DIVERSITY - THE ONLY ASSURANCE AGAINST GENETIC VULNERABILITY TO DISEASE IN MAJOR CROPS 1/

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I am honored to address this distinguished group interested in and dedicated to the improvement of forest trees. There is precedent for a small grain pathologist's addressing tree breeders, as at least two forestry symposia, in 1964 and 19 69 , included papers by small grain workers. The implication was that small grain breeders and pathologists had shown the way, and that tree scientists should learn from successes with small grains. The successes have been many but, unfortunately, there also have been failures with small grains and other intensively bred crops. The message I hope to leave tonight is that breeders and pathologists of both field crops and trees can learn from the failures as well as the successes, and that both can profit from emulating or maintaining natural ecosystems such as most forests represent.

The proneness to disease of genetically homogeneous crops has long been a concern of a few plant pathologists and plant breeders, but as far as recognition by the public and the scientific community, the concept of genetic vulnerability had its belated birth in 1970 - and southern corn leaf blight was its midwife. Fortunately for corn, wheat, rice and, especially, man, its gestation period was not longer. But the birth was legitimate, and the certificate to that effect was published in 1972 by the National Academy of Sciences. I don't need to tell you about the 1970 corn extravaganza; anyone who read the popular and scientific press then couldn't have escaped it. Suffice it to say that all was rosy in 1970 when suddenly panic hit the Corn Belt, there was apprehension in Washington, pandemonium characterized the commodities market, and southern corn leaf blight was a household phrase. The reason: Corn, America's great energy crop, basis of a multi-billion dollar beef and pork industry, indigenous to this hemisphere and long considered safe from disease, was threatened. The problem, or so people were told, was a microscopic fungus, Helminthosporium maydis race T.

But race T was not the basic problem. It was present, obviously, as it had been for several years previously. But a pathogen alone does not make a pandemic. It is obvious also that the environment was highly favorable, which allowed race T to surface in 1970.

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The real cause of the corn pandemic can be learned from the science of comparative epidemiology. To illustrate, we will compare disease development in a typical, modern agro-ecosystem with that in a check population that is an indigenous ecosystem. From our particular comparison, "the oat model" for disease devastation and control will emerge. I believe that, had lessons been learned from experiences with oats and other small grains, the 1970 corn debacle might have been avoided.

Any wilderness area should serve as our indigenous ecosystem but we haven't studied the plants and their pathogens well enough yet in most wilderness areas and a hungry world that wants food from stable, pollution-free agro-ecosystems cannot await the outcome of such studies. Hence, we will study an area where the host plants or their wild relatives are well known - then conclusions are immediately meaningful. in terms of present world literature, and immediately applicable to known agro-ecosystems. No group of plants has had its diseases more researched than the small grains, so let's go quickly to the Eastern Mediterranean where progenitors of cultivated species of barley, wheat, and oats are indigenous, and where they have been studied intensively.

Wild oats, Avena sterilis, progenitor of our cultivated oats, are abundant near the Sea of Galilee where our cultural and agricultural histories merge in a very small area. For instance, on the shore of the Sea of Galilee there stands the Church of the Multiplication of the Loaves and Fishes where tradition says Jesus performed the miracle of feeding the five thousand. Mosaic tile dating from Byzantine times depict the loaves and fishes, and on a small altar the Bible is opened to John 6:9 where it says very clearly that the loaves were made of barley. What a thrill it was, then, to go immediately outside and see Dr. Y. Aikster, an Israeli plant pathologist, collecting wild barley, Hordeum spontaneum, almost 2,000 generations removed from that gathered for baking barley loaves in Jesus' time.

The primary habitat of wild barley is the so-called Fertile Crescent (Harlan and Zohary 1966). The same area serves also for wild oats, wild emmer, and wild einkorn, except that the last is concentrated more in Turkey and not south into Israel.

Centuries of overuse of this area by man resulted in the indigenous forests being replaced by strong colonizing species to form an open Quercus-woodland belt. Rhamnus, the alternate host of the oat crown rust fungus, and Quercus are two important components of this ecosystem. Other important, well known components are wild barley, wild flax, and Ornithogallum or Star of Bethlehem, the alternate host of the barley leaf rust fungus. Wild emmer, a progenitor of cultivated wheat, is a less conspicious component. Now, especially since I mentioned alternate hosts, none of you would expect this heterogeneous popula-

tion to be composed just of higher plants, and it isn't. For instance, Puccinia hordei occurs on barley and, of course, on the alternate host, Ornithogallum. H. murinum is another species of wild barley and on it are two different rust fungi, Pucciania striiformis and Uromyces sp.

Certain valleys are especially rich in oat disease resistance genes. Rhamnus plants abound on the surrounding hills and the floras of the small valleys are very diverse. In one, Dr. Isaac Wahl's group found a crown rust resistance gene in 30% of the oat plants collected. In that small but diverse area, one finds oat plants with and without crown rust. One finds some plants with Erysiphe graminis, the powdery mildew fungus, but the plants still are vigorous and productive. Some plants will be systemically infected with Ustilago avenge, the oat loose smut fungus, but not many. And some plants, but again not many, show symptoms of infection by the barley yellow dwarf virus. Most plants have some stem rust, but they remain green and productive, as apparently a different balancing mechanism has evolved for that host-parasite interaction than for the others. Apparently genes for specific or vertical resistance, backstopped by general resistance and dispersed at random in the population to form a natural multiline, protect the population from crown rust, powdery mildew, and smut, while uniform but general resistance keeps stem rust in balance.

The periphery of this wonderful collecting area is bordered with encroaching agriculture and highway construction. How long before this valuable, living gene park or preserve is gone forever? Probably not long. Yet once gone, it is gone forever. Extinction is an irreversible biological phenomenon. Such living gene parks, where coevolution in host and pathogen can continue, should be established for all our major crops.

The Valley of Elah is where tradition says David slew Goliath. The Philistines advanced from one side of the valley while Saul and young David advanced from the other. You know the rest, except that the wild barley and wild oats that young David used for sustenance abound. My Israeli friends look at this, consider the history of the area, and seem not at all surprised that scientists are finding genes for high protein, high yield, disease resistance, and many other desirable characters in the Israeli grains. After all, they say, it was dense stands of these robust and vigorous grains that provided energy for the rise of civilization in this area. It wasn't until over grazing by Arab flocks was brought under control by the young state of. Israel that the extent of these spontaneous stands was realized. But Harlan and Zohary (1966) say there was no need to cultivate here, for man tilling the soil could not have improved on the stand densities provided by nature. Rather, cultivation must have begun on the fringes of the Fertile Cresent.

This should have given you a feeling for the dynamic balance in a Vavilov center of diversity or origin of one of our major crop species. We saw homozygous and heterozygous plants mixed in a very heterogeneous population that included also their heterogeneous pathogens and, doubtless, antagonists of those pathogens. Most plant diseases about which we have any concern are crowd diseases - they are serious only because like host genotypes are crowded together. This common agricultural practice simultaneously homogenizes the pathogen population and aids its dispersal. In populations such as we described, both host and pathogen were present in a favorable environment, yet epidemics of crowd diseases were virtually impossible. This, then, is a natural gene-management system that results in a well buffered plant population that can be said to have "population resistance."

Such interacting populations are buffered and protected by several mechanisms:

1. General, non-host resistance
2. General, horizontal, race-nonspecific, polygenic resistance 3/
3. Race-specific, vertical, oligogenic resistance 3/
4. Tolerance
5. Antagonists
6. Population resistance or buffering, and
7. Homeostatic tendencies in the pathogen population

General, non-host resistance is the kind of resistance that makes plants resistant to most pathogens, and most pathogens avirulent on most plants. For instance, wheat is highly resistant to most Quercus pathogens - a fact that is little thought of since we can't yet cross Triticum and Quercus and so study or use this type of resistance. I tell my students that a possible challenge for the wheat breeder may be to make wheat like oaks as far as disease resistance is concerned and still be able to make angel food cake from the acorns. Of course, one would hope the new wheat would not be susceptible to oak wilt, either. I'll have no more to say about this type of resistance, but keep in mind that it is operating in our check population.

I wish that we had time to discuss fully each of these resistance mechanisms; instead, we must move on in our comparative epidemiology study in which most of them will reemerge. We saw what an indigenous ecosystem is like. What happened to the agro-ecosystems that had their genesis in such stable indigenous ecosystems?

Barley and wheat were domesticated on the fringes of the Fertile Crescent and taken by primitive man to Europe and Asia. Oats were carried as a weed, domesticated in Europe, and eventually brought by early settlers to North America. They were grown in the USA in 1632, and in 1786 George

3/For purposes of simplicity, I am equating the adjectives in (2) and (3), respectively.

Washington had 580 acres of oats. Oats, like most other major field crops, went through three stages of improvement - introduction, selection, and hybridization. In many cases, selection preceded hybridization by 300-500 years and resulted in land varieties that were in balance with local pathogens. These varieties, where they remain, constitute a valuable source of diversity that should be preserved in gene banks and gene parks. Hybridization of oats brought a significant increase in potential yield, but it brought very little increase in actual yield because of diseases. This should have caused scientists to stop and reevaluate, but it did not. Oat workers, like other workers on other crops that Harlan (1972) describes as having a "pure line mentality", were filled with optimism that the next gene would do the job, and they continued to pursue the dream of obtaining that pure line of greatest value. Let's see what happened.

The first pure line oats of hybrid origin, the Victoria-Richlands, were released in 1942 and were immediately popular. They replaced varieties developed by pure line selection more rapidly than hybrid corn had replaced open pollinated corn varieties. They rapidly rose to 98% of Iowa's 5.3 million acres of oats and also covered vast acreages from Texas to the Prairie Provinces of Canada. They controlled crown rust, stem rust, and the smuts, as intended, but a new disease, Victoria blight, hit them and caused their rapid decline.

Victoria blight, caused by Helminthosporium victoriae, was controlled quickly - farmers simply switched from Victoria derivatives to Bond derivatives that, fortunately, were waiting in the wings. Iowa's oat acreage went from 86% Victoria derivatives in 1947 to 95% Bond derivatives in 1948.

What happened during this period of rapidly shifting varieties and pathogens is an instructive portion of "the oat model" of disease devastation and control. H. victoriae proved to be a seed-transmitted, soil-borne pathogen. It produced a pathotoxin that was highly specific in its host range, and the pattern of resistance and susceptibility to the toxin and to the pathogen was the same. This specificity carried over to the field. (In all these aspects, H. victoriae and H. maydis race T were very similar.)

Bond derivatives were resistant to Victoria blight and, like the Victoria derivatives, they were resistant initially to crown and stem rust. But the extensive plantings first of Victoria derivatives, then those from Bond, quickly selected for variants in the population with virulence for those respective host genotypes. Further, Bond and Victoria derivatives, because of genetic linkages, were essentially "opposites" in the spectrum of crown and stem rust races to which they were susceptible. Thus, when the dust had settled, H. victoriae had come and been controlled, but crown and stem rust remained in the picture as major threats to oat production - the only difference being that different races

of the pathogens were involved. Summarized by Murphy (1965) and Browning and Frey (1969), this illustrates what Johnson (1961) called "man-guided evolution." Now recognized as a general phenomenon, it has been the same in Canada, Australia, Kenya, and the USA, on wheat and oats, whenever genes for specific resistance have been used. Most recently it was illustrated with Aurora and Kavkaz wheats in the USSR. But nowhere is it more dramatically and succinctly illustrated than with "the oat model" because of the rapid, H. victoriae-forced changeover of varieties that were "opposite" in disease susceptibility.

Rapid control of the "new" pathogen, H. victoriae, was, and still is, considered a major achievement. It still is cited, correctly, as an example of the permanent control of a major pathogen through breeding for disease resistance. But the main plot of the story gave much less cause for applause than the H. victoriae subplot. Rust continued to plague us. First race 202 negated the Bond resistance. We added Landhafer resistance to control race 202, only to lose this resistance to race 290. We were on a vicious circle. We knew it, but we could live with it. But then, in 1957, a new race was found by Dr. M. D. Simons - race 264 - that could attack all our hexaploid sources of seedling resistance. This, at long last, jarred us at Iowa State University into reevaluating our whole program and situation. Finally, we looked hard at the history of oat improvement and realized what had happened. Man had taken oats from the balanced equilibrium in their center of origin, selected them rigorously, always narrowing their genetic base, added genes for specific resistance, inadvertently rejected genes for general resistance, then tried to grow the resultant varieties in a pure and extensive monoculture. But nature abhors a vacuum. So first H. victoriae, then one rust race or another attempted to fill the void, as nature tried to return a semblance of balance to the population. The pandemics were the result.

Thus, we realized, from the perspective of Sunday-morning quarterbacking, that H. victoriae had not been the basic problem back in the mid 40's - it was only a symptom of the basic problem. The basic problem was extreme homogeneity in the absence of general resistance. The symptom was controlled very rapidly, simply by not growing Victoria oats, but the problem remained. Recurring epidemics caused by different races of crown and stem rust were just other symptoms of the same basic problem.

Thus, we saw that we were going to have to seek totally different means of disease control in oats, and at that time Dr. Simons began his long range basic study of tolerance, seeking a polygenic type of control, and Dr. K. J. Frey and I committed our program to the development of multiline cultivars, seeking an alternate way of using genes for vertical resistance.

How does this apply to corn? In 1970 there were about 58 million acres of corn - over 1 trillion virtually identical plants - with Texas male sterile cytoplasm. This was like a tinder-dry prairie waiting for a spark to ignite it. H. maydis race T was the spark, the pandemic the result.

Race T was controlled as easily as H. victoriae, but the problem of excessive homogeneity in corn remains. This is like whitewashing the pump when it rusts from disuse because the well water is contaminated; we only treat the symptom. And in doing so we gamble that the environment will not be favorable for another spark in this tinder-dry prairie.

Concerned plant scientists must ask, "What next awaits corn? Fungicides?" Each year the USA uses some 3.6 million pounds of active ingredients of fungicides to control diseases on some 1.4 million acres of potatoes. If we applied fungicides at a proportionate rate on corn, 150 million pounds of active ingredients, suspended in at least 625 million gallons of water, would be necessary for low gallonage aerial application on some 60 million acres of corn. To be sure, corn is not potatoes - but there are similarities. A field of a single cross is just as homogeneous as a clonally propagated potato variety, and many fungicides have been tried on corn since 1970 with a view of controlling disease. But I think the sheer magnitude of these figures suggests that the quantities of conventional fungicides that might be required for our major field crops stagger the imagination and that fungicides should be avoided if at all possible.

Is there an alternative for corn? Yes! Simply broaden the genetic base! The National Academy of Sciences' (1972) study on genetic vulnerability makes the point that, "It is possibly not accidental that all of our serious widespread corn epidemics have occurred since 1960. These include: (1) The maize dwarf mosaic virus and corn stunt mycoplasma attacks of 1964; (2) Phyllosticta leaf blight in 1968; and (3) Southern corn leaf blight in 1970." Since that was written, additional fungal and bacterial problems have shown up on corn. My suggestion, therefore, is simply to progress back to the level of genetic diversity that existed in the USA corn crop about 1960. If I am not mistaken, this will be the level of diversity that exists in double-cross as compared to single-cross hybrids. I am optimistic that, with population improvement through recurrent selection, the requisite level of diversity can be obtained without undue sacrifices in yield and other desirable characteristics.

How much diversity may be needed in a population? Let me answer this in part by presenting the rest of "the oat model."

We ran our first experiments at Iowa State University on disease development in diverse populations in 1954 using the oat stem rust fungus on mixtures of contemporary oat varieties. We could easily pick out the susceptible plants by the rust on them. But the important thing is, there was not nearly as much rust on susceptible plants in a mixed stand as in a pure stand. Obviously, by some mechanism the resistant plants protected the susceptible plants from this crowd disease. To make a long story short, because there are so many benefits from mixing per se, we had to develop near isogenic lines before we could test the true contribution of diversity to disease control.

Since crown rust is our major oat disease, we began, in 1957, development of lines isogenic for different crown rust resistance genes. When adequate seed stocks were available we ran experiments in many 50' x 50' plots arranged in an east-west direction to minimize interplot spread of spores. Our best measure of disease development in the different treatments was yield of the pathogen as measured with Rotorod spore traps downwind from the center of each plot. We trapped spores, counted them, converted the data to number of spores per liter of air, and ran the data through a computer that drew disease progress curves (Figure 1) .

The disease progress curves fit the logistic equation that describes growth of any organism in a limiting environment. The series of curves in Figure 1 (Cournoyer 1970) , from an experiment that involved our first commercially available multilines, fell into four groups: (1) the resistant midseason cultivar, (2) the susceptible midseason cultivar, (3) the susceptible early cultivar, and (4) the four multilines and the resistant early cultivar. Two very important facts are evident: First, a diverse multiline population was buffered against a diverse population of rust under the environmental conditions of this experiment. Second, the advantage of the early susceptible over the midseason susceptible cultivar was due to background general resistance in the early line.

After many experiments, we satisfied ourselves that multilines would protect Iowa oats against their major disease, crown rust, in our agro-ecosystem, and for several years this gene-management system has been used successfully on over half a million acres of oats annually. But our colleagues who work on other crops wanted to know if diversity would protect against disease in a longer, more severe disease season than that on oats in Iowa. To test this, last winter Dr. M. E. McDaniel of Texas A. & M. University and I grew Texas and Iowa oat lines in isolated 1 1/4-acre fields on the Texas Coastal Plain near Corpus Christi. (This work was sponsored by The Perry Foundation of Robstown, Texas.) Puccinia coronata overwinters in South Texas. Our experimental fields were inoculated only naturally by P. coronata inoculum from a diffuse source. Fogs and heavy dews were extremely favorable for rust development throughout the 4-5 month disease season. For present purposes,

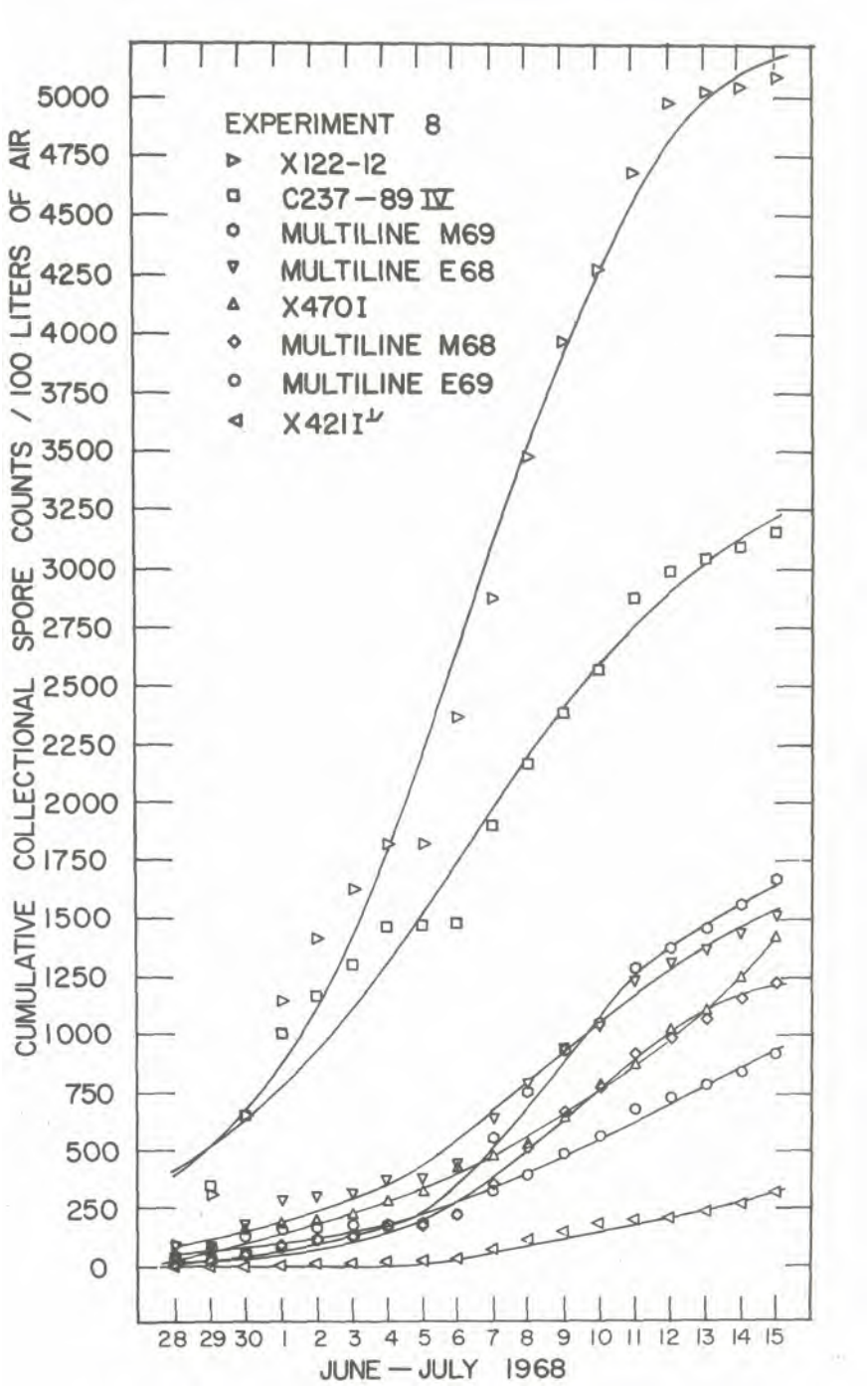

Figure 1. Disease progress curves of cumulative collectional crown rust urediospore counts/100 liters of air for eight oat cultivars in early and midseason maturity classes. X122-12 and X421I are susceptible and immune midseason lines, respectively. C237-89 IV and X470I are susceptible and resistant early lines, respectively. Multiline M68 and Multiline M69, and Multiline E68 and Multiline E69 are commercially available multilane cultivars that are midseason and early, respectively, in maturity and that are isogenic with the above cultivars in the respective maturity classes.

1/The curve for X421I, an immune line, represents "background noise." It could not be fit by the logistic equation and is, consequently, a curve of best visual fit. (From Cournoyer 1970) .

the important conclusion drawn from the experimental results was that the Iowa multilines stood up even in that long, severe disease season and that their ordering was the same in South Texas as in Iowa. Again, lines with background general resistance yielded fewer spores than lines without general resistance. Also, indicator plants showed that a more diverse rust population was active in the multilines than in the pure lines, just as the Israelis have shown.

In an interesting study in Israel, Wahl and Eyal (1972) obtained data that suggested how much resistance may be necessary to protect a population. They collected seed from wild oat plants along transects without regard to the rust reaction of the mother plant. Then they tested the seedlings with race group 264, the most prevalent and virulent race group in the population. Surprisingly, they found 29% of the plants resistant to race 264! Thus, when used as part of a diverse population, a frequency of only 29% can be considered adequate protection against the most virulent strain of the pathogen. Notice, too, that susceptible plants were not eliminated as many writers assume will happen with continuous host-parasite coevolution. As Day (1974) says, "during evolution - - - parasites have been kept in check by the requirement to conserve their hosts for their own future survival."

Thus, it seems conclusive that specific resistance, wisely managed, will buffer against a diverse pathogen population in an indigenous ecosystem as in Israel, in an agro-ecosystem with a short disease season as in Iowa, and in an agro-ecosystem with a long, very severe disease season as on the Texas Coastal Plain. Further, the pathogen population is kept more innocuous in all three ecosystems by being kept more diverse.

We think of small grains as being autogamous and, thus, some may think that lessons from these populations will not apply to non-autogamous crops. But, in fact, outcrossing in these indigenous populations is so common, and heterozygosity so prevalent, that I am confident lessons from the natural ecosystem will apply to other crops regardless of mating system.

I could spend more time on our project's efforts to find other ways of managing genes for vertical resistance (such as interregional diversity via gene deployment) so that, as in nature, man can retain the effectiveness of this valuable natural resource. Also, I could spend much time on our project's efforts towards obtaining polygenic tolerance or generalized resistance in a superior recurrent parent. Otherwise, this essentially completes "the oat model." No other crop, to my knowledge, has suffered more destruction at the hands of homogeneity, nor progressed further down the road to recovery and, therefore, is a better model for discussions of genetic vulnerability.

How did we get into this problem of homogeneity? Were warnings not sounded? They were. Dr. H.H.K. Thwaites , Superintendent of the Royal Botanic Gardens in Ceylon, said even before coffee rust broke out there in 1869: "Ceylon will be ruined. Any country that gambles its future against the quick wealth that comes from a single crop is inviting bankruptcy" (National Academy of Sciences 1972) . Too bad Brazil didn't read his warning before coffee rust broke out there in the late 1960's. Also, one wonders what Dr. Thwaites would say about Iowa's $7.5 billion agricultural industry that is based on a very limited number of varieties of corn and soybeans!

Stevens (1939) associated disease loss in several field crops with mode of reproduction and concluded that buckwheat, corn, and rye, being naturally cross pollinated, had great protection from genetic diversity per se . He said, "Any one who is unwilling to accept the significance of a correlation between the striking freedom from disease and the fact that the plant can reproduce only by crossing should at least advance some other hypothesis."

Again, in 1948, Stevens (1948) stated, "Against the proven ability of the various types of parasites to vary and mutate, the best natural defense would seem to be the ability of the host plant to vary at least as often and as much. This can occur only with frequent cross pollination. From an evolutionary point of view, there is no substitute for this condition. No control measure yet used equals in efficiency on a broad scale the natural ability of a cross-pollinated crop to protect itself." Instead of heeding him, we made our main cross-pollinated crop, corn, as dependent on man for protection as clonally propagated potatoes have always been.

Jones (1958) , who made the first double cross in corn and who perfected the use of Tcms , said, "The first double crosses were designed to overcome the handicaps that the single crosses had in seed production. It actually turned out that the genetic homeostasis was far more important. (Genetic homeostasis) is the gyroscope that holds the ship steady in a surging sea." We sank the double cross, and the sea got pretty rough in 1970.

Of self fertilized crops, Jones (1958) said: "Genetically uniform pure line varieties are very productive and highly desirable when environmental conditions are favorable and the varieties are well protected from pests of all kinds. When these external factors are not all favorable, the result can be disastrous."

How well were these warnings heeded? Harlan (1972) described early plant improvement procedures and then said, "Whatever the procedure, the net effect was to narrow the genetic base of the crop as grown in production fields. Generalized adaptation and general resistance to pests were both sharply reduced. A large proportion of the genes of the old land races was discarded, and modern varieties represent only a fraction of the germ plasm once grown.

"A pure line mentality, convinced that variation was bad, uniformity was good, and off-types in the field somehow immoral, developed. Symptoms of this mental climate could be found in crop judging contests, ribbons awarded at county and state fairs, crop improvement associations, seed certifying agencies, and in some provisions of state and federal seed acts. It did not seem to occur to anyone that a deliberate mixture of cultivars could be a useful alternative to pure line culture. Although grain is frequently mixed in the elevator anyway, a mixture in the field was considered bad husbandry and a slightly less than mortal sin to be kept hidden on the back forty off the road. Varieties adapted to very specific conditions were considered desirable, and the ultimate goal of some plant breeders was to develop a different variety for every field."

"Thus it was that we laid ourselves open to epiphytotics of serious dimensions. Each pure line invited a race of pathogen to attack it. The plant breeders set up crash programs to find and incorporate a gene for resistance to that race, only to invite a new race to increase and attack the new variety. The cycle went on and still goes on from crisis to crisis without any real hope of a permanent solution, as long as the gene base is limited and the pure line mentality prevails."

But let's look at some actual data from the report of the National Academy of Sciences (1972) . In the USA, for corn, six major varieties make up 71% of 66 million acres. These are not all unrelated and, of course, in 1970 Tcms was common to all of them.

For sorghum, while the exact data are hidden in files of commercial companies, male sterility is used so that all sorghum varieties have only one kind of sterility-inducing cytoplasm, and present parents of hybrids have at most five or six varieties in their parentage. Sorghum, then, is much like corn was in 1970.

For wheat, nine cultivars make up half of 44 million acres. Scout alone is sown on 8 million acres in the southern and central Great Plains. Again, these nine contain some common factors for disease susceptibility.

For soybeans, six cultivars make up 56% of 42 million acres. Harlan (1972) adds, "we produce 75% of the world's supply of soybeans . . . . with a significant portion of the world's supply of oil and protein for human nutrition, yet the entire American soybean industry traces back to only six accessions introduced from the same part of Asia."

These are figures for the USA - those for the world at large are more alarming. The USA and other developed countries formerly did the crop improving, frequently using land-races in developing countries and wild races in the Vavilov centers to make repairs, but this has changed drastically.

For wheat, Dr. Norman E. Borlaug's program has been so successful that 24 million acres of his semidwarf wheats are being grown in South Asia alone, 15 million of them in India. This is a calculated gamble of which Dr. Borlaug is well aware, and so far it has not backfired seriously. Yet these wheats are known to have several difficulties associated with the dwarfness character. Foremost are susceptibility to Septoria tritici and S. nodorum, root rots, insect damage, and poor seedling vigor. A requisite of cultivars from a center of cultivar improvement such as that in Mexico is photoperiod insensitivity. Yet this character in wheat seems associated with susceptibility to root rots, the straw-breaker disease, and frost sensitivity (National Academy of Sciences 1972).

Rice is an especially interesting case. So variable is the rice crop around the world that large-scale failures due to disease and insect attacks have not occurred in the recorded history of the crop with the exception of the great Bengal famine of 1942-43. However, the new IR varieties may be changing this. As for wheat, the short, highly fertilized and irrigated rice canopy provides a much more favorable microclimate for disease and insect development than the old plant types. As a result, IR 8 in the Philippines was severely damaged by a virus disease called tungro. A newspaper article even said that this disease of "American-developed" rice was one reason that President Marcos declared martial law in the Philippines. The National Academy of Sciences' (1972) study says that the market demands uniformity, but that when something goes wrong, the scientist gets the blame. In this case, a new rice cultivar was developed by a highly international staff at the International Rice Research Institute (IRRI) - but when something went wrong, it was "American developed." I should note that recent reports from the IRRI claim resistance to tungro.

Such extensive use of the same genotypes for dwarfness, photoperiod insensitivity, or other characters, no matter how desirable on the surface, is a gamble I hate to see us take. Eighty percent of the world population depends on rice and wheat for food. Man is dependent on these crops, along with corn, sorghum, and potatoes. Yet man has so modified these plants that they are dependent on his husbandry for survival. Thus, we and the plants are mutually dependent on each other. If man has endangered their survival, and he has, he has simultaneously endangered himself (Browning 1972).

We can stand a pandemic of our chestnuts or elms. We can survive if rust wipes out South American coffee. We can even survive a corn pandemic in our American agriculture. But who could survive a real pandemic in wheat and rice on which 80% of the world depends?

Do dangers accompany the intensive and extensive culture of narrow-based genotypes of field crops? We might as well ask if atomic warfare is dangerous. But let us again learn from history by looking at what happened after the first green revolution.

The introduction of Solanum tuberosum to Northern Europe from Chile was a green revolution - probably the first green revolution. Much of Northern Europe had a diversified agriculture but, unfortunately, Ireland did not. The Irish, victims of an atrocious English social system, soon became dependent on the potato. It thrived there, and they thrived on it. But, whereas some 2 million Irishmen could have lived in plenty with the potato, they chose to use its energy to double their population twice and, instead of 2 million people living in plenty, 8 million people lived in poverty. Then Phytophthora infestans changed even that. You know the rest. An estimated million Irishmen died horrible deaths from famine and malnutrition and disease, and another million emigrated, many of whom died before reaching foreign shores. There are at least three lessons here: (1) a microscopic plant pathogen can destroy man's food, cause governments and whole political systems to tumble, and even destroy man himself; (2) a population dependent on a single, homogeneous crop for survival is headed for catastrophe - the only question is, "How soon? "; (3) a green revolution without population control is doomed to failure. Dr. Borlaug recognizes this and says his Green Revolution has only given two or three more decades to get population under control. More recent predictions are not even that optimistic.

The Irish weren't the only ones hit by late blight. For instance, in Germany in 1915, 700,000 people died of its ravages. This helped the Allies win World War I which, of course, made it alright and so we seldom hear of this catastrophe.

Thus, some 2 million people died as a result of two late blight pandemics in Ireland and Germany. By way of contrast, only 105,000 people died as a result of two atomic bombs we dropped on Japan to end World War II. Is it a fair question to ask if atomic bombs might not have been more merciful to the Irish and Germans than was P. infestans ? But the point is, plant disease pandemics and atomic warfare are both gambles man cannot afford to take. Yet modern man continues to live under the shadow of the threat of both and few voices sound in alarm.

CONCLUDING REMARKS

I described the indigenous *Avena* populations as protected from stem rust by general, horizontal resistance. Dr. Wahl's group has found general resistance against crown rust common in wild oats, and against leaf rust common in wild barley. Such experiences, with that from agro-ecosystems, has made many plant pathologists and plant breeders consider general resistance the control of choice against most pathogens. This may be justified. But to rely *only* on general resistance, especially from one source, is again to put all our eggs into one basket. As Day (1974) says, there is no way of knowing *a priori* that a cultivar has general resistance. At best, general resistance is conceptual (Browning and Frey 1969) . We can count on it only against strains of pathogens against which it has been tested, leaving homogeneous cultivars potentially vulnerable to new or previously undetected pathogenic strains. Therefore, use of general resistance from different sources is indicated.

Nature uses vertical resistance very successfully to protect indigenous populations. Why has experience with vertical resistance in agro-ecosystems generally been so unsatisfactory? In nature, vertical resistance is used only in diverse populations; so must it be in agro-ecosystems. Further, vertical and horizontal resistance are miscible in all proportions and should be so used by man, as in indigenous populations.

The indigenous populations we examined and our multiline varieties truly are phenomena much greater than the sum of their parts, and the key to this is their diversity. Kucera (1973) , in his interesting book, "The Challenge of Ecology", says that, "Diversity prevents the development of excessive pressures on any part of the ecosystem and the threat of unstable conditions is thus minimized." Diversity, thus, is the key to stability, especially against plant pests, for it is the only protection against the unknown, against a future disease risk situation.

The National Academy of Sciences' (1972) report on genetic vulnerability says, "If uniformity be the crux of genetic vulnerability, then diversity is the best insurance against it." Unfortunately, in the Academy's conclusions and recommendations - the only part most decision makers will probably ever read - the Academy doesn't even hint at recommending the most logical thing of all, namely, diversify!

The Academy report seems to justify this position by saying that the market place demands uniformity and scientists provide it. I don't think the market place should be allowed to make that decision. Plant scientists, not farmers and processors, have degrees "Doctor of Philosophy." They should stop being plant technicians, putting out another variety or testing another fungicide, and

use some of that philosophy to say, "No! You can't grow that genotype so extensively or use that hazardous pesticide indiscriminately. It isn't a safe practice for you, your children, or your fellowman."

We don't give the public the freedom to treat diseases of people or of their animals with antibiotics and certain other drugs. Why should we give the public the freedom to endanger the plants they cultivate? We should not. The stakes are too high.

No one in the financial world would want all stocks or all bonds if he wanted a well managed portfolio. Even so in the biological world. But scientist brought up in the pure-culture, pure-line tradition balk at diversity. Purity is absolute. When one deviates from it, where does one stop? One is afraid to plan a diverse population for fear he will get a parade of cultivars as diverse and unpredictable as the parades of people at the height of the Viet Nam and civil rights protest marches. I can understand this. I, too, went through a painful evolution in my thinking before I could accept diversity in principle.

What do I recommend now? What should our parade of varieties of the future be like? They should be like a parade of high school bands! This is another way of saying that people are diverse, and that diversity is one of the greatest things going for them.

I marvel when I see a high school band. Kids of all sizes, as long as they can get into a uniform that's invariably the wrong size, are eligible. But after all, members of a high school marching band only need four things in common: they need to start together, produce together, stop together, and look more or less alike from a respectable distance. They can be diverse for all other characteristics.

Similarly, all components of a cultivar must be planted at the same time, produce during the same growing season, be harvested the same day, and look more or less the same from a respectable distance.

Now I cannot seriously suggest that breeders consider making our cultivars as diverse as a high school band. But I do challenge them to heed the lessons of the indigenous population and, instead of asking, "How pure can I make this cultivar?" ask "How much diversity can I retain without due sacrifices in yield and quality?" just the slight change in philosophy represented by rephrasing this question, you see, makes a tremendous difference.

In closing, just for fun, let's assume that 100 identical people could be obtained and that they could be made into a band. Can you imagine what it would be like? They might all play base drums, or base horns, or trumpets, or trombones. Or they might all be drum majorettes. But one thing is certain, if the band were in a long parade on a hot afternoon and one member fainted of heat prostration, they all would.

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