sub-plots. Treatments were: 1- Ridomyl applied at sowing time @ 2.5 kg/ha, 2- one time Ridomyl applied at flowering @ 2.5 kg/ha, 3- Ridomyl applied at flowering and again after 28 days, 4- maneb applied three times, at flowering and then at 10 days intervals, 5- Control. After 1, 2 and 3 months of storage, healthy tubers were artificially inoculated with the pathogen and incubated at 27°C for 6 days. Tubers were then cut and after the appearance of pink colour, fungal progress was measured. 20 tubers were used in each test. Statistical analysis of data showed significant differences between treatments in all tests (p=0.1%). Two applications of Ridomyl at a 28-day interval after one month of storage gave best results in inhibiting pathogen progress. One Ridomyl application was less effective. Treatments 4 and 5 had no effect in inhibiting the pathogen and fell in the same group in all tests. It seems that induced resistance decreases in time, as the mean difference between pathogen progress of treatments 3 and 5 after 1, 2 and 3 months of storage were 2.35, 1.02 and 0.64 cm respectively. There were no significant differences between varieties.

Innovative disease control strategies

34.1 INHIBITORY EFFECT OF ALUMINIUM CHLORIDE AND SODIUM METABISULFITE ON *FUSARIUM SAMBUCINUM*.

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Aluminium chloride and sodium metabisulfite have been shown to possess antimicrobial activity against several plant pathogens. In an effort to develop new methods to control the fungus *Heterobasidion annosum*, cause of a major root rot of conifers in temperate regions, the objectives of this study were (1) to evaluate the inhibitory effect of aluminium chloride and sodium metabisulfite on conidial viability of the pathogen and (2) to visualise the effect of these salts on fungal membrane integrity using the SYTOX Green fluorochrome and electron microscopy. Results demonstrated that as little as 1 mM of sodium metabisulfite and 10 mM of aluminium chloride were sufficient to kill *H. annosum* conidia. Fluorescence analysis with SYTOX Green indicated that both salts affect fungal cells apparently through the loss of cellular membrane integrity. Ultrastructural observations confirmed that these salts cause plasmalemma invagination and/or rupture and cytoplasmic aggregation and/or leakage. This study points to the possibility of exploiting these salts as antifungal agents for controlling annosum root rot through their application to cut tree stumps.

34.2 NUTRIENTS TO ENHANCE THE BIOCONTROL ACTIVITY OF YEASTS AGAINST BLUE MOULD OF APPLES – TOWARDS RESTORATIVE BIOLOGICAL CONTROL.

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A number of studies have shown that nutrient supplements enhance the biocontrol activity of yeasts against a range of pathogens, including *Penicillium expansum,* one of the causal organisms of blue mould of apples. The aim of this research was to identify in the laboratory, nutrients that improve the performance of yeasts. From the results of previous laboratory studies, using an apple wound assay, calcium chloride ($CaCl_2$, 20 mg/ml) and calcium carbonate ($CaCO_3$, 20 mg/ml) were chosen and evaluated against *P. expansum* at 10^5 spores/ml using three yeasts known to inhibit lesion development. Cell suspensions were prepared in sterile distilled water (SDW) and in the nutrient at 10^5, 10^6 and 10^7 CFU/ml. The nutrient-alone treatment was also tested against the pathogen. When yeasts were prepared in SDW, a yeast cell concentration of 10^7 CFU/ml reduced disease incidence by an average of 85% compared with the pathogen control. Disease incidence was not reduced at 10^5 or 10^6 CFU/ml. Calcium chloride alone reduced disease incidence by 36%. When yeast cell suspensions were prepared in $CaCl_2$, disease incidence was reduced by 80% and 85% at 10^5 or 10^6 CFU/ml, respectively. Calcium carbonate alone reduced disease incidence by 82% and addition of yeasts did not improve this. As a combination of the yeast and the nutrient supplement $CaCl_2$ improved performance of both the yeast alone and the nutrient alone; this nutrient will be trialled in the field as a step towards a new disease management strategy termed restorative biological control.

34.3* GENOTYPE ROTATION FOR MANAGEMENT OF RESISTANCE TO THE SORGHUM ANTHRACNOSE PATHOGEN *COLLETOTRICHUM SUBLINEOLUM*.

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The high variability of *Colletotrichum sublineolum* in Brazilian conditions poses a continuous threat to the control of anthracnose through genetic resistance. Several alternatives such as dilatory resistance, cultivar mixtures, and gene pyramiding have been evaluated as ways to stabilize pathogen populations in the different sorghum-growing areas of the country. This work reports results on the rotation of sorghum genotypes in time as a strategy to increase the durability of genetic resistance and to stabilize populations of *C. sublineolum*. Anthracnose progress and the pathogen population virulence structure were evaluated, for three consecutive years, on sorghum genotypes BR008, BR005, BR009, and CMSXS210 both in continuous and alternating plantings in a no-tillage system, totaling 14 treatments. Specific responses of the pathogen and the predominance of less complex races (as indicated by the number of virulence genes) were observed in response to each sorghum genotype. Disease progress in each treatment was influenced by the virulence structure of the pathogen population developed in response to the genotype cultivated in the previous year. Higher disease severities were observed in treatments in which the same genotypes were cultivated continuously in the same area, as compared to the genotype rotation treatments. These results indicate that this can be a viable alternative to manage resistance to *C. sublineolum* in Brazil.

34.4* WHAT CAUSED THE RECENT EMERGENCE OF ANTHRACNOSE DISEASE ON GOLF COURSE GREENS IN NORTH AMERICA?

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Grasses cultivated as turf make up a major component of the North American landscape. Beginning in the 1990s, anthracnose caused by the fungus *Colletotrichum cereale* emerged as one of the most destructive diseases of golf course turf, with its incidence, severity and geographic range greatly expanded. The sudden emergence of this disease on greens is puzzling. Was the spread of turfgrass anthracnose due to the introduction of novel genotypes or did recent environmental/cultural change provide an opportunity