Symposium: Genetic Aspects of Breeding for Resistance Against Soilborne Plant Pathogens

Nonspecific Genetic Resistance to Soilborne Fungi

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Plant physiology and anatomy are partially controlled by genetics; this paper presents some examples of nonspecific genetic resistance to soilborne fungi based on whole-plant physiology, or "macrophysiology." For years, many plant pathologists and physiologists have devoted their efforts to studies of specific resistance, particularly when isogenic lines of a host were available. Much physiologic research has also been devoted to elucidation of the mechanisms involving host-specific toxins. A part of the aim of this paper is to encourage greater emphasis upon nonspecific resistance and macrophysiology. Those who studied Pythium root rots; damage to maize seed and seedlings in cold, wet soil; maize stalk rots; snow molds; and strawbreaker foot rot of winter wheat were unable to determine much about the genetics of resistance. Greater knowledge of the ways by which plants resist these diseases and of the inheritance of resistance is needed.

Pythium root rot of sugarcane. In the late 19th and early 20th century, Java in the Dutch East Indies was the world's leading exporter of sugar. In about 1882, an infectious disease of uncertain etiology, known as sereh, began to threaten *Saccharum officinarum* L. (16), the most important cane in Java. In 1887, the Proefstation Oost Java (Experiment Station of East Java) was established at Pasoeroean to study this threat. *Saccharum barberi* Jeswiet was introduced from India, but the planters rejected it because it had thinner stalks and produced less sugar per hectare than *S. officinarum*. Kobus (as mentioned in reference 16) crossed Chunee, a cultivar of *S. barberi*, with *S. officinarum*. This interspecific cross resulted in P.O.J. 36, P.O.J. 213, and P.O.J. 234. These canes became popular in other parts of the world, but were rejected in Java even though they were resistant to sereh. An expensive system of special seed nurseries and roguing kept the *S. officinarum* cane productive until a second disease, sugarcane mosaic, began to spread.

A natural hybrid of *S. officinarum* and *S. spontaneum* L. 'Kassoer' was found near Pasoeroean. Kassoer was resistant to both mosaic and sereh. When Kassoer was crossed with *S. officinarum*, the resultant offspring were still too thin, fibrous, and low in sugar. It was not until Jeswiet (as mentioned in reference 16) crossed P.O.J. 2364 [(*S. officinarum* × *S. spontaneum*) × (*S. officinarum*)] again with *S. officinarum* (+ approximately 7/8 *S. officinarum*, 1/8 *S. spontaneum*) that satisfactory canes resistant to mosaic and sereh were produced. In 1922, a line that became known as P.O.J. 2878 was a single plant. By 1929, it occupied 90% of the cane land in Java. P.O.J. 2878 was grown extensively in many countries after that.

The interspecific hybrids developed to resist sereh and mosaic produced more sugar than pure *S. officinarum*. The increased yield was attributed to increased tillering and a more vigorous root system. Evans (cited in reference 37) in Mauritius studied the roots of many canes and found that *S. officinarum* in general had less vigorous roots than the other *Saccharum* spp. P.O.J. 2878 differed from *S. officinarum* in that it produced new roots throughout the growing season (see pages 123–161 in Van Dillewijn [37] for an extensive review of research on roots of sugarcane).

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Rands and Dopp (33) studied yield decline in Louisiana before the discovery of ratoon stunt. They concluded that *Pythium arrhenomanes* Drechs. was important on the 40% of Louisiana cane lands that were heavy and poorly drained. They rated Co. 290 (6/8 *S. officinarum*, 1/8 *S. spontaneum*, 1/8 *S. barberi*), Kassoer (1/2 *S. officinarum*, 1/2 *S. spontaneum*), and Uba (*S. sinensis* Roxb.) as most resistant (Table 1). The noble canes (*S. officinarum*) were in the most susceptible class. P.O.J. 2878 was intermediate. The discovery of ratoon stunt and its dramatic effect upon yield decline overshadowed the role of Pythium root rot, but I believe the observations of Rands and Dopp are valid.

Teakle et al (36) reported a high correlation between rate of water flow in xylem with susceptibility to ratoon stunt. Co. 290, a cane rated resistant to Pythium root rot, has a high water flow rate. G. T. A. Benda and H. Koike kindly searched the records and provided the ratoon stunt data presented in Table 1. There is no correlation between the Pythium root rot rankings of Rands and Dopp with the reaction of these canes to ratoon stunt.

Interspecific hybridization in sugarcane was initiated to control sereh and sugarcane mosiac, but it resulted in elevating cane yields to higher levels than were ever attained with *S. officinarum*. Part of this elevation is due to the increased root vigor of interspecific hybrids, which helped to reduced losses due to Pythium root rot.

The inheritance of most traits in cultivated sugarcane is lost in complexity. *S. officinarum* may be an octoploid (3) with a somatic chromosome number of 80. The *S. spontaneum* of Java has 112 somatic chromosomes. Most interspecific hybrids that are about 7/8 *S. officinarum* and 1/8 *S. spontaneum* have about 114 somatic chromosomes. Some of the finest cytologists and genetists have worked with sugarcane, but much remains to be learned.

Seedling blight of maize. Maize (*Zea mays* L.) is favored by a long, warm growing season. Far-sighted researchers believed that yields in much of the North American maize belt would benefit from a lengthened season and that the only way to achieve a lengthened season was to plant maize reasonably early. Early planting, however, entailed the risk of seeding in cold, wet soils, which is unfavorable to maize.

Dickson (11) and Dickson and Holbert (13) began to test maize in cold soils for resistance to *Gibberella zeae* (Schw.) Petch. Early studies involved *G. zeae* and *Diplodia zeae* (Schw.) Lev., but emphasis soon changed to *Pythium* spp.

Dickson et al (12) in 1923 and Dickson and Holbert (13) in 1926 reported that maize seedling grown between 8 and 20 C had thin, water cell walls compared to those grown under higher temperatures. Resistance was attributed to maintenance of "balanced metabolism" over a wider range of temperatures with a better balance between carbohydrate and nitrogenous substances, resulting in seedlings with thicker cell walls.

Hooker and Dickson (21) studied excised embryos and found that those from lines that survived best in cold, wet soils were more resistant to *Pythium debaryanum* Hesse. Resistance was inherited from either the male parent or the female parent. The F_1 embryos were intermediate in resistance. Crane (9) studied several inbred lines and reciprocal F_1s and found that part of the resistance to *P. ultimum* Trow was in the pericarp (maternal tissue) and part was in the embryo. There was no evidence of cytoplasmic inheritance of resistance. Hooker (19) tested maize against eight *Pythium* spp. and resistance to all eight species was correlated, which is evidence for a common resistance mechanism. Line 159 had a resistance rating of 2 and line 991 a rating of 31 of a possible 75. A rating of 31 of 75 is low, but this difference is important under field conditions. Hooker (20) further found significant correlations between resistance to seedling blights caused by *G. zeae, D. zeae*, and *Pythium* spp.

Early seeding is of value even in Illinois, which is in the heart of the maize belt, not on its northern fringes. Pendleton (32) emphasized the importance of early seeding in obtaining high yields in much of the USA, and his data indicate a gain of from 0.5 to 2.0 t/ha when maize is seeded from 19 April to 4 May over seedings 2 wk later.

Pythium root rot of spring barley. *Pythium arrhenomanes* was severe on barley (*Hordeum vulgare* L.) growing in an infertile, sandy-clay soil in south central South Dakota (4). On this soil, Trebi and Odessa (mid-maturity) outyielded Plains and Feebar (early maturity) in four consecutive seasons by amounts ranging from 19 to 49%. In nurseries containing from 24 to 99 entries, the mid-late class yielded most (5). This was contrary to expectations. In this part of South Dakota, water limits yield and early maturity should have been advantageous.

When Odessa (mid-maturity) and Feebar (early maturity) were compared on natural and fumigated soil, Odessa yielded the most on untreated field soil; Feebar yielded the most on fumigated soil (4). Without root rot, earliness was advantageous. With root rot, earliness was a handicap.

Inoculation experiments in the greenhouse involving 9- and 18-hr light periods indicated a strong interaction between day length and susceptibility to Pythium root rot. Varieties from North Africa were inferior to those from Manchuria in the field plots. Barley is seeded in November in North Africa and in the spring in Manchuria (as in South Dakota). Lack of adaptation to photoperiod predisposed North African barleys to root rot (5). Carbohydrate transfer to roots (though not meaured) must have been inadequate in those barleys forced into early maturity by response to photoperiod, resulting in an inability to replace damaged roots.

So far as I know, no one knows much about the inheritance of resistance to Pythium root rot in barley, but inheritance of response to photoperiod is not simple (2).

Gibberella and Diplodia stalk rots of maize. Susceptibility to these stalk rots usually increases at about the time of pollination or shortly after (27). Highly productive lines of maize put much of the total dry matter into grain. Part of resistance to these stalk rots depends upon delaying senescence of stalk tissue in late stages of development. Pappelis and Smith (31) reported that live pith cells high in water content restricted the spread of *Diploida maydis*, which this pathogen spreads in cells already dead. Pappelis (30) found a similar correlation between senescent or dead pith cells in the stalks and the spread of *Gibberella zeae*.

Wall and Mortimer (38) found that single-cross hybrids susceptible to root and stalk rot were characterized by cessation of vegetative growth at pollination and a rapid senescence of leaves at or shortly after physiologic maturity. In resistant hybrids, dry matter in vegetative parts increased for several weeks after pollination. Resistant single crosses differed from susceptible types in that their shift from vegetative to reproductive growth was less distinct. They (38) postulated a major role for growth-regulating substances in determining resistance.

Craig and Hooker (8) and Mortimore and Ward (28) found that resistant stalks had higher levels of soluble sugars at physiologic maturity than did susceptible stalks. Gates (17) found a high correlation between response to tetrazolium chloride, a vital stain, in pith cells 0–10 days before maturity and resistance to stalk rot. Dodd (14) studied plants of the same genetic constitution growing under the same conditions and found that stalks with the most

TABLE 1. The reaction of canes to Pythium root rot according to Rands and Dopp (33) and their reaction to ratoon stunt (provided by G. T. A. Benda and H. Koike, U.S. Sugarcane Field Laboratory, Houma, LA)

Cane	Pythium root rot	Ratoon stunt
Co. 290	1[a]	Susceptible
P.O.J. 2364	2	Very susceptible[b]
P.O.J. 2878	3	Susceptible
P.O.J. 213	3	Susceptible
Chunnee	4	Susceptible
P.O.J. 234	5	Susceptible
Co. 281	5	Very susceptible
Badilla[c]	6	Resistant
Louisiana Purple[c]	6	Susceptible

[a] 1 is the most resistant class, 6 is the most susceptible.
[b] Data by personal communication from O. W. Sturgess, director of Bureau of Sugar Experiment Stations, Indooroopilly, Queensland, Australia 4068.
[c] Noble canes—believed to be pure *S. officinarum*.

kernels had more stalk rot than stalks with fewer kernels, ie, that size of the grain sink influenced stalk rot.

The above studies support the general hypothesis that life within the stalks must be maintained at a certain critical minimum for them to remain resistant to Gibberella and Diplodia stalk rots. White (39) reported correlations of 0.87 and 0.81 in early and late-maturing inbreds, respectively, and their susceptibility to *D. zeae* and *G. zeae*. The underlying resistance mechanisms of resistance have much in common.

Kappelman and Thompson (23) concluded that inheritance of resistance to Diplodia stalk rot is not as complex as that of yield, but that it is sufficiently complex to require the same testing procedures (ie, evaluate inbreds and test crosses, single crosses, and double crosses).

The magnitude of progress in stalk strength and resistance to stalk rots is apparent in Fig. 1. W. A. Russell (as mentioned in reference 1), Iowa State University, Ames, assembled hybrid corns grown during 10-yr intervals and grew them for 2 yr, 1971 and 1972, under the same conditions. Increased standability is essential to increased yield.

Fusarium and Typhula snow molds of winter wheat. *Fusarium nivale* (Fr.) Ces. and three *Typhula* spp. rot the leaves and crowns of winter wheat (*Triticum aestivum* L.) under deep snow on unfrozen soil. Deep snow prevents photosynthesis; if snow cover persists for 100 days or more, respiration by the wheat plant depletes the carbohydrate reserves of the host and severe losses can occur. In 1960, R. Sprague in Washington and D. Sunderman in Idaho began to search the World Cereal Collection of the USDA for wheats with useful levels of resistance. Both workers found a few wheats that survived under severe snow mold conditions. All sources of resistance had very weak straw. It was obvious that resistance had been transferred in the F_2 generation (7), and by 1973, a soft white wheat named Sprague (CI 15376) was available for release to farmers. In the winter of 1973–1974, the snow persisted 150 days on unfrozen soil. Sprague was the only commercial wheat that survived and it survived only if seeded early, between 1 and 26 August. When seeded late (26 September) it died.

Resistance to snow mold depends upon genetic constitution, and upon size (6) and physiologic condition of the plant. Resistant wheats respire more slowly than susceptible wheats when attacked by *T. idahoensis* (24). Efforts to determine the inheritance of resistance have been unsuccessful, but it is believed to be complex. It is nonspecific because it functions against both *F. nivale* and the *Typhula* spp. of the region.

Strawbreaker foot rot of winter wheat. Foot rot develops most severely on early seeded winter wheat in regions of mild, humid winters. The pathogen, *Pseudocercosporella herpotrichoides* (Fron) Deighton, produces spores that splash from infested stubble to growing wheat. Infection occurs from late autumn until spring. The fungus advances slowly from leaf sheath to leaf sheath until it penetrates the culm at or near the ground line. The culms of many wheats are so rotted that the straw breaks after the wheat has headed, justifying the common name of strawbreaker.

Strawbreaker, or eyespot as it is called in Britain, has been studied most extensively in northern Europe. The most resistant wheats have been developed in Europe. The leading wheats of the Pacific Northwest in the USA are susceptible, and to date no satisfactory resistant wheats have been developed for this region.

Pseudocercosporella herpotrichoides has no known races of marked differential virulence within *T. aestivum*. Doussinault (15) studied several wheat cultivars in France from seedling to adult stages and found three components affecting resistance: probability of infection, resistance of the leaf sheath to penetration, and resistance of stems to attack. Schaffnit (34) in 1933 reported that the hypodermis of a resistant wheat was 71 μm in width and those of the two susceptible wheats were 55 and 61 μm wide. Murray and Bruehl (29) found a high correlation between the width of the hypodermis in the first elongated internode of mature wheats culms and resistance.

Cappelle-Desprez has resisted strawbreaker foot rot for at least 20 yr in Britain and has been used as a parent in developing other resistant wheats. Law et al (25) studied the inheritance of the resistance to foot rot using Cappelle-Desprez (resistant), Chinese Spring aneuploids (susceptible), and Mara (highly susceptible). Chromosome substitution lines, with single Cappelle-Desprez chromosomes added to Chinese Spring, enabled these workers to determine that chromosomes 1*A* and 7*A* affected resistance. In tests with Mara, the use of chromosome substitution and aneuploid analysis techniques provided evidence for resistance genes on chromosomes 7*A*, 2*B*, and 5*D*. Resistance is dominant. Law et al (25) concluded that resistance to strawbreaker is probably inherited in a complex manner, even though chromosome 7A is important.

The resistance of Cappelle-Desprez is not adequate to prevent losses under extreme conditions. Sprague (35) in 1936 reported that *Aegilops ventricosa* Tausch was very resistant. Mme. Ometz crossed *Aegilops ventricosa* (genome DDM^vM^v) with *Triticum persicum* Vav. (genome *AABB*) in 1953 and obtained fertile hybrid amphidiploids by doubling the chromosomes with colchicine (26). Ecochard backcrossed the above amphidiploid twice with *T. aestivum* 'Marne' (*AABBDD*). After selfing for six generations, a relatively stable 42-chromosome line, VPM-1, was proven foot rot resistant by Maia (26) in 1967 (VPM = *V*entricosa, *P*ersicum, and *M*arne). The resistance of VPM-1 is greater than that of any "pure" *T. aestivum* and it has been used extensively as a source of resistance. It possesses a type of hypersensitivity (18) and it possesses the widest hypodermis found by Murray and Bruehl (29). Delibes et al (10) believe that the resistance of VPM-1 is simply inherited. Experience in Washington State indicates that this is unlikely. The hypersensitivity is probably governed by either one or just a few genes, but not by the hypodermal width.

Jahier et al (22), using monosomic and disomic analysis, agreed with Law et al (25) that resistance was conferred by chromosomes 7*A*, 2*B*, and 5*D* by Cappelle-Desprez, and they found that chromosome 7*D* of *A. ventricosa* was important, thus confirming earlier speculation that the *D* genome was important (10,25). To date, foot rot resistance is known to be influenced by genes on four chromosomes.

SUMMARY

The pathogens in this paper are pathogenically unspecialized. The most resistant hosts are not immune. The geneticists and pathologist work with shades of gray, not black and white, and disease classes are arbitrary. In none of these cases, to my

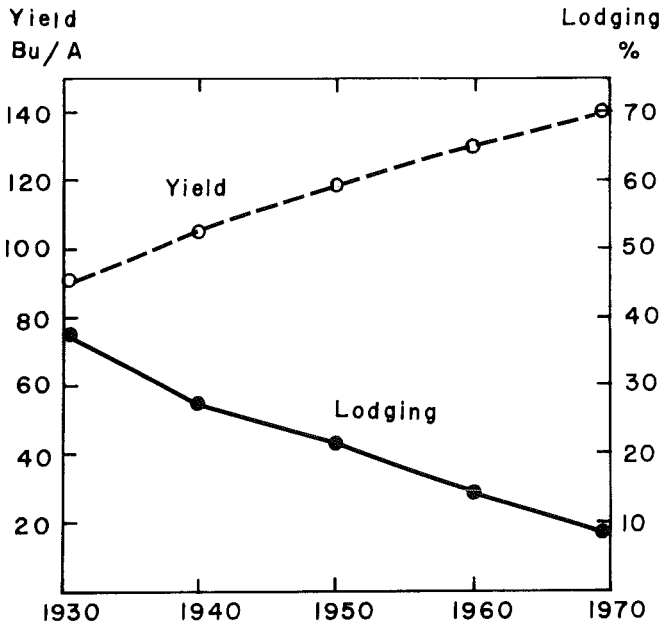

Fig. 1. Yield in bushels per acre and percent lodging of hybrids grown in Iowa in 1930, 1940, 1950, 1960, and 1970, when grown at the same time (1971, 1972) under the same cultural conditions (90 bu/a = 5,647 kg/ha; 140 bu/a = 8,784 kg/ha). Reproduced, by permission, from page 38 of Aldrich et al (1).

knowledge, does anything resembling precise knowledge of the genetics of resistance exist.

Resistance to Pythium root rot of sugarcane involves the vigor of interspecies hybrids. Resistance in maize to seedling blight involves maternal tissue (pericarp) and embryos, and balanced metabolism. Resistance to Gibberella and Diplodia stalk rots of maize and Pythium root rot of barley involve the balance between vegetative and reproductive processes. Resistance to snow molds involves the rate of respiration of winter wheat in the dark at near 0 C. Resistance to strawbreaker foot rot of winter wheat is influenced by the thickness of the hypodermal layer in the stem, about which little genetic information exists.

If we visualize the host plant as a machine, the mechanisms of resistance to these diseases involve the engine and alloys of the machine. Fundamental processes within the host are involved. In contrast, in most cases of specific resistance to highly specialized pathogens, the resistance gene(s) usually plays no known essential role in the basic workings of the plant. In the absence of the pathogen, isogenic lines of the host may be indistinguishable. Study of isogenic lines of wheat versus rust is comparable to studying the paint on the machine, not the engine or the metal.

Improvements in most cultivars represent horizontal movements, ie, they represent better quality, specific resistance to disease, earlier maturity, or advances in some other characteristic, but the yield plateau is usually the same as in the previous cultivars. Vertical movements to new yield plateaus are few and far between, but studies of nonspecific resistance may help achieve new yield levels.

Nonspecific resistance to nonspecialized pathogens is difficult to manipulate, both by the breeder and by the pathologist. Resistance is seldom high, and it is often found in plants of poor agronomic type or poorly adapted to the area in which commercial production is intended. After years of effort the result is usually a compromise, with only a portion of the resistance present in the source plant having been transferred to commercially useful plants. Increases in resistance often come in rather small increments. The pathologist usually cannot develop sufficient precision in testing to identify individual genes or their effects, so that the results are difficult to analyze genetically. Consolation and satisfaction result from the assurance that most of these advances in resistance have been permanent. New races of pathogens seldom negate the progress, and the most of these advances in disease resistance have resulted in sustained higher production.

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