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PERSPECTIVES

Reconsidering the Lessons Learned from the 1970 Southern Corn Leaf Blight Epidemic

Peter Balint-Kurti[1,2,†] and Jerald Pataky[3]

[1] Plant Science Research Unit, U.S. Department of Agriculture-Agricultural Research Service, Raleigh, NC 27695
[2] Department of Entomology and Plant Pathology, North Carolina State University, Box 7616, Raleigh, NC 27695
[3] Department of Crop Sciences, University of Illinois, Urbana, IL 61801
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Abstract

The southern corn leaf blight epidemic of 1970 caused estimated losses of about 16% for the U.S. corn crop, equivalent to about $8 billion in current terms. The epidemic was caused by the prevalence of Texas male sterile cytoplasm (*cms*-T), used to produce most of the hybrid corn seed planted that year, combined with the emergence of a novel race of the fungus *Cochliobolus heterostrophus* that was exquisitely virulent on *cms*-T corn. Remarkably, the epidemic lasted just a single year. This episode has often been portrayed in the literature and textbooks over the last 50 years as a catastrophic mistake perpetrated by corn breeders and seed companies of the time who did not understand or account for the dangers of crop genetic uniformity. In this perspective article, we aim to present an alternative interpretation of these events. First, we contend that, rather than being caused by a grievous error on the part of the corn breeding and seed industry, this epidemic was a particularly unfortunate, unusual, and unlucky consequence of a technological advancement intended to improve the efficiency of corn seed production for America's farmers. Second, we tell the story of the resolution of the epidemic as an example of timely, meticulously applied research in the public sector for the public good.

Keywords: Art Hooker, genetic uniformity, race T, southern leaf blight

Notes on Prior Literature and Nomenclature

The 1970 southern corn leaf blight (SCLB) epidemic has been reviewed in the scientific literature (Bruns 2017; Horsfall 1975; Ullstrup 1972) as well as in a number of textbooks. Although our purpose here is not a comprehensive retelling of the story of the epidemic, we recapitulate the main points and refer the reader back to these previous publications and to webinar by The American Phytopathological Society (https://www.apsnet.org/edcenter/resources/Webinars/Pages/CornBlight1970.aspx) for aspects we have missed. We have used a number of quotations throughout, and some nomenclature is not consistent. The Latin binomial for the causal agent of SCLB currently is *Cochliobolus heterostrophus* (anamorph, *Bipolaris maydis*). Previously, the anamorph stage of the fungus was called *Helminthosporium maydis*. The disease, SCLB, has also been referred to as Helminthosporium leaf spot. Likewise, Texas male sterile cytoplasm (*cms*-T) has been referred to as T-cytoplasm and T-*cms*. We use the terms *C. heterostrophus*, SCLB, and *cms*-T here, but in quoting from previous sources, we use the original language.

†Corresponding author: P. Balint-Kurti; Peter.Balint-Kurti@usda.gov

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A Brief History of the 1970 SCLB Epidemic: Changes in Technology Set the Stage for Unexpected Problems

Average annual corn yields in the United States have been estimated for nearly 160 years (Fig. 1). For more than 70 years from the time of the U.S. Civil War until World War II, average U.S. corn yields were less than 30 bushels/acre (bu/A) and did not vary appreciably. During this period, farmers primarily grew open-pollinated cultivars. Then, between 1937 and 1945, U.S. farmers switched from growing open-pollinated cultivars to newly available hybrid corn that had been developed in the 30 years following the discovery of heterosis in 1908. Coinciding with the adoption of hybrids, the average U.S. corn yield began to rise at a rate of about 1 to 2 bu/A/year, a trend that has continued through today. By the late 1960s, average yields were over 80 bu/A, nearly three times higher than yields from the era of open-pollinated cultivars. Hybrids were not the sole reason for this dramatic yield increase. Changes in pro-

duction practices, especially improved fertilization, higher plant populations, and mechanization, contributed significantly. Nevertheless, the continual development of new hybrids allowed growers to achieve maximum advantage from other changes in corn production. Throughout the world, hybrid corn was viewed as something of a miracle crop. With remarkable productivity and uniformity, hybrid corn was a prime example of the improvement science could bring to agriculture.

As with all new technologies, hybrid corn was not without its own set of new problems. Foremost among these was the production of hybrid seed. Besides multiple years of inbreeding to develop ear-parent (female) and pollen-parent (male) inbreds, seed production fields had to be isolated and arranged in a manner that allowed female inbreds to be pollinated only by pollen from male inbreds. Among other restrictions, this required removal of pollen-producing tassels from all female plants prior to pollen shed. Detasseling could be done by either of two methods: mechanically or by hand. Both had drawbacks that affected seed quality.

Mechanical detasseling used machinery that physically cut off the top third to half of the female inbred plant. In doing so, tassels were removed, but so was a considerable amount of leaf tissue, thus reducing the photosynthetic capacity of the plant. With a reduction in photosynthesis, fewer carbohydrates were produced to fill developing corn kernels, thus reducing seed quality. Smaller seed with reduced carbohydrate reserves was particularly problematic when hybrid seed was planted in weather conditions adverse to rapid germination and vigorous seedling growth.

Hand detasseling required large groups of laborers, usually rural teenagers, to physically pull tassels from the uppermost leaf whorl prior to pollen shed. Timing and thoroughness were critical to ensure that all seed produced was that of the hybrid. Any tassels that remained on female plants after hand detasseling could shed pollen, which would result in a portion of seed that would be the female inbred rather than the hybrid. Each seed of a female inbred was a contaminant in hybrid seed lots and caused a loss in yield when that hybrid seed was planted in growers' fields. As might be expected when employing thousands of teenagers for a hot, dirty, boring, and physically demanding job, quality control was a constant issue.

The problems associated with mechanical or hand detasseling were solved for the corn seed industry by academicians in a series of discoveries in the 1930s and 1940s. Cytoplasmic male sterility in corn was first discovered in the 1930s (Rhoades 1931). Subsequently, scientists in academia identified a source of male sterility discovered in Texas, known as Texas male sterile cytoplasm or *cms*-T (Rogers and Edwardson 1952), and nuclear genes (*Rf*) that restored male fertility in progeny of male sterile *cms*-T plants (Duvick 1956; Jones 1951). The process of producing hybrid corn seed with *cms* and *Rf* technology was then patented by an academician, Donald Jones, following his discovery of the *Rf* genes (Jones 1956). This technology allowed for the production of hybrid seed without the need for manual or mechanical detasseling. Extensive testing of *cms*-T undertaken by the hybrid seed industry in the 1950s, before the technology was widely used, produced little evidence that plants with *cms*-T and *Rf* genes differed from their normal cytoplasm counterparts (Duvick 1965). Thus, female inbred parents used for hybrid seed production were converted to *cms*-T; and *Rf* genes were backcrossed into male inbreds (Wych 1988).

By the mid-1960s, corn hybrids could be separated into three categories based on method of seed production: (i) normal cytoplasm hybrids (seed produced on a normal cytoplasm female inbred using the detasseling method), (ii) *cms*-T hybrids (seed produced on a *cms*-T female pollinated by a male carrying the *Rf* genes, thus producing a pollen fertile hybrid), and (iii) blends (seed produced on a *cms*-T female pollinated by a male without the *Rf* genes, thus producing a pollen infertile hybrid but blended with normal cytoplasm seed of the same hybrid to provide pollen in the grower's field). In 1970, about 85% of corn seed planted commercially in the United States was produced by one of the two methods using *cms*-T (Campbell 1970; Ullstrup 1972).

Conditions Conducive for an Epidemic on Hybrid Corn

Any time a plant disease increases or decreases appreciably in prevalence or severity, one or more of the elements of the disease triangle (i.e., host, pathogen, environment) has changed. In the case of SCLB in 1970, all three elements played a role.

During most of the twentieth century, SCLB was not considered to be a major problem of corn in the United States. Only rarely

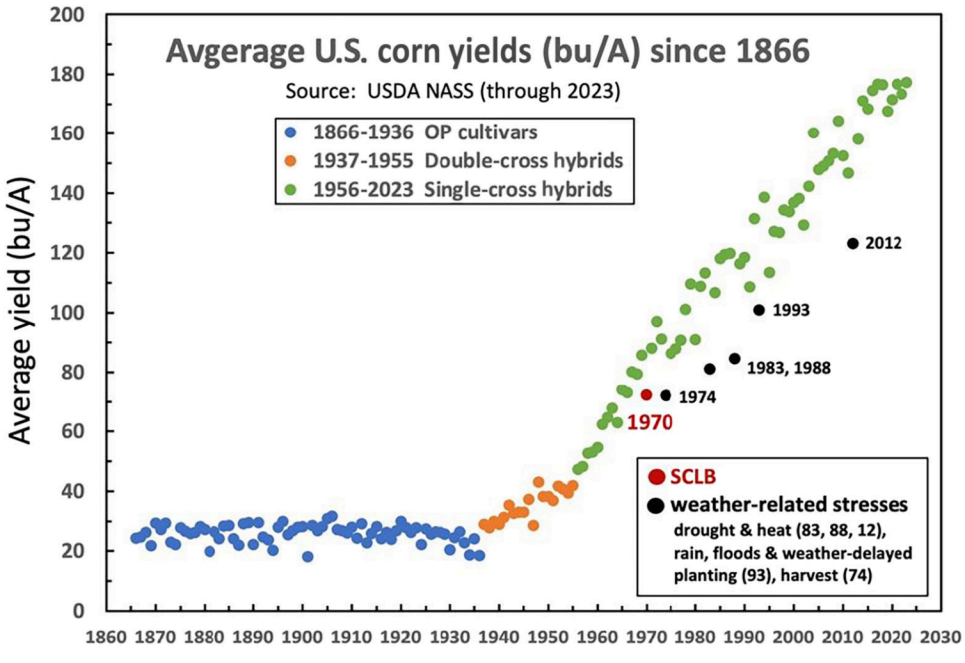

FIGURE 1
Average U.S. corn yields in bushels per acre (bu/A) since 1866, including periods in which the crop was planted with open-pollinated (OP) cultivars, double-cross hybrids, or single-cross hybrids. In the past 70 years, average yield was considerably lower than expected six times: five as a result of weather-related stresses and once in 1970 due to southern corn leaf blight (SCLB). These are indicated by black and red circles, respectively.

and in limited geographies did SCLB reach levels of severity that resulted in substantial damage to corn yield. Favored by warm, damp weather, the disease was endemic in the Southern United States and occasionally was found in the northern Corn Belt. During the period from April to August in 1970, average U.S. temperature and precipitation were within 1°F and 0.5 inches of historic means. These relatively normal weather conditions, by themselves, were unlikely to cause unusually severe levels of SCLB in 1970. However, humidity throughout the South and Midwest was reported as being favorable for a rapid increase of inocula (Wallin 1970), which created an adequately favorable environment for the epidemic development of SCLB considering the recently altered status of the host and the pathogen.

Unknown at the time, the mitochondrial gene *T-urf13* that caused male sterility in *cms*-T (Dewey et al. 1987; Levings 1990) also, unfortunately, caused extreme susceptibility to a previously unknown race of *C. heterostrophus* (Dewey et al. 1988). Race T, which the new race was labeled, produced a toxin to which *cms*-T plants were exquisitely sensitive (Lim and Hooker 1971; Yoder and Gracen 1975). The pervasive use of *cms*-T carrying the *T-urf13* gene and the sudden, widespread appearance of the toxin-producing *C. heterostrophus* race T in conjunction with conducive weather, created the conditions for a severe epidemic in the U.S. corn crop.

Although there were some indications prior to 1970 (described below) that *cms*-T conferred increased disease severity, the potential scale and economic impact of the problem were not appreciated prior to the 1970 growing season. During that summer and continuing through the harvest season, the U.S. agricultural community was near panic as SCLB appeared suddenly and severely in fields from Florida to Minnesota (Fig. 2). Ultimately, the U.S. corn harvest was nearly 700 million bushels lower than expected, which represented a yield reduction of about 16% with an economic value of more than $1 billion (about $8 billion current value). This lost income for America's farmers remains the greatest economic damage ever in a single season from a plant disease on an American row crop. In addition to concern over a $1 billion loss of on-farm revenue, the SCLB epidemic created doubts in the fall of 1970 about the long-term continued use of hybrid corn and gains in yield associated with hybrids. As Ullstrup (1972) noted, "During the winter, many dire predictions of failure were made for the 1971 corn crop. A report was circulated that normal-cytoplasm corn died soon after planting in soil taken from a field where the blight was severe in 1970."

As serious as it was in 1970, the epidemic lasted just a single season. The rapid resolution of the epidemic, described below, enhanced the scientific credibility of plant pathologists and their colleagues in the corn seed industry and is one of the best examples of agricultural scientists rising to the task of solving growers' problems.

Resolution of an Epidemic

In the most frequently cited accounts of the epidemic and in the subsequent summaries based on those accounts, the story of how the epidemic was resolved is not as well told as that of the occurrence of the epidemic itself. We feel that details about the research that resulted in the discovery of the cause and resolution of the epidemic deserve more attention because some of the most important lessons from the epidemic are based on that work.

There were two early warnings of disease susceptibility associated with *cms*-T. The first warning that SCLB might be problematic on *cms*-T corn came from observations made in the Philippines in the late 1950s and published in the early to mid-1960s (Aala 1964; Lantican et al. 1963; Mercado and Lantican 1961; Villareal and Lantican 1965). Villareal and Lantican (1965) wrote, "It is evident … that the cytosterile lines were … much more infected with the disease than their normal counterparts," and "it is hypothesized that the T-cytoplasm carries a factor which is responsible for the induction of extreme susceptibility to Helminthosporium leaf spot."

The Philippine observations were not ignored. In fact, they prompted additional testing in North America to compare *cms*-T and normal cytoplasm corn. In 1963, Art Hooker, the corn pathologist at the University of Illinois, tested the "Philippine hypothesis" by comparing *cms*-T and normal cytoplasm corn for reactions to *C. heterostrophus* using isolates from Illinois (Smith et al. 1970). He observed no differences. Likewise, in a 1965 review article on male sterile cytoplasm, Don Duvick, the director of research for Pioneer Hybrids, summarized various company trials of *cms*-T and normal cytoplasm corn, none of which identified differences in reactions to SCLB (Duvick 1965). Duvick suggested, "It may be that the increased susceptibility in the Philippines is a secondary effect of reduced plant vigor, accentuated by the Philippine environment." Unfortunately, Duvick's hypothesis was incorrect. In fact, no one in North America at that time proposed the correct explanation for the Philippine observations, that is, a new race of *C. heterostrophus*, distinct from the predominant race in North America, was re-

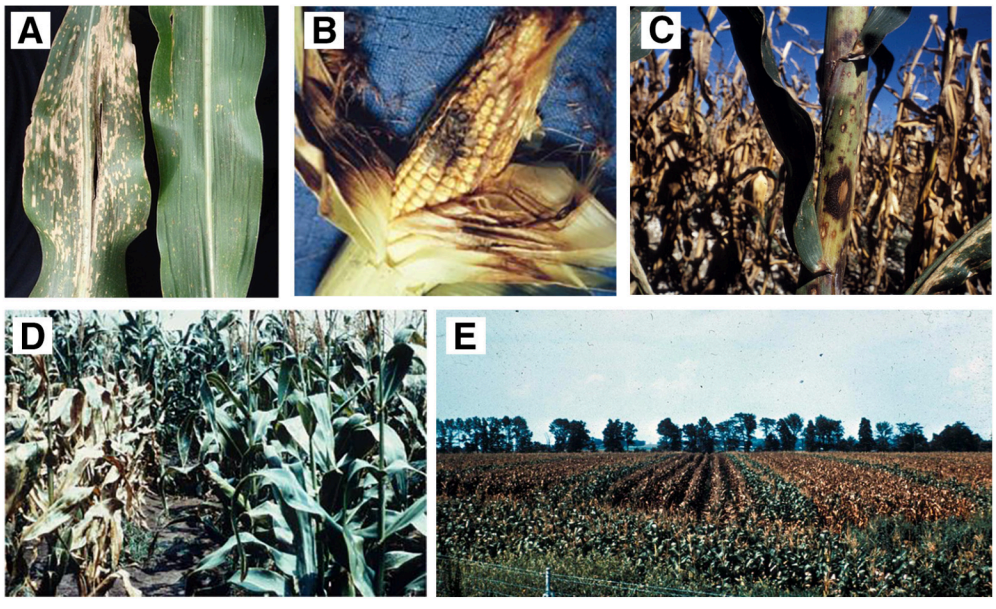

FIGURE 2
Symptoms of southern corn leaf blight caused by *Cochliobolus heterostrophus* race T: **A,** leaf infections on plants with *cms*-T (left) and normal (right) cytoplasm; **B,** ear infection of *cms*-T corn; **C,** leaf sheath infection of *cms*-T corn; **D,** female inbred W64a *cms-T*, left, and normal cytoplasm W64a, right; and **E,** seed production field in 1970: *cms*-T female rows susceptible, normal cytoplasm male rows resistant.

sponsible for the increased susceptibility of *cms*-T material in the Philippines.

The second warning occurred in 1967 when Deane Arny, a pathologist at the University of Wisconsin, observed unusually severe yellow leaf blight (YLB), caused by *Phyllosticta maydis*, on *cms*-T corn in Wisconsin. Originally identified from Native American flint corn in southern Illinois in 1930 (Stout 1930), YLB occurred rarely and was of no importance on corn previously. Subsequently, Arny and pathologists at Pennsylvania State University demonstrated that corn inbreds and hybrids were predominantly resistant to YLB but that "resistance may be totally or partially lost upon conversion to Texas male-sterile cytoplasm" (Arny et al. 1970; Ayers et al. 1970). This unusual susceptibility of *cms*-T to YLB prompted Hooker to retest the Philippine hypothesis in 1969, once again using isolates of *C. heterostrophus* from central Illinois. As before, Hooker observed no difference in reactions of *cms*-T and normal cytoplasm corn to SCLB (Smith et al. 1970).

Late in the 1969 growing season, unusually severe, localized infections of SCLB were seen in regions of southern Iowa, Indiana, and Illinois (Ullstrup 1972). Art Hooker was one of the pathologists to make these observations. As Hooker drove across central Illinois to teach an off-campus course in western Illinois, he stopped at several fields to collect leaf samples (D. R. Smith, *personal communication*). Early in 1970, using isolates from those collections, Hooker and his associates at Illinois, David Smith and Sung Lim (Fig. 3), carried out an experiment to retest the Philippine hypothesis, the third such test by Hooker since 1963. Upon observing greater susceptibility of *cms*-T corn compared with normal cytoplasm corn, Hooker, Smith, and Lim expanded the experiments to include additional sources of cytoplasmic male sterility, additional corn inbred and hybrid lines, and additional isolates of *C. heterostrophus*. They also began experiments to test why isolates might differ in their ability to infect *cms*-T and normal cytoplasm corn. These conceptually simple but well-defined and carefully conducted experiments not only identified the cause of the yet-to-occur SCLB epidemic but also outlined its solution and identified a toxin produced by the fungus that accounted for the unique susceptibility of *cms*-T. As noted above, Hooker had tested *cms*-T and normal cytoplasm lines twice before and observed no differences in SCLB susceptibility. The crucial difference in the new trial was, of course, that they now had isolated the toxin-producing race T from the leaf samples collected in 1969.

As the epidemic developed during the 1970 growing season, only three articles on SCLB appeared in the scientific literature. The first was authored by a pathologist, Gordon Scheifele, and two corn breeders, Wayne Whitehead and Cledith Rowe, with PAG Seeds. Scheifele had just recently completed an MS degree at Pennsylvania State University, working on *cms*-T and YLB before taking a job with PAG in Champaign, Illinois. Upon joining PAG, he approached Hooker about the possibilities of simultaneously working on a Ph.D. degree at Illinois. As a part-time Ph.D. student, Scheifele was well aware of the work being done by Hooker, Smith, and Lim with the new isolates of *C. heterostrophus* (D. R. Smith, *personal communication*). Scheifele and his co-authors reported field observations from PAG nurseries in 1969 showing increased susceptibility of *cms*-T maize to SCLB after anthesis (Scheifele et al. 1970). They wrote that "an apparent relationship between the Texas male sterile cytoplasm and susceptibility to *Helminthosporium maydis* … was observed in inbreds and hybrids of maize"; however, no additional experiments tested the hypothesis proposed from these observations.

FIGURE 3
Plant pathologists who in the spring of 1970 experimentally identified race T of *Cochliobolus heterostrophus* and cautioned of the potential for a southern corn leaf blight epidemic on *cms*-T corn. **A,** Art Hooker; **B,** Dave Smith (left) and Sung Lim (right).

The other two articles appearing in the scientific literature as the epidemic occurred were based on greenhouse experiments completed by Hooker, Smith, and Lim early in 1970 using the new isolates. They described the increased susceptibility of *cms*-T to SCLB (Hooker et al. 1970) and the existence of a new race of *C. heterostrophus*, which they named race T, that was responsible for this susceptibility (Smith et al. 1970). Additional work on the discovery and inheritance of T-toxin production in race T was completed as the epidemic occurred and reported the following winter (Lim and Hooker 1971). An assay using T-toxin was also developed to differentiate seed with *cms*-T and normal cytoplasm (Lim et al. 1971). The T-toxin test was particularly useful in determining the percentage of *cms*-T in seed lots that were blends of the two cytoplasms, which comprised about 36% of the seed available for the 1971 growing season (Campbell 1970).

The first of the two 1970 papers from the group at Illinois noted that "inbred lines with T or P cytoplasm for male sterility were susceptible to *Helminthosporium maydis*" (Hooker et al. 1970). Prophetically, writing in the spring of 1970, they also noted that "although many different inbred lines are used, thus giving genetic variability among the different hybrids in the US, the cytoplasm of the American corn crop is essentially uniform. **A majority of America's most valuable crop is now uniformly susceptible and exposed to a pathogen capable of developing in epiphytotic proportions** [bold emphasis added]." At the time, Hooker and his colleagues could not have known the severity to which the SCLB epidemic would develop. They concluded by proposing the eventual solution, saying, "Hopefully, genetic resistance to *H. maydis* can be found … **Other alternatives are the production of seed on plants with regular cytoplasm by the detasseling method** [bold emphasis added]."

Differential races of the SCLB pathogen were identified in the second article (Smith et al. 1970). Smith et al. (1970) reported the following: "Two physiologic races of *Helminthosporium maydis* were distinguished … Seedlings of inbreds with T or P cytoplasm for male sterility inoculated with Race T were susceptible. All seedlings … inoculated with Race O were resistant." They went on to explain the negative results from previous tests of the Philippine hypothesis: "The existence of two races explains the inability to distinguish … corn lines with T cytoplasm from their regular counterparts in Illinois prior to 1969."

As the full impact of the epidemic was becoming evident in the summer of 1970, this set of rather simple but well-executed studies were published presenting a clear, experimentally based, scientifically sound explanation of what was happening in America's corn fields. The solution to the epidemic, which of course was to stop using *cms*-T for hybrid corn seed production, was also clearly evident from those studies. The clarity and timeliness of this research provided scientifically credible evidence of the cause of the blight. Corn seed companies realized the need to immediately begin producing normal cytoplasm hybrids to avoid another growing season marred by a catastrophic epidemic.

Fortunately, corn seed companies had a sufficient supply of seed of normal cytoplasm female inbreds to quickly facilitate the

conversion of the corn crop back to normal cytoplasm hybrids. Seed production in South America and Hawaii that winter increased the supply of normal cytoplasm hybrids for the areas that were most vulnerable to SCLB, as well as increasing the seed of normal cytoplasm female inbreds to be used in 1971 to produce the hybrid seed crop for 1972. Corn production recovered in 1971 due both to the normal cytoplasm seed that had been expeditiously produced and cool weather conditions that were less favorable for SCLB. By 1972, nearly all hybrid corn seed contained normal cytoplasm, the epidemic was truly over, and average U.S. corn yields attained a new record high of 97 bu/A (Fig. 1).

The Epidemic Could Have Been Prolonged

The method by which the seed industry deployed *cms*-T technology allowed for the rapid resolution of the epidemic without a loss in the genetic gains for yield that had been achieved by corn breeders during the years *cms*-T was being used. Because *cms*-T could not be incorporated directly into breeding populations without causing male sterility, it was backcrossed into elite female inbred lines. Normal cytoplasm versions of *cms*-T female inbreds were maintained to provide pollen for production of seed of *cms*-T females and were therefore available to facilitate the rapid conversion back to normal cytoplasm hybrids. Had the susceptibility to race T been caused by pleiotropy in a gene that did not require maintenance of the original normal versions of lines and populations, fixing the problem may have taken considerably longer.

Interestingly, commercial corn breeding programs currently incorporate genetically modified organism (GMO) traits in a similar manner. Rather than convert entire breeding programs to GMOs, massive backcrossing programs are used, at considerable costs, to convert elite inbred lines for GMO traits. By doing so, seed companies are exercising caution and mitigating a degree of risk that might be associated with unforeseen problems arising from new GMO technology.

Societal Consequences of the Epidemic

Beyond its economic impact, the epidemic had a profound societal and cultural impact. It was the subject of numerous media reports in the national broadcast news and printed press. It remained a dominant concern of the U.S. agricultural community well into the 1971 growing season. In the fall of 1970 when the ramifications of the epidemic were evident, more than $10 million was appropriated by the U.S. government specifically for the study of SCLB. That funding supported multiple research and extension projects that produced a thorough, scientific understanding and explanation of the epidemic, prompting the popular concept that the solution to the epidemic resulted from the cooperative effort of many scientists. Although some of the funded projects made significant scientific contributions, few, if any, impacted the rapid solution to the epidemic. The cause of the epidemic was well defined by the research completed in the spring of 1970 before the epidemic occurred, and the solution was in the process of being implemented by the hybrid corn seed industry before any federal funding was allocated.

Following the epidemic, a National Academy of Sciences select committee was created to examine the risks associated with genetic uniformity. Their report, titled "Genetic Vulnerability of Major Crops" (National Research Council 1972) included the following conclusion: "The key lesson of 1970 is that genetic uniformity is the basis of vulnerability to epidemics." In the past five decades, this conclusion and other aspects of the 1970 SCLB epidemic have been used as justification for continued funding of activities that enable the diversification of the genetic bases of our crops and for funding of programs that aim to help us understand and ameliorate future disease problems. This work entails projects such as plant introduction stations, seed storage facilities, pathogen collections, national and regional disease monitoring efforts, and quarantine programs.

Interestingly, in discussing genetic uniformity, the National Academies report also included the following prophetic passage: "Clearly the market wants uniformity. If one breeder or one farmer fails to provide it, the market will turn to another that will. The irony is that if this uniformity encourages an epidemic, the scientist, not the market, tends to receive the blame." In fact, as noted below, placement of blame on corn breeders and the corn seed industry has occurred repeatedly in several recollections of the 1970 SCLB epidemic.

The Misleading Lesson Most Frequently Cited from the Epidemic

Probably the most influential account of the epidemic in the scientific literature was written by one of the most prominent corn pathologists of the time, Arnold Ullstrup. Ullstrup's article, titled "The impacts of the southern corn leaf blight epidemic of 1970-1971" (Ullstrup 1972), provided a history of the development of *cms*-T technology, the development of the epidemic, and the panic that gripped the agricultural community in its wake. The reader is directed to this work for a detailed description of these issues.

Perhaps surprisingly, Ullstrup did not emphasize the important role of the research published in 1970, in his own discipline of plant pathology, which helped bring the epidemic to a rapid end, although he does cite these papers. Rather, he chose to emphasize what he evidently saw as mistakes that were made by the corn breeding and seed industry, ending the paper with the following statements: "Never again should a major cultivated species be molded into such uniformity that it is so universally vulnerable to attack by a pathogen, an insect, or environmental stress. Diversity must be maintained in both the genetic and cytoplasmic constitution of all important crop species" (Ullstrup 1972).

This portentous passage, in particular, has, we believe, colored some of the subsequent commentary regarding the epidemic. It has been cited verbatim a number of times in the scientific literature and the popular press, including in the educational materials offered by The American Phytopathological Society. It has helped promulgate the idea that the epidemic was an avoidable consequence of poor planning and complacency by corn breeders and seed companies. The need to avoid genetic uniformity in favor of genetic diversity has become the key take-home lesson from the SCLB epidemic. For example, prominent textbooks make this point when discussing the epidemic:

> "The big mistake was to develop the most popular cultivars of the period using the so-called Texas male-sterile cytoplasm." *Plant Pathology*: *Principles and Practice* (Jones 1987)

> "This corn disease is an excellent example of the problems that can arise from genetic uniformity and a narrow view of an agricultural ecosystem … Genetic uniformity was the first step on the road to disaster." *Hungry Planet* (Schumann and D'Arcy 2012)

This view is also found in articles published recently in the scientific press:

> "This serves as a warning to the seed production business never to purify the genetics of our crops to such an extent as this again and to preserve genetic diversity." *Agronomy Journal* (Bruns 2017)

> "It is also a textbook example of what not to do" and "a lesson we should not have been forced to learn in 1970." *Phytopathology News* (Harveson 2023)

Although it is undeniable that the 1970 SCLB epidemic would not have occurred without the genetic uniformity created by the

widespread use of *cms*-T, it does not logically follow that genetic uniformity for other traits is likely to result in similar epidemics or that maintaining a level of genetic diversity at all loci that would be necessary to lower the risk of an unexpected epidemic is worth the costs of decreased levels of crop productivity that accompany that diversity. Stated differently, who can predict which widely deployed, beneficial allele, if any, might be pleiotropic for susceptibility to a new race of a previously minor pathogen, such as what occurred with *T-urf13* and *C. heterostrophus* race T?

An Alternative Consideration of the Benefits and Risks of Genetic Uniformity

We should first consider that phenotypic uniformity for important agronomic traits is a fundamental basis of modern agricultural production. Uniformity for traits such as yield, standability, flowering, grain or fruit quality, and resistance to environmental and biological stresses allows farmers to plant, manage, and harvest crops in an efficient manner. Phenotypic uniformity, of course, implies a certain degree of genetic uniformity, and this in turn implies a certain vulnerability to disease epidemics, as was noted in the National Academies report (National Research Council 1972) and repeated many times since.

We should also consider that the SCLB epidemic was caused by genetic near-uniformity with respect to a single cytoplasmic gene, *T-urf13*, that resides in *cms*-T, rather than by uniformity of the entire nuclear genome, as has been suggested in some publications. Although it is hard to determine the breadth of the genetic base of the commercial U.S. maize crop in 1970, it appears to have been substantial (Darrah and Zuber 1986; Smith et al. 1985a, b). As Hooker noted in 1970, "Many different inbred lines are used, thus giving genetic variability among the different hybrids in the US" (Hooker et al. 1970). The *T-urf13* gene was introduced as a technological advancement, cytoplasmic male sterility, that facilitated efficient production of hybrid corn seed and, in doing so, delivered higher quality seed to growers. At the time of the development and introduction of *cms*-T, as Ullstrup (1972) noted, "It obviously could not be foreseen that this type of cytoplasm carried with it hyper-susceptibility to a then unknown physiologic race of *Helminthosporium maydis*."

Genetic uniformity is prevalent in crops grown in modern agriculture. There are numerous cases of the successful adoption of new genetic technology in the form of the introduction of favorable alleles widely into a crop species. Perhaps the most celebrated is the introduction into wheat varieties of mutant *Rht* alleles (especially *RhtB1* and *RhbD1*) conferring gibberellin insensitivity and dwarfing (Silverstone and Sun 2000). The "green revolution" was dependent upon these alleles, and they are still widely used today (Pearce 2021). Ironically, for his part in this work, Norman Borlaug was awarded the Nobel Peace Prize in 1970, the year that genetic uniformity for *T-urf13* contributed to the SCLB epidemic. Other examples of genetic uniformity come from sorghum, including the *ma1* allele that eliminates the photoperiodic response (W. Rooney, *personal communication*), the *dw2* and *dw3* dwarfing alleles in grain sorghum (Brown et al. 2008), and the male sterility system based on A1 cytoplasm and *Rf1* and *Rf2* nuclear restorer genes (Bohra et al. 2016).

Interesting examples of allelic uniformity also occur in corn. All sweet corn hybrids grown in North America are homozygous for one of two recessive, endosperm mutation genes, either *sugary-1* (normal sugary sweet corn) or *shrunken-2* (supersweet sweet corn) that inhibit conversion of sugars to starch in the developing kernel, thus conferring sweetness. In another case, genetic uniformity in corn is actually essential for avoiding a catastrophic disease. Resistance to northern corn leaf spot caused by *Cochliobolus carbonum* race 1 is conferred by a nearly ubiquitous presence of the gene *Hm1* in corn, as well as in other grass crops (Sindhu et al. 2008). If mutations occur in the dominant *Hm1* gene, infection by the toxin-producing *C. carbonum* race 1 can be more severe than in the SCLB epidemic of 1970, as has been seen occasionally on female inbred lines in seed production fields (Fig. 4).

These examples are illustrative but merely representative of a much larger category of genetic uniformity within cultivated crops. In every case, except for *T-urf13*, these predominant alleles have not caused catastrophic susceptibility to a novel pathogen. Therefore, it seems to us that the susceptibility of *cms*-T corn to *C. heterostrophus* race T was an unforeseeable and unlucky consequence of the introduction of new agricultural technology to improve the method of producing hybrid corn seed. We argue that this approach of discovering and incorporating genes and technologies that are thought to be beneficial is one of the major factors responsible for current high levels of agricultural productivity. It is an approach that has, rightly, continued, despite the 1970 epidemic.

Additional Thoughts on Genetic Diversity and Reducing the Risk of Disease Epidemics

Genetic diversity plays an important role in reducing the risk of epidemics in at least two different, but related, ways. Deploying a genetically diverse group of cultivars in commercial crops theoreti-

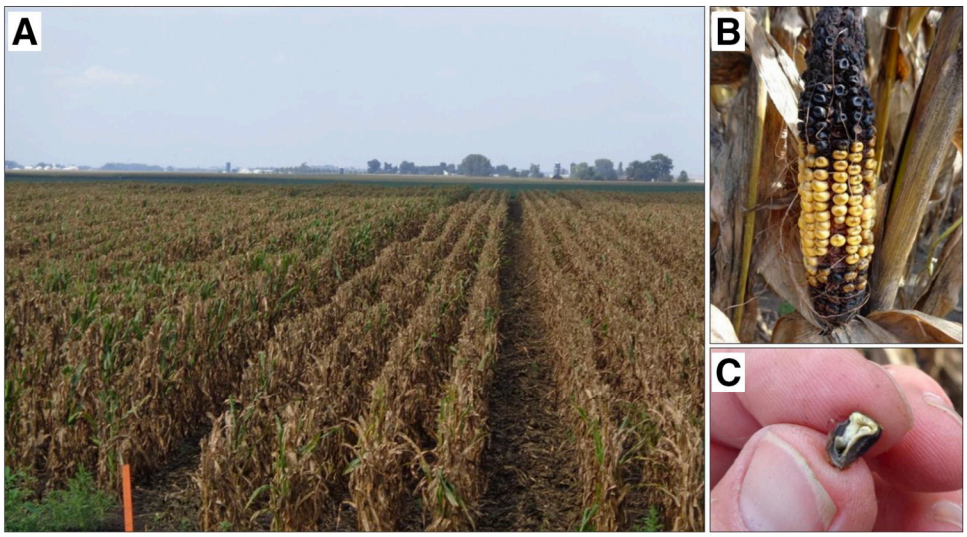

FIGURE 4
Fields in Farmer City, IL, in 2014 in which lines homozygous for a mutation in Hm1 had been inadvertently planted. The disease developed to catastrophic levels on all the aboveground organs, including the **A,** leaves and stem and the **B,** ears, **C,** causing the loss of endosperm in individual kernels.

cally reduces the impact of epidemics by increasing the probability that some cultivars will not be as severely infected as others. Alternatively, maintaining the genetic diversity of a crop species through various types of germplasm collections serves as a resource from which to identify and deploy genes conveying disease resistance to pathogens that become prevalent. SCLB and the 1970 epidemic provide examples of both.

Many different hybrids were grown in 1970, thus giving rise to genetic diversity in the 1970 corn crop in spite of widespread uniformity for the *T-urf13* cytoplasmic gene. Several hybrids that were grown from blended seed of *cms*-T and normal cytoplasm versions of a hybrid were evaluated in 1970 for severity of SCLB on their *cms*-T and normal cytoplasm components. SCLB ratings ranged from 2.5 to 5 (0 to 5 scale) among *cms*-T components of various hybrids, whereas only a few lesions were seen on the lower leaves of the normal cytoplasm components of those hybrids (Josephson et al. 1971). Likewise, yield reductions on *cms*-T components of the blends ranged from 30% to 76% among various hybrids when ear weights of *cms*-T and normal cytoplasm components were compared within hybrids. Thus, even though all *cms*-T components of hybrids were susceptible to *C. heterostrophus* race T, a range of susceptible reactions occurred among the many different *cms*-T hybrids being grown.

Germplasm collections also served as a source of SCLB resistance. In the years immediately prior to the epidemic, chlorotic lesion resistance to SCLB was identified from an inbred line, 024-2-4, derived from Kenyan population AFRO.P.59.289 (Craig and Daniel-kalio 1968). This resistance was inherited as a single recessive gene, *rhm*, or as two closely linked recessive genes (Chang and Peterson 1995; Craig and Fajemisin 1969; Smith and Hooker 1973). Subsequently, the *rhm* gene was not widely deployed because, as a recessive, it would need to be incorporated into both male and female inbreds and/or breeding populations from which male and female inbreds were derived. Also, improved levels of resistance were unnecessary to prevent severe SCLB on most normal cytoplasm hybrids. As Hooker (1977) noted when discussing breeding and research efforts devoted to a specific disease, "There is little merit in doing work where work is not needed. Work is better expended where achievements can be expected."

Ideally, these two aspects of genetic diversity, identifying multiple sources of disease resistance and deploying that resistance in genetically diverse cultivars grown as commercial crops, can be combined to provide long-term control of prevalent, yield-reducing diseases. In fact, this combination is most likely the basis for durable, polygenic resistance that is prevalent throughout the corn crop today (Yang et al. 2017). The multiple sources of polygenic resistance that are currently deployed were brought together almost exclusively through phenotypic selection. As genome-wide selection and other advances in molecular genetics introduce new technology and methods to select for resistance, it will be interesting to observe if resistance eventually becomes more genetically uniform and less durable. If these scientific advances that change technology once again create a new disease problem, rather than place blame on molecular geneticists and the corn seed industry, it should be recognized that encountering and solving new problems is simply part of the process of making progress.

Conclusions

It is, of course, inarguable that the immediate cause of the 1970 SCLB epidemic was genetic uniformity with respect to a cytoplasmic gene that conferred susceptibility to an emerging race of *C. heterostrophus*. Whenever a disaster such as this occurs, it is a natural human instinct to search for an entity on which to place blame and to declare "never again." However, we believe that, in this case, there is no blame to be ascribed; rather, the research and measures taken to address the epidemic promptly should be celebrated.

Rather than being a "textbook example of what not to do" (Harveson 2023), the process of scientific discovery that led to the introduction of *cms*-T was a case of research providing a new technology that reasonably would be expected to facilitate more efficient food production. The process of incorporating new scientific discoveries into agriculture has proved successful many times before and since, and it has been one of the main reasons for the spectacular yield gains and abundance of food and fiber that have been achieved since the beginning of the twentieth century. Specifically for corn, this process has been instrumental in increasing the average U.S. yield from less than 30 bu/A to nearly 180 bu/A. Unfortunately, in the case of *cms*-T, there was an unforeseeable problem associated with the technology. In other words, rather than saying "never again," we should continue to use the approach of introducing new technologies that have the potential to advance agriculture in the manner that has been overwhelmingly successful. In doing so, we do of course need to recognize that sometimes, despite careful testing and monitoring, bad things happen that are unforeseeable. Therefore, we need to be diligent in monitoring for unintended, deleterious consequences associated with change.

So, we believe that the, often repeated, lesson that we should never again allow our crops to be genetically uniform is an incorrect conclusion from the epidemic. However, several other lessons can be derived from this episode.

- Widespread change and new technologies can have unintended consequences.

Any widely adopted new technology, agricultural or otherwise, may bring with it unintended, unexpected secondary consequences that can be advantageous, neutral, or deleterious. As Ayers and others noted in follow-up work on *cms*-T and YLB, "Yellow leaf blight and southern leaf blight clearly illustrate the influence of changing technology on the occurrence and severity of plant diseases" (Castor et al. 1977).

Another interesting example of unintended consequences from adoption of new methods concerns Diplodia diseases of corn: During the first half of the twentieth century, Diploida was the most prevalent stalk rot of corn, and Diplodia ear rot was also common (Koehler and Boewe 1957). Following World War II, farmers in the Corn Belt began to adopt the practice of fall moldboard plowing, which lifts and turns the soil after harvest, thus burying crop residue in the process. An advantageous, unintended consequence of moldboard plowing was the decrease in incidence of Diplodia ear and stalk rot because the causal fungus (*Stenocarpella maydis*) has no other host and overwinters on corn debris (Hooker and White 1976; Munkvold and White 2016). However, moldboard plowing also had the unintended, deleterious consequence of increasing soil erosion. The recognition of this problem led to the adoption of conservation tillage beginning in the mid-1970s and a subsequent reemergence of Diplodia diseases, as well as increases in other diseases, such as gray leaf spot, whose causal agents overwinter on debris (Anderson and White 1987; Latterell and Rossi 1983).

As this example and the SCLB epidemic illustrate, we must continually address and solve unexpected problems that may arise with new technology and methods—and then move on. However, we would be impeding progress if we failed, after adequate testing, to use a potentially advantageous new technology, genetic or otherwise, in fear that it may introduce some previously unforeseen problem.

- "Freedom from epidemics is purchased at the price of vigilance" (National Research Council 1972).

This statement from the National Academy of Sciences report on the SCLB epidemic explicitly describes one of the most important lessons to be learned from an examination of the scientific work that led to the rapid resolution of the 1970 epidemic. The Philippine hypothesis that *cms*-T carried factors conveying extreme suscepti-

bility to SCLB was tested multiple times by Hooker, Duvick, and possibly others. Using isolates of *C. heterostrophus* that were prevalent in North America throughout most of the 1960s, the hypothesis was rejected, but retesting the hypothesis with new isolates from 1969 that were race T resulted in a definitive explanation of the cause of the epidemic and the potential solution. Questions of importance, such as this, are worthy of repeated testing exemplified by this degree of watchfulness.

Vigilance is a never-ending job. Yesterday's, today's, and tomorrow's problems are unlikely to be the same. As an example of such change, consider that three of the most important corn diseases in North America in the past two decades, anthracnose stalk rot, gray leaf spot, and Goss's wilt (Mueller et al. 2020), were of little or no importance in 1970 when the SCLB epidemic occurred. Similarly, some of the most important corn diseases two decades from now are unlikely to be the same as those from two decades ago. Change should be anticipated. To solve problems quickly, we must be on the lookout for the initial occurrence of new problems and design the appropriate experiments to determine their cause, potential impact, and ultimate solutions.

- Open communication between scientists in the public and private sectors must be maintained and facilitated.

In responding to the SCLB epidemic, breeders and seedsmen in the private sector acted quickly on information provided by public sector researchers, partly because previous interactions among those groups established a high degree of credibility and trust. In the decades prior to the SCLB epidemic, corn breeders and pathologists in academia and industry generally shared a common sense of purpose. Several inbred lines used widely in commercial hybrids or as parents in commercial breeding populations were developed in the public sector at various agricultural experiment stations (e.g., A619, A632, B14, B37, C103, C123, Mo17, N28, Oh43, Va26, and W64a). Likewise, many corn pathology programs in the public sector were focused on disease resistance. Ideas and information often were exchanged at meetings such as the American Seed Trade Association Corn and Sorghum Conference, the Illinois Corn Breeders' School, and the Maize Genetics Conference. In fact, one of the earliest reports of the susceptibility of *cms*-T to SCLB in the Philippines appeared in the Maize Genetics Cooperative Newsletter (Lantican et al. 1963). Hooker was highly regarded among commercial breeders and seedsmen for discovering the *Ht1* gene conveying resistance to northern corn leaf blight (Hooker 1963) and for backcrossing that gene into all the public inbreds listed above, as well as several others, prior to releasing those lines to the private sector for commercial use. Interestingly, many present-day commercial hybrids still carry the *Ht1* gene despite virulence against it becoming prevalent in the 1980s and 1990s (Leonard et al. 1989; Smith and Kinsey 1980; Thakur et al. 1989; Turner and Johnson 1980) and little or no selection specifically for this gene in the past three decades.

Today, geneticists, breeders, and pathologists in the public and private sectors still interact, to a degree, at professional meetings and elsewhere, but their specific objectives and overall goals are less aligned than those of 50 or more years ago. Little, if any, public germplasm is used by commercial breeders, and the private sector rarely looks to the public sector for sources of disease resistance. Scientists in the private sector are reluctant to share information that may impart an advantage over their competitors in the marketplace. Research in the public sector often is not undertaken with the goal that it may be of applied, practical use to colleagues in the private sector. Developing ways to facilitate greater communication, common goals, and sharing of ideas and information about potential disease threats among scientists in both sectors will help in reducing the risk of and finding solutions to future disease problems.

- Monitoring for potential new disease problems requires a global perspective and coordinated effort.

The exact origin of *C. heterostrophus* race T in North America is unknown. In the fall of 1970, some pathologists speculated that race T was present in the United States as early as the mid-1950s (Nelson et al. 1970), although subsequent evidence indicates this is unlikely (Leonard 1971). Regardless, the pathogen clearly was present in the Philippines a decade before it caused an epidemic in the United States. A few months after that epidemic, *cms*-T hybrids were severely infected by race T in South America, particularly in Brazil. Race T was global in its occurrence.

A puzzling aspect of the SCLB epidemic is why no one speculated in the early 1960s that a new race of *C. heterostrophus* might account for the occurrence of severe SCLB on *cms*-T germplasm in the Philippines. Questions of importance such as this might have been asked and answered if, at that time, there existed a well-coordinated, cooperative global effort to monitor for potential new problems. A global monitoring system of this sort involving the public and private sectors with adequate government funding may be even more important today, as global commerce and travel increase the potential for dissemination of pathogens and pests.

- Genetic uniformity is, to some extent, inherent and inevitable in modern agriculture, leaving our crops vulnerable to epidemics that will occur in an unpredictable fashion. We should continue to take measures to ameliorate these risks.

Although genetic diversity within a crop may be desirable with respect to making the crop more resilient to unexpected stresses, a substantial degree of phenotypic, and therefore genetic, uniformity is an inevitable consequence of the modern agricultural system. In fact, phenotypic selection for favorable alleles at specific loci played a substantial role in the domestication of crops. Rather than say "never again should a major cultivated species be molded into such uniformity," a more accurate statement might be "never could we have modern agriculture without a degree of genetic uniformity within our major crops." This uniformity has allowed for the enormous increases in production seen in the modern era. Genetic uniformity does make crops more vulnerable to disease and pest epidemics, but we should be careful not to fall into thinking that, for this reason, genetic uniformity is an unalloyed negative. We should strive to maintain sufficient genetic diversity among cultivars being grown at the local and regional levels to minimize the risks of epidemics and other biological and environmental stresses. However, this is not straightforward to achieve and, in some cases, as with some of the examples listed above, it is not even beneficial. In situations in which multiple breeders in the public or private sector are intensively selecting on the same set of traits, a certain amount of genetic uniformity at certain loci seems to be an inevitable consequence. Although it is not realistic to expect we can prevent agricultural epidemics from occurring, we should ensure we have a robust infrastructure that enables us to identify and respond to them rapidly.

- The rapid resolution of the SCLB epidemic should be regarded as a triumph for agricultural science and for partnership between the commercial and academic sectors.

The rapid resolution of the SCLB epidemic was the result of keen observation and timely research completed by a few knowledgeable plant pathologists in academia who followed the scientific method. The credibility of their work led to the rapid response of the corn seed industry required to resolve the epidemic. Moreover, their ability to recognize significant phenomena and to understand the potential importance of those observations led them to ask the correct question more than once. This level of expertise, vigilance, and diligence is indicative of the attributes necessary in the scientists who will be responsible for rapidly resolving major disease problems that are nearly certain to arise again in the future.

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