Resurgence of Cucurbit Downy Mildew in the United States: A Watershed Event for Research and Extension

Gerald J. Holmes

California Polytechnic State University, San Luis Obispo 93407

Peter S. Ojiambo

North Carolina State University, Raleigh 27695

Mary K. Hausbeck

Michigan State University, East Lansing 48824

Lina Quesada-Ocampo

North Carolina State University, Raleigh 27695

Anthony P. Keinath

Clemson University, Charleston, SC 29414

In 2004, an outbreak of cucurbit downy mildew (CDM) caused by the oomycete *Pseudoperonospora cubensis* (Berk. & M. A. Curtis) Rostovzev resulted in an epidemic that stunned the cucumber (*Cucumis sativus* L.) industry in the eastern United States. The disease affects all major cucurbit crops, including cucumber, muskmelon, squashes, and watermelon. Although the 2004 epidemic began in North Carolina, the cucumber crop from Florida to the northern growing regions in the United States was devastated, resulting in complete crop loss in several areas. Many cucumber fields were abandoned prior to harvest. The rapid spread of the disease coupled with the failure of fungicide control programs surprised growers, crop consultants, and extension specialists. The epidemic raised several fundamental questions about the potential causes for the resurgence of the disease. Some of these questions revolved around whether the epidemic would recur in subsequent years and the possible roles that changes in the host, pathogen, and environment may have played in the epidemic.

2014 marked the tenth anniversary of the 2004 resurgence of CDM and offered a unique opportunity to assess our current understanding of this dynamic pathosystem and the sociological aspects of plant disease epidemics. Lessons from the 2004 CDM epidemic can be applied to other diseases that threaten our food supply and to how plant pathologists, growers, and stakeholder organizations can respond to mitigate losses.

CDM in the United States Prior to 2004

Cucurbit downy mildew is one of the most important diseases of cucumber worldwide, especially in regions with high rainfall and high relative humidity. The first report of the disease was from a noncultivated cucurbit in Cuba in 1868 (13). The earliest published record of the disease in the United States is from 1889 on greenhouse cucumber in New Jersey (27). Subsequently, CDM was found on field-grown cucumber (*Cucumis sativus* L.), pumpkin (*Cucurbita* spp.), and squash (*Cucurbita pepo* L.) in New Jersey and on cucumber

in Florida and Texas in 1889 (21,28). Ramsey et al. (62) reported that the disease was rare on watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai) and, although common on squashes, it was seldom a serious problem. In the 1940s, C. J. Nusbaum (51,52) conducted a study on the long-distance transport of *P. cubensis* using a network of plant pathologists who recorded the dates and places they detected the disease. From the 1940s to the 1960s, cucumber breeding efforts by W. Barnes led to the release of several cultivars that demonstrated a high level of resistance to CDM (7,8). This resistance, derived primarily from the cultivar Chinese Long and Plant Introduction (PI) 197087, was so effective that CDM on cucumber was largely controlled without the use of fungicides (9,72).

While *P. cubensis* was not absent from the southeastern United States, the cucumber crop did not require fungicide protection, and CDM on other cucurbits (e.g., squash) was managed successfully with fungicides. In Michigan, CDM had not been diagnosed on any cucurbit crop for decades (M. Hausbeck, *personal observation*). In Ohio, the disease would routinely appear late in the summer after most cucumber growers had harvested. Disease resistance was still effective in pickling and slicing cucumbers, and the disease was more of a problem on squash and pumpkin. On *Cucurbita* species, growers managed the disease with fungicides such as mefenoxam plus chlorothalonil or an ethylene bis-dithiocarbamate, such as mancozeb. Other states, including New York and Maryland, occasionally experienced CDM late in the growing season. Typically, fungicides were not necessary to reduce losses but when necessary, the disease was effectively managed with mefenoxam.

In the late 1990s, muskmelon and squashes were commonly affected by CDM in North Carolina, Georgia, and South Carolina. Effective new fungicides, such as propamocarb, were identified in yearly field trials on squash (32) and recommended to cucurbit growers. However, cucumber growers did not include these fungicides in their spray programs since CDM was not considered a threat to their crop. Thus, although fungicide efficacy information was available at the time of the regional epidemic in 2004, it was not used in the cucumber industry given the lack of disease on cucumber in previous years.

In 1998, a CDM forecasting effort was initiated at North Carolina State University based on a framework of the atmospheric transport

Accepted for publication 11 November 2014.

<http://dx.doi.org/10.1094/PDIS-09-14-0990-FE>
© 2015 The American Phytopathological Society

and dispersion models established by C. E. Main for tobacco blue mold (18) and the pioneering model launched by C. J. Nusbaum (51). A network of plant pathologists affiliated with land grant institutions was established throughout the United States to report new CDM outbreaks and disseminate forecasts using an Internet-based system (31,54). This new forecasting system was made possible because of the existing infrastructure of the tobacco blue mold forecasting system. At that time, CDM occurred primarily on squashes, and local cucurbit grower organizations and stakeholders could not financially support the forecasting effort. Eventually, lack of operating funds became so limiting that the forecasting effort was abandoned for a year in 2004; coincidentally, this was the year of the regional epidemic.

Resurgence

On June 3, 2004, G. J. Holmes, an extension plant pathologist at North Carolina State University, along with A. Thornton, county agent, identified *P. cubensis* on cucumber in a field in Sampson County, North Carolina. Symptoms on the plant were initially mistaken for spray burn (off-target, phytotoxic crop damage by a chemical application). Upon closer examination, the affected plants exhibited classic symptoms of CDM: angular, chlorotic leaf spots that became necrotic; and older foliage more severely diseased than younger foliage, causing a striping effect down the center of the plant row. The lower leaf surfaces revealed signs of the pathogen observable with a 20× hand lens, which allowed for a positive identification in the field (Figs. 1 and 2). Within a week, additional cucumber fields were diagnosed with CDM and the pathogen spread so rapidly that some fields were abandoned without harvesting. In response to the CDM outbreak on cucumber, growers turned to fungicides. Initially, mefenoxam- and strobilurin-based fungicides were applied but they proved to be ineffective, especially when applied postinfection.

The scenario that played out in North Carolina, i.e., late application of ineffective fungicides resulting in dramatic losses in cucumber yields, was also observed in other states. Extension specialists in neighboring states to the north (e.g., Virginia, Delaware, New Jersey, etc.) were warned about the disease, but growers were reluctant to take preventive action against the disease on such short notice and without certainty that CDM would in fact cause losses in their states. In 2004, aggressive *P. cubensis* strains also were found on cucumbers in Maryland and Delaware. Mefenoxam had little effect, and growers attempted to modify their fungicide programs based on the then current North Carolina trial results. In New York, pumpkin, rather than cucumber, was the crop primarily affected by CDM in 2004. While mefenoxam is currently ineffective at managing the

disease (57), it still was effective during the 2004 epidemic. Growers in Michigan escaped the effects of CDM that year.

By the end of the 2004 growing season, severe to complete crop losses occurred in Georgia, South Carolina, North Carolina, Virginia, Delaware, Maryland, and New Jersey. Many production fields were abandoned without harvesting (33). Even where growers applied fungicides, the disease was not effectively controlled despite multiple applications during the growing season (53). Many long-time cucumber growers feared that if this disease scenario repeated in 2005, they would be driven out of business. Growers turned to state- and county-based extension for assistance and answers. That winter, several meetings were held to specifically address growers' concerns. Attendance at these meetings quadrupled from that of previous years and cucumber growers expressed great interest in learning details about the history, biology, and management of CDM.

In Michigan, before 2005, the primary, yield-limiting pathogen on cucurbits was *Phytophthora capsici*, a soilborne oomycete responsible for plant death and fruit rot (25). Historically, cucumber production in Michigan rarely required fungicide applications unless the field was infested with *P. capsici*. Diseases with symptoms similar to those of CDM include angular leaf spot (caused by *Pseudomonas syringae* pv. *lachrymans*) and occasionally herbicide injury (e.g., paraquat). Throughout the previous decade, growers submitted symptomatic plant tissue samples to ensure that observed symptoms were not caused by CDM.

In early August 2005, the M. Hausbeck lab at Michigan State University was contacted regarding a foliar disease on cucumber. The symptoms were classic for downy mildew and the pathogen was identified by Amanda Gevens, a Ph.D. student at the time. Cognizant of the devastation wrought by CDM in the southeastern United States during the previous year, M. Hausbeck and A. Gevens immediately contacted county extension educators, growers, scouts, and cucurbit processors to alert the industry and provide management recommendations. Emergency meetings were held within the primary cucurbit growing regions to distribute fact sheets and to scout fields for the presence of CDM. Within 2 weeks of the initial CDM confirmation, the disease was confirmed across the entire state. Some Michigan growers had planted a second cucumber crop and the pathogen was detected on the cotyledons of recently emerged plants. One of these plantings was selected for a fungicide efficacy experiment to determine which fungicides would provide acceptable disease control in Michigan (22). Some growers were reluctant to expend resources to protect their cucurbit crops or did not understand the urgency and threat of the situation. Growers who aggressively sprayed fungicides on cucurbit crops that were newly infected were able to fully harvest the produce, whereas those who delayed their spray program suffered significant yield losses.

Fig. 1. Cucumber field in Sampson County, North Carolina, showing severe downy mildew on June 3, 2004. This was the first field where cucurbit downy mildew was detected in 2004. (Photo courtesy G. J. Holmes)

Fig. 2. Close up of downy mildew symptoms on cucumber from the field in Fig. 1. (Photo courtesy G. J. Holmes)

Whereas Ohio was prepared for an epidemic in 2005, the disease was not confirmed on cucumbers until July 2006 in Wayne County. Sally Miller at Ohio State University released an alert to growers the same day the disease was confirmed and worked closely with grower organizations to spread awareness. Despite their best efforts, growers suffered significant losses because available fungicides were ineffective at managing the disease, similar to North Carolina in 2004.

Response

Short-term. Late in the 2004 growing season, a fungicide efficacy trial was established with a grower cooperator at the epicenter of the initial outbreak in Sampson County, North Carolina. Fungicide treatments were selected based on previous CDM trials on squash and with input from growers. Due to the severity of the epidemics, cucumber seedlings were treated beginning at the four-true-leaf stage, with subsequent sprays applied every 5 to 7 days for a total of 11 applications. Nontreated plants and those treated with ineffective fungicides were severely stunted and chlorotic, while plants treated with effective fungicides were healthy, dark green, and produced good yields (Fig. 3). Results from this trial provided an important foundation for fungicide recommendations in subsequent years (77). Given their direct experience with failed management due to ineffective fungicide programs, growers were reluctant to implement control recommendations based on fungicide efficacy trials. Growers argued that even if fungicides were effective, the added cost of application would erode an already narrow profit margin for pickling cucumbers. Efficacy data from one or more growing seasons was required to convince growers that CDM could be managed successfully if effective fungicides were applied preventively and reapplied every 5 to 7 days.

Adjacent to the fungicide trial in Sampson County, North Carolina was a volunteer yellow summer squash plant that remained CDM-free, even though the disease had affected squash in the area in previous years. This raised the question of whether a new pathotype or race of *P. cubensis* had developed or been introduced to the region. This became one of the central questions that emerged from the epidemic and is addressed below in greater detail.

Fungicides. The 2004 CDM epidemic created a new market for products active against oomycete diseases. Consequently, many agro-chemical companies became very interested in establishing themselves as having the product(s) of choice. This led to intense scrutiny by many interested groups of data and recommendations from fungicide trials. This marketing opportunity led several companies to promote products that had little to no efficacy against CDM. A more normal equilibrium of testing viable candidate fungicides and scrutiny of results ensued only after several additional years of fungicide testing.

Since the 2004 CDM fungicide trial conducted on cucumbers in North Carolina, more than 140 trials on cucumber have been reported from the eastern United States through 2014 (based on a count of the number of published Plant Disease Management Reports, September 2014). In these performance trials, registered and nonregistered fungicides were evaluated in various combinations, rates, and application intervals to maximize crop protection and minimize fungicide inputs. While growers were initially reluctant to adopt new fungicide programs, such programs currently are more readily adopted once they are shown to be effective in research trials. University researchers gain growers' trust by consistently providing reliable information over several years. Such trusting relationships help to minimize the confusion that develops when growers are influenced by the bias of agrichemical salespeople. Appropriate checks and balances are in place when growers, university researchers, and the agricultural products and services industries exchange information freely.

Host plant resistance. Before 2004, host plant resistance to CDM was an exceptional success story for plant breeding; cucumbers were grown with minimal fungicide inputs. However, the 2004 epidemic stimulated cucumber breeders to re-establish durable CDM resistance as a top priority for cucumber-breeding programs. Initially, seed companies were blamed for the "loss of resistance" in their CDM-resistant cultivars, but this falsity was gradually replaced with an understanding of pathogen biology and the ability of *P. cubensis* to overcome host plant resistance. This gradual education took place at local and national meetings where unbiased and

Fig. 3. Cucumber (cv. Jackson) plants showing the effects of fungicide treatment on the day of the 5th and 10th fungicide application. Left column: nontreated; right column: fungicide treated (Tanos 50DF, 560 g/ha + Manzate 75DF, 367 g/ha alternated with Curzate 60DF, 224 g/ha + Bravo Weather Stik 6SC, 383 ml/ha). Top row: Photos taken on October 1, 2004; bottom row: photos taken on October 26, 2004. (Photos courtesy of G. J. Holmes)

knowledgeable scientists conveyed a clear and consistent message grounded in reality.

Numerous studies have evaluated cucurbit germplasm for resistance to CDM to identify new sources of durable resistance and to determine the genes and mechanisms moderating susceptibility (2,10,40,59). While disease resistance has been identified, breeders must integrate this into commercially acceptable cultivars. New cucumber cultivars (e.g., SV3462CS and SV4719CS from Monsanto) with improved resistance to CDM began to be released in 2012. However, the level of resistance in these new cultivars was not sufficient to supplant the use of fungicides. Host plant resistance is still a major component of CDM management programs, but currently must be complemented with fungicides in order to maximize yields (10). Given that fungicides are expensive, maximizing their efficiency through improved timing of their applications is an important component in the integrated management of CDM.

Disease forecasting for CDM. In 2006, Pickle Packers International, a stakeholder group for pickling cucumbers, funded a grant to develop the CDM forecasting system and to support fungicide efficacy trials. Smaller grower groups in six states and in the province of Ontario, Canada, also supported the CDM forecasting system. In 2008, funding from stakeholder groups was leveraged to compete for federal funding from the USDA to establish the Cucurbit Downy Mildew Integrated Pest Management Pest Information Platform for Extension and Education (CDM ipmPIPE) forecasting system.

The CDM ipmPIPE system was established primarily to provide end users with nearly realtime information and the flexibility to make management decisions for CDM (54). For example, the CDM ipmPIPE alert system allows registered users to receive disease alerts directly via mobile phones or e-mail when a new disease outbreak emerges within a given radius of their specified area of operation. This forecasting system derives from the biological understanding of *P. cubensis* as an airborne pathogen that can be disseminated over long distances via wind currents (53). Historically, wind speed and direction can be predicted fairly accurately and therefore, dissemination of *P. cubensis* sporangia from identified sources can also be predicted accurately. A network of approximately 40 state collaborators across 23 states in the United States and Ontario, Canada, reports the time and location of CDM outbreaks online via the project website (<http://cdm.ipmpipe.org/>). Wind speed and direction at inoculum sources determine where the inoculum is likely to be transported within the next 6 to 48 h. Specific environmental thresholds for a disease outbreak are based on a combination of predicted temperatures and durations of leaf wetness (3,50). Maps of cucurbit acreage are layered with predicted trajectories of spore transport and weather conditions (temperature and leaf wetness duration) to produce an estimate of risk for cucurbit growers along the predicted trajectory of spore transport (Fig. 4).

Pathogen biology. Answers to questions about the biology of *P. cubensis* were necessary to render forecasting more accurate, reliable, and efficient. For example, for how long do sporangia survive

Fig. 4. Sample forecast on April 24, 2007, from a source of cucurbit downy mildew in southeastern Florida. Top box: black star shows the source of disease; red line is the center of the spore trajectory; each red triangle represents where the trajectory will be at 6-h intervals. Bottom box shows the same trajectory in terms of its elevation.

as they are transported via air currents? How much inoculum is produced per unit area of infected cucumber foliage? How much inoculum is released from an infected field over the course of an epidemic? Although oospores are rarely found in nature, what role, if any, do they play in initiating epidemics? Can greenhouses serve as a “green bridge” during winters in areas where the host is absent during winter months? While these questions were often pondered prior to 2004, the resurgence of CDM intensified efforts to address and answer them. New research techniques in population biology and ecology coupled with availability of funding put answers to these questions within reach.

The resurgence of CDM in the United States prompted studies to determine possible changes or introductions of new biotypes into pathogen populations (16,61,64). Unfortunately, since *P. cubensis* was not intensively sampled before 2004, a direct comparison of pre-epidemic versus postepidemic populations was not possible. However, a characterization of the current pathogen populations infecting cucurbit crops in the United States and other countries

(61) revealed that identifiable subpopulations of the pathogen were found more frequently in cucumber than in other cucurbit hosts in some states in the United States (Fig. 5). Population genetics analyses also indicate that some states such as North Carolina and Florida have highly diverse *P. cubensis* populations compared with other regions (61). This information has been helpful in highlighting the importance of accounting for pathogen genetic diversity when developing regional and local management strategies for CDM. Population studies have also provided useful information on pathogenicity and virulence. A recent study by Cespedes-Sanchez et al. (11) showed populations in Michigan differed in pathogenicity from *P. cubensis* populations in South Carolina. Another study by Lebeda et al. (43) showed that virulence and pathogenicity could change for populations of *P. cubensis* in Europe from year to year.

Another effort prompted by the 2004 epidemic was the sequencing of the genome of a *P. cubensis* isolate collected from cucumber in Ohio (67). The genome provided much needed information to: i)

Fig. 5. Population structure of *P. cubensis* within the United States. Bayesian clustering analysis of 465 isolates revealed six genetic clusters present in the United States (each represented by a different color). Each isolate is represented by a vertical bar, which is partitioned into colored segments that represent the individual's proportionate genetic membership in a given cluster. Some genetic clusters (green, yellow, purple) were found more frequently in cucumber than in other hosts, and other clusters (blue) were found more frequently in noncucumber hosts than in cucumber.

develop markers for population genetics studies; ii) better understand the pathogen through comparative genomics; and iii) perform gene expression analysis to characterize host-pathogen interactions (2,68). For example, genomic analyses revealed over 2,000 genes that are differentially expressed by the pathogen during cucumber infection. Most of those genes become active within the first day of infection, indicating the importance of early host-pathogen interaction events (67).

Aerobiology of *P. cubensis*

Nusbaum's 1940s reports of CDM outbreaks along the Atlantic coast led to the assumption that *P. cubensis* can be dispersed over long distances (51,52) from overwintering sources in southern Florida. On the other hand, it was observed that CDM outbreaks in the eastern United States mainly follow the normal planting dates of cucurbits from south to north. However, given that *P. cubensis* is an obligate parasite and does not overwinter in areas above the 30th latitude, the south-to-north pattern of disease outbreaks is possible only if the pathogen is disseminated from overwintering sources each year. The presence of local inoculum in the form of oospores has not yet been documented as a possible source of initial inoculum in regions of the United States where *P. cubensis* is unable to survive during winters.

The epidemic of 2004 provides insight into the pathogen's potential for long-distance dispersal. The timing of subsequent disease outbreaks north of the affected fields in North Carolina followed the initial CDM confirmation in North Carolina, which was approximately 8 weeks earlier than normal. Moreover, the pathogen possessed similar phenotypic features (i.e., aggressive on commercial cucumber cultivars and resistant to mefenoxam- and strobilurin-based fungicides) wherever it occurred. The observation that severe epidemics of the disease were not reported in Michigan until 2005 provides anecdotal information on the extent of the spatial dissemination and establishment of this "new" strain of the pathogen within a single growing season.

Spore dispersal and transport. The resurgence of CDM in the United States in 2004 reignited efforts to address basic questions on the epidemiology and aerobiology of the disease pertaining to the long-distance dispersal from overwintering sources in the southern United States. Like many aerially transmitted plant pathogens, the long-distance dispersal of *P. cubensis* is fundamental to the spread of CDM from field to field for susceptible hosts. This attribute of the pathogen allows it to overcome management efforts that limit disease development via local control measures, such as crop rotation. Given that CDM must be reintroduced annually into northern states, questions to be addressed related to factors that affected the probability of dispersal of *P. cubensis*. The odds that CDM infections will occur in a given field are directly related to the concentration of viable spores in the air around a given cucurbit field. Aerial concentration is determined by spore dispersal, which is composed of i) number of spores released at the source field, ii) dilution of the spore cloud by turbulence, and iii) survival of spores during transport (4). Once determined, these aerobiological factors can be integrated with information on the concurrent temporal development of the disease to quantify infection probabilities and rates of disease spread at different spatial scales (5).

In response to the resurgence of CDM in 2004, the CDM ipmPIPE was established in 2008 with funding from the U.S. federal government (54). The project was designed to address aspects of the epidemiology of CDM and to provide answers to aerobiological questions that influenced the probability of transmission of *P. cubensis*. The pathogen is a prolific producer of spores and approximately 2×10^8 spores can be produced from 1 ha of a moderately diseased field of cucumber (49). A majority of these spores are usually liberated into the atmosphere between 0800 and 1100 h EDT (23,49). Production of spores at the source by *P. cubensis* is also dependent on disease severity, aerial spore concentration, and escapes from the canopy, and follows a lognormal distribution with a log-mean of 15% disease severity (49). An implication of the latter is that disease severity at the source is useful in estimating sporangial escape from

a source field, instead of direct measurement of spore escape in the field with spore traps. This is particularly important since quantification of spores is labor intensive, time consuming, and impractical for regional disease forecasting programs.

Spores liberated at a source must escape the canopy and move into the atmosphere above an affected field to be aerially transported over long distances to disease-free fields. The proportion of *P. cubensis* spores produced in the field that eventually escape the canopy into the atmosphere is approximately 17%, with the remaining 83% of the spores unable to escape (49). During transport in the atmosphere, the volumetric concentration of spores in the air can be reduced by atmospheric dispersion. Some spores are able to move far from their original source, and the probability that they will cause disease upon arrival at a distant host depends on their survivability during transport and the weather conditions at the point of spore deposition.

Solar radiation is the primary physical variable that negatively affects the survival of *P. cubensis* spores in the atmosphere, while temperature and relative humidity have only minimal effects on spore survival (37). The effect of solar radiation on spore survival of *P. cubensis* is best described by an exponential decay function with a solar radiation dose of about 29.5 MJ m^{-2} decreasing viability by 95%. The critical dose of irradiation before *P. cubensis* spores begin to lose viability is 9.5 MJ m^{-2} . This critical dose, which results in a 63% decrease in spore survival, is usually attained within 4.5 h of exposure on sunny days (37). Other oomycetes have lower critical doses of solar radiation. For example, the critical dose for *Phytophthora infestans* is 2.4 MJ m^{-2} (63), while that of *Peronospora tabacina* is 0.9 MJ m^{-2} (48). The exponential decay function for the effects of solar radiation on spore survival has now been integrated into spore transport models used in disease forecasting as a multiplier function to generate realistic estimates of the number of viable spores available for deposition to a disease-free cucumber field. This correction for spore survival is expected to improve the accuracy of forecasts for the risk of downy mildew outbreaks.

Epidemic spread. Given the assumption that *P. cubensis* can be disseminated over long distances through the atmosphere, a logical step was to determine the rate of spread of the disease in the eastern United States. Establishing the rate of spread of the disease would determine: i) whether disease spread over long distances is limited more often by pathogen establishment than by long-distance dispersal, and ii) if other possible overwintering niches for the pathogen exist in the eastern United States. These two aspects of the epidemiology of the disease can have significant implications in disease management. For example, confirmed records of the first observation of disease symptoms and signs of the pathogen (i.e., disease onset) on a distance-time continuum are routinely used as a measure of the effective dispersal of a pathogen (42).

As mentioned previously, a CDM disease-monitoring network was established in 2008 as part of CDM ipmPIPE to monitor and record onset of CDM in sentinel plots located in 23 states in the eastern United States from Florida to Connecticut (54). Sentinel plots were nonsprayed, fixed plots that were planted early and designated a priori for regular, visual surveillance (at least once a week) within the monitoring network. Weekly monitoring for CDM in sentinel plots was necessary to avoid underestimation of time of arrival of the pathogen in a plot by at least one latent period (i.e., 5 to 7 days for *P. cubensis*). The weekly monitoring schedule also minimized the possibility of the disease symptoms going unnoticed for a longer time. In subsequent work, first reports of disease onset were used to estimate the rate of northward spread of the disease wave front, assuming that the time lag between pathogen arrival and disease detection was the same in all locations. Rates of disease spread were calculated by dividing the maximum linear distance between two cases across the 24 states by the duration of the epidemic. Based on those calculations, the CDM wave front is expected to advance northwards every year at a rate of about 10 km/day (53). These rates of spread for CDM are comparable to those reported for tobacco blue mold estimated from disease onset data (6). Disease reports from the sentinel-plot network have also been pivotal in providing, for the first

time, some sense of uncertainty of the risk of CDM outbreaks in the United States (55). In Michigan, airborne spores are monitored using a spore trapping surveillance system. Since 2005, spore counts are reported weekly as a resource to help growers determine when to begin spraying. Once spores are consistently detected at levels of $>20/\text{m}^3/\text{h}$, growers are alerted and fungicide programs commence (24).

Pathotype or Race

Host specialization in *P. cubensis* was first studied in the United States by Doran (19) and Hughes and Van Haltern (35). Later, Thomas et al. (76) described five pathotypes based on compatibility between the pathogen and six host taxa. Eight isolates of *P. cubensis*—two from the United States, four from Japan, and two from Israel—were used to describe five pathotypes. Each pathotype was compatible with cucumber and muskmelon, but not compatible with other types of melons, watermelon, or squash and pumpkin (Table 1). Thomas et al. (76) did not include European isolates in their study. In subsequent work conducted in the Czech Republic (44), seven more cucurbit differentials were added to the set of five used by Thomas et al. (76). In the study conducted in the Czech Republic, it was observed that European populations of *P. cubensis* were highly variable and generally do not fall neatly into one of the five pathotypes described by Thomas et al. (76). In addition, 34 different host compatibility patterns (or pathotypes) were defined when 12 cucurbit hosts were inoculated with 42 isolates of *P. cubensis* (44).

Following the 2004 epidemic, Wehner (88) screened cucumber cultigens under field conditions in North Carolina and reported that CDM was aggressive on all host genotypes, rather than on specific genotypes. Since a novel race of the pathogen would show a genotype-specific reaction with *P. cubensis*, subsequent research efforts focused on establishing the pathotype(s) of this “new population.”

Colucci (15) used the 12 differential cucurbit hosts of Lebeda and screened 32 *P. cubensis* isolates collected from 11 states from 2004 to 2007. All 32 isolates produced a unique pattern of host compatibility, or 32 pathotypes. As reported in other studies in the United States (11,19,35,76), all isolates were also highly compatible with cucumber and *C. melo* subsp. *melo* (muskmelon). A majority of the isolates tested were also highly compatible with the remaining *Cucumis* spp., where 75% were compatible with *C. melo* var. *conomon* (honeydew melon) and 84% were compatible with *C. melo* var. *acidulous* (bitter melon). In addition, 81 and 72% of the isolates were compatible with two squash differentials, *Cucurbita pepo* var. *texana* and *C. pepo* subsp. *fraterna*, respectively. Similar to the findings in the Czech Republic (44), *C. pepo* var. *pepo* was the most resistant *Cucurbita* genotype, forming highly compatible reactions with only 7 of the 32 isolates (22%).

These results indicated that *P. cubensis* was highly variable in its compatibility with cucurbit hosts. However, the current system of describing pathotypes by their compatibility with 12 host differentials finds almost as many pathotypes as isolates. A more rapid and

reliable method is needed to consistently and accurately identify pathotypes of *P. cubensis*. Understanding host specialization in *P. cubensis* is an important area for future research that has implications for both basic and applied aspects of the cucurbit-*P. cubensis* pathosystem.

Ultimately, since we have very little information about the population structure of *P. cubensis* in the United States prior to the 2004 epidemic, we do not know if the 2004 epidemic was caused by a novel pathotype. Our working hypothesis is that a new biotype was introduced into the United States that was virulent on previously resistant cucumber genotypes and resistant to mefenoxam and strobilurin fungicides.

Lessons Learned

Not since the Southern corn leaf blight epidemic in 1970 and 1971 (78) has there been such a dramatic “failure” of host plant resistance. Despite efforts to mitigate the losses caused by plant diseases, pathogens adapt and overcome control measures. The durability and effectiveness of host plant resistance in cucumber led growers and researchers to become complacent about CDM and its potential to cause crop damage.

Rapid response. Extension plays a critical role in recognizing and combating crises in disease management. The ability to recognize an event as *atypical* requires experience and knowledge of what is *typical*. Inexperience leads to the pursuit of every lead where scarce resources are squandered and little is achieved. Over time, experience allows one to quickly discriminate what is worthy of pursuit from what is not. Likewise, rapid turnover in extension positions prevents accumulation of experience and increases the likelihood that a true crisis will go unrecognized.

The ability of *P. cubensis* to disseminate widely in a relatively short period of time requires a rapid response to protect cucurbits with fungicides prior to infection. Within two weeks, CDM can progress from a few scattered leaf spots to death of 90% of the foliage in a field of pickling cucumber. If a CDM epidemic is at an advanced stage of development, little can be done to salvage a crop, but alerting others of the threat can increase their chances of controlling the disease. In order to deal with a crisis effectively and promote adoption of control strategies, well-established and effective communication tools among growers, county-based extension educators, and state-based extension specialists are critical. The level of confidence and trust among individuals must be well established *before* a crisis in order to effectively partner *during* a crisis (70). Thus, establishing relationships should be among the first orders of business for county- and state-based extension personnel. For example, at the start of the epidemic, North Carolina and Michigan had online tools to quickly communicate outbreaks and fungicide efficacy results through the NC Pest News (http://ipm.ncsu.edu/current_ipm/pest_news.html) and the M. Hausbeck lab website (<http://veggies.msu.edu>). However, information on these sites was beneficial only to those growers who used them. Active and effective communication with stakeholders is a key necessity in the face of a destructive epidemic.

Portable, digital communication technology allowed rapid reporting of the 2004 and 2005 CDM outbreaks. In 2004, digital cameras were fairly common and photographs were used (30) to provide a rapid diagnosis of CDM over a wide area. Training in CDM diagnosis was accomplished rapidly via e-mail and the Internet using digital photographs showing characteristic symptoms. Diagnostic guides were also produced and distributed to help diagnose the disease on the major cucurbit hosts and communicate the importance of preventive fungicide applications for disease management (Fig. 6).

The challenge of funding research for specialty crops. The role of extension professionals is to address current and major problems within their assigned focus area. The use of modern communication technologies has fostered stronger, direct linkages between growers and extension specialists (36). When a new disease is detected, the scope of the research and extension effort often depends on the immediate or short-term funding available. Observing field symptoms,

Table 1. Compatibility of *Pseudoperonospora cubensis* pathotypes with cucurbit hosts^a

Host	Common name	Pathotype				
		1	2	3	4	5
<i>Cucumis sativus</i>	cucumber	+	+	+	+	+
<i>C. melo</i> var. <i>reticulatus</i>	cantaloupe	+	+	+	+	+
<i>C. melo</i> var. <i>conomon</i>	honeydew	-	+	+	+	+
<i>C. melo</i> var. <i>acidulus</i>	oriental pickling melon	-	-	+	+	+
<i>Citrullus lanatus</i>	watermelon	-	-	-	+	+
<i>Cucurbita</i> spp.	squashes	-	-	-	-	+

^a Table reproduced from Thomas, 1996 (75).

interacting with industry, and providing simple assessments of disease severity or incidence can be accomplished with minimal funding. In contrast, addressing fundamental questions similar to those raised following the 2004 epidemic of CDM requires a substantial investment in research funding.

In the case of the CDM epidemic, short-term, year-by-year, contract funding was obtained from agro-chemical companies to test registered and experimental fungicides for their efficacy against CDM. In company-sponsored performance trials, specific treatment protocols are usually followed. In some instances (e.g., numbered compounds in early development), restrictions on publication or presentation of results are mandated. The IR-4 Project supported the registration of several promising experimental fungicides for CDM some of which will soon be commercially available (e.g., oxathiapiprolin).

Established applied research programs often have access to fungicide products and grower cooperators, which facilitates quick establishment of an emergency fungicide efficacy trial. In 2004 and 2005, on short notice, North Carolina and Michigan growers provided fields of pickling cucumber in the heart of affected areas for fungicide evaluations (22,77). Time was devoted to this project after the recognition that this was a true crisis that could not wait another year for a response. Any extension program that was not already active in downy mildew research and did not have strong ties to county-

based extension and the agro-chemical industry was not able to mobilize a quick response.

Regional cooperation among the authors of this Feature article and other vegetable pathologists in the eastern United States began in 2006 with funding from the USDA Southern Region IPM Center to monitor pathotypes and fungicide sensitivity of *P. cubensis* (15). This project included two sets of field plots in seven locations, one set with 12 differential cucurbit genotypes and another set with pickling cucumber sprayed with four fungicide treatments (38,46). Data collected as part of this project provided the necessary justification to obtain the national funding awarded in 2008 for the CDM ipmPIPE research and extension project.

USDA competitive grant programs require applicants to involve stakeholders (e.g., growers) in projects awarded for funding. A strong and active collaboration on applied research, on-farm experiments, or extension outreach is often a requirement for receiving a grant award. Commodities and states with active grower organizations theoretically have an advantage over commodities and states with no organized grower groups. Established relationships among researchers and commodity boards increase the likelihood of funding. In addition to funding, grower groups can provide forums, newsletters, and websites for information delivery, assist with contacting members to increase outreach, and provide matching funds for research and extension.

Fig. 6. Symptoms of downy mildew on four major cucurbit hosts at early, intermediate, and late stages of disease development. Photographs such as these were used to assist growers and county educators in diagnosing downy mildew while emphasizing the importance of early detection and preventive fungicide applications. (Photos Courtesy of G. J. Holmes)

Documenting economic impact. The decision to shift federally provided extension funds from formula funds to competitive grants means that sufficient economic justification is required to obtain funding (20). Up to 100% reduction in yield is possible when CDM strikes early and fungicides are not used (77). If fungicides are applied one week after symptom appearance, yield is reduced approximately 50% (17) (Fig. 7). As early as 1897, Stewart (73) noted the dramatic impact that CDM has on the marketable yield of cucumber: “after the disease is once thoroughly established, very few cucumber are produced, although the plants may continue to grow profusely. The few cucumbers which are formed grow slowly and become misshapen so that they are unsaleable.” Colucci (15) showed that yield loss is strongly and significantly correlated to the area under disease progress curve (Fig. 8).

In the early years of CDM resurgence, growers shared estimates on yield losses and costs associated with protecting their crop from the pathogen. Through the USDA, National Agricultural Statistics Service (NASS) yearly crop reports (78,79), CDM epidemics can be associated with yield reductions to illustrate economic impacts. For example, pickling cucumber yields in North Carolina were 13.5 to 14.0% lower during 2004 and 2005 (years with CDM epidemics), compared with 2003 (prior to pathogen resurgence). In South Carolina, pickling cucumber yield was 30.3% lower in 2005 than in 2003 to 2004 (Table 2). The national figures show an 8.4% mean reduction in yield per hectare in 2005 and 2006 relative to 2003 and 2004. In 2006, dramatic reductions in acreage planted to cucumber of 43 and 31% occurred in North Carolina and South Carolina, respectively, reflecting growers who “gave up” trying to manage CDM.

Indirect evidence of economic impact can be found in the USDA NASS Agricultural Chemical Use Database by examining the data on fungicides recommended for CDM (80). The acres treated with these fungicides may provide an estimate of the acres requiring protection from CDM. Although the database does not include disease information, trends relevant to CDM are apparent between 2002 and 2010 for pickling cucumber (Table 3). For example, the amount of chlorothalonil applied per year and the acres treated with chlorothalonil increased by 2.3 (United States) and 9.3 (Michigan) times between 2002 to 2004 and 2006 to 2010. Chlorothalonil is widely recommended as an early-season protectant against CDM and mixed with CDM-specific fungicides. Likewise, pickling cucumber acres treated with mancozeb in the United States increased by 9.3 times during the same period. Although mancozeb was not applied to pickling cucumber in Michigan or North Carolina in 2002 or 2004, it was used on 78 and 32% of the acreage, respectively, in 2010. The greater use of mancozeb in Michigan compared with North Carolina reflects the impact of

the product’s 5-day preharvest interval on harvest practices, which differ between the two states. In Michigan, the crop is harvested once because growers utilize a mechanical harvester, whereas North Carolina growers hand-harvest at 2- to 3-day intervals. Thus, it is easier to schedule the use of a fungicide with a relatively long preharvest interval if the crop is harvested once. The fungicide propamocarb was first registered on cucurbits in the United States in 2005 and by 2006 it was applied to 78 and 41% of the pickling cucumber acreage in Michigan and the United States, respectively (Table 3).

The economic value of a crop determines in large measure the level of awareness of specific disease epidemics. For example, the same year that CDM was found on pickling cucumbers previously resistant to the disease, Asian soybean rust (ASR) was found at the end of the growing season (November 2004) in Louisiana, the first detection in the United States. Thirteen months later, EPA had approved 20 fungicides for treating ASR on soybeans, including 12 that had emergency exemptions (79). In contrast, a search of the Federal Register found no pesticide tolerances approved for CDM fungicides in 2005 or 2006, one and two years after CDM was found in the southeastern and midwestern United States, respectively. Fortunately, four new fungicide active ingredients with activity against CDM: famoxadone, cymoxanil, fenamidone, and propamocarb hydrochloride, had been approved for use in 2003 and 2004 (79–86).

Current Research and Outlook

The resurgence of CDM represents a significant threat to cucurbit production in the United States. Although host resistance provided protection until 2004, severe epidemics have subsequently occurred each year. Whether this resurgence of the disease is due to a change in the local pathogen population or entry of a new population into the United States, climate change that has allowed for expansion of the geographic area of the pathogen, or a combination of these factors is currently unknown. Nevertheless, significant progress has been made in understanding host-pathogen interactions, the genetic structure of pathogen populations, environmental factors that affect the spread and outbreak of CDM, and resistance management approaches to improve the efficacy of fungicides.

Overwintering of *P. cubensis*. The initial source of *P. cubensis* inoculum in the United States has been a central focus of epidemiological studies. Accurate knowledge on the sources of initial inoculum at the start of the growing season increases the efficiency of disease monitoring and improves the timing of fungicide applications. The latter significantly improves the efficiency of fungicides in the control of CDM. Like other downy mildew pathogens, *P. cubensis* is an obligate parasite that can only survive within its living host or

Fig. 7. Example of a PowerPoint slide presented to growers showing the dramatic impact that delaying two different fungicide programs has on yield. In the case of mixing and alternating fungicides (Tanos + Manzate alternated with Previcur Flex + Bravo), a delay of 7 days (3 applications) resulted in a 54% reduction in yield compared with 4 applications. A delay of 14 days (2 applications) yielded the same as the nontreated (0 applications).

Fig. 8. Correlation data between disease severity (Area Under Disease Progress Curve; AUDPC) and weight of marketable yield from the delay of fungicide application experiment in 2006 in Sampson County, North Carolina.

as resting oospores as a result of sexual reproduction, which usually occurs at the end of the growing season (47). In the United States, cucurbit hosts can only survive year-round south of the 30th latitude in southern Florida and along the Gulf of Mexico, where winters are mild and this allows for cultivation of cucurbits or survival of wild cucurbits. Based on yearly epidemic trends, inoculum for initial CDM infection of cucurbits in the continental United States originates every year from overwintering sources in southern Florida and moves north during the growing season. A recent study based on disease records collected within the sentinel plot network of the CDM ipmPIPE linked disease occurrence in southern Florida to CDM outbreaks in northern states (53). Greenhouse crops of cucurbits grown as winter crops could also potentially serve as an important source of primary inoculum for fields (58). However, systematic studies are still needed to directly link inoculum production in greenhouses to disease outbreaks in local fields in the United States. Cucurbit weeds and wild hosts have also been shown to harbor the pathogen, but their role in overwintering in the United States is still unclear (65,87).

Production of oospores for *P. cubensis* has been reported in Israel (14), China (12,90), and India (71). Previous efforts to find oospores in cucurbit fields or under controlled conditions in the United States were not successful. Recently however, Thomas et al. (74) reported, for the first time, the presence of A1 and A2 mating types of *P. cubensis* and production of oospores under controlled conditions in the laboratory. The oospores were observed in crosses between opposite mating types for isolates originating from the southeast, mid-Atlantic, and Great Lakes regions. In that study, the A1 mating type was primarily associated with cucumber, while the A2 mating type was associated with cucurbits other than cucumber (74). Thus, it is likely that *P. cubensis* could overwinter as oospores in the United States in the absence of its living host. Planting cucumber in close

proximity to other cucurbits would increase the likelihood of sexual recombination and production of oospores. Thus, it is conceivable that oospores could serve as a local inoculum source at the start of the growing season in areas north of the 30th latitude in the continental United States. Studies are underway to determine the viability of the oospores and their survival under different simulated field conditions. Additional studies have also been initiated to investigate whether oospore production occurs under field conditions.

Early detection and diagnostics. Whole-genome sequencing of several *P. cubensis* isolates is key to generating markers, such as single nucleotide polymorphisms or simple sequence repeats, which will be useful for disease diagnosis and analysis of pathogen population structure. Efforts are underway to sequence *P. cubensis* isolates from diverse cucurbit hosts, including wild cucurbit hosts (89), which will facilitate development of robust and specific molecular diagnostics. Diagnostic markers are desirable, since accurate and timely identification of CDM in noncucumber hosts (e.g., watermelon) can be difficult (Fig. 6). In addition, developing molecular diagnostics would allow for pathogen detection prior to symptom onset via spore traps. Delivering a user-friendly diagnostic tool to stakeholders, such as those available for *Phytophthora* species (45), would significantly improve diagnostic capabilities for CDM.

Breeding. Host-pathogen transcriptome analyses (2,67) coupled with the increasing cucurbit genomic resources (26,34,60) has allowed for identification of pathogen genes and host markers that could be used to accelerate breeding efforts. Linking a *P. cubensis* genotype to a specific host phenotype is also becoming feasible for *P. cubensis*. Systems for in planta culture and long-term storage of *P. cubensis* are now well established, which allows for characterization of traits such as virulence that can later be associated to genotypic data via genome sequencing. Characterizing host-pathogen interactions and identifying pathogen genes that allow it

Table 2. Production and yield of pickling cucumber in the eastern United States from 2003 (prior to cucurbit downy mildew epidemics) to 2008^a

Location	Area harvested (ha)						Production (Mg/kg)					
	2003	2004	2005	2006	2007	2008	2003	2004	2005	2006	2007	2008
Michigan	13,568	13,973	15,390	13,446	14,175	12,353	164,076	156,458	165,437	156,584	168,249	171,514
N. Carolina	7,047	6,683	6,480	3,848	4,334	3,807	72,596	62,855	62,402	34,466	38,339	38,366
S. Carolina	1,620	1,620	1,661	1,134	972	851	16,689	20,680	13,015	10,920	8,707	9,524
United States	48,114	45,968	44,753	41,715	41,108	39,123	588,126	538,649	489,853	458,207	490,896	514,360

^a Data taken from United States Department of Agriculture, National Agricultural Statistics Service (79,80).

Table 3. Use of selected fungicides on pickling cucumber with activity against cucurbit downy mildew in the eastern United States between 2002 and 2010^a

Fungicide	Location	Amount of fungicide applied (kg/ha)				Percentage of hectares treated			
		2002	2004	2006	2010	2002	2004	2006	2010
Chlorothalonil	Michigan	9,192	3,363	61,543	79,927	12	4	71	94
Chlorothalonil	North Carolina	nd ^b	23,653	14,237	16,142	nd	71	64	42
Chlorothalonil	United States	41,589	45,064	100,105	148,084	16	23	50	79
Mancozeb	Michigan	nd	nd	88,111	88,671	nd	nd	65	78
Mancozeb	North Carolina	nd	nd	* ^c	5,493	nd	nd	*	32
Mancozeb	United States	14,349	1,569	99,993	111,203	9	1	45	58
Propamocarb	Michigan	nr ^d	nr	43,719	42,150	nr	nr	78	95
Propamocarb	North Carolina	nr	nr	*	6,838	nr	nr	*	40
Propamocarb	United States	nr	nr	45,849	64,345	nr	nr	41	69

^a Data taken from United States Department of Agriculture, National Agricultural Statistics Service (81).

^b nd = no data. Fungicide use was not reported in that year.

^c Too few respondents to report values, but value was >0.

^d nr = not registered. Fungicide was not yet registered in that year.

to infect and cause disease will facilitate development of durable host resistance.

Chemical control. In a synthesis of fungicide efficacy trials conducted between 2000 and 2008, it was observed that fungicide efficacy was significantly lower during the 2004–2005 season than the season prior to the resurgence of the disease (56). It was also observed that early and frequent fungicide applications (every 5 to 7 days) are required for maximum control when environmental conditions favor CDM. In addition, the 5- to 7-day preharvest interval required for some fungicides limits product choices.

The high potential of *P. cubensis* to develop resistance to fungicides has made chemical control challenging (66). In the United States, *P. cubensis* is resistant to mefenoxam, strobilurins, and mandipropamid (38,46,57). In recent years, new fungicides including fluopicolide (Presidio, Valent Corporation, Walnut Creek, CA) and cyazofamid (Ranman, FMC Corporation, Philadelphia, PA) were registered and play an important role in CDM management. In 2008, the fungicide program of cymoxanil plus famoxadone (Tanos 50DF, DuPont, Wilmington, DE) + mancozeb alternated with propamocarb (Previcur Flex 6F, Bayer CropScience, Research Triangle Park, NC) + mancozeb provided only 40% control of CDM on pickling cucumber, a significantly lower level of control than observed in earlier studies (39). Results from 2012 and 2013 trials in Georgia (41), Michigan (29), and North Carolina (1) indicate that fluopicolide has reduced field performance against *P. cubensis* compared with previous years. Judicious mixing and rotation of fungicide active ingredients and yearly evaluation of registered fungicides and those in development are key to integrating new chemistries into existing disease control programs and prolonging the efficacy of these fungicides. Populations of *P. cubensis* in major production regions should be continually monitored for the development of fungicide resistance, so that growers can be alerted immediately if and when key fungicides are no longer effective against CDM. To this end, establishing baseline sensitivity of all active ingredients, especially prior to launch, is essential to determining when a shift in sensitivity has occurred.

Improving disease forecasting. Growers use fungicides intensively to limit the progress of CDM. This drives the need for disease forecasting to determine “if” and “when” to apply fungicides. Judicious fungicide scheduling can also reduce the development of fungicide resistance and negative impacts of fungicides on the environment. For example, fewer fungicide applications may be needed in the mid-Atlantic and northeastern regions of the United States if arrival of the pathogen into these regions can be predicted based on CDM monitoring and detection in the southeastern region (54). The CDM ipmPIPE forecasting system has had a clear impact on the management of CDM (54). However, it has not been demonstrated that inoculum produced at a known source location is responsible for a disease outbreak at another, distant location. Thus, efforts are currently underway to validate the disease forecasting system in seven states in the eastern United States where CDM is an annual problem. Validation involves inoculum detection by trapping airborne spores and collecting and testing rainwater and relating spore detection to predicted spore arrival and observations of first disease symptoms in sentinel plots within the CDM ipmPIPE network.

Decision support systems are also needed to guide in-season application of fungicides after the initial spray is recommended. Currently, growers initiate their spray program based on historical dates of first CDM detection, CDM tracking information from the CDM ipmPIPE, or local atmospheric sporangial concentrations, as is the case in Michigan. Subsequent spray applications are made using a prophylactic or calendar-based approach, which can be inefficient when environmental conditions do not favor the development of CDM. Recently, a project was launched to develop a model to predict within-season infection risk of cucurbits by *P. cubensis* to guide fungicide applications (P.S. Ojiambo and A. Keinath, *personal communication*). When completed, this within-season model will represent a final step in developing a comprehensive decision support system for CDM to facilitate rational fungicide applications at the start as well as during a growing season. This is expected to decrease costs associated with managing CDM using fungicides and

reduce the risk of *P. cubensis* to rapidly develop resistance to currently used fungicides.

Future focus. Research is underway to answer several key questions. For example, what is the role of natural (i.e., wild host) or artificial reservoirs (i.e., greenhouses) in disease epidemiology, what is the overwintering potential of the pathogen, and how can host resistance and chemical control be preserved in a rapidly evolving and diverse pathogen such as *P. cubensis*? Answering these and other related questions is expected to lead to the development of effective and sustainable disease management measures for CDM, thereby protecting the U.S. cucumber industry. Synergistic partnerships among academics, industry, and funding agencies will be needed to blend applied and fundamental research to generate solutions that can be readily implemented. Ideally, durable genetic resistance would be incorporated into varieties with desirable horticultural traits. New genome editing technologies such as clustered regularly interspaced short palindromic repeats (CRISPR) could provide a way to transfer disease resistance to preferred cultivars (69). Nonetheless, acceptance of genetically modified vegetables and the high cost of developing a CRISPR system for cucurbits remains an obstacle. For now, an integrated management strategy that combines CDM alert systems, cultural practices, host plant resistance, and chemical controls is needed in order ensure the long-term economic viability of U.S. cucumber growers.

Acknowledgments

Research in the Holmes and Ojiambo laboratories was supported by grants from the USDA Pest Information Platform for Extension and Education (PIPE) program Award 528923-02208, USDA National Institute of Food and Agriculture; Regional IPM Competitive Grants Program (Southern Region) Award No. 2010-41530-21134, 201-34103-19622, and 201-34103-19623 and Pickle Packers International. Research in the Hausbeck laboratory was supported by Pickle Packers International, and the Michigan Pickle and Pepper Ad Hoc Group, and Project GREEN (Michigan State University AgBioResearch). Research in the Quesada laboratory was supported by Pickle Packers International, the USDA Animal and Plant Health Inspection Service (APHIS) Award 13-8130-0254-CA, and the USDA North Carolina Department of Agriculture (NCDA) Specialty Crop Block Grant Program (SCBGP) Award 12-25-B-16-88. This material was based upon work supported by NIFA/USDA under project numbers NC02418 and SC-1700446.

Literature Cited

1. Adams, M. L., and Quesada-Ocampo, L. M. 2014. Evaluation of fungicides for control of downy mildew on cucumber, Kinston 2013. Plant Dis. Manag. Rep. 8:V240. Online publication.
2. Adhikari, B. N., Savory, E. A., Vaillancourt, B., Childs, K. L., Hamilton, J. P., Day, B., and Buell, C. R. 2012. Expression profiling of *Cucumis sativus* in response to infection by *Pseudoperonospora cubensis*. PLoS ONE 7:e34954.
3. Arauz, L. F., Neufeld, K. N., Lloyd, A. L., and Ojiambo, P. S. 2010. Quantitative models for germination and infection of *Pseudoperonospora cubensis* in response to temperature and duration of leaf wetness. Phytopathology 100: 959-967.
4. Aylor, D. E. 1986. A framework for examining inter-regional aerial transport of fungal spores. Agric. For. Meteorol. 38:263-288.
5. Aylor, D. E. 1996. Comparison of the seasonal pattern of airborne *Venturia inaequalis* ascospores with the release potential of *V. inaequalis* ascospores from a source. Phytopathology 86:769-776.
6. Aylor, D. E. 2003. Spread of plant disease on a continental scale: role of aerial dispersal of pathogens. Ecology 84:1989-1997.
7. Barnes, W. C. 1948. The performance of Palmetto, a new downy mildew resistant cucumber variety. Proc. Am. Soc. Hortic. Sci. 51:437-441.
8. Barnes, W. C. 1966. Development of multiple disease resistant hybrid cucumbers. Proc. Am. Soc. Hortic. Sci. 89:390-393.
9. Beste, C. E., Dively, G., Everts, K., Linduska, J., Fournier, A., and Brown, A. 2000. Crop Profile for Cucumbers (Pickling) in Maryland. National Information System for the Regional IPM Centers. Online, <http://www.ipmcenters.org/cropprofiles/docs/MDcucumbers-pickling.pdf>.
10. Call, A. D., Wehner, T. C., Holmes, G. J., and Ojiambo, P. S. 2013. Effects of host plant resistance and fungicides on severity of cucumber downy mildew. HortScience 48:53-59.
11. Cespedes-Sanchez, M. C., Naegle, R. P., Kousik, C. S., and Hausbeck, M. K. 2014. Field response of cucurbit hosts to *Pseudoperonospora cubensis* in Michigan. Plant Dis. (in press). doi: 10.1094/PDIS-05-14-0500-RE.
12. Chen, C. P., Sung, C. C., and Ho, C. C. 1961. 1959. A brief report of the discovery of oospores of downy mildew of cucumber *Pseudoperonospora cubensis* (Berk. and Curt.). Rost. Rev. Appl. Mycol. 40:393-394.

13. Clinton, G. P. 1905. Downy mildew, or blight, *Peronosplasmopara cubensis* (B. & C.) Clint., of musk melons and cucumbers. Conn. (New Haven). Agr. Exp. Stn. Rep. 28:329-362.
14. Cohen, Y., Rubin, A. E., and Galperin, M. 2011. Formation and infectivity of oospores of *Pseudoperonospora cubensis*, the causal agent of downy mildew in cucurbits. Plant Dis. 95:874.
15. Colucci, S. J. 2008. Host range, fungicide resistance and management of *Pseudoperonospora cubensis*, causal agent of cucurbit downy mildew. MS Thesis, Department of Plant Pathology, North Carolina State University, Raleigh.
16. Colucci, S. J., and Holmes, G. J. 2007. Fungicide insensitivity and pathotype determination of *Pseudoperonospora cubensis*, causal agent of cucurbit downy mildew. (Abstr.). Phytopathology 97:S24.
17. Colucci, S. J., Thornton, A. C., Adams, M. L., and Holmes, G. J. 2006. Delayed fungicide application to cucumber and its effect on downy mildew severity and yield. 2005. Fungic. Nematic. Tests 61:V104. Online publication.
18. Davis, J. M., and Main, C. E. 1986. Applying atmospheric trajectory analysis to problems in epidemiology. Plant Dis. 70:490-497.
19. Doran, W. L. 1932. Downy mildew of cucumbers. Mass. Agric. Exper. Stat. Bull. No. 283.
20. Everts, K. L., Osborne, L., Gevens, A. J., Vasquez, S. J., Gugino, B. K., Ivors, K., and Harmon, C. 2012. Extension plant pathology: Strengthening resources to continue serving the public interest. Phytopathology 102: 652-655.
21. Galloway, B. T. 1889. New localities for *Peronospora cubensis* B. & C. J. Mycol. 5:216.
22. Gevens, A. J., and Hausbeck, M. K. 2006. Control of downy mildew of cucumbers with fungicides, 2005. Fungic. Nematic. Tests 61:V062. Online publication.
23. Granke, L. L., and Hausbeck, M. K. 2011. Dynamics of *Pseudoperonospora cubensis* sporangia in commercial cucurbit fields in Michigan. Plant Dis. 95: 1392-1400.
24. Granke, L. L., Morrice, J. J., and Hausbeck, M. K. 2014. Relationships between airborne *Pseudoperonospora cubensis* sporangia, environmental conditions, and cucumber downy mildew severity. Plant Dis. 98:674-681.
25. Granke, L. L., Quesada-Ocampo, L. M., Lamour, K. H., and Hausbeck, M. K. 2012. Advances in research on *Phytophthora capsici* on vegetable crops in the United States. Plant Dis. 96:1588-1600.
26. Guo, S., Zhang, J., Sun, H., Salse, J., Lucas, W. J., Zhang, H., Zheng, Y., Mao, L., Ren, Y., Wang, Z., Min, J., Guo, X., Murat, F., Ham, B. K., Zhang, Z., Gao, S., Huang, M., Xu, Y., Zhong, S., Bombarely, A., Mueller, L. A., Zhao, H., He, H., Zhang, Y., Zhang, Z., Huang, S., Tan, T., Pang, E., Lin, K., Hu, Q., Kuang, H., Ni, P., Wang, B., Liu, J., Kou, Q., Hou, W., Zou, X., Jiang, J., Gong, G., Klee, K., Schoof, H., Huang, Y., Hu, X., Dong, S., Liang, D., Wang, J., Wu, K., Xia, Y., Zhao, X., Zheng, Z., Xing, M., Liang, X., Huang, B., Lv, T., Wang, J., Yin, Y., Yi, H., Li, R., Wu, M., Levi, A., Zhang, X., Giovannoni, J. J., Wang, J., Li, Y., Fei, Z., and Xu, Y. 2013. The draft genome of watermelon (*Citrullus lanatus*) and resequencing of 20 diverse accessions. Nat. Genet. 45:51-58.
27. Halsted, B. D. 1889. *Peronospora* on cucumbers. Bot. Gaz. 14:152-153.
28. Halsted, B. D. 1889. Some notes upon economic Peronosporae for 1889 in New Jersey. J. Mycol. 5:201-202.
29. Hausbeck, M. K., and Linderman, S. D. 2014. Evaluation of fungicides for control of downy mildew of cucumber, 2013. Plant Dis. Manag. Rep. 8: V304. Online publication.
30. Holmes, G. J., Brown, E. A., and Ruhl, G. 2000. What's a picture worth? The use of modern communications in diagnosing plant diseases. Plant Dis. 84: 1256-1265.
31. Holmes, G. J., Main, C. E., and Keever, Z. T. 2004. Cucurbit downy mildew: a unique pathosystem for disease forecasting, Pages 69-80 in: Advances in Downy Mildew Research, volume 2. P. Spencer-Phillips and M. J. Jeger, eds. Kluwer Academic Publishers, Boston.
32. Holmes, G. J., Pollard, D. W., and Darden, J. B. 1998. Evaluation of fungicides for control of downy mildew and powdery mildew of squash, 1997. Fungic. Nematic. Tests 53:239.
33. Holmes, G., Wehner, T., and Thornton, A. 2006. An old enemy re-emerges. Am. Veg. Grower 54:14-15.
34. Huang, S., Li, R., Zhang, Z., Li, L., Gu, X., Fan, W., Lucas, W. J., Wang, X., Xie, B., Ni, P., Ren, Y., Zhu, H., Lin, K., Jin, W., Fei, Z., Li, G., Staub, J., Kilian, A., van der Vossen, E. A. G., Wu, Y., Guo, J., He, J., Jia, Z., Ren, Y., Tian, G., Lu, Y., Ruan, Y., Qian, W., Wang, M., Huang, Q., Li, B., Xuan, Z., Cao, J., Wu, Z., Zhang, J., Cai, Q., Bai, Y., Zhao, B., Han, Y., Li, Y., Li, X., Wang, S., Shi, Q., Liu, S., Cho, W. K., Kim, J. Y., Xu, Y., Heller-Uszynska, K., Miao, H., Cheng, Z., Zhang, S., Wu, J., Yang, Y., Kang, H., Li, M., Liang, H., Ren, X., Shi, Z., Wen, M., Jian, M., Yang, H., Zhang, G., Yang, Z., Chen, R., Liu, S., Li, J., Ma, L., Liu, H., Zhou, Y., Zhao, J., Fang, X., Li, G., Fang, L., Li, Y., Liu, D., Zheng, H., Zhang, Y., Qin, N., Li, Z., Yang, G., Yang, S., Bolund, L., Kristiansen, K., Zheng, H., Li, S., Zhang, X., Yang, H., Wang, J., Sun, R., Zhang, B., Jiang, S., Wang, J., Du, Y., and Li, S. 2009. The genome of the cucumber, *Cucumis sativus* L. Nat. Genet. 41: 1275-1281.
35. Hughes, M. B., and Van Haltern, F. 1952. Two biological forms of *Pseudoperonospora cubensis*. Plant Dis. Rep. 36:365-367.
36. Jacobsen, B. J., Burrows, M. E., and Ong, K. L. 2008. Extension plant pathology: Bringing our service to the public, a century of service. Online. APSnet Feature.
37. Kanetis, L., Holmes, G. J., and Ojiambo, P. S. 2010. Survival of *Pseudoperonospora cubensis* sporangia exposed to solar radiation. Plant Pathol. 59:313-323.
38. Keinath, A. P., DuBose, V. B., and Lassiter, A. W. 2008. Evaluation of fungicides to manage downy mildew on pickling cucumber in Charleston, South Carolina, 2007. Plant Dis. Manag. Rep. 2:V024. Online publication.
39. Keinath, A. P., DuBose, V., Lassiter, A., and Baccari, G. 2009. Evaluation of fungicides to manage downy mildew on pickling cucumber and summer squash, 2008. Plant Dis. Manag. Rep. 3:V033. Online publication.
40. Kozik, E. U., Klosinska, U., Call, A. D., and Wehner, T. C. 2013. Heritability and genetic variance estimates for resistance to downy mildew in cucumber accession Ames 2354. Crop Sci. 53:177-182.
41. Langston, D. B., Jr., and Sanders, F. H., Jr. 2013. Evaluation of fungicides for control of downy mildew on cucumber in Georgia I, 2012. Plant Dis. Manag. Rep. 7:V108. Online publication.
42. Lawson, A. B. 2013. Bayesian Disease Mapping: Hierarchical Modeling in Spatial Epidemiology, 2nd Ed. Chapman & Hall/CRC, FL.
43. Lebeda, A., Pavelková, J., and Sedláková, B. 2013. Structure and temporal shifts in virulence of *Pseudoperonospora cubensis* populations in the Czech Republic. Plant Pathol. 62:336-345.
44. Martin, F. N., Abad, Z. G., Balci, Y., and Ivors, K. 2012. Identification and detection of *Phytophthora*: reviewing our progress, identifying our needs. Plant Dis. 96:1080-1103.
45. McGrath, M. T., and Fox, G. M. 2008. Assessment of field resistance to pyraclostrobin- and metenoxam-based fungicides for managing downy mildew in cucumber, 2007. Plant Dis. Manag. Rep. 2:V149. Online publication.
46. Michelmores, R. W. 1981. Sexual and asexual sporulation in the downy mildews. Pages 165-181. in: The Downy Mildews. D. M. Spencer, ed. Academic Press, London.
47. Mizubuti, E. S. G., Aylor, D. E., and Fry, W. E. 2000. Survival of *Phytophthora infestans* sporangia exposed to solar radiation. Phytopathology 90:78-84.
48. Neufeld, K. N., Isard, S. A., and Ojiambo, P. S. 2013. Relationship between disease severity and escape of *P. cubensis* sporangia from a cucumber canopy during downy mildew epidemics. Plant Pathol. 62:1366-1377.
49. Neufeld, K. N., and Ojiambo, P. S. 2012. Interactive effects of temperature and leaf wetness duration on sporangia germination and infection of cucurbit hosts by *Pseudoperonospora cubensis*. Plant Dis. 96:345-353.
50. Nishimura, S., Lebeda, A., and Gadasová, V. 2002. Pathogenic variation of *Pseudoperonospora cubensis* in the Czech Republic and some other European countries. Proceedings of the 2nd International Symposium on Cucurbits. S. Nishimura, ed. ISHS. Acta Hort. 588:137-141.
51. Nusbaum, C. J. 1944. The seasonal spread and development of cucurbit downy mildew in the Atlantic coastal states. Plant Dis. 28:82-85.
52. Nusbaum, C. J. 1948. A summary of cucurbit downy mildew reports from Atlantic coastal states in 1947. Plant Dis. 32:44-48.
53. Ojiambo, P. S., and Holmes, G. J. 2011. Spatiotemporal spread of cucurbit downy mildew in the eastern United States. Phytopathology 101:451-461.
54. Ojiambo, P. S., Holmes, G. J., Britton, W., Keever, T., Adams, M. L., Babadoost, M., Bost, S. C., Boyles, R., Brooks, M., Damicone, J., Draper, M. A., Egel, D. S., Everts, K. L., Ferrin, D. M., Gevens, A. J., Gugino, B. K., Hausbeck, M. K., Ingram, D. M., Isakeit, T., Keinath, A. P., Koike, S. T., Langston, D., McGrath, M. T., Miller, S. A., Mulrooney, R., Rideout, S., Roddy, E., Seebold, K. W., Sikora, E. J., Thornton, A., Wick, R. L., Weyand, C. A., and Zhang, S. 2011. Cucurbit downy mildew ipmPIPE: a next generation web-based interactive tool for disease management and extension outreach. Plant Health Prog. Online, doi: 10.1094/PHP-2011-0411-01-RV.
55. Ojiambo, P. S., and Kang, E. L. 2013. Modeling spatial frailties in survival analysis of cucurbit downy mildew epidemics. Phytopathology 103: 216-227.
56. Ojiambo, P. S., Paul, P. A., and Holmes, G. J. 2010. A quantitative review of fungicide efficacy for managing downy mildew in cucurbits. Phytopathology 100:1066-1076.
57. Olaya, G., Kuhn, P., Hert, A., Holmes, G., and Colucci, S. 2009. Fungicide resistance in cucurbit downy mildew. (Abstr.). Phytopathology 99:S171.
58. Papadopoulos, A. P., and Gosselin, A. 2007. Greenhouse vegetable production in Canada. Chron. Horticult. 47:23-28.
59. Percheval, L., Bardin, M., Dogimont, C., and Pitrat, M. 2005. Relationship between loci conferring downy mildew and powdery mildew resistance in melon assessed by quantitative trait loci mapping. Phytopathology 95: 556-565.
60. Qi, J., Liu, X., Shen, D., Miao, H., Xie, B., Li, X., Zeng, P., Wang, S., Shang, Y., Gu, X., Du, Y., Li, Y., Lin, T., Yuan, J., Yang, X., Chen, J., Chen, H., Xiong, X., Huang, K., Fei, Z., Mao, L., Tian, L., Stadler, T., Renner, S. S., Kamoun, S., Lucas, W. J., Zhang, Z., and Huang, S. 2013. A genomic variation map provides insights into the genetic basis of cucumber domestication and diversity. Nat. Genet. 45:1510-1515.
61. Quesada-Ocampo, L. M., Granke, L. L., Olsen, J., Gutting, H. C., Runge, F., Thines, M., Lebeda, A., and Hausbeck, M. K. 2012. The genetic structure of *Pseudoperonospora cubensis* populations. Plant Dis. 96: 1459-1470.

62. Ramsey, G. B., Wiant, J. S., and Link, G. K. K. 1938. Market diseases of fruits and vegetables: crucifers and cucurbits. USDA Miscellaneous Publication No. 292.
63. Rotem, J., Wooding, B., and Aylor, D. E. 1985. The role of solar radiation, especially ultraviolet, in the mortality of fungal spores. *Phytopathology* 75: 510-514.
64. Runge, F., Choi, Y. J., and Thines, M. 2011. Phylogenetic investigations in the genus *Pseudoperonospora* reveal overlooked species and cryptic diversity in the *P. cubensis* species cluster. *Eur. J. Plant Pathol.* 129:135-146.
65. Runge, F., and Thines, M. 2009. A potential perennial host for *Pseudoperonospora cubensis* in temperate regions. *Eur. J. Plant Pathol.* 123:483-486.
66. Russell, P. E. 2004. Sensitivity baselines in fungicide resistance research and management. FRAC Monograph 3. Fungicide Resistance Action Committee, CropLife International.
67. Savory, E. A., Adhikari, B. N., Hamilton, J. P., Vaillancourt, B., Buell, C. R., and Day, B. 2012. mRNA-Seq analysis of the *Pseudoperonospora cubensis* transcriptome during cucumber (*Cucumis sativus* L.) infection. *PLoS ONE* 7:e35796.
68. Savory, E., Zhou, C., Adhikari, B., Hamilton, J. P., Buell, C. R., Shiu, S. H., and Day, B. 2012. Alternative splicing of a multi-drug transporter from *Pseudoperonospora cubensis* generates an RXLR effector protein that elicits a rapid cell death. *PLoS ONE* 7:e34701.
69. Shan, Q., Wang, Y., Li, J., Zhang, Y., Chen, K., Liang, Z., Zhang, K., Liu, J., Xi, J. J., Qiu, J. L., and Gao, C. 2013. Targeted genome modification of crop plants using a CRISPR-Cas system. *Nat. Biotechnol.* 31: 686-688.
70. Sherman, J., and Gent, D. H. 2014. Concepts of sustainability, motivations for pest management approaches, and implications for communicating change. *Plant Dis.* 98:1024-1035.
71. Singh, P. P., and Sokhi, S. S. 1989. 1st report of occurrence of oospores of *Pseudoperonospora cubensis* on 2 cucurbitaceous hosts. *Curr. Sci.* 58: 1330-1331.
72. Sitterly, W. R. 1972. Breeding for disease resistance in cucurbits. *Annu. Rev. Phytopathol.* 10:471-490.
73. Stewart, F. C. 1897. The downy mildew of the cucumber: what it is and how to prevent it. *N.Y. Ag. Exp. Sta. Bullt.* 119:154-183.
74. Thomas, A., Carbone, I., and Ojiambo, P. S. 2013. Occurrence of the A2 mating type of *Pseudoperonospora cubensis* in the United States. *Phytopathology* 103:S2.145.
75. Thomas, C. E. 1996. Downy mildew. Pages 25-27. in: *Compendium of Cucurbit Diseases*. T. A. Zitter, D. L. Hopkins, and C. E. Thomas, eds. American Phytopathological Society, St. Paul, MN.

Gerald Holmes

Peter Ojiambo

Mary Hausbeck

**Lina
Quesada-Ocampo**

Anthony Keinath

Dr. Holmes is director of the Strawberry Sustainability Research & Education Center at California Polytechnic State University in San Luis Obispo. He received his Ph.D. in plant pathology from the University of California Riverside. Current research is focused on diseases and pests of strawberries in California. From 2009 to 2014, he was product development manager for fungicides with Valent USA Corporation. From 1997 to 2009, Dr. Holmes was assistant and associate Professor of Plant Pathology at North Carolina State University where he had statewide responsibility for diseases of vegetable crops. His research program at NC State focused on the biology, epidemiology, and management of economically important diseases of sweetpotatoes and cucurbits. Dr. Holmes was the founding project director of the Cucurbit Downy Mildew ipmPIPE, a program that provides growers with near real-time information to efficiently control cucurbit downy mildew.

Dr. Ojiambo is an associate professor in the Department of Plant Pathology at North Carolina State University. Dr. Ojiambo received his B.S. degree (first class honors) in agriculture in 1994 and M.S. degree in plant pathology in 1996 from the University of Nairobi, Kenya. He received his Ph.D. in plant pathology from the University of Georgia in 2004. Dr. Ojiambo has a 100% research appointment and his program focuses on theoretical and applied aspects of the ecology and epidemiology of plant diseases. A particular interest of his research program is to understand how epidemics change in time and space and to apply knowledge thereof to manage plant diseases. Dr. Ojiambo is the current project director of the Cucurbit Downy Mildew ipmPIPE.

Dr. Hausbeck is professor and extension specialist of plant pathology in the Department of Plant, Soil and Microbial Sciences at Michigan State University (MSU). She received her Ph.D. in plant pathology from Pennsylvania State University. She joined the faculty at MSU in 1990, where she has statewide extension (60%) and research (40%) responsibilities in field and greenhouse vegetables, greenhouse ornamentals, hop, and ginseng. Recently, Dr. Hausbeck was elected a Fellow of the American Phytopathological Society (APS) and received the

William J. Beal Outstanding Faculty/All University Award from MSU. Earlier, she received the Excellence in Extension Award from APS, the IR-4 Meritorious Service Award, the Alex Laurie Award for Research and Education from the Society of American Florists, and the Master Farmer Associate Award from the Michigan Vegetable Council. Her current research emphasis includes downy mildews and *Phytophthora* spp. Her overall program goal is to develop novel and integrated disease management strategies for growers and to train graduate students. She has served as major professor for 33 students receiving advanced degrees in plant pathology.

Dr. Quesada is an assistant professor in the Department of Plant Pathology at North Carolina State University. Her research program uses a broad range of tools from field studies to genomics to improve management strategies of vegetable diseases, especially those caused by oomycete pathogens such as the downy mildews and *Phytophthora*. She received her B.Sc. degrees in microbiology and biology from Universidad de Los Andes, Bogota, Colombia. She then joined the Ohio State University and worked on genomics of *Phytophthora infestans*. She received her Ph.D. in Plant Pathology from Michigan State University (MSU) and her doctoral work focused on the population structure of *Phytophthora capsici*. Later she studied worldwide populations of *Pseudoperonospora cubensis* as a postdoctoral researcher at MSU, and Fusarium diseases in corn as a NIFA postdoctoral fellow.

Dr. Keinath is a professor of plant pathology at Clemson University with an 80% research and 20% extension appointment in vegetable pathology. He received his B.S. in botany from Michigan State University in 1982 and his M.S. and Ph.D. in plant pathology from Cornell University in 1985 and 1988, respectively. After postdoctoral work with the USDA, ARS in Beltsville, MD, he joined Clemson's Coastal Research and Education Center in Charleston, SC, in 1991. He conducts applied research on management, ecology, and epidemiology of pathogens on cucurbits and brassicas. Current research projects include fungicide efficacy and resistance, grafted cucurbits, and Fusarium wilt of watermelon.

76. Thomas, C. E., Inaba, T., and Cohen, Y. 1987. Physiological specialization in *Pseudoperonospora cubensis*. *Phytopathology* 77:1621-1624.
77. Thornton, A. C., Holmes, G. J., and Adams, M. L. 2005. Evaluation of fungicides for control of downy mildew of cucumber, 2004. *Fungic. Nematic. Tests* 60:V059. Online publication.
78. Ullstrup, A. J. 1972. The impacts of the Southern corn leaf blight epidemics of 1970-1971. *Annu. Rev. Phytopathol.* 10:37-50.
79. United States Department of Agriculture, National Agricultural Statistics Service. 2006. *Agricultural statistics 2006*. Chapter IV. Statistics of vegetables and melons. Online, http://nass.usda.gov/Publications/Ag_Statistics/2006/CHAP04.PDF.
80. United States Department of Agriculture, National Agricultural Statistics Service. 2009. *Agricultural statistics 2009*. Chapter IV. Statistics of vegetables and melons. Online, http://www.nass.usda.gov/Publications/Ag_Statistics/2009/chp04.pdf.
81. United States Department of Agriculture, National Agricultural Statistics Service. 2011. 2010 Vegetable chemical use, pesticides, cucumbers, processing, pickles. Online, http://www.nass.usda.gov/Data_and_Statistics/Pre-Defined_Queries/2010_Vegetables/index.asp.
82. United States Environmental Protection Agency. 2003. Famoxadone; pesticide tolerance. *Fed. Regist.* 68:39462-39471.
83. United States Environmental Protection Agency. 2003. Cymoxanil; pesticide tolerance. *Fed. Regist.* 68:41927-41936.
84. United States Environmental Protection Agency. 2004. Propamocarb hydrochloride; pesticide tolerance. *Fed. Regist.* 69:47013-47022.
85. United States Environmental Protection Agency. 2004. Fenamidone; pesticide tolerance. *Fed. Regist.* 69:58058-58066.
86. United States Government Accountability Office. 2006. Agriculture production. USDA Needs to Build on 2005 Experience to Minimize the Effects of Asian Soybean Rust in the Future
87. Wallace, E., Adams, M., Ivors, K., Ojiambo, P. S., and Quesada-Ocampo, L. M. 2014. First report of *Pseudoperonospora cubensis* causing downy mildew on *Momordica balsamina* and *M. charantia* in North Carolina. *Plant Dis.* 98:1279.
88. Wehner, T. C., and Ellington, T. L. 2005. Summer (Stage 4) Pickling Cucumber Trial. Online: <http://cuke.hort.ncsu.edu/cucurbit/cuke/cktrials/ckrpt05pk4.html>
89. Withers, S., Gongora-Castillo, E., Bowman, M. J., Childs, K. L., Gent, D., Ojiambo, P., and Quesada-Ocampo, L. M. 2014. Developing genomic resources for species-specific molecular diagnostics of cucurbit downy mildew. *Phytopathology* 104:397.
90. Zhang, Y., Pu, Z., Zhou, X., Liu, D., Dai, L., and Wang, W. 2012. A study on the overwintering of cucumber downy mildew oospores in China. *J. Phytopathol.* 160:469-474.