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Project title: How does *VENturia Inaequalis* VIrulenCe emerge worldwIde ?

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Key-words: *Venturia inaequalis*, Effectoromics, Virulence, Durable resistance

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Summary:

The use of disease-resistant apple cultivars represents an environmentally friendly alternative to chemicals treatments. Resistant cultivars carry resistance (R) genes, and the products of these genes recognize the products of avirulence (*AVR*) genes secreted by pathogens (known as gene-for-gene interactions). However, plant pathogens can rapidly adapt to circumvent this recognition in agrosystems, and thus it is crucial to understand how *AVR* genes evolve to optimize plant resistance management. In the *V. inaequalis*/Malus pathosystem, 18 gene-for-gene interactions have been described. Most R genes have been overcome, however, three remain very promising; *Rvi15, Rvi5* and *Rvi12*. In the case of *Rvi15* no resistance-breaking strains have been detected to date, while with *Rvi5,* despite reports of resistance-breaking strains, the disease level remains very low in orchards where cultivars with *Rvi5* are grown. For *Rvi12*, resistance has only been overcome in Canada. *Rvi6, Rvi15* and *Rvi18* have now been cloned and candidate genes for *Rvi5* and *Rvi12* have also been identified. On the pathogen side, we have one *AvrRvi5* candidate and have also identified 70 genes that encode small secreted proteins (SSP) that are highly expressed during first hours after infection. Like other *AVR* genes from other plant-pathogenic fungi, high expression of these 70 genes are making them good *AVR* gene candidates. These resources enable us to test whether fitness **c**ost and selective pressure exerted on the *AVR* genes *AvrRvi5*, *AvrRvi12*, *AvrRvi15* and *AvrRvi18* can be used to explain why the *R* genes *Rvi5*, *Rvi12*, *Rvi15* and *Rvi18* seem to confer durable resistance to scab. Screening for recognition by corresponding R proteins will be carried out using transient expression assays in *Nicotiana benthamiana,* as has already been done successfully in our laboratory for the identification of AvrRvi6*.* Once the *AVR* genes are identified we will (1) analyse their fitness cost to the fungus by producing deletion mutants and (2) determine evolutionary pressures exerted on these genes by analyzing polymorphism at each locus on a worldwide collection. This study will help to identify mechanisms involved in *V. inaequalis* virulence emergence worldwide. If our hypotheses are validated, we will build a strong knowledge base from which to identify durable R genes in resistant Malus genotypes already identified at IRHS.