Jerald K. Pataky
University of Illinois, Department of Crop Sciences, Urbana

Martin M. Williams II
USDA-ARS, Global Change and Photosynthesis Research Unit, University of Illinois, Urbana

John M. Headrick
Monsanto Company, Seminis Vegetable Seeds, St. Louis, MO

Claude Nankam
World Vision International, Malawi

Lindsey J. du Toit
Washington State University, Department of Plant Pathology, Mount Vernon

Phillip M. Michener
Terral Seeds, Greenville, MS

Observations from a Quarter Century of Evaluating Reactions of Sweet Corn Hybrids in Disease Nurseries

Host resistance is one of the most effective and efficient ways to control plant diseases. Resistance eliminates or minimizes losses from diseases and reduces the need for other control tactics. Resistance also can be integrated easily with other pest management practices.

Resistance and susceptibility are two extremes of a continuum of host reactions to diseases. Resistance is a measure of the ability of the host to reduce the growth, reproduction, and/or disease-producing abilities of the pathogen, thus resulting in less severe disease symptoms. Major genes for resistance, such as the *Rp1-D*, *Ht1*, or *Mdm1* genes in maize, can prevent or substantially limit disease development if specific virulence (i.e., races) occurs infrequently in pathogen populations. Consequently, cultivars with major gene resistance usually have clearly distinguishable phenotypes. Major gene resistance may become ineffective if specific, complementary virulence becomes prevalent in the pathogen.

Aside from major gene resistance, disease reactions usually vary among cultivars, often ranging from highly resistant to highly susceptible based on the genetic background and level of polygenic or partial resistance of each cultivar. Based on the quantitative nature of this variation, cultivar responses to pathogens do not separate into distinct categories. However, descriptions and comparisons of

cultivars are more pragmatic and easily understood if disease reactions are placed into distinct, ordinal classes. Thus, cultivar reactions often are grouped broadly as: resistant (R), moderately resistant (MR), moderate (M), moderately susceptible (MS), and susceptible (S) based on severity of disease symptoms. Sometimes these categories are designated numerically, e.g., from 1 to 9. While this categorization produces statistically “overlapping” groups without obvious boundaries (e.g., cultivars with the least severe symptoms in the MR class do not differ significantly from cultivars with the most severe symptoms in the R class), a consistent response of a cultivar in multiple trials can produce a reasonable estimate of the disease reaction of that cultivar relative to the response of other cultivars. These reactions can then be used to assess the potential for diseases to become severe enough to affect yield.

Since 1984, nearly 3,700 commercial or pre-commercial sweet corn hybrids were evaluated for disease reactions in nurseries at the University of Illinois (www.sweetcorn.illinois.edu). Each year, hybrids were evaluated for reactions to four diseases that consistently were prevalent on sweet corn grown in North America, including common rust caused by *Puccinia sorghi* Schwein., northern corn leaf blight (NCLB) caused by *Exserohilum turcicum* (Pass.) Leonard & Suggs, Stewart’s wilt caused by *Pantoea stewartii* (Smith) Dye (syn. *Erwinia stewartii*), and maize dwarf mosaic (MDM) caused by *Maize dwarf mosaic virus* or *Sugarcane mosaic virus*. In some years, hybrids also were evaluated for reactions to diseases that occurred more sporadically on sweet corn, including southern corn leaf blight (SCLB) caused by *Bipolaris maydis* (Nisikado & Miyake) Shoemaker, southern rust caused by *Puc-*

Dr. Pataky’s address is: University of Illinois, Department of Crop Sciences, Urbana, IL 61801; E-mail: j-pataky@illinois.edu

Table 1. Seed companies and breeding programs submitting entries to the University of Illinois sweet corn hybrid disease nursery from 1984 to 2010

Seed company or breeding program	Number of entries	Seed company or breeding program	Number of entries
Abbott and Cobb, Inc.	158	Musser Seeds	7
Advanta Semillas/Pacific Seeds	9	Nunhems Seeds	4
Asgrow	232	Park Seeds	2
Basso Semillas	20	Pop Vriend	2
Centest Inc.	26	Rispens Seeds	5
Charter Seed	5	Robson Seed Farm	92
Crookham Company	284	Rogers/Syngenta	493
Del Monte	15	Sakata Seed Co.	38
Escagenetics	1	Seed Savers	37
Ferry Morse	97	Seedway	48
Galilee Seeds	38	Seminis/Monsanto	530
Green Giant	198	Snowy River/HSR Seeds	99
Harris Moran Seed	298	Stokes Seeds	60
Harris Seeds	2	Sun Seed	58
Illinois Foundation Seeds Inc.	559	Top Corn	2
Johnny's Selected Seeds	9	University of Delaware	8
Liberty Seed Co.	11	University of Florida	8
Market More	94	University of Hawaii	18
Mesa Maize	96	University of Illinois	8
Mikado Kyowa Seed	6	Wisconsin Crop Improvement	10

cinia polysora Underw., anthracnose leaf blight (ALB) caused by *Colletotrichum graminicola* (Ces.) G.W. Wils., gray leaf spot (GLS) caused by *Cercospora zea-maydis* Tehon & E.Y. Daniels, and Goss's wilt caused by *Clavibacter michiganensis* subsp. *nebraskensis* (Vidaver & Mandel) Davis et al. Beginning in 2002, hybrids in the Illinois nurseries also were evaluated for responses to selected P450-metabolized, postemergence herbicides, including: bentazon, carfentrazone, dicamba plus diflufenzopyr, foramsulfuron, halosulfuron, mesotrione, nicosulfuron, primisulfuron, tembotrione, and topramezone.

This article presents a summary of observations and trends from more than a quarter century of evaluating sweet corn hybrids in University of Illinois disease nurseries.

Nursery Protocols

Hybrids evaluated. Nearly 3,700 sweet corn hybrids have been tested in University of Illinois disease nurseries in the past 27 years, including commercial and pre-commercial hybrids from 40 private and public sweet corn breeding programs located in North America, South America, Asia, and Europe (Table 1). Hybrids were evaluated annually for reactions to five to eight diseases and for specific races of some pathogens. Each disease or pathogen race was evaluated in a separate trial with two or three replicates of single rows of hybrids with 12 to 18 plants per row. Trials were planted each year on the University of Illinois South Farm, Champaign-Urbana, IL. In some years, additional trials were located on the Central Illinois Irrigated Growers farm near Havana, IL or on the Del Monte USA Agriculture Research farm in Rochelle, IL.

From 1984 to 1991, the number of hybrids evaluated each year increased from 75 to over 200 (Fig. 1). Since 1992, the number of entries evaluated annually averaged about 350 and ranged from 247 to 486 except for the Silver Anniversary nursery in 2008 in which 565 hybrids were evaluated. From 1984 to 1988, most hybrids were available commercially at the time they were first entered in the Illinois trials. Since then, a portion of each nursery consisted of pre-commercial hybrids that had not yet been released for sale at the time they were submitted for evaluation.

The elevated sugar content of most sweet corn hybrids grown in North America is the result of one of two recessive, endosperm mutant genes: *sugary-1* (*su1*), or *shrunk-2* (*sh2*). Most sweet corn hybrids in North America are classified into one of three endosperm types: sugary, sugary enhancer, or shrunk-2 (14). Sugary sweet corn has been grown in the Americas since pre-Columbian times (40). The sugar content of sugary sweet corn can be elevated further by recessive, sugary enhancer genes (*se*), thus forming the sugary enhancer endosperm type. Sugary enhancer

Fig. 1. Number of entries in the University of Illinois sweet corn hybrid disease nurseries from 1984 to 2010.

varieties tend to be extremely tender due to thin pericarps. Similar to the sugary endosperm types, sugary enhanced hybrids have a creamy, sweet corn texture as a result of kernel polysaccharides. Discovered at the University of Illinois in the late 1960s by A. M. "Dusty" Rhodes, the sugary enhancer trait was released in the inbred IL677a in the mid-1970s (4). The elevated sugar characteristic of corn homozygous for the *sh2* gene was first identified at the University of Illinois in 1953 by John Laughnan (12). In addition to having high levels of kernel sugars, shrunk-2 varieties are unique in their ability to maintain a sweet flavor over an extended period because they lack ADP-glucose pyrophosphorylase, an enzyme involved in starch synthesis. "Supersweet" hybrids based on the *sh2* endosperm mutation were first released by Laughnan in 1961 (13).

Because these endosperm mutations are recessive, cross pollination between shrunk-2 hybrids and sugary or sugary enhanced hybrids results in starchy corn. Therefore, in the Illinois disease nursery, hybrids were placed into one of two main plots based on their endosperm type. Hybrids with the sugary or sugary enhancer endosperm mutations were placed together in one main plot. Hybrids with the shrunk-2 endosperm mutation (aka supersweets) formed the other main plot. Hybrids that had various combinations

Table 2. Disease and herbicide reactions of sweet corn hybrids included as standards in the University of Illinois nursery in 2010

SdCo ^b	ET ^c	KC ^d	RM ^e	Hybrid	Com. rust ^f	Rust races				Ht	St. wilt ^f	MDM	SLB	So. rust ^f	GLS	ALB	Goss's wilt	Herbicide reactions ^a		
						avir	D-vir	G-vir	NLB									Callisto	Laudis	Accent
IFS	sh2	B	3	277A	6 ^{23g,h}	6 ⁸	6 ⁹	7 ⁶	5 ¹⁵		3 ¹²	9 ¹²	4 ¹⁰	7 ¹	6 ³			3 ⁸	1 ²	2 ⁵
Cr	se	B	3	Ambrosia	6 ³⁰	6 ¹⁵	5 ⁹	6 ⁶	5 ²¹		2 ¹⁹	9 ¹⁴	6 ¹⁴	6 ³	5 ³	6 ³		1 ⁸	1 ²	2 ⁵
Cr	se	Y	3	Bodacious	5 ²⁹	4 ¹⁶	5 ⁸	6 ⁵	5 ²³		5 ²¹	9 ¹²	5 ¹⁵	7 ³	6 ³	3 ³		1 ⁶	1 ¹	1 ⁴
Rog	su	Y	5	Bonus	Rp ²⁸	0 ¹²	4 ¹⁰	0 ⁶	5 ¹⁸	Ht	1 ¹⁴	2 ¹⁴	7 ¹³	7 ³	5 ³	3 ¹		2 ⁸	1 ²	2 ⁵
Sem	su	Y	4	El Toro	Rp ²⁸	0 ¹²	6 ¹⁰	0 ⁶	6 ¹⁸		3 ¹⁴	3 ¹⁴	3 ¹³	7 ³	5 ³			3 ⁸	1 ²	2 ⁵
Cr	su	Y	4	Eliminator	Rp ³¹	0 ¹⁵	6 ¹⁰	0 ⁶	6 ²¹		2 ¹⁷	2 ¹⁴	6 ¹⁵	8 ³	6 ³	4 ³		2 ⁸	1 ²	2 ⁵
Rog	sh2	Y	3	Garrison	Rp ¹⁴	0 ⁵	0 ⁵	0 ⁴	2 ⁶	Ht	2 ⁵	2 ⁵	4 ⁵	7 ¹				2 ⁴	1 ²	1 ²
Rog	su	Y	4	Jubilee	5 ³⁷	5 ²¹	6 ¹⁰	6 ⁶	8 ²⁷		9 ²⁵	9 ¹⁴	4 ¹⁵	7 ³	6 ¹	7 ⁵	9 ³	3 ⁷	1 ²	2 ⁵
Sem	su	Y	4	Merit	8 ¹⁷	8 ⁸	8 ⁵	7 ⁴	6 ¹⁰	Ht	5 ⁹	9 ⁵	5 ⁶		6 ²	4 ³		9 ⁴	9 ²	9 ²
MM	se	Y	5	Merlin	3 ²⁴	3 ¹²	3 ⁸	3 ⁴	4 ¹⁸		2 ¹⁵	9 ¹²	2 ¹²	4 ³	5 ³	3 ¹		2 ⁶	1 ¹	1 ⁴
Cr	se	Y	4	Miracle	3 ³⁶	2 ²¹	4 ⁹	3 ⁶	3 ²⁸		2 ²⁶	9 ¹⁴	4 ¹⁶	6 ³	6 ³	3 ⁵	1 ³	2 ⁸	1 ²	1 ⁵
Sem	sh2	B	3	Obsession	Rp ²¹	0 ⁷	4 ⁸	0 ⁶	3 ¹²	Ht	3 ¹⁰	9 ¹⁰	3 ⁹	9 ¹	7 ²			1 ⁷	1 ²	1 ⁴
Rog	sh2	Y	5	Overland	Rp ¹²	0 ⁴	0 ⁴	4 ⁴	1 ⁶	Ht	2 ⁵	7 ⁵	4 ⁵	5 ¹				2 ⁴	1 ¹	1 ³
Sem	se	B	4	Sensor	4 ²⁹	3 ¹⁴	4 ⁹	4 ⁶	4 ²¹		5 ²⁰	9 ¹³	3 ¹⁴	5 ²	5 ³	3 ¹		1 ⁸	1 ²	1 ⁵
MM	se	Y	4	Tuxedo	3 ¹⁹	3 ⁷	4 ⁷	4 ⁵	3 ¹²		3 ¹¹	9 ⁸	2 ¹⁰	2 ¹	6 ²			2 ⁶	1 ²	1 ³

^a Herbicide reactions: tolerant (1-2), intermediate (3-6), sensitive (7-9).

^b SdCo - seed source: Cr - Crookham Company, IFS - Illinois Foundation Seeds, MM - Mesa Maize, Rog - Syngenta Rogers Brand, Sem - Monsanto Seminis Vegetable.

^c ET - endosperm type: su - sugary 1, se - sugary enhancer, sh2 - shrunken-2 (others classified within these three major endosperm types).

^d KC - kernel color: B - bicolored, W - white, and Y - yellow.

^e RM - relative maturity: 1 - first early, 2 - second early, 3 - mid-season, 4 - main season, and 5 - full season.

^f Com. rust, common rust; St. wilt, Stewart's wilt; So. rust, southern rust.

^g Disease reactions: 1 - resistant, 3 - moderately resistant, 5 - moderate, 7 - moderately susceptible, 9 - susceptible; Rp and Ht - major gene resistance.

^h Example: 5¹⁵ indicates that a hybrid has a moderate (5) reaction to that disease based on 15 trials (superscript indicates number of trials).

Fig. 2. Sectors of asymptomatic and symptomatic leaf tissue on a sweet corn hybrid inoculated with *Maize dwarf mosaic virus* and *Sugarcane mosaic virus*. Incidence of plants with "sectoring" symptoms can range from 0 to 70% among hybrids with the *Mdm1* resistance gene.

of these mutations were placed in the most appropriate main plot, e.g., synergistic hybrids (*susu sese sh2SH2*) were placed with the sugary and sugary enhanced hybrids, while sugary-shrunken hybrids (*susu SESE sh2sh2*) were placed with the supersweet hybrids.

Each trial included one or two sets of 8 to 15 standard hybrids (i.e., checks) with a range of reactions to each disease being evaluated. Standard hybrids have been evaluated for several years and have been observed to have relatively consistent responses to diseases (Table 2). The set of standard hybrids stayed nearly the same between consecutive years, although over decades the set varied as newer hybrids gradually replaced older standards.

Inoculation and disease ratings. To assure adequate and uniform disease pressure, plants were inoculated at least twice and sometimes as often as five or six times beginning at the two- to three-leaf stage or whenever leaf whorls were large enough to hold inocula. Although all experimental units were treated the same, inoculum varied somewhat with each inoculation (e.g., concentration, volume applied). Production of inocula and methods of inoculation were unique for each pathogen.

Inoculation served a different purpose for each of the various diseases. For the foliar fungal diseases, plants were inoculated with the purpose of infecting lower leaves sufficiently so that an abundance of secondary inocula was produced. Hybrids were then rated for severity resulting mostly from secondary infection. Consequently, results were unaffected by moderate variation in the amount of initial disease that resulted from inoculation of each experimental unit. For diseases for which symptoms developed predominantly from initial infection (e.g., MDM, Stewart's wilt, and Goss's wilt), ratings were based primarily on the reaction of plants to inoculation, i.e., incidence of systemic viral infection or intra-plant movement of bacteria from wounds inflicted by the method of inoculation. Therefore, greater uniformity in inoculation procedures was necessary to more accurately separate reactions of hybrids to these diseases.

Diseases were rated with binomial, ordinal, or discrete quantitative scales. On an individual plant basis, MDM ratings were binomial, i.e., symptomatic or asymptomatic plants. Plants with intermediate responses, i.e., sectors of symptomatic and asymptomatic leaf tissue (Fig. 2), were considered symptomatic even though these symptoms were associated with the presence of an MDM-resistance gene. Incidence (%) of MDM-infected plants was then calculated for each row. Stewart's and Goss's wilt were rated on a 1 to 9 ordinal scale that reflected the extent to which bacteria spread from inoculation wounds or the severity of systemic infection (39). To account for variation among individual plants in a row, each row received three bacterial wilt ratings which were averaged for the row. For the foliar fungi, different rating scales were used depending on the amount and uniformity of secondary infection. Total leaf area infected, from 0 to 100%, was rated on a row basis when secondary infection was abundant and symptoms were relatively uniform among plants within a row (Fig. 3). When secondary infection was less severe, often resulting in bands of infected leaf tissue, an ordinal scale of 1 to 9 was used to rate the size and density of lesions and the overall extent of infection (Fig. 4). For the foliar fungi, each row was given two ratings in order to avoid spending an inordinate amount of time estimating an exact severity or ordinal rating for the row (e.g., a row was rated 25 and 30% if the estimated severity was somewhere between those values). For every disease except MDM, each row usually was rated by two people; and means were calculated from all ratings. The

Fig. 3. Northern corn leaf blight, caused by *Exserohilum turcicum*, on rows of sweet corn hybrids with different levels of resistance or susceptibility. When levels of disease are relatively uniform among plants within rows, severity is rated from 0 to 100% on a row basis.

bacterial wilts and MDM were rated prior to anthesis, usually about 10 to 20 days after inoculation. The foliar fungal diseases were rated about 2 to 4 weeks after pollination near the fresh market harvest stage of sweet corn.

The discrete, quantitative ratings or ordinal ratings that were used to assess symptom severity were treated as quantitative data when analyzed statistically. The quantitative nature of these data resulted in information about hybrids that did not separate into discrete categories. However, descriptions and comparisons of hybrids are more pragmatic and easily understood by growers and personnel in the seed industry if characteristics such as reactions to diseases are placed into distinct, ordinal categories (e.g., R, MR, M, MS, S). Classifying disease reactions on an ordinal level also helps to account for variation in the range of disease severity that frequently occurs among trials, often associated with differences in environmental conditions.

Disease reactions of hybrids. Disease reactions of hybrids were classified from resistant to susceptible (1 to 9, respectively) based on categorization of disease ratings which in some cases also used a 1 to 9 ordinal scale. Despite the same numerical values, the 1 to 9 categories of disease reactions were not the same as the 1 to 9 ratings of symptom severity.

For each disease, reactions of hybrids in each annual nursery were categorized from resistant to susceptible based on mean separation of disease ratings (e.g., least significant difference values), standardized z-scores of ratings (i.e., standard deviations from the trial means), and ratings in that trial for the set of standard hybrids. Using this procedure, disease reactions were based on the response of a hybrid relative to all other hybrids in a trial rather than on exact values of disease ratings. Thus, in a trial with a mean disease severity of 20% and a standard deviation of 8%, a hybrid with a rating of 18% severity may have been classified moderate (M) because severity on this hybrid was near the mean severity of all hybrids in that trial; whereas, in a different trial with a mean disease severity of 32% and a standard deviation of 8%, a hybrid with a rating of 18% severity may have been classified as resistant (R) because severity on that hybrid was nearly 1.75 standard deviations below the trial mean.

During the first decade of the nursery, hybrids were placed in one of five categories, R, MR, M, MS, or S. Although the exact boundaries between categories were somewhat arbitrary, the range of disease ratings usually was five or more times greater than the mean separation value for those ratings. Thus, hybrids with the lowest ratings in one disease reaction category usually were significantly different from hybrids with the lowest ratings in the next higher category. Conversely, hybrids with the highest ratings in one disease reaction category were not significantly different from hybrids with the lowest ratings in the next higher category. Al-

Fig. 4. Bands of southern corn leaf blight lesions, caused by *Bipolaris maydis*, on sweet corn hybrids with different levels of resistance or susceptibility. When secondary infection is limited, an ordinal scale of 1 to 9 is used to rate disease based on the size and density of lesions and the overall extent of infection.

though classification criteria differed among trials depending largely on the variation in the trial, z-scores for hybrids rated R often were below -1.2 ; MR hybrids were below -0.4 ; M hybrids were between -0.4 and 0.5 ; MS hybrids were above 0.5 ; and S hybrids were above 1.4 .

Classification of the disease reaction of a hybrid varied somewhat among trials. Therefore, after a few years of evaluation, the range of the disease reaction of a hybrid may have been broad and relatively uninformative, e.g., R to MS. To account for that variation, R to S disease reaction categories were replaced in the 1990s with a 1 to 9 system where 1 = R, 3 = MR, 5 = M, 7 = MS, and 9 = S, as illustrated by classification of NCLB reactions in the 2009 nursery (Fig. 5). The means of 1 to 9 reactions from all trials were then used to describe the disease response of a hybrid with superscript numbers indicating the number of trials from which the mean reaction was calculated (Table 2). Thus, the 1 to 9 classification of disease reactions allowed for an easy method to combine data from multiple trials; even though occasionally, 1 to 9 disease reactions of hybrids were misinterpreted as 1 to 9 disease ratings of symptoms in a specific trial. Although not used, means of annual z-scores also were evaluated as an alternative method to calculate disease reactions of hybrids from multiple trials. Means of 1 to 9 reactions and means of z-scores produced similar results.

All hybrids were not evaluated each year. Also, classifications of individual hybrids varied among years, as illustrated by NCLB classifications of three standard hybrids, Miracle, Ambrosia, and Jubilee (Fig. 6). Therefore, an estimate of the number of trials necessary to effectively classify hybrid reactions and an estimate of the confidence associated with disease reactions based on single or multiple trials was sought. Using parameters from distributions of disease ratings from over 12 years of trials, Monte Carlo simulation methods in combination with Bayesian theory were used to determine the posterior probability ($P(\Theta_i/z_k)$) associated with assigning hybrids to disease reaction categories based on differing numbers of trials (17). The simulation model was programmed to calculate 1,000 simulated sample means for each hybrid in each simulated trial. Four years of trials were simulated using the most and least variable disease nursery data for each disease. Simulations were done for common rust, NCLB, and Stewart's wilt.

Posterior probabilities ($P(\Theta_i/z_k)$) of assigning a hybrid to the same 1 to 9 category as the true disease reaction of that hybrid (i.e., the reaction assigned to the hybrid for the simulation) were very low from a single trial ($P(\Theta_i/z_k)$ ranged from 0.39 to 0.76) and relatively low for mean reactions based on three trials ($P(\Theta_i/z_k)$ ranged from 0.39 to 0.95) (17). This was expected considering that mean separation values from individual trials usually overlapped by two or

Fig. 5. Distribution of northern corn leaf blight (NCLB) severity ratings of 387 sweet corn hybrids evaluated in the 2009 University of Illinois disease nursery; and classification of hybrid reactions from 1 to 9 (resistant to susceptible) for NCLB. Mean disease reactions of five standard hybrids were based on multiple evaluations, e.g., Garrison = 1.5 (see Table 2 and text).

Fig. 6. Distributions of annual disease classifications (1 to 9) of three standard hybrids, Miracle, Ambrosia, and Jubilee, for reactions to northern corn leaf blight (NCLB) based on 28, 21, and 27 trials, respectively, in the University of Illinois sweet corn disease nursery. Mean reaction of each hybrid is given in parentheses.

three of the 1 to 9 disease reactions categories (e.g., Fig. 5). Conversely, posterior probabilities ($P(\Theta_{i+1}/z_k)$) of assigning a hybrid to within one category ($i \pm 1$) of the true disease reaction of that hybrid when disease reaction means were based on three trials was usually greater than 0.95 (Fig. 7). When simulations used the most

variable nursery trial data, posterior probabilities ($P(\Theta_{i+1}/z_k)$) of assigning a hybrid to within one category of the true disease reaction were above 0.95 except for four cases (Fig. 7A). When simulations used the least variable nursery trial data, all posterior probabilities ($P(\Theta_{i+1}/z_k)$) of assigning a hybrid to within one category of the true disease reaction for that hybrid were above 0.98 (Fig. 7B). Thus, with 3 years of trials, there is a relatively high probability that the mean disease reaction assigned to a hybrid is within one category of the true disease reaction.

Z-scores as a measure of changes in resistance. As sweet corn breeders develop new hybrids with improved disease resistance, disease reactions of standard, check hybrids may change over time. For example, if a check hybrid has a moderate disease reaction (i.e., disease rating close to the mean of the group of hybrids evaluated), but over a period of time, most new hybrids have better levels of resistance, the disease rating for the check hybrid will become higher than the mean of the new group of hybrids; and the disease reaction of the check hybrid will appear to be moderately susceptible relative to the group of new hybrids. Thus, shifts in groups of hybrids to higher or lower levels of resistance can be measured by changes in z-scores of standard, check hybrids.

Z-scores of disease ratings (i.e., standard scores) are a measure of raw scores in units of standard deviations from the mean of the distribution in which the raw score occurs, i.e., $Z\text{-score} = (\text{raw score} - \text{mean}) / \text{standard deviation}$. Z-scores of disease ratings of standard hybrids in annual nurseries were used to determine if the distribution of hybrid reactions to a specific disease shifted over time toward greater resistance or susceptibility. If the distribution of reactions of all hybrids in the nursery shifted toward greater

Fig. 7. Posterior probabilities, $P(\Theta_{i\pm}/z_k)$, of assigning a disease reaction to within one of the 1 to 9 categories of the true reaction of a sweet corn hybrid based on mean reactions from three simulated sample means (i.e., trials) for northern corn leaf blight (NCLB), common rust, or Stewart's wilt: **A**, input data for simulated sample means based on the most variable Illinois disease nursery trial data, and **B**, input data for simulated sample means based on the least variable Illinois disease nursery trial data.

resistance, z-scores of standard hybrids increased; whereas z-score of standard hybrids decreased with a shift of the distribution toward greater susceptibility. Evaluated over time, z-scores of standard hybrids serve as a measure of changes in resistance or susceptibility of the group of sweet corn hybrids being evaluated in the nursery.

Trends in Sweet Corn Reactions During the Past Quarter Century

Longevity of data. Information on the disease reaction of a hybrid is useful to the public only as long as the hybrid is sold commercially. For the 312 hybrids that were newly entered in the nursery in 2009 and 2010, only 55% were available commercially in 2010. A few more of these hybrids may be released for sale after 2010, but a large portion will not be sold due to unacceptable deleterious traits which may include disease reactions. Considering that pre-commercial hybrids may be evaluated in the disease nursery for an initial year or two, the half-life of information from any annual Illinois disease nursery appears to be slightly more than 5 years (Table 3). Only 31% of the 653 hybrids that were new entries during the 5-year period from 2004 to 2008 were available for sale in 2010. The percentage of hybrids available for sale in 2010 decreased to 20, 10, and 6% for new entries submitted during each 5-year period prior to 2003 (Table 3). Interestingly, about 9% of the 398 hybrids that were evaluated during the first 5 years of the

Table 3. Sweet corn hybrids submitted to the University of Illinois disease nursery that were available for sale in 2010

	Number of new hybrids submitted to disease nursery	Hybrids available for sale in 2010	
		Number	%
1984-1988	398	35	9
1989-1993	750	45	6
1994-1998	890	92	10
1999-2003	684	134	20
2004-2008	653	202	31
2009-2010	312	172	55

Fig. 8. Percentage of sugary, sugary enhancer, and shrunken-2 endosperm types among new hybrids submitted to the University of Illinois sweet corn disease nursery during 5-year periods beginning in 1984.

nursery (1984 to 1988) are still available commercially. This may be due to hybrids with remarkable longevity, such as Merit developed by Asgrow Seed Company in the early 1960s and Jubilee bred by Frank Blankenburg and first listed in the Rogers Bros. seed catalog in 1960. Surprisingly, Merit is among the most susceptible sweet corn hybrids for common rust (mean disease reaction of 7.9), and Jubilee is among the most susceptible for NCLB and Stewart's wilt (mean disease reactions of 8 and 8.7, respectively). The longevity of these two hybrids and others may be due to what some have called "hybrid inertia". Once a hybrid has proved itself to be successful, growers often become accustomed to that hybrid and accepting of its flaws. Being somewhat risk-averse and conservative, growers may elect to grow a less-than-perfect hybrid with known flaws, rather than risk growing a new hybrid whose deficiencies are yet to be discovered.

Shifts in endosperm mutants. The percentage of hybrids in the Illinois disease nursery with each of the three endosperm types (sugary, sugary enhancer, and shrunken-2) has changed from 1984 to 2010 (Fig. 8). Of the initial new 398 hybrids evaluated from 1984 to 1988, about 40% each were sugary or shrunken-2 endosperm types, and slightly more than 20% were sugary enhancer types. Since 1988, the percentage of new sugary hybrids submitted to the nursery has remained at about 15 to 20%. Nearly 93% of the new sugary hybrids in the nursery since 1988 had yellow kernel color. Since 1993, no new bi-colored, sugary hybrids and only 26 new white, sugary hybrids have been entered in the nurseries. From 1989 to 2003, the percentage of new shrunken-2 hybrids in the nursery increased to 50 to 60%, while sugary enhancer hybrids comprised 20 to 30% of new entries. Since 2003, the percentage of new shrunken-2 hybrids in the nursery increased to about 75% and the percentage of new sugary enhancer hybrids dropped to less

Fig. 9. Sweet corn hybrids with susceptible and Rp-resistant reactions to the common rust fungus, *Puccinia sorghi*. **A**, Sporulating uredinia typical of a susceptible reaction. **B**, Chlorotic flecks conditioned by Rp resistance. **C**, Susceptible and Rp-resistant hybrids in the Illinois disease nursery.

Fig. 10. Percentage of Rp-resistant hybrids in Illinois sweet corn disease nurseries from 1984 to 2010. Rp-resistance in all hybrids evaluated prior to 1999 was susceptible to an Rp1D-virulent race of *Puccinia sorghi* that became widespread in North America in 1999. A more diverse set of Rp genes has been used since 2000.

than 10%. At least part of the change in the prevalence of endosperm types during the past decade resulted from the introduction of “high quality” shrunken-2 (HQ-sh2) hybrids that have multiple endosperm mutations (e.g., homozygous *sh2* in combination with homozygous or heterozygous *su* and/or *se*). The HQ-sh2 hybrids combine elevated levels of kernel sugars and extended shelf life (i.e., persistent quality) derived from Laughnan’s shrunken-2 endosperm type with tender pericarp and creamy texture from Rhodes’ sugary enhancer and/or standard sugary endosperm types. As a larger number of HQ-sh2 hybrids have been introduced, fewer sugary enhancer hybrids have been evaluated.

Reactions to common rust. A number of different single, dominant Rp genes convey chlorotic-fleck, hypersensitive reactions to *P. sorghi* resulting in little or no formation of urediniospores (Fig. 9). Most of the Rp genes were identified by Art Hooker and colleagues in the 1960s (7,9). “Compound” rust resistance genes also have been developed by Scot Hulbert and colleagues through the identification of cross-over events in which Rp genes that occur in very close proximity on chromosome 10S are linked in coupling phase (8).

Possibly, the greatest change in disease reactions of sweet corn hybrids in the past quarter century has been the widespread use of Rp genes to control *P. sorghi* (Fig. 10). Of 75 hybrids in the first Illinois disease nursery in 1984, only three (aRRestor, Excellency, and Prevailer) were Rp resistant. A decade later, 20% of all hybrids in the nursery were Rp resistant; and by 1999, over a third of the nursery entries had Rp resistance (Fig. 10). In 1999, isolates of *P. sorghi* virulent against the *Rp1D* gene (i.e., the D-virulent race) became widely prevalent in North America (32,35,37). None of the Rp-resistant sweet corn hybrids evaluated in the Illinois disease nursery prior to 1999 were resistant to this race; however, the *Rp1D* gene conveyed a level of control proportional to the frequency of virulence in mixed populations of *P. sorghi* when D-virulent isolates were below 40% of the initial inocula (24,26).

Although the use of the *Rp1D* gene increased in sweet corn from 1984 to 1999, it is unlikely that this gene deployment was the selection pressure that caused the increased frequency of D-virulent isolates in the North American population of *P. sorghi*. Sweet corn comprises less than 1% of the total corn acreage in North America; and sweet corn hybrids with the *Rp1D* gene were grown during this period primarily in the upper Midwest, New York, and Ontario, Canada. Even if virulence had been selected in these plantings, the virulent populations would not have survived to infect the subsequent year’s crops. Teliospores produced on Rp-resistant hybrids would have been of little effect because the fungus does not survive the winter on the alternate host, *Oxalis* spp., in this region. Likewise, it is unlikely that urediniospores or teliospores produced on Rp-resistant hybrids in the upper Midwest, New York, or Canada would have been disseminated to Florida, Mexico, or the southern United States where inocula originate for the next year’s crop. A more likely cause of the increased frequency of D-virulent isolates was the increased use of the *Rp1D* gene in field corn grown in the southern United States following severe epidemics of common rust that occurred in conjunction with unusual conditions associated with floods throughout the Corn Belt in 1993 (21).

In the past decade, sweet corn breeders have developed hybrids with multiple Rp genes in order to combat this “new” race of *P. sorghi* (Fig. 10). The *RpG*, *Rp1E*, *Rp1I*, *Rp1K*, and *Rp5* genes have been used individually or in hybrid combination with *Rp1D* (i.e., different Rp genes coming from each inbred parent of a hybrid). Alternatively, some sweet corn hybrids carry “compound” rust resistance genes in which very closely linked Rp genes on the short arm of chromosome 10 occur in coupling phase and segregate as a

single gene (8). Presently, about two-thirds of the hybrids evaluated annually in the Illinois disease nursery have Rp resistance, of which nearly 50% is based on the *Rp1D* gene, 25% is based on other Rp genes, and 25% is based on the combination of the *Rp1D* gene and other Rp genes.

Since 2001, hybrids in the Illinois disease nurseries have been evaluated separately for reactions to three populations of *P. sorghi*: (i) “avirulent” isolates which do not have known virulence against the *Rp1D*, *RpG*, *Rp1E*, *Rp1I*, and *Rp1K* genes, (ii) “D-virulent” isolates which are virulent against the *Rp1D* gene as well as the *Rp1A*, *Rp1C*, *Rp1F*, *Rp1J*, *Rp4A*, *Rp4B*, and *Rp5* genes, and (iii) “G-virulent” isolates which are virulent against the *RpG*, *Rp1E*, *Rp1I*, and *Rp1K* genes. Comparing results from trials using D-virulent, G-virulent, and avirulent isolates identifies Rp-resistant hybrids with differential reactions (e.g., Table 2). These trials also provide an assessment of the background rust reaction of hybrids that may have ineffective Rp-resistance when virulence is present in naturally occurring populations of *P. sorghi* (e.g., Table 2).

Excluding hybrids with Rp-resistance, distributions of rust reactions of hybrids evaluated in the Illinois nurseries have remained similar during the past 27 years, as indicated by the lack of significant changes in z-scores of R, MR, M, and MS standard hybrids (Fig. 11). Very few sweet corn hybrids have high levels of partial rust resistance (i.e., general resistance or polygenic resistance); and partial rust resistance has remained relatively unimproved during the past quarter century. For example, rust ratings for the yellow, sugary enhancer hybrid Miracle ranked 1st, 11th, and 6th in the first three nurseries from 1984 to 1986 and 9th, 6th, and 9th in the last three nurseries from 2008 to 2010. Several other sugary enhancer hybrids have been among the group of hybrids with the best levels of partial rust resistance, although this resistance may be simply inherited from one of the original sources of the sugary enhancer trait, inbred IL677a (11).

Z-scores of the original rust susceptible standard hybrids (e.g., Florida Staysweet) decreased during the first decade of the nursery, possibly in association with the introduction of a large number of shrunken-2 hybrids in the early 1990s. The susceptibility of many of the shrunken-2 hybrids introduced during this period may have originated from the rust susceptible, sugary inbred Ia2132 (23). Inbreds FaHt32B and FaHt32C, developed in Florida by Emil Wolf, are rust susceptible, shrunken-2, sister-line conversions of Ia2132 (48). These inbreds also have the *Ht1* gene for resistance to *E. turcicum* (48). Many supersweet hybrids developed in the 1980s and 1990s had a 2132-derived, shrunken-2 inbred parent related to Fa32. Other supersweet hybrids introduced during this time were extremely susceptible to rust, such as two hybrids that became rust susceptible standards, Snow White and Sweet Magic.

Reactions to northern corn leaf blight. The *Ht1* gene discovered by Hooker and other simply-inherited Ht genes result in resistant reactions to NCLB with qualitatively distinct phenotypes (6,42). Lesions of NCLB are chlorotic and reduced in size when plants with the *Ht1*, *Ht2*, or *Ht3* genes are infected by *E. turcicum* race 0 (Fig. 12A). When plants with the *Ht1* gene are infected by *E. turcicum* race 1, which is virulent against the *Ht1* gene, NCLB lesions are necrotic (Fig. 12A). The *HtN* and *Hm1* genes, which delay the frequency of lesions and the rate of lesion development, produce a resistant phenotype that is not differentiated by qualitatively distinct, chlorotic lesions (Fig. 12B).

Chlorotic lesions typical of reactions conveyed by the *Ht1* gene against *E. turcicum* race 0 occurred on 25 to 45% of the hybrids in the Illinois disease nurseries during the past 20 years (Fig. 13). The *Ht1* gene lowers severity of NCLB by as much as two-thirds depending on the frequency of avirulent and virulent races of *E. turcicum* (27). Severity of NCLB is lower on the group of sweet corn hybrids with Ht1-reactions than on hybrids without the *Ht1* gene even in trials in which inoculum consists entirely of *E. turcicum* race 1 (Fig. 14). Thus, the use of *Ht1* and other Ht genes in sweet corn appears to occur frequently in combination with moderate to high levels of polygenic resistance (Fig. 12B). This is particularly true for hybrids with a 2132-derived, shrunken-2 in-

Fig. 11. Z-scores of A, common rust, B, northern corn leaf blight, and C, Stewart's wilt ratings for a set of standard hybrids included in the University of Illinois sweet corn hybrid disease nurseries from 1984 to 2010. Z-scores for common rust are based on hybrids that do not have Rp-resistance. Mean z-scores of resistant (R), moderately resistant (MR), moderate (M), moderately susceptible (MS), and susceptible (S) standard hybrids are given in parentheses.

bred parent because Wolf purposefully incorporated the *Ht1* gene and selected for polygenic NCLB resistance while converting the sugary inbred Ia2132 to its shrunken-2 version, Fa32 (48). The *Ht1* gene also can be found in hybrids that are very susceptible to *E. turcicum* in which case NCLB can be quite severe even when inoculum consists entirely of *E. turcicum* race 0 and lesions are chlorotic (Fig. 15).

Z-scores of standard hybrids with R, MR, M, and MS reactions to NCLB increased as much as 0.65 to 0.85 during the past quarter century, which is indicative of a shift toward greater levels of NCLB resistance among the group of hybrids being evaluated in the Illinois nurseries (Fig. 11). For example, hybrids that were originally considered to be among the most resistant (e.g., Florida

Fig. 12. Resistance to northern corn leaf blight (NCLB) conveyed by *Ht* genes. **A**, Chlorotic lesions of NCLB result from infection of plants with the *Ht1* gene by *Exserohilum turcicum* race 0, whereas infection by *E. turcicum* race 1 results in necrotic lesions. **B**, Delayed lesion development resulting from the *HtN* gene or the *Htm1* gene in combination with high levels of polygenic resistance (right) is easily distinguished from very susceptible NCLB reactions (left).

Fig. 13. Percentage of hybrids in University of Illinois sweet corn disease nurseries with chlorotic, Ht-reactions to *Exserohilum turcicum* race 0.

Fig. 15. When northern corn leaf blight susceptible sweet corn hybrids with the *Ht1* gene are inoculated with *Exserohilum turcicum* race 0, substantial leaf necrosis and large chlorotic lesions occur in spite of the single-gene resistance resulting in the chlorotic-lesion response.

Fig. 14. Distributions of severity (% leaf area symptomatic) of northern corn leaf blight (NCLB) on sweet corn hybrids with and without the *Ht1* gene in trials inoculated with *Exserohilum turcicum* race 0 or race 1. Data are from University of Illinois sweet corn hybrid disease nurseries in 2009 and 2010.

Staysweet and Crisp n Sweet 710) were grouped with moderately resistant hybrids by the mid-1990s when they were discontinued commercially. Replacing these hybrids as the most resistant to NCLB was a group of hybrids such as Day Star, Twin Star, Holiday, Tribute, Legion, Garrison, and ACX 7641W. In addition to

better levels of NCLB resistance among the most resistant hybrids, the shift toward higher levels of NCLB resistance probably was due in part to an increasingly larger proportion of shrunken-2 hybrids in the nursery. Supersweet hybrids with the *sh2* gene, particularly those derived from a 2132-background, tend to have greater resistance to NCLB than many other hybrids in the Illinois nurseries; whereas susceptibility to NCLB is more prevalent among many of the sugary hybrids grown primarily for processing. Z-scores of standard hybrids susceptible to NCLB (e.g., Jubilee) remained near 1.5 as hybrids in Illinois nurseries have shifted toward greater NCLB resistance.

Reactions to Stewart's wilt. Reactions to Stewart's wilt among sweet corn hybrids range from highly resistant to highly susceptible (Fig. 16); and the incidence of systemically infected plants increases as hybrid reactions go from R to S (3,18). Yield reductions due to Stewart's wilt are associated with systemic infection (3).

Hybrids with superior resistance to Stewart's wilt have been released in the past 25 years (e.g., Bonus, Mirai 336 BC, 179A, and Eliminator), while hybrids extremely susceptible to Stewart's wilt (e.g., Jubilee) have lost market share in areas where the disease was epidemic, particularly in the late 1990s. Despite these changes, the distribution of hybrid reactions to Stewart's wilt in the Illinois disease nursery appears to have remained unchanged for a quarter

Fig. 16. Reactions of sweet corn hybrids to Stewart's wilt range from highly resistant (left) to susceptible (right). Yield reductions are directly associated with the incidence of plants systemically infected by *Pantoea stewartii*.

Fig. 17. Susceptible and resistant reactions of sweet corn hybrids to maize dwarf mosaic, caused by *Maize dwarf mosaic virus* and/or *Sugarcane mosaic virus*, are differentiated by the incidence of symptomatic plants.

century. Z-scores of standard hybrids were unaltered from the first nurseries in the 1980s (Fig. 11). Three possible explanations for an overall lack of improvement in Stewart's wilt resistance in sweet corn are: (i) the geographic area over which the disease is extremely problematic is limited, (ii) levels of resistance in many hybrids are adequate to prevent yield reductions, and (iii) an increased use of neonicotinoid insecticide seed treatments has lowered the need for resistance.

Stewart's wilt does not occur or is a minor problem in many of the largest sweet corn growing regions of North America (e.g., Wisconsin, Minnesota, Florida, the Pacific Northwest, and New England). Improved levels of Stewart's wilt resistance would be of little or no value in hybrids grown in these regions. Likewise, many of the hybrids grown in areas where the disease is endemic have levels of resistance that prevent yield reductions, thus negating the need to breed for additional resistance. In areas where the disease is endemic (e.g., the DelMarVa peninsula, the Ohio River valley, and southern areas of the midwestern Corn Belt), the flea beetle vectors of *P. stewartii* can be controlled moderately well with neonicotinoid insecticide seed treatments (20,30). The reduction in the incidence of Stewart's wilt on hybrids treated with these insecticides is equivalent to about one level of host resistance, i.e., incidence of Stewart's wilt on an M hybrid treated with a neonicotinoid insecticide seed treatment equals that of an untreated MR hybrid (29). The widespread use of these insecticides in field corn very likely has reduced the incidence of Stewart's wilt dramatically throughout the North American Corn Belt. Although flea beetles

Fig. 18. Percentage of maize dwarf mosaic (MDM)-resistant hybrids in the University of Illinois sweet corn disease nurseries from 1997 to 2010. **A**, A range of reactions (resistant to moderate) among MDM-resistant hybrids is based on the incidence of symptomatic plants. **B**, The percentage of MDM-resistant hybrids that also have Rp-resistance to common rust has increased during the past decade.

may feed on other grass hosts after emerging from overwintering, a large proportion of these vectors of *P. stewartii* are killed after feeding on field corn seedlings grown from seed treated with systemic, neonicotinoid insecticides. Thus, a lower amount of Stewart's wilt occurs in sweet corn that is typically planted later than field corn.

Correlations between Stewart's wilt ratings and Goss's wilt ratings of 0.7 to 0.8 were observed among sweet corn hybrids evaluated in the Illinois nurseries from 1984 to 1986. Subsequently, correlations of a similar strength were observed when other materials (e.g., inbred lines or mapping populations) were evaluated for reactions to these two bacterial diseases. Weaker correlations ranging from 0.4 to 0.6 between Stewart's wilt ratings and NCLB severity have been observed consistently among hybrids in the Illinois nurseries. From 1984 to 1986 when Goss's wilt was evaluated in the nursery, correlations between Goss's ratings and NCLB severity also ranged from 0.35 to 0.55. Since all three pathogens invade vascular tissues, these phenotypic correlations may be indicative of a xylem-mediated mechanism that affects symptom development and host resistance.

Reactions to maize dwarf mosaic. Since the mid-1990s, hybrids in the Illinois disease nursery have been screened for reactions to maize dwarf mosaic (MDM) following inoculation with *MDMV-A* and/or *SCMV* (Fig. 17). The percentage of MDM-resistant hybrids has increased gradually from about 10 to 20% in the late 1990s to about 40% in 2009 (Fig. 18A). Concurrently, the percentage of MDM-resistant hybrids that also are Rp-resistant to

common rust has increased from about 50% prior to 2000 to over 90% since 2004 (Fig. 18B). Clearly, resistance to these two diseases is being selected in combination during backcross conversions of elite inbred lines or in the development of new inbreds. Resistances to both diseases are relatively simply inherited.

Most, if not all, MDM resistance in sweet corn is based on the *Mdm1* gene located on chromosome 6S and first identified in the field corn inbred line Pa405 (15). Mark Mikel and colleagues developed several MDM-resistant, sugary enhancer S₃ and S₄ lines from crosses involving IL677A and Pa405 (19). These lines have been important sources from which MDM resistance has been incorporated into commercial sweet corn hybrids. MDM resistance also has been incorporated into sweet corn from field corn inbreds Pa405, Oh7B, Oh514, and B68 and from Mexican lowland corn (P. M. Richter and W. F. Tracy, *personal communications*). Some of the tropical and semitropical sweet corns developed in Hawaii by Jim Brewbaker based primarily on the brittle-1 mutation rather than shrunken-2 also are excellent sources of MDM resistance in sweet corn.

Unidentified genes in addition to *Mdm1* appear to be involved in reactions of sweet corn hybrids to *MDMV-A* and *SCMV*. In a study by Jones et al. (10), MDM resistance was associated with the *Mdm1* gene or closely linked genes in 42 of 43 field corn lines, although other loci also affected resistant reactions. This also appears to be the case with sweet corn. Unlike Pa405, no sweet corn hybrid is known to produce 100% asymptomatic plants when inoculated at early seedlings stages with *MDMV* and *SCMV*. A mixture of symptomatic and asymptomatic plants occurs when MDM-resistant sweet corn hybrids evaluated in the Illinois nurseries were inoculated at early seedling stages (Table 4). Inoculated at later growth stages, plants of these hybrids were mostly asymptomatic. Incidence of symptomatic plants of MDM-resistant hybrids ranged from 0 to 75% in the nurseries, and consequently, MDM-resistant hybrids were classified from 1 to 7 (R to MS) based on incidence. In many cases, development of symptoms is delayed in plants of MDM-resistant hybrids compared to susceptible hybrids. Also, plants of MDM-resistant hybrids often display sectors of asymptomatic and symptomatic leaf tissue as if cell-to-cell movement of the virus is inhibited in meristematic tissues (Fig. 2).

Reactions to common smut. From 1992 to 1994, hybrids in the UI nursery were evaluated for reactions to *Ustilago maydis* (DC) Corda (= *U. zeae* (Beckm.) Unger) following inoculation in the silk channel with compatible sporidial isolates or following natural infection (31). Although hybrid reactions to smut could be separated within trials based on incidence and severity of kernel galls, hybrid reactions were inconsistent among trials. The basis for this inconsistency became evident when Lindsey du Toit observed that silk maturity and pollination reduced the incidence of kernel galls and Karen Snetselaar and colleagues demonstrated that kernels were protected from infection by the formation of a silk abscission layer within 24 h after fertilization (2,38). Because fertilized ovaries escaped infection after pollination, inoculation methods used in the UI nursery appear to have resulted in detection of subtle differences in maturity among hybrids at the time silks were inocu-

lated. In subsequent work, an association between the rapidity of silk abscission and smut resistance provided additional circumstantial evidence that silk dynamics and other aspects of maize floral biology play important roles in resistance to infection of kernels (34). These observations are consistent with a mechanism of resistance first proposed by Marion Griffiths in the 1920s that "resistance or susceptibility is largely a matter of relative accessibility of the susceptible parts to the invading organisms" (5). In other words, resistance to *U. maydis* appears to be conditioned by morphological and phenological characteristics that allow plants to avoid infection.

Although common smut is an important disease of sweet corn and hybrids differ in reactions to this disease, distinguishing resistant and susceptible hybrids in standard disease nurseries appears to be nearly impossible. Incidence of natural infection is an unreliable method to differentiate smut reactions of a large, diverse group of hybrids because natural infection is affected by plant maturity at the time environmental conditions are conducive for infection. Thus, disease reactions are confounded with hybrid maturity. Inoculation procedures evaluated to date have not alleviated this problem. Methods of inoculation that do not involve wounding plants or injection of compatible sporidia have been unreliable. Conversely, if plants are inoculated at the proper time with an injection method similar to the silk channel procedure, all hybrids are severely infected because avoidance mechanisms are circumvented. Thus, silk channel inoculation is an excellent method to produce edible galls of *U. maydis*, known as cuitlacoche, but it will not differentiate resistant and susceptible reactions (25,31,41). Presently, observations of the incidence of smut galls or an absence of the disease in multiple plantings over many environments is the most reliable way to determine if a sweet corn hybrid is resistant or susceptible to common smut.

Reactions to other diseases. Southern leaf blight, southern rust, ALB, GLS, and Goss's wilt occur sporadically on sweet corn in North America. Hybrid reactions to these diseases have been evaluated in the Illinois disease nurseries on an infrequent basis, usually following years in which the disease caused concern within the sweet corn industry. Therefore, a limited amount of information is available on hybrid reactions to these diseases, but due to limited resources, hybrids have not been evaluated as consistently or extensively for these "minor" diseases.

Reactions to postemergence herbicides. At the request of companies in the sweet corn seed and food processing industries, evaluations of hybrid responses to applications of postemergence herbicides were added to the Illinois sweet corn nursery in 2002. Hybrids were screened initially for reactions to carfentrazone, halosulfuron, and mesotrione, which represent three different classes of herbicide modes of action: protoporphyrinogen oxidase (PPO)-inhibitors, acetolactate synthase (ALS)-inhibitors, and 4-hydroxyphenylpyruvate dioxygenase (HPPD)-inhibitors, respectively. In subsequent trials, other ALS- and HPPD-inhibiting herbicides were included (e.g., foramsulfuron, nicosulfuron, primisulfuron, tembotrione, and topramezone) as well as bentazon, a photosystem II (site B)-inhibitor, and dicamba plus diflufenzopyr, a

Table 4. Incidence (%) of symptomatic plants of maize dwarf mosaic (MDM)-resistant and MDM-susceptible sweet corn hybrids inoculated at different growth stages in 2009 field trials

Reaction ^a	n	Incidence of MDM (%) and growth stage at inoculation					
		Mean			Range		
		2-3 leaf	4-6 leaf	7-9 leaf	2-3 leaf	4-6 leaf	7-9 leaf
R	10	4.6	0.8	0.7	1-9	0-5	0-5
R-MR	38	26.3	1.1	0.4	10-39	0-11	0-6
MR-M	27	57	4.4	0.6	40-79	0-31	0-9
M-MS	2	91.5	73	49	85-95	62-81	48-50
S	3	100	100	99	100	100	96-100

^a R, resistant; MR, moderately resistant; M, moderate; MS, moderately susceptible; S, susceptible.

growth regulator combination. Each of these compounds is metabolically detoxified in corn by cytochrome P450 enzymes. Cross-sensitivity of sweet corn to multiple P450-metabolized herbicides in the Illinois nursery and other trials led to the confirmation of a “super P450” as proposed initially by Barrett (1,22,46,47). The response of sweet corn hybrids to recommended usage rates of these herbicides is affected principally by the presence of functional or mutant cytochrome P450 (CYP) alleles at a single locus, or possibly, at one of four closely linked loci on chromosome 5S (22,33,43). Hybrids homozygous for functional CYP alleles are tolerant of the herbicides and usually do not show symptoms of injury. Hybrids homozygous for mutant CYP alleles are sensitive to the herbicides and can be severely injured or killed depending on the herbicide chemistry. Hybrids heterozygous for a functional and a mutant allele metabolize these herbicides at an intermediate rate and have phenotypes that are intermediate between tolerant and sensitive hybrids, although phenotypes of heterozygotes often are more similar to tolerant hybrids than sensitive hybrids (Fig. 19). Consequently, responses of heterozygous hybrids are more variable and tend to be affected by other factors such as environments, adjuvants, usage rates, and herbicide safeners (16,28,44,45). A common genetic basis for herbicide cross-sensitivity occurs in hybrids marketed across all segments of the sweet corn industry in North America (36). Based on evaluations in the Illinois sweet corn nurseries, fewer than 25 hybrids are sensitive (i.e., homozygous for mutant CYP alleles) but 20% or more of the hybrids evaluated in

the nursery since 2002 had intermediate responses indicative of heterozygous CYP genotypes.

Resistance within market segments. About 800 of the 3,700 hybrids evaluated in the Illinois nurseries are currently available commercially or are in pre-commercial stages of development. These hybrids are divided approximately into 15% sugary, 15% sugary enhancer, and 70% shrunken-2 endosperm types (Fig. 20). About 90% of the sugary hybrids have yellow-colored kernels, and most are grown for processing, i.e., canning or freezing. About 60% of the sugary enhancer hybrids are bi-colored, and 20% each have yellow or white kernels. The sugary enhancer types are grown primarily for fresh market, local consumption. Kernel color among the supersweet, shrunken-2 hybrids is divided about 60, 25, and 15% into yellow, bi-colored, and white, respectively. Supersweet hybrids are grown for all three primary uses: processing, fresh market local consumption, and fresh market shipping (e.g., grocery store chains).

Simply-inherited disease resistance occurs primarily among the sugary and shrunken-2 hybrids (Fig. 21). Nearly 70% of sugary hybrids and over 50% of shrunken-2 hybrids have Rp-resistance to common rust. Similarly, more than 50% of sugary hybrids and nearly 40% of shrunken-2 hybrids are MDM-resistant. Chlorotic-lesion responses typical of the *Ht1* gene occur in 40% of shrunken-2 hybrids and about 25% of sugary hybrids. Conversely, Rp-resistance occurs in less than 20% of sugary enhancer hybrids, and only 5% of sugary enhancer hybrids have MDM-resistance or *Ht1*-reac-

Fig. 19. Injury from cytochrome P450-metabolized herbicides on sweet corn plants that differ genetically for functional and mutant alleles at a cytochrome P450 locus on chromosome 5S. **A**, Injury from a 4-hydroxyphenylpyruvate dioxygenase (HPPD)-inhibiting herbicide, mesotrione: left to right: homozygous functional (tolerant), heterozygous (intermediate), and homozygous mutant (sensitive). **B**, Injury to ears from an acetolactate synthase (ALS)-inhibiting herbicide, nicosulfuron: left, homozygous functional (tolerant); right, heterozygous (intermediate). Plants homozygous for mutant alleles are killed by nicosulfuron and produce no ears.

Fig. 20. Percentage of sugary, sugary enhancer, and shrunken-2 hybrids with white, bi-color, and yellow kernel color among the 800 sweet corn hybrids evaluated in the Illinois nurseries from 1984 to 2010 that were available commercially or were in pre-commercial stages of development in 2010.

Fig. 21. Percentage of sugary (su), sugary enhancer (se), and shrunken-2 (sh2) hybrids with major gene resistance for common rust (Rp-resistance), maize dwarf mosaic, and northern corn leaf blight (Ht chlorotic reactions) among the 800 sweet corn hybrids evaluated in the Illinois nurseries from 1984 to 2010 that were available commercially or were in pre-commercial stages of development in 2010.

tions. Apparently, during the development of sugary enhancer hybrids, selection has focused primarily on high quality, eating characteristics rather than on simply-inherited disease resistance.

Polygenic disease resistance and susceptibility also vary among the endosperm types. Hybrids with polygenic rust resistance are rather rare among each of the endosperm types (Fig. 22A). Susceptibility to rust is somewhat more common among the sugary enhancer types, probably because Rp-resistance has been incorporated in many of the rust susceptible sugary and shrunken-2 hybrids (Fig. 21). Polygenic resistance to NCLB occurs in about twice the percentage of shrunken-2 hybrids as sugary or sugary enhancer hybrids (Fig. 22B). This may be due, partly, to the continued use of NCLB-resistant, 2132-derived, shrunken-2 inbreds. Over 40% of sugary hybrids are susceptible to NCLB. Many of

these hybrids are grown for processing in the Midwest where NCLB can be quite problematic. Clearly, this market segment would benefit from incorporation of higher levels of NCLB resistance in widely grown hybrids. Over 20% of sugary enhanced and shrunken-2 hybrids also are susceptible to NCLB. Within the shrunken-2 endosperm types, NCLB susceptibility is somewhat more prevalent among the relatively new, HQ-sh2, supersweet hybrids that usually combine two or more endosperm mutations. Improved levels of NCLB resistance might be particularly useful if the HQ-sh2 hybrids become more prominent in spring plantings in the Florida and Georgia shipper markets, where the disease is endemic. Levels of Stewart's wilt resistance that typically prevent yield reductions occur in about 17 to 27% of hybrids with various endosperm mutations (Fig. 22C). Susceptibility to Stewart's wilt is somewhat more prevalent among sugary enhancer hybrids than sugary or shrunken-2 hybrids, which is curious because hybrids with the sugary enhancer trait historically have been popular in an area where Stewart's wilt is endemic from the Ohio River valley eastward to the DelMarVa peninsula.

Summary

Host plant resistance often plays an important role in the control of diseases of sweet corn. Resistance prevents substantial reductions in yield and quality that occur when conditions are favorable for diseases to become severe (Fig. 23). Nevertheless, the commercial success or failure of a disease-resistant sweet corn hybrid is based largely on sales of seed. Ultimately, the widespread use and value of disease resistance in sweet corn is determined by free market forces, i.e., whether or not resistance increases seed sales.

Fig. 22. Percentage of sugary (su), sugary enhancer (se), and shrunken-2 (sh2) hybrids with resistant (1 to 3) or susceptible (7 to 9) reactions to: **A**, common rust, **B**, northern corn leaf blight, or **C**, Stewart's wilt among the 800 sweet corn hybrids evaluated in the Illinois nurseries from 1984 to 2010 that were available commercially or were in pre-commercial stages of development in 2010. (Common rust data excludes hybrids with Rp resistance.)

Fig. 23. Effects of severe maize dwarf mosaic (MDM) and common rust on an Rp- and MDM-resistant version and a susceptible version of the sweet corn hybrid Bodacious: **A**, ears from the resistant and susceptible version of the hybrid, **B**, one row each of the resistant and susceptible version of the hybrid in the Illinois disease nursery, and **C**, small plots of the resistant and susceptible version of the hybrid about 1 week after the fresh market harvest stage.

Hybrids with resistance to prevalent diseases have clear advantages over hybrids with susceptible or moderate disease reactions in certain segments of the sweet corn industry. For example, most of the sugary and many of the shrunken-2 hybrids grown for processing in the Midwest have Rp resistance to common rust and, in many instances, MDM resistance. This combination of resistance, or at least Rp resistance, usually is necessary for new hybrids to replace existing hybrids in that market.

While disease resistance is a necessary trait for hybrids in some markets, resistance alone will not sell a hybrid. In order to be widely grown, a hybrid must have several essential horticultural quality and agronomic traits which typically are of greater importance than disease resistance. These traits have been the focus of most sweet corn breeding programs.

Essential traits of sweet corn hybrids differ among the various uses and types of sweet corn (i.e., processing, local fresh market, fresh market shipping; yellow, white, bi-colored; sugary, sugary enhancer, shrunken-2). These traits also differ with an assortment of production practices used throughout different areas of North America and the world (e.g., machine versus hand harvest). Thus, sweet corn is an extremely diverse crop with many different market segments, each with a unique set of requirements for new hybrids. It should come as no surprise that the 500 to 600 sweet corn hybrids available for sale each year have a wide range of reactions to several diseases that may be important in some, but not all, of the market segments. Like other important traits, the need for disease resistance is market specific.

Inbreds with superior combining ability for several traits that are essential in various markets are used in multiple hybrids. While it is extremely difficult to develop an elite, superior inbred, it is very easy to lose that superiority when attempting to improve an elite inbred for a specific trait, such as disease resistance. Thus, sweet corn breeders have relied heavily on simply-inherited resistances when they are effective because simply-inherited resistance can be backcrossed into elite materials with less risk of losing the superiority of the recurrent parent. Improvement in polygenic resistance has been more difficult but may occur more frequently in the future as marker-assisted selection is used to improve elite sweet corn lines by incorporating quantitative trait loci with major effects for disease resistance. As new, improved inbreds are used in hybrid combinations, field trials will continue to be necessary to confirm the phenotypic performance of these new hybrids. Trials similar to the Illinois disease nursery will be required to continue to provide the sweet corn industry with documentation of the relative levels of resistance to economically important diseases among new sweet corn hybrids.

Acknowledgments

The University of Illinois sweet corn disease nursery has been supported financially and with gifts-in-kind by nearly every participating company in the sweet corn seed and food processing industries (Table 1). Grants from the Midwest Food Processors Association have supported the nursery since 1988. The number of people who have assisted with day-to-day operations is too numerous to mention, but their hard work has been appreciated greatly. The following people (listed chronologically) have co-authored annual nursery reports: Snook Pataky, John Headrick, Suparyono, John Gantz, Payam Fallah, Annette Meyer, Claude Nankam, Mike Kerns, Lindsey du Toit, Phil Michener, Noah Freeman, Molly Pate, Mirian Gonzalez, Andrea Campaña, Mohammad Babadoost, Loyd Wax, Christy Sprague, Tatjana Ledençan, Jon Nordby, Mark Bogner, Clint Mapel, Marty Williams, Ryan Hasty, John Frihauf, Valdimir Casteñada, Alicia Chávez, Mike Meyer, Bryan Warsaw, and Jim Moody.

Literature Cited

- Barrett, M., Bradshaw, L. D., Polge, N. D., Baerg, R. J., and Poneleit, C. G. 1994. Evidence for multiple herbicide metabolizing cytochrome P450 from maize. *Weed Sci. Soc. Am. Abstr.* 34:60.
- du Toit, L. J., and Pataky, J. K. 1999. Effects of silk maturity and pollination on infection of maize ears by *Ustilago maydis*. *Plant Dis.* 83:621-626.
- Freeman, N. D., and Pataky, J. K. 2001. Levels of Stewart's wilt necessary to prevent yield reductions in sweet corn hybrids. *Plant Dis.* 85:1278-1284.
- Gonzales, J. W., Rhodes, A. M., and Dickinson, D. B. 1974. A new inbred with high sugar content in sweet corn. *HortScience* 9:79-80.
- Griffiths, M. A. 1928. Smut susceptibility of naturally resistant corn when artificially inoculated. *J. Agric. Res.* 36:77-84.
- Hooker, A. L. 1963. Inheritance of chlorotic-lesion resistance to *Helminthosporium turcicum* in seedling corn. *Phytopathology* 53:660-662.
- Hooker, A. L. 1969. Widely based resistance to rust in corn. Pages 28-34 in: *Disease consequences of intensive culture of field crops*. Iowa Agric. Home Econ. Exp. Stn. Spec. Rep. 64.
- Hu, G., and Hulbert, S. H. 1996. Construction of 'compound' rust resistance genes in maize. *Euphytica* 87:45-51.
- Hulbert, S. H. 1997. Structure and evolution of the rp1 complex conferring rust resistance. *Annu. Rev. Phytopathol.* 35:293-310.
- Jones, M. W., Redinbaugh, M. G., and Louie, R. 2007. The *Mdm1* locus and maize resistance to *Maize dwarf mosaic virus*. *Plant Dis.* 91:185-190.
- Kim, S. K. and Brewbaker, J. L. 1987. Inheritance of resistance of sweet corn inbred IL677a to *Puccinia sorghi*. *HortScience* 22:1319-1320.
- Laughnan, J. R. 1953. The effect of *sh2* factor on carbohydrate reserves in the mature endosperm of maize. *Genetics* 38:485-499.
- Laughnan, J. R. 1961. Super sweet, a product of mutation breeding in corn. *Seed World* 13:18-19.
- Marshall, S. W., and Tracy, W. F. 2003. Sweet corn. Pages 537-569 in: *Corn: Chemistry and Technology*, 2nd ed. P. J. White and L. A. Johnson, eds. Am. Assoc. Cereal Chemists, Inc., St. Paul, MN.
- McMullen, M. D., and Louie, R. 1989. The linkage of molecular markers to a gene controlling symptom response in maize to maize dwarf mosaic virus. *Mol. Plant-Microbe Interact.* 2:309-314.
- Meyer, M. D., Pataky, J. K., and Williams, M. W., II. 2010. Genetic factors influencing adverse effects of mesotrione and nicosulfuron on sweet corn yield. *Agron. J.* 102:1138-1144.
- Michener, P. M. 2006. The relative efficiency of replicates in sweet corn hybrid disease nurseries and posterior probabilities as a measure of confidence in assigning sweet corn hybrids to disease reaction categories. Ph.D. thesis. University of Illinois, Urbana, IL.
- Michener, P. M., Freeman, N. D., and Pataky, J. K. 2003. Relationships between reactions of sweet corn hybrids to Stewart's wilt and incidence of systemic infection by *Erwinia stewartii*. *Plant Dis.* 87:223-228.
- Mikel, M. A., D'Arcy, C. J., Rhodes, A. M., Carey, E. E., and Juvik, J. A. 1983. Sugary (su) sweet corn germplasm with resistance to the maize dwarf mosaic virus. *HortScience* 18:964-965.
- Munkvold, G. P., McGee, D. C., and Iles, A. 1996. Effects of imidacloprid seed treatment of corn on foliar feeding and *Erwinia stewartii* transmission by the corn flea beetle. *Plant Dis.* 80:747-749.
- Munkvold, G. P., and Yang, X. B. 1995. Crop damage and epidemics associated with 1993 floods in Iowa. *Plant Dis.* 79:95-101.
- Nordby, J. N., Williams, M. M., II, Pataky, J. K., Riechers, D. E., and Lutz, J. D. 2008. A common genetic basis in the sweet corn inbred Cr1 for cross-sensitivity to multiple cytochrome P450-metabolized herbicides. *Weed Sci.* 56:376-382.
- Pataky, J. K. 1987. Reaction of sweet corn germplasm to common rust and an evaluation of Rp resistance in Illinois. *Plant Dis.* 71:824-828.
- Pataky, J. K., and Campaña, M. A. 2007. Reduction in common rust severity conferred by the *Rp1D* gene in sweet corn hybrids infected by mixtures of Rp1D-virulent and avirulent *Puccinia sorghi*. *Plant Dis.* 91:1484-1488.
- Pataky, J. K., and Chandler, M. A. 2003. Production of huitlacoche, *Ustilago maydis*: Timing inoculation and controlling pollination. *Mycologia* 95:1259-1268.
- Pataky, J. K., Gonzalez, M., Brewbaker, J. L., and Klopers, R. 2001. Reactions of Rp-resistant sweet corn hybrids to populations of *Puccinia sorghi* virulent on corn with the *Rp1-D* gene. *HortScience* 36:324-327.
- Pataky, J. K., and Ledençan, T. 2006. Resistance conferred by the *Ht1* gene in sweet corn infected by mixtures of virulent and avirulent *Exserohilum turcicum*. *Plant Dis.* 90:771-776.
- Pataky, J. K., Meyer, M. D., Bollman, J. D., Boerboom, C. M., and Williams, M. M., II. 2008. Genetic basis for varied levels of injury to sweet corn hybrids from three cytochrome P450-metabolized herbicides. *J. Am. Soc. Hortic. Sci.* 133:438-442.
- Pataky, J. K., Michener, P. M., Freeman, N. D., Weinzierl, R. A., and Teyker, R. H. 2000. Control of Stewart's wilt in sweet corn with seed treatment insecticides. *Plant Dis.* 84:1104-1108.
- Pataky, J. K., Michener, P. M., Freeman, N. D., Whalen, J. M., Hawk, J. A., Weldekidan, T., and Teyker, R. H. 2005. Rates of seed treatment insecticides and control of Stewart's wilt in sweet corn. *Plant Dis.* 89:262-268.
- Pataky, J. K., Nankam, C., and Kerns, M. R. 1995. Evaluation of a silk inoculation technique to differentiate reactions of sweet corn hybrids to common smut. *Phytopathology* 85:1323-1328.
- Pataky, J. K., Natti, T. A., Snyder, E. B., and Kurowski, C. J. 2000. *Puccinia sorghi* in Sinaloa, Mexico virulent on corn with the *Rp1-D* gene. *Plant Dis.* 84:810.
- Pataky, J. K., Nordby, J. N., Williams, M. M., II, and Riechers, D. E. 2006. Inheritance of cross-sensitivity in sweet corn to herbicides applied post-emergence. *J. Am. Soc. Hortic. Sci.* 131:744-751.
- Pataky, J. K., and Richter, P. M. 2007. Silk abscission in two sweet corn (*Zea mays* L.) hybrids that differ in susceptibility to common smut infection of ears. *HortScience* 42:1409-1412.
- Pataky, J. K., and Tracy, W. F. 1999. Widespread occurrence of common

- rust, caused by *Puccinia sorghi*, on Rp-resistant sweet corn in the midwestern United States. Plant Dis. 83:1177.
36. Pataky, J. K., Williams, M. M., II, Riechers, D. E., and Meyer, M. D. 2009. A common genetic basis for cross-sensitivity to mesotrione and nicosulfuron in sweet corn hybrid cultivars and inbreds grown throughout North America. J. Am. Soc. Hortic. Sci. 134:252-260.
 37. Pate, M. C., Pataky, J. K., Houghton, W. C., and Teyker, R. H. 2000. First report of *Puccinia sorghi* virulent on sweet corn with the *Rp1-D* gene in Florida and Texas. Plant Dis. 84:1154.
 38. Snetselaar, K. M., Carfioli, M., and Cordisco, K. A. 2001. Pollination can protect maize ovaries from infection by *Ustilago maydis*, the corn smut fungus. Can. J. Bot. 79:1390-1399.
 39. Suparyono and Pataky, J. K. 1989. Influence of host resistance and growth stage at the time of inoculation on Stewart's wilt and Goss's wilt development and sweet corn hybrid yield. Plant Dis. 73:339-345.
 40. Tracy, W. F. 1997. History, genetics, and breeding supersweet (*shrunk2*) sweet corn. Plant Breed. Rev. 14:189-236.
 41. Valverde, M. E., Fallah Moghaddam, P., Zavala-Gallardo, M. S., Pataky, J. K., Paredes-Lopez, O., and Pedersen, W. L. 1993. Yield and quality of huitlacoche on sweet corn inoculated with *Ustilago maydis* (DC) Corda. HortScience 28:782-785.
 42. Welz, H. G., and Geiger, H. H. 2000. Genes for resistance to northern corn leaf blight in diverse maize populations. Plant Breed. 119:1-14.
 43. Williams, M., Sowinski, S., Dam, T., and Li, B. L. 2006. Map-based cloning of the *nsf1* gene of maize. Program Abstr. 48th Maize Genetics Conf., Pacific Grove, CA.
 44. Williams, M. M., II, and Pataky, J. K. 2008. Genetic basis of sensitivity in sweet corn to tembotrione. Weed Sci. 56:364-370.
 45. Williams, M. M., II, and Pataky, J. K. 2010. Factors affecting differential sensitivity of sweet corn to HPPD-inhibiting herbicides. Weed Sci. 58:289-294.
 46. Williams, M. M., II, Pataky, J. K., Nordby, J. N., Riechers, D. E., Sprague, C. L., and Masiunas, J. B. 2005. Cross-sensitivity in sweet corn to post-emergence applications of nicosulfuron and mesotrione. HortScience 40:1801-1805.
 47. Williams, M. M., II, Wax, L. M., Pataky, J. K., and Meyer, M. D. 2008. Further evidence of a genetic basis for varied levels of injury to sweet corn hybrids from cytochrome P450-metabolized herbicides applied postemergence. HortScience 43:2093-2097.
 48. Wolf, E. A. 1978. Florida Staysweet. Circ. Fla. Agric. Exp. Stn. S-259.

**Jerald K. (Snook)
Pataky**

**Martin M.
Williams II**

John M. Headrick

Claude Nankam

Lindsey J. du Toit

**Phillip M.
Michener**

Dr. Pataky is a professor emeritus of plant pathology in the Department of Crop Sciences, University of Illinois. He earned B.S. degrees in advertising and agronomy and an M.S. degree in plant pathology from the University of Illinois. His Ph.D. in plant pathology is from North Carolina State University. During 28 years on the faculty at Illinois, Dr. Pataky taught more than 50 classes, including: introductory statistics, plant disease control, plant disease epidemiology, and diseases of field crops. His research focused on practical application of epidemiology and breeding for host plant resistance to control important diseases of sweet corn. He evaluated hybrid reactions in the Illinois sweet corn disease nursery for 27 consecutive years.

Dr. Williams is a weed ecologist with the USDA-ARS Global Change and Photosynthesis Research Unit at the University of Illinois. He earned a B.S. in biology at Truman State University and M.S. and Ph.D. degrees in weed science at the University of Nebraska. Prior to joining the USDA in 2003, Dr. Williams was an assistant professor of weed science at Washington State University. The objective of his research is to develop weed management systems that are economically viable and environmentally sound. During the past decade, Dr. Williams expanded the scope of the Illinois sweet corn disease nursery by incorporating herbicide evaluations.

Dr. Headrick is the Global Product Lead, Large Seeded Vegetable Crops, Monsanto Company, Seminis Vegetable Seeds, St. Louis, MO. He earned a B.S. in agronomy from the University of Missouri, M.S. and Ph.D. degrees in plant pathology from the University of Illinois, and an MBA from Washington University-St. Louis. During a career at Monsanto, Dr. Headrick has worked in fungicide development, transgenic improvement of small grain crops and field corn, and vegetable product development. During the first five years of the Illinois sweet corn disease nursery, Dr. Headrick helped develop and establish many of the inoculation and rating protocols and experimental procedures that have been used since.

Dr. Nankam is the Regional Food Security Adviser for Southern Africa for World Vision International, Malawi. He earned an Ingenieur Agronome Degree from the University

of Dschang, Cameroon, an M.S. in plant pathology from Iowa State University, and a Ph.D. in plant pathology from the University of Illinois. Dr. Nankam's career with Christian-based, non-government organizations, such as World Vision, has focused on improving agricultural production and food security in underdeveloped nations of Africa. Dr. Nankam suggested improvements in inoculation and rating protocols and helped with the transition to a 1 to 9 classification system implemented at the beginning of the second decade of the Illinois sweet corn disease nursery.

Dr. du Toit is an associate professor of plant pathology in the Department of Plant Pathology at Washington State University (WSU). She earned a B.S. honors in plant pathology from the University of Natal-Pietermaritzburg, South Africa. Her M.S. and Ph.D. degrees in plant pathology are from the University of Illinois. Dr. du Toit has a research and extension appointment at the WSU Northwestern Washington Research & Extension Center in Mount Vernon, where her research focuses on the epidemiology and management of diseases affecting small-seeded, vegetable seed crops in the Pacific Northwest. Dr. du Toit helped revise protocols for reporting reactions from multiple years of the Illinois sweet corn disease nursery and initiated a study using Bayesian theory to examine the number of trials necessary to effectively classify hybrid reactions.

Dr. Michener is a corn breeder and product development manager for Terral Seeds, Greenville, MS. He earned a B.S. in biology from the University of Evansville, and M.S. and Ph.D. degrees in plant pathology from the University of Illinois. At Terral Seeds, Dr. Michener oversees the corn breeding program as well as yield and disease testing programs at locations throughout the southern United States. Dr. Michener helped devise procedures to conduct trials using specific races of pathogens in the Illinois sweet corn disease nursery. Nursery procedures also were modified following Dr. Michener's use of a Monte Carlo simulation in combination with Bayesian theory to explore the relative efficiency of differing numbers of replicates and to determine the posterior probability associated with assigning hybrids to disease reaction categories based on differing numbers of trials.