a) found that 18 uredia/tiller equals 1% and thus 1.8 uredia/tiller = 0.1%, 1.8 uredia/10 tillers = 0.01%, etc. The number of crown rust and rye leaf rust uredia per 1% would be similar to wheat leaf rust and for barley leaf rust the number would be larger. Disease severity is correlated with numbers of spores impacted just above the canopy (Burleigh *et al.*, 1969a), which can also be used to estimate disease losses.

D. ENVIRONMENTAL FACTORS

Many workers have studied the effect of environmental factors on epidemics in North America (Table II). It would appear that nearly every possible combination of temperature—maximum, minimum, winter, summer, $\sin^2$ transformed, weekly means, monthly means, annual means, degree days, etc.; precipitation—days of rain, hours of

Table II

Major Studies of the Effect of Environmental Factors on Cereal Rust Epidemics in North America

Disease	Factor(s) studied	Reference
Wheat stripe rust	Temperature, dew	Burleigh and Hendrix, 1970
	Temperature, precipitation	Line, 1976
	Temperature, precipitation	Coakley, 1979
	Temperature	Coakley and Line, 1981b
	Temperature, precipitation, degree days	Coakley and Line, 1981a
Wheat stem rust	Temperature	Walster, 1921
	Temperature, precipitation	Tehon and Young, 1924
	Temperature	Stakman and Lambert, 1928
	Temperature, wind, precipitation, rainy days	Lambert, 1929
	Temperature, precipitation, rainy days	Peltier, 1933
	Temperature, humidity, precipitation	Atkins, 1936
	Temperature, hours of sun, wind, precipitation	Craigie, 1945
	Temperature, rain	Wallin, 1964a,b
	Temperature	Katsuya and Green, 1967
	Dew	Prabhu and Wallin, 1970
	Temperature, precipitation, dew	Burleigh *et al.*, 1972
Oat stem rust	Temperature, precipitation	Roelfs and Long, 1980
Wheat leaf rust	Temperature, precipitation	Tehon, 1927
	Temperature, precipitation, dew	Eversmeyer and Burleigh, 1970

rain, amount of rain, monthly means, etc.; relative humidity; and hours of free moisture have generally failed to provide a means of explaining a major proportion of the variation when host resistance–pathogen virulence is not a factor. Perhaps the recent work on stripe rust of wheat by Coakley (1979) and Coakley and Line (1981a,b) will prove to be an exception to these failures. Their findings of differences in winter and spring temperatures are not that far from Chester's (1943) "critical month." In most of the other studies, the disease studied was wheat stem rust in the prairies of the central third of the United States. Here, wide variations of temperatures can occur within a kilometer or even a few meters at night, and precipitation can vary even more (e.g., 1 cm in a single storm or by 6–10 cm within a growing season). Temperature and hours of free moisture vary with height within the canopy. Thus, even if temperature and precipitation play a very important part in epidemic development as most epidemiologists believe, the variation between points within the canopy and over an area no bigger than a field is such that some points are very favorable for, and others very unfavorable for, disease development at any given time. Although additional measurements may solve this problem in small uniform plots, there are severe financial and labor restrictions in conducting such measurements in commercial fields.

VIII. The Future

Attempting to study disease epidemiology on a national basis when over 30 million hectares are grown presents a mammoth problem. Methods are available for determining the race-specific resistances in the 300 cultivars grown; however, because the change over of cultivars grown annually is near 10%, continued work is required. The effect of resistance not known to be race-specific resistance is poorly characterized currently in most cultivars. Virulence of the pathogen populations of stem rust and crown rust in the United States and Canada are well characterized, as is wheat leaf rust in Canada. The other diseases are not regularly studied or published data are lacking. Only hints exist on aggressiveness differences in any of the pathogen populations, and the stability of the pathogen both genetically and environmentally remains to be investigated. Little methodology is available for this latter type of study.

The effects of the race-specific genes for resistance on epidemic development under field conditions needs to be studied throughout the plant growth period. Studies of effects of multilines, mixtures of host genotypes in a field, as well as mixtures of different host cultivars or crops in adjacent fields over large areas need to be conducted.

Studies of disease in commercial fields are now limited by our inability to cover large areas and still detect small foci or low severities over local areas. Perhaps spore trapping would solve some of these problems if the counting could be done by mechanical means. Another potential method for measuring disease severity in the future may be by remote sensing (Colwell, 1956; Kanemasu *et al.*, 1974) as that technology develops.

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