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Southern Corn Leaf Blight: A Story Worth Retelling

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ABSTRACT

The southern corn (*Zea mays* L.) leaf blight (SCLB) epidemic of 1970–1971 was one of the most costly disease outbreaks to affect North American agriculture, destroying 15% of the crop at a cost of US$1.0 billion (≥$6.0 billion by 2015 standards.). It resulted from an over reliance on cytoplasmic Texas male sterile (cms-T) lines in hybrid seed production and a natural mutation of a race of SCLB *Bipolar maydis* that for years was seldom of economic importance. This mutation discovered in the Philippines in 1961 first appeared in the Corn Belt in 1969, damaging not only leaves, but stalks, ears, and developing kernels of hybrids containing cms-T genetics. A favorable environment, combined with >85% of the hybrids grown being of cms-T genetics set the stage for an epidemic. The cms-T was discontinued in 1971 and hybrid seed production returned to using detasseling for the female parent. This serves as 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.

Core Ideas

- A history of corn leaf blight and its host.
- A synopsis of southern corn leaf blight.
- Lessons for the future.

There are few active practitioners of our profession left who recall first-hand the harsh lesson Mother Nature inflicted on agronomists and North American agriculture in 1970 and 1971. The maize disease SCLB (*Bipolar maydis* T.) (earlier known as *Helminthosporium maydis* T.) reached epidemic proportions in the United States and southern Canada, destroying an estimated 15% of the corn crop at an estimated loss of $1.0 billion at that time (Ullstrup, 1972). It was not uncommon for some growers to suffer 80 to 100% losses and some multiple county regions in the Corn Belt to have average losses ranging between 35 and 50% of their crop. In some southern states where the disease first appeared, relative losses were also high with those in Mississippi estimated to be between 30 and 40% (Moore, 1970a.)

This "perfect storm" of a plant disease epidemic was a prime example of the Disease Triangle coming together in a devastating way; a relatively new race of an existing disease being introduced, a host crop of which >85% had a vulnerable common genetic background, and environmental conditions exceptionally favorable for infection and growth of the pathogen. Obviously humans have little or no control over the evolution of pathogenic organisms. Like any living thing, fungi along with all other pests, will find a way to survive and propagate. Paraphrasing the character Dr. Ian Malcom played by Jeff Goldblum in the movie "Jurassic Park" when informed that all of the dinosaurs in the park were female and through the controlled cloning program would be denied natural reproduction, Dr. Malcom warned "You can't deny a species the ability to reproduce. Life will find a way".

Corn has always been a major feed grain in the United States since colonial times, with >80% going for livestock feed and the remainder for food and industrial products. The United States in 1969 was exporting about 15.5 Tg of corn overseas with a value of ≃ $690 million. Currently exports are more than 56 Tg valued at ≃ $8.7 billion (Index Mundi, 2016). The lead up to the 1970–1971 SCLB race-T epidemic had its roots with the commercialization of hybrid seed corn, and its wide acceptance by American growers. Prior to 1930, virtually all corn produced in the world utilized open-pollinated cultivars with nearly 1000 different cultivars being grown in the United States alone

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Abbreviations: SCLB, southern corn leaf blight.

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(Martin and Leonard, 1967). During this era of open-pollinated corn production, varietal improvements in yield were essentially unsuccessful. In 1909 two genetists with an interest in corn production both expressed in separate but similar observations that inbreeding to establish pure lines, then crossing these inbreds to produce cultivars, improved yields (East, 1909; Shull, 1909). However, the seed yield of the parents used in these crosses was low and the costs to make a sufficient volume of single-cross seed was prohibitive.

Jones (1918) determined that a larger volume of seed with superior yield similar to the single-crosses could be obtained by crossing two superior single-cross lines to produce a double-cross hybrid with sufficient seed volume to make commercial production of these genetically improved lines available to corn growers. Such knowledge led to Henry A. Wallice founding the very successful commercial seed corn company "Pioneer Hi-Bred" in 1926. This resulted in him becoming quite wealthy and eventually being appointed Secretary of Agriculture which later led to him being elected the 33rd Vice President of the United States. The term "Hi-Bred" quickly became nicked to "hybrid" and is now an accepted term used to describe any specific cross of plants, or animals, that results in an increase in heterosis of a progeny.

Many other seed corn companies were started and prospered from what Jones (1918) had reported, combined with the rapid adoption of hybrid cultivars by American corn producers (Hallauer et al., 1988). By 1960 the relative amount of hybrid corn produced in the United States had increased to nearly 90.0% and today is likely to be ^{3}97% of the total hectarage of the major corn-producing regions of the world. Besides yellow hybrids, used mainly for animal feed and industrial products, virtually all food corn, sweet, pop, and white are hybridized for improved yields, pest resistance, and greater palatability. Lately hybrids have been developed with more fermentables for the specific purpose of increased levels of ethanol production for use in the renewable fuels industry.

One of the greatest labor requirements to produce hybrid seed corn in the beginning was the removal of the tassel from the female parent to acquire the desired genetic makeup of the seed to be sold. Prior to discovery of male sterile genotypes, detasseling of seed fields was accomplished by manually breaking the tassel off the intended female parent which would comprise about 6 to 12 rows of plants. Usually two rows of the male parent would then be planted on either side, with this pattern repeated over the entire field. Detasseling had to occur when the tassels first emerged and before they began to shed pollen. Time was of the essence in the process and labor could be expensive even though much of it was performed by local teenagers working for minimum wage.

THE DISCOVERY OF CYTOPLASMIC MALE STERILITY

Male sterility in corn was first discovered in Peru from seedings of an ear of an open-pollinated cultivar grown by R.A Emerson and F.D. Richey (Duvick, 1965). A cross was made to a plant from Chile that was unrelated, producing 45 F_1 kernels that were all male sterile but, when fertilized with viable pollen, produced well-filled ears with no indication of female sterility. Rhoades (1931, 1933), published an analysis of this discovery, stating that the observed male sterility was due exclusively to factors contributed by the female parent and that chromosomal genes had no effect on this expression. Unfortunately further genetic studies were not made on the Peruvian source of cytoplasmic sterility and it has since been lost. Another source of cytoplasmic male sterility was reported in an open-pollinated cultivar (*amargo blanco*) in Argentina by Gini (1940), but like the one from Peru it too has been lost (Duvick, 1965).

In 1945 reports from Kentucky and two neighboring states of poor kernel set in white corn pointed to possible male sterility as the cause. Studies conducted on the inbred parents concluded that male sterility occurred in an early inbred 33-16. Further research with single-, double-, and three-way crosses concluded that 33-16 contributed something through the cytoplasm of the egg to impart male sterility and that the degree of sterility was influenced by contributions from the male parent (Josephson and Jenkins, 1948). Progeny of (*iojap* × *teapod*) from an Iowa source, grown by M.T. Jenkins, while a USDA employee, was found to have a cytoplasmic-based pollen sterility and is often referred to as USDA source sterility (cms-S) (Jones et al., 1957).

The origin of cms-T in corn began with a discovery of male sterility in a field of white corn Mexican June, a cultivar developed by P.C. Manglesdorf and used for both food and feed (Rogers and Edwardson, 1952). Manglesdorf later developed the cultivars Golden June and Honey June through backcrossing of Mexican June, and found that male sterility was displayed in the two new cultivars. Rogers and Edwardson (1952), observed male sterility in an inbred line developed from Golden June and from it was initiated the development of other cytoplasmic male sterile inbreds. The most prominent of these was from a single male sterile plant that occurred in a cross with T×203. Repeated backcrossing eventually resulted in an inbred designated as T×203Ms which was maintained by crossing with the fertile strain of T×203. Later research by Forde et al. (1978), Dewey et al. (1986, 1987), and Wise et al. (1987) determined that a mitochondrial gene (*T-urf13*) was responsible for male sterility in cms-T plants. The *T-urf13* gene encodes for a protein of 13 kDa, URF-13, that is a component of the cristae of the mitochondria and uniquely associated with the suppression of pollen production.

Two traits that made cms-T plants appealing to commercial hybrid seed corn producers was its stability in retaining male sterility across a range of environmental conditions and the relative ease to be genetically returned to male fertile in the final stage of seed production (Coe et al., 1988). The presence of "restorer" genes "*Rf*" can overcome cytoplasmic male sterility. For cms-T plants, two restorer genes Rf_1 and Rf_2, are known to be involved in restoring phenotypic male fertility. A plant that is heterozygous $Rf_1\ rf_1\ Rf_2\ rf_2$ at both loci will produce viable pollen. The Rf_1 gene has been found to suppress the production of the URF-13 protein previously mentioned, by $\simeq$ 80% but activity by Rf_2 is required for restoration of male fertility (Dewey et al., 1987). Duvick (1965) pointed out that cms-T female parent plants tended not to exsert anthers, making it easy to visually determine if plants in a seed field were in fact male sterile. These factors combined to make cms-T the overwhelming choice of male sterility in seed corn production to where by 1970 it was part of the genetic background

of between 75 and 90% of hybrid cultivars being grown in the United States (National Research Council, 1972).

A third cytoplasmic male sterile group cms-C, discovered in a Brazilian cultivar Charrua, was reported by Beckett (1971) after the 1970–1971 SCLB-race T epidemic. This particular cytoplasm has been used in more modern hybrids but has succumbed to SCLB race-C, which was first described by Wei et al. (1988). Presently this race is confined to China and has not yet been identified in other regions of the world.

A SYNOPSIS OF SOUTHERN CORN LEAF BLIGHT

Southern corn leaf blight is found in all corn production areas of the world. It is most prevalent though in the warm temperate and humid subtropical regions. According to Robert (1953), SCLB (no race designation) was first discovered in the United States in 1923. It killed the green tissue of the leaves, effectively reducing the photosynthetic source area of the plant. With the exception of isolated outbreaks, prior to the 1970 epidemic, SCLB was not considered a major pest of corn in the United States (Carson, 2016).

Race O of SCLB, as stated previously, will attack the leaf blade tissue of corn causing the development of small tan lesions usually 0.6 by 2.5 cm (Agrios, 1997). They can be so numerous to almost cover the leaf blades and effectively "defoliate" the plant (Carson, 2016). Another factor of foliar fungal infections is a disruption of normal source-sink relationships in developing plants. Fungal lesions not only reduce effective photosynthetic leaf area, they can become strong sinks for what photosynthate is produced along with elevated respiration of infected tissue, and rob developing economic yield of needed organic nutrition, further adding to the damage (Kosuge, 1978).

Race T of SCLB is more destructive to host plants than race O because of its tendency to form lesions on the leaf sheaves, ear husk, developing grain on the ear, as well as leaf blades (Carson, 2016). Both races though can predispose the host plants to secondary infections of various stalk rotting diseases increasing harvest losses further due to stalk lodging and dropped ears. Race T's ability to infect developing grain can cause substantial losses at harvest by direct destruction of the kernels. Concern arose in the 1970 epidemic about feeding SCLB damaged grain to livestock (Ullstrup, 1972). Feeding studies using a variety of livestock species were conducted in some southeastern States as soon as the severity of the disease was recognized. However, no toxicity symptoms were recorded in these preliminary trials which were later confirmed by similar tests conducted in Indiana (Foster, 1971).

A detailed description of SCLB race-C is currently limited in part to its present confinement to China. Some references refer to cms-C as being "China male sterile". However as pointed out earlier the discovery of cms-C was reported in 1971 and was from a Brazilian cultivar Charrua. It just so happens this particular male sterile has been used extensively in China and research thus far on SCLB race-C shows it to be every bit as destructive to susceptible hybrids as SCLB race-T was to cms-T hybrids during the epidemic of 1970–1971 in North America. Wei et al. (1988) concluded that a major epidemic of SCLB race-C similar to that in North America could occur in China or elsewhere if the majority of hybrids being grown are of cms-C geneotype.

THE MAKING OF A CALAMITY

As stated earlier, cms-T was widely adopted by the hybrid seed corn industry of the United States during the 1960s because it was plainly and simply a "money maker" for the industry. It was reliable and a labor saver. It freed up more money for hybrid development, and corn growers were eager and willing to rapidly adopt the new hybrids because of the improved yields they were experiencing. The entire industry was putting more and more of its "eggs" into one genetic basket. Although a few scientists may have wondered in silence about the potential for a major crop failure following this formula, most failed to see the punch headed their way or refused to acknowledge its possibility.

The first warning sign of a problem was reported in the Philippines by Mercado and Lantican (1961) with later confirmations by Aala (1964) and Villareal and Lantican (1964, 1965). However, these observations were during below average rainfall seasons and differences between cms-T and normal cytoplasm for SCLB were not readily apparent. Hybrid seed corn companies did and continue to use areas of the Philippines and other tropical and subtropical regions as winter nurseries and areas for seed production increases. This allows for continuous hybrid development and increases in seed stockpiles for sale to producers in North America and elsewhere. The exact source of inoculum that entered the United States will never be known, but it most likely entered by way of contaminated seed either from breeding material or production seed stocks produced in an infected area of the world. Some speculated at the time that it may have occurred from a mutation of the pathogen already here in the United States (Ullstrup, 1972). Foley and Knaphus (1971), reported finding the disease on isolations from stored corn in Iowa that had been harvested in 1968.

In the central Corn Belt states of Illinois, Indiana, and Iowa in 1969, an unidentified disease was infecting the leaves and ears of plants in several production fields. Examinations by Ullstrup (1972), of a seed field in southern Iowa showed that *H. maydis* was involved in the rotting of the ears. The infection occurred in the later part of the growing season and besides the rotting of ears, stalk rots had set in, resulting in extensive lodging. Hybrids without cms-T showed no ear or stalk infections and very few lesions on the leaves. Scheifele et al. (1970) reported the same disease in corn growing in southern Illinois and undocumented observations were received from Iowa (Ullstrup, 1972). By the autumn of 1969 it was obvious that two edges of the disease triangle, a virulent pathogen and a susceptible host, were in place. All that was needed now to trigger a major plant disease outbreak was a favorable environment for the fungus.

That third edge of the disease triangle fell in place in midwinter of 1970. As early as late February and early March reports and plant specimens from southern Florida began to come into plant pathologists' laboratories with a leaf disease causing serious damage to cms-T corn (Moore, 1970b) (Fig. 1). Leaf tissue was dying and necrotic lesions were appearing on ears, developing kernels, and stalks. With early spring planting in Alabama and Mississippi the warm and unseasonably wet environmental conditions were conducive to the disease's spread to where by mid-June it had infected corn along the gulf coast up through the Mississippi Delta into Kentucky

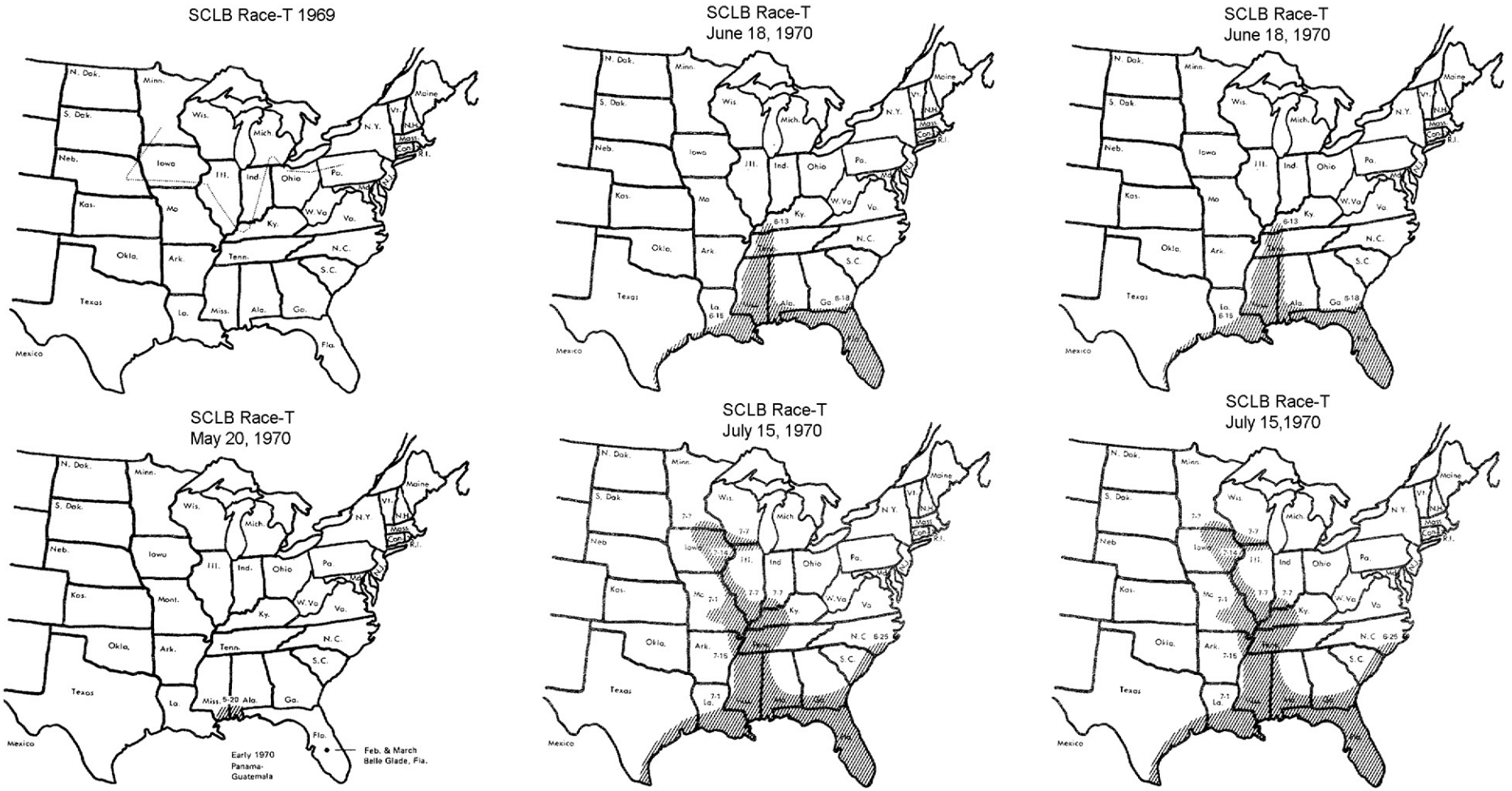

Fig. 1. Progression of southern corn leaf blight Race-T in North America from 1969 through 1970 (Moore, 1970b).

and the Missouri Bootheel. By mid-July the disease had spread along the Atlantic Coast into southern Virginia and up the Mississippi River Valley to northern Iowa and Wisconsin. By 15 August it had engulfed most all of the southern Corn Belt and within 2 wk it had spread as far West as the eastern half of Nebraska, eastern Kansas, southern Minnesota, and all of the United States East of there as well as southern Ontario, Canada.

The disease became a national headline in many newspapers. According to Ullstrup (1972) the *Chicago Tribune* published 37 articles on SCLB between August and November 1970. The *Wall Street Journal*, *The New York Times*, and several weekly news magazines all carried articles about SCLB. On the Chicago Board of Trade corn futures increased from $1.35 in late July to $1.68 per bushel by mid-September. Prices paid for other feed grains increased around the world due to a shortage of corn from the United States and the need to replace it with other cereals. Numerous University Extension meetings about the disease's impact, and more importantly what could be done for next year were being held throughout the corn-growing regions of the United States.

The severity of the disease locally was likely due in part to the time of initial infection. The earlier in the season the infection occurred, the greater the damage to the crop. As stated earlier some southern locations saw yield losses of between 30 to 50% of their crop. However, there were fields in the northern Corn Belt where the disease did not strike until later and still suffered total losses. A personal observation I made while working on the Missouri Corn Hybrid Evaluation Program was that by mid-August, in tests located in middle and northern Missouri, some hybrids had already died. Stalk rots were so severe that merely thumping a stalk with your index finger would cause the plant to lodge. Some of these hybrids had dropped up to 15% or more of their ears by then which were only $\simeq$ 5.0 cm in diameter. This situation made for poor harvesting conditions. More plant material had to be taken into the combine due to the lodging. This slowed down the harvest, increased the amount of residue being passed though the machine and increased chances of the lighter kernels resulting from the disease being thrown from the combine before falling through the sieves and being collected in the hopper.

An estimate of the total loss of corn from the 1970 epidemic in North America was 254 million hectoliters of grain (Tatum, 1971). The overall financial loss, as stated earlier was about $1.0 billion at that time (≥ $6.0 billion by 2015 standards). The total calories of feed/food energy lost to SCLB in 1970 was greater than that lost due to the potato (*Solanum tuberosum* L.) leaf blight in Ireland in the 1840s. However, the potato leaf blight resulted in massive starvation and a large emigration to the United States and Canada because the disease affected a primary food crop of a small and economically challenged island country (APS, 2016). The United States with its diversified agriculture and its overall wealth was able to absorb most of the impact of the SCLB epidemic. However, some of the corn futures markets were tweaked and some increases in meat, dairy, and poultry prices occurred, despite the fact farmers have never had any input on setting such prices because of their inability to pass along increases in production costs.

A huge challenge brought on by the SCLB of 1970 was, what to do for the following growing season so as to avoid or prevent another calamity of this magnitude. It was evident that the disease appeared virulent only to plants grown from seed developed using cms-T and that plants from seed that had been grown from detasseled female parents of normal cytoplasm were relatively unharmed. However, there were not enough normal cytoplasmic seed stocks to even begin to fill the upcoming demand. Unfortunately many of the seed production fields in 1970 used cms-T female parent stock that was either decimated by SCLB or would produce seed stock susceptible to another outbreak of the disease. The decision was made by virtually all seed companies to offer a blend of seed stock in which approximately 50% of the bag would contain normal cytoplasmic corn and 50% cms-T developed seed. On a whole the seed that was available for growers in 1971 was about 25% normal cytoplasm hybrids, 25% cms-T hybrids, and 50% blended seed (Ullstrup, 1972). All the major seed companies returned to detasseling seed fields in 1971 and discarded cms-T as a source of female parent in seed production. Everyone though had their fingers crossed that SCLB would not make a reappearance in 1971.

The hopes of not seeing another outbreak of SCLB in 1971 were dashed in June when disease activity was again reported in the Corn Belt (*Chicago Tribune*, 11 June 1971). The corn futures market climbed by $0.35 in 4 wk beginning in late May, on news of the disease reappearing. The introduction of a new Cu-based fungicide "Citcop 4E" (Southern Agricultural Insecticides, Inc., Pallmeto, FL) offered a means of chemical control but the costs to purchase and apply the pesticide did not readily justify its use except on seed fields. By July of 1971 temperatures and humidity levels were not as conducive to disease development and generally yields and total corn production were near record levels, with only some losses due to SCLB. Since 1971 SCLB Race T has not been a factor in corn production. Later research determined that the mitochondrial gene (*T-urf13*) that was responsible for inducing male sterility in cms-T corn was also responsible for its susceptibility to SCLB race T (Forde et al., 1978; Dewey et al., 1986, 1987; Wise et al., 1987). Levings (1990), found that an interaction between URF-13, the protein coded for by T-urf13, and the fungal toxins of SCLB race T accounted for the specific susceptibility of cms-T genotypes to fungal attack.

It is difficult to ascertain but undoubtedly this epidemic had a major impact on the seed corn industry as many smaller companies were either purchased or merged with larger ones, with some brands failing and disappearing completely within the next 5 yr. Very few seed companies marketing seed corn in 1970 are in existence today. Those that are have usually undergone mergers and are divisions of multi-national industries with other interest besides agriculture.

LESSONS FOR THE FUTURE

Nearly two generations have passed since the SCLB epidemics of 1970–1971 and about the only mention of it is in the study of epidemics in plant pathology classes. A few term papers are probably written about it every semester in universities across the country. But like most experiences, unless you lived through it, it has a diminished meaning for you. Ullstrup's warning is as true today as it was in 1972 "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".

With this in mind where are we at today? Are the lessons of the SCLB epidemic of the 1970s being applied or largely ignored? The National Research Council (1972) warned that the usual practice of selecting "good hybrids" as genetic resources for developing improved lines will ensure the eventual reduction of the genetic base of a crop and lead to increased risks of economic loses such as those experienced in the SCLB epidemic of 1970. Machines have since been developed that mechanically detassel corn plants thus reducing labor costs and making hybrid seed production without male sterility much more economical.

Darrah and Zuber (1986) in a survey of germplasm resources determined that normal cytoplasm hybrids (seed produced by detasseling) accounted for 87% of the available seed in 1984 while cms-C hybrids were 8% and cms-S type hybrids were only 3%. They also concluded that 88% of all hybrids being grown at publication had Reid yellow dent in their parentage. Shortly thereafter Smith (1988) reported that about 60% of privately released hybrids analyzed in his study had one of three inbred lines, B73, Mo17, or A632 either as a direct parent or major contributor to their genotype. Mikel and Dudley (2006) pointed out that current corn hybrids at the time of publication were being produced using inbred lines that were proprietary and protected by the U.S. Plant Variety Protection Act and/or U.S. Patent. They stated that at that time, much of the corn being grown was derived from seven progenitor lines, two of which were Mo17 and B73, which have been available for more than 40 yr. They state that the tendency for corn breeders to continue to recycle inbreds may, over the long-term, decrease genetic diversity in the crop.

With the development of biotechnology, specifically genetic engineering, one wonders if we are setting ourselves up for another fall by increasing genetic uniformity of our crops. We are already experiencing problems with some herbicide-resistant crops by facilitating the evolution of biotypes of weed species that were once susceptible to specific herbicides but are now resistant to them, that is, glyphosate [*N*-(phosphonomethyl)glycine]-resistant Amaranth (*Amaranthus palmeri* S. Wats.), Mare's tail [*Conyza canadensis* (L.) Cronq.], and Johnsongrass [*Sorghum halepense* (L.) Pers.]. Insect control developed by incorporating genes from *Bacillus thuringiensis* (*Bt*) to cause a crop, usually corn or cotton (*Gossypium hirsutum* L.), to produce a δ endotoxin that is lethal to certain lepidopteran species was introduced in 1996. Prior to the release of *Bt* crops for production, the governments of Canada and the United States adopted a "high dose/refuge" model of an insecticide resistance plan (IRP) (Ostlie et al., 1987; Gould, 1998). Huang et al. (2011) reported in their review, that the high dose/refuge model has been successful in delaying the development of *Bt* resistance in susceptible insect pests and continues to allow use of this form of insect control in several important crops. However, reports by Tabashnik et al. (2013) of a survey over five continents of *Bt* crops, found that 5 of 13 major insect pests had developed "field resistance" to *Bt* at the time of the survey, compared to only one in 2005.

The banana (*Musa acuminata* Colla) industry is currently facing the possibile extinction of their sole species (Sedgeman, 2015). From the late 19th century until the mid-1950s one variety of banana Gros Michel was the only one eaten as a fruit in the developed world. But, during the early 1950s a soil-borne wilt disease, plantation blight (*Fusarium oxysporum*) virtually destroyed the species because the plants were all clones of one plant and can only be propagated asexually. The variety was then replaced with variety Cavendishii which has since been the sole variety grown and is now threatened with extinction due to a mutation of the same fungus that destroyed Gros Michel. Every Christmas Americans are reminded by a favorite song made famous by Nat King Cole of a tradition that is no longer possible, roasting chestnuts [*Castanea dentata* (Marsh.) Borkh.] on an open fire. Chestnut blight or Asian bark fungus (*Cryphonectria parasitica*) was accidently imported to the United States in the early 20th century and proceeded to destroy the American chestnut tree in its native habitat along the eastern half of the country (Davis, 2005).

This begs the question, "Would it be possible to lose a crop species to disease or insect pests"? Probably not. But, the SCLB epidemic, the potato leaf blight in Ireland in the 1840s, the boll weevil (*Anthonomus grandis*) infestation during the late 19th and early 20th centuries through the American Cotton Belt, and the current problem with the world's banana crop are all reminders that we need to be mindful of that possibility and think of potential solutions or alternatives if any of our crops become threatened. In other words we always need to "keep our options open" and make or have plans in place to overcome and/or avoid such disasters.

Conflicts of Interest

The author declares there are no conflicts of interest regarding the procedures used or data presented for publication of this manuscript.

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