

Genetic and biochemistry analyses of the natural resistance of the fungicide fenhexamid in the phytopathogenic fungus Botrytis pseudocinerea

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POSTER SESSION ABSTRACTS Session CS8 Adaptation to xenobiotics CS8W8

Wednesday 6th April 14:00 - 16:00

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Genetic and biochemistry analyses of the natural resistance of the fungicide fenhexamid in the phytopathogenic fungus *Botrytis pseudocinerea*

The Botrytis species complex responsible for grey mould disease on grapevine is composed of two species: Botrytis cinerea the major one (about 90%) and Botrytis pseudocinerea. Despite their genetic polymorphism, these species cannot be morphologically distinguished. However, they do differ in their response to several fungicides, especially to the sterol biosynthesis inhibitor fenhexamid. While B. cinerea is sensitive to this hydroxyanilide, B. pseudocinerea is naturally resistant. Enzyme assays showed that in B. pseudocinerea the fenhexamid target enzyme, the sterol 3-ketoreductase was less sensitive to fenhexamid. In addition, a synergic effect between fenhexamid and sterol 14Ademethylation inhibitors (DMIs) known to inhibit Cyp51, a cytochrome P450 monooxygenase was observed in B. pseudocinerea. These results could suggest detoxification of fenhexamid by cytochromes P450. The cyp684 gene showing the strongest similarity to cyp51 among all B. cinerea cytochrome P450 genes was found strongly overexpressed in the presence of fenhexamid in B. pseudocinerea. In this work, we studied separately the effect of B. pseudocinerea erg27 polymorphism, erg27 encoding 3-ketoreductase, and of the recently identified cytochrome P450 gene, cyp684, on resistance to fenhexamid. The objective is to determine their respective implication in Experiments were conducted by exchange between erg27B.cinerea resistance. erg27B.pseudocinerea in B. cinerea and by cyp684 deletion in B. pseudocinerea. In parallel, metabolization studies are conducted to identify metabolites and test their activity on Botrytis spp.