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**94. Impact and biology of *Phytophthora ramorum*.** Grunwald, N. J. *Phytopathology* 97(7)Suppl:S168. 2007.

1860 acres were significantly damaged due to HPV infection, causing a reported 45–50% reduction in yield. Additional damage was found in 2005. There is no labeled control for the vector, and control measures have been limited to cultural practices, particularly disruption of the vector life cycle and planting of resistant varieties.

**Pitch canker in California: Is the invasion over?** T. R. GORDON. Dept. of Plant Pathology, University of California, Davis, CA 95616. *Phytopathology* 97:S168.

Pitch canker, caused by *Gibberella circinata*, was discovered in California in 1986. Over the next several years, the disease became more widespread and it is now found in at least 18 coastal counties. Monterey pine (*Pinus radiata*) continues to be the most severely affected host species, with damage occurring in both native and planted stands. However, where pitch canker is well established, disease severity appears to have stabilized. This may be attributed to a loss of highly susceptible individuals and recovery of many trees that were once severely diseased. Recovery results from containment of existing infections, which are often obscured by vigorous growth of healthy branches subtending those that were symptomatic, and a lack of new infections. The absence of new infections can be explained by systemic resistance induced by prior exposure to the pathogen. This hypothetical explanation is supported by field observations and by results of experiments conducted under both field and controlled conditions. It remains to be seen how long trees showing recovery will remain resistant to pitch canker and it has yet to be determined if the observed resistance is specific to this disease.

**Impact and biology of *Phytophthora ramorum*.** N. J. GRUNWALD. Horticultural Crops Research Laboratory, USDA ARS, Corvallis, OR. *Phytopathology* 97:S168.

*Phytophthora ramorum*, causal agent of sudden oak death (SOD) on oaks and Ramorum blight on ornamentals such as rhododendron, is an emerging pathogen with significant impact on both natural oak forest ecosystems and the nursery industry. The pathogen was simultaneously discovered in Germany and California in the mid 1990s. Oak mortality in CA and OR forests, where SOD is either established or being eradicated, is severe. Nursery crops can also be severely affected, although levels of resistance to *P. ramorum* on ornamentals such as *Rhododendron* and *Viburnum* vary considerably. Nurseries are also impacted due to restrictions on interstate movements of SOD hosts. The *P. ramorum* population in the US is clonal and consists of three major lineages. Both the European and North American populations are reproductively isolated and have gone through a genetic bottleneck. There currently is no evidence for sexual recombination in the field.

***Togninia*: Newly discovered pathogen causes centuries-old disease.** W. D. GUBLER, S. N. Rooney-Latham, and A. Eskalen. Department of Plant Pathology, University of California, Davis, CA 95616. *Phytopathology* 97:S168.

The grapevine disease esca was first described by the Greeks and Romans and was referred to in the Bible. For centuries the disease occurred in vineyards while etiology was unknown. The disease was described in California over 100 years ago. Early research attributed the disease to two basidiomycetes but symptoms could not be reproduced using the suspect pathogens. Recently, a group of fungi belonging to the genus *Phaeoacremonium* (teleomorph: *Togninia*) were shown to reside in grapevines as endophytes. Pathogenicity studies showed these fungi to be capable of causing both vascular and foliar symptoms of esca. Fruit symptoms also now have been reproduced with multiple species of *Togninia*. While spore trapping of the pathogens showed *Togninia* spp. spores were being released during periods of rainfall in the fall and winter, fruiting bodies had not been observed. Subsequently, perithecia of *Togninia minima* and 3 other species of *Togninia* were detected in pockets of decayed vascular tissue on grapevine. These same species were subsequently found on native trees in California; in rotted vascular tissue indicating that these fungi are endophytes in more than one host. For thousands of years these pathogens have gone undetected in grapevines while causing severe disease in Mediterranean climates. The disease cycle is now known and control measures are being investigated.

**Resistance to Wheat streak mosaic virus in perennial wheat.** L. A. HARRISON and T. D. Murray. Dept. Plant Pathology, Washington State University, Pullman, WA 99164. *Phytopathology* 97:S168.

Anticipated benefits of perennial wheat include reduced planting costs, improved wildlife habitat, reduced soil erosion and increased water retention. *Wheat streak mosaic virus* (WSMV) has potential to reduce vigor and regrowth of perennial wheat. The objective of this work was to identify perennial wheat lines with effective resistance to WSMV. Thirty-three lines derived from *Thinopyrum* sp. were evaluated in a growth chamber for

resistance to WSMV. Lines 03JP031 and 03JP039 had reduced virus replication, as indicated by lower ELISA values, and less severe symptoms, compared to a susceptible control. In field plots, 03JP039 had no symptoms and WSMV was detected in only 7% of plants, compared with two susceptible annual wheat controls, in which 46 to 91% of plants were symptomatic and positive for WSMV by ELISA. In the second year, 59% of 03JP039 plants developed symptoms and virus was detectable when temperatures rose above 32°C, suggesting temperature-sensitive resistance, compared with 100% symptomatic plants in five other perennial lines. These results show that resistance to WSMV is present in perennial wheat and should be useful to plant breeders.

**Strategies to ameliorate glyphosate immobilization of Mn and its impact on disease.** D. M. HUBER. Botany & Plant Pathology, Purdue University, West Lafayette, IN 47907. *Phytopathology* 97:S168.

The widely used herbicide glyphosate [N-(phosphonomethyl)glycine] in root exudates changes the rhizosphere biology to reduce Mn availability for crop uptake. Mn deficiency compromises plant resistance mechanisms mediated through the shikimate pathway, and take-all, head scab, *Corynespora* root rot, citrus variegated chlorosis, and other diseases increase after glyphosate application. Research to minimize the soil and plant immobilization of Mn by glyphosate has evaluated Mn source, time of application, and glyphosate formulation on Mn utilization by glyphosate-resistant corn and soybean. The K salt of glyphosate was less reactive than the isopropylamine formulation, and inorganic salts were more reactive than chelated or organic micronutrient sources. Normal translocation and utilization was observed only when Mn was applied 8 or more days after glyphosate. Glyphosate reduced Mn-reducing organisms in soybean rhizospheres, increased Mn-oxidizers, and predisposed soybeans to *Corynespora* root rot. Lower rates and fewer applications of glyphosate should be considered to minimize impacts on the soil environment and predisposition to disease.

**Influences of application timing on sporulation of the biocontrol product *Aspergillus flavus* AF36 in cotton fields of Arizona.** R. JAIME-GARCIA and P. J. Cotty. USDA-ARS, Dept. Plant Sciences, University of Arizona, Tucson. AZ 85721. *Phytopathology* 97:S168.

Aflatoxins are toxic, carcinogenic metabolites produced by certain fungi in the genus *Aspergillus*. Atoxigenic *A. flavus* strains are used as biocontrol to reduce aflatoxin contamination. To be effective, atoxigenic strains must reproduce during crop development when environmental conditions are conducive to aflatoxin contamination. However, optimal timing of atoxigenic strain application has not been determined. Commercial AF36 biocontrol product was applied in five cotton fields in each of two locations in 2004 and three locations in 2005. Applications were made biweekly following emergence. Incidence of product sporulation and amount of spores produced were determined weekly for four weeks after each application. Results indicate significant differences among application dates. Applications before June had little sporulation. Optimal sporulation within one week occurred for applications during July and August following canopy closing. Prior to application, an environment favorable for fungal growth is required for optimal dispersal from the biocontrol formulations.

**Wilt/decline of *Acacia koa* caused by *Fusarium oxysporum* in Hawaii.** R. L. JAMES (1), N. S. Dudley (2), and R. Sniezko (3). (1) USDA Forest Service, Forest Health Protection, Coeur d'Alene, ID 83815; (2) Hawaii Agriculture Research Center, Aiea, HI 96701; (3) USDA Forest Service, Dorena Genetic Resource Center, Cottage Grove, OR 97424. *Phytopathology* 97:S168.

An important wilt/decline disease of native *Acacia koa* was first described in Hawaii in 1985; the disease currently causes varying impacts on the four major Hawaiian Islands: Oahu, Kauai, Maui, and Hawaii. Disease etiology was investigated using Koch's Postulates; *Fusarium oxysporum* was repeatedly shown to cause disease symptoms. Other *Fusarium* spp., particularly *F. solani*, are also commonly isolated from diseased trees. Large survival differences among koa families in young field trials on infested sites indicate that genetic variation in disease resistance likely exists. Current efforts are underway to determine biological and non-biological factors associated with disease severity, determine disease intensity and impact throughout Hawaii, assess genetic variation and develop molecular markers for detecting and monitoring pathogens associated with the disease, and develop/implement screening protocols to rapidly evaluate a large number of koa families for genetic resistance to this disease.

**Secondary spread of *Verticillium* wilt in perennial mint fields: Evidence from spatial analysis.** D. A. JOHNSON (1), H. Zhang (2), and J. R. Alldredge (2). (1) Dept. of Plant Pathology, Washington State University; (2) Dept. of Statistics, Washington State University, Pullman, WA 99164. *Phytopathology* 97:S168.

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