

Plant Pathology Master's Symposium

Talley Student Union
4140 Governance Chamber
November 20, 2015
10:00 am - 12:00 noon

SPEAKERS:

Emma Wallace
Andrew Scruggs
Megan Miller
Nathan Miller
Michael Cannon

WELCOME & INTRODUCTIONS
DR. MARC CUBETA
9:55 AM

SESSION I
10:00 AM - 11:00 AM

EMMA WALLACE
"Trends in North Carolina populations
of *Pseudoperonospora cubensis* on
commercial and non-commercial
cucurbits"
10:00-10:20

ANDREW SCRUGGS
"Improving Control of Sweetpotato
Postharvest Diseases through Integrated
Strategies"
10:20-10:40

MEGAN MILLER
"Thermal Inactivation of *Calonectria*
pseudonaviculata, the Causal Agent of
Boxwood Blight"
10:40-11:00

BREAK
11:00

SESSION II
11:20 AM - 12:00 NOON

NATHAN MILLER
"Evaluation of fungicides for chemical
management of Fusarium wilt of
watermelon"
11:20-11:40

MICHAEL CANNON
"Quantifying Inoculum Density, Disease
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Blight in Peanut"
11:40-12:00

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TRENDS IN NORTH CAROLINA POPULATIONS OF *PSEUDOPERONOSPORA CUBENSIS* ON COMMERCIAL AND NON-COMMERCIAL CUCURBITS

Emma C. Wallace¹ and Lina M. Quesada-Ocampo¹

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Pseudoperonospora cubensis is an obligate oomycete pathogen of plants in the Cucurbitaceae family. The pathogen causes foliar lesions and sporulation on the abaxial leaf surface creates a characteristic purple-brown ‘downy’ appearance. In 2004, the pathogen overcame host resistance resulting in major crop losses particularly on cucumber that year. Understanding population diversity of *P. cubensis* on a regional level can help optimize management strategies. This may be particularly useful in North Carolina, which has higher genetic diversity of *P. cubensis* than in other parts of the United States. Additionally, several wild and non-commercial cucurbit hosts prevalent in North Carolina and the southeast may contribute to disease epidemics and population diversity. Microsatellites were used to understand local populations of the pathogen from commercial and non-commercial cucurbits. Microsatellites were identified from the predicted transcriptome of *P. cubensis* and primers flanking these sequences were designed. Of the 2,088 primers generated, 100 were screened across a diverse panel of *P. cubensis* isolates to evaluate fidelity to *in silico* mining results and also to establish informative markers. It was determined that 91% of the markers validated corresponded to predicted products. Fragment analysis revealed 11 markers to be reliable and polymorphic, exhibiting an average heterozygosity of 0.38. The greatest genetic differentiation observed was between isolates from *Cucumis* species and isolates from *Momordica* species. Using these informative markers, basic genetic differentiation can be evaluated based on time, geography, and host.

IMPROVING CONTROL OF SWEETPOTATO POSTHARVEST DISEASE THROUGH INTEGRATED STRATEGIES

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Sweetpotato production is limited by several postharvest diseases in North Carolina. Two of the most common postharvest diseases are Rhizopus soft rot (caused by *Rhizopus stolonifer*) and Fusarium root rot (caused by *Fusarium solani* and *F. proliferatum*). Traditionally, these two diseases have been largely controlled by the use of dicloran fungicide dips. However, due to tighter residue restrictions in Europe and changes in consumer preferences, use of dicloran is now limited for export markets, creating a need for integrated strategies to control these diseases. The effects of storage temperature (13, 18, 23, 29, and 35°C), relative humidity (80, 90, and 100%), and initial inoculum level (3, 5, and 7 mm-diameter mycelia plug) were examined for their effect on progression of Rhizopus soft rot and Fusarium root rot on 'Covington' sweetpotatoes. The intensity of both diseases was significantly ($P < 0.05$) reduced at lower temperatures (13°C) and Fusarium root rot intensity was significantly ($P < 0.05$) reduced at lower relative humidity levels (80%) and initial inoculum levels. Sporulation of *F. proliferatum* and *R. stolonifer* was also reduced under the same conditions. The efficacy of chlorine dioxide fumigation and postharvest dips in alternative control products were also investigated for control of Rhizopus soft rot. Qualitative mycotoxin analysis of roots infected with one of six *Fusarium* species revealed the production of fumonisin B1 by *F. proliferatum* when infecting sweetpotato. A survey of *Fusarium* species infecting sweetpotato in North Carolina identified six species contributing to disease, with *F. solani* as the primary causal agent. Understanding the etiology and epidemiology of postharvest diseases and identifying alternative control strategies will allow for more improved recommendations to limit postharvest losses in sweetpotato.

THERMAL INACTIVATION OF *CALONECTRIA PSEUDONAVICULATA*, THE CAUSAL AGENT OF BOXWOOD BLIGHT

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Calonectria pseudonaviculata (*Cps*) is a fungal pathogen of boxwood that causes lesions and cankers on leaves and stems. Disease symptoms that develop as dark pinpoint lesions on infected leaves or stems of partially resistant boxwood cultivars are often challenging to recognize and may increase the probability of introducing infected plants into production systems. Current disease management strategies focus on the use of preventative fungicides and planting partially resistant cultivars. Studies are needed to develop methods to mitigate the chance of spreading the boxwood blight pathogen during propagation of boxwood cuttings. To address this need, we conducted research to explore the potential utility of hot water treatment to inactivate *Cps*. The objectives of this research were to: 1) predict the thermal death kinetics of *Cps* conidia, and 2) determine the ability of commonly grown commercial boxwood cultivars to survive exposure to hot water. To address the first objective, conidia of three field isolates of *Cps* were treated at five temperatures ranging from 45°C to 55°C for 0 to 20 min and their percent germination assessed 24 h after treatment. Regression analysis indicated that a longer time was needed to kill 90% of the spores at 45°C. To address the second objective, cuttings of six boxwood cultivars were submerged in water at 45°C, 50°C, and 55°C for 0 to 60 min and evaluated for rooting and phytotoxicity after 86 days. The ability of boxwood cuttings to survive treatment at 45°C and 50°C varied by cultivar and no cultivars survived treatment at 55°C.

EVALUATION OF FUNGICIDES FOR CHEMICAL MANAGEMENT OF FUSARIUM WILT OF WATERMELON

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Fusarium wilt of watermelon, caused by *Fusarium oxysporum* f. sp. *niveum*, is an economically important disease in the United States and worldwide. Current disease management options rely heavily on crop rotation and host resistance, but new races of the pathogen have overcome previously resistant cultivars. Although use of fungicides to control Fusarium wilt in watermelon is currently limited in practice, this study examines the efficacy of new and existing fungicides in reducing disease severity. Fungicide efficacy was evaluated *in vitro* using a fungicide amended media assay and *in vivo* in a small plot field trial conducted over the summer of 2015. Different concentrations of ten fungicides were evaluated in an *in vitro* test, and sensitivity to two fungicides was screened across fifty isolates of *Fusarium oxysporum*. The most effective fungicides were prothioconazole and an experimental chemical, MI-6. None of the isolates were found to be insensitive to these fungicides. Field-testing with these two fungicides at two concentrations and application methods indicated that both products were significantly ($P < 0.05$) different from the untreated control, but a drench treatment at transplant followed by a foliar spray was effective in reducing Fusarium wilt incidence in comparison to either product used only as a drench treatment at transplant. The two fungicides could effectively complement existing strategies in Fusarium wilt management in watermelon.

QUANTIFYING INOCULUM DENSITY, DISEASE INCIDENCE AND YIELD LOSS OF SCLEROTINIA BLIGHT IN PEANUT

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Sclerotinia blight caused by the soilborne fungus *Sclerotinia minor* is a serious disease of peanut. Disease incidence and resulting yield-loss are both highly spatially variable within and among fields, probably due to variations in soil inoculum density. Current methods for quantifying inoculum density after wet sieving are impractical for routine use. The current assay could be simplified by 1) using volatiles to stimulate germination of extracted sclerotia or 2) plating extracted sclerotia and debris on a semi-selective medium. In the first simplification, remoistened dried peanut leaves, lettuce leaves, or methanol were tested as sources of volatiles. Sclerotia were placed on moistened field soil in a sealed container containing the test material. Lettuce leaves slightly stimulated germination, but germination was less than on agar controls. In the second simplification, growth of *S. minor* and *Trichoderma*, a common contaminant, was tested on seven fungicides. Chlorothalonil at 1.0 ppm inhibited growth of *Trichoderma* by 47.4%, while inhibiting *S. minor* by 18.3%. To characterize disease incidence and yield, two cultivars were planted at two locations. Fluazinam was applied at six timings to create variation in disease incidence: early- (56 days after planting; DAP), mid- (84 DAP), late-season (112 DAP); delayed (84, 112 DAP), full (56, 84, 112 DAP), and an untreated control. Although disease incidence varied across plots and treatments, treatments did not affect yield at either location. Additionally, yield was not significantly correlated ($r = -0.22$; $P = 0.1422$) with disease incidence.