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## Comparison of resistance mechanisms to pyrrolnitrin and to iprodione in *Botrytis cinerea*

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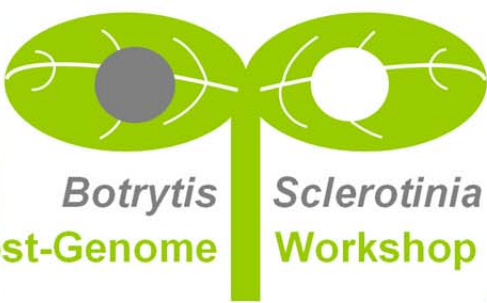
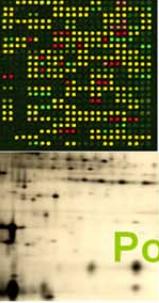
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**Botrytis**  
**Post-Genome**

**Sclerotinia**  
**Workshop**

# BSPGW 2011

## **Botrytis-Sclerotinia Post-Genome Workshop**

**15th - 17th September 2011, Lyon, France**

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**«The Most Studied Necrotrophic Fungal Plant Pathogens»**

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## 02.2-COMPARISON OF RESISTANCE MECHANISMS TO PYRROLNITRIN AND TO IPRODIONE IN *BOTRYTIS CINEREA*

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In a recent study, we reported the development of resistance to pyrrolnitrin in laboratory-induced mutants of *Botrytis cinerea*, suggesting a possible loss of efficacy of pyrrolnitrin-producing biological control agents. Pyrrolnitrin is a structural analogue of phenylpyrrole fungicides and it was shown that highly-resistant mutants to phenylpyrroles generally acquire resistance to iprodione, a dicarboximide fungicide. In the present study we compared the mechanisms of resistance to pyrrolnitrin and to iprodione in *B. cinerea*. This knowledge may be helpful to predict the risk related to the loss of efficacy of pyrrolnitrin-producing biological control agents against *B. cinerea* in the field. Pyrrolnitrin-induced mutants and iprodione-induced mutants of *Botrytis cinerea* were produced *in vitro* on media containing pyrrolnitrin or iprodione. For the pyrrolnitrin-induced mutants, high level of resistance to pyrrolnitrin was associated with a high level of resistance to iprodione, for the four tested isolates. For the iprodione-induced mutants, the high level of resistance to iprodione detected generated variable levels of resistance to pyrrolnitrin, for the five tested isolates. All selected mutants showed hypersensitivity to high osmolarity, suggesting a defect in the osmosensing signal transduction pathway. The sequences of the osmosensing class III histidine kinase encoding gene *bos1* showed different mutations in both types of mutants, although different isolates derived from a same strain displayed sometimes identical mutations suggesting an influence of the genetic background on the selected mutation. Almost all mutations affect the signal transduction or HAMP-domains of the histidine-kinase including yet undetected replacements. Altogether our analyses show that each of the six HAMP domains is important for signal-transduction. The impact of the replacements on HAMP structure and function will be discussed.