

Comparative QTL mapping for fire blight resistance

Thomas Wöhner¹, Isabelle Vogt¹, Klaus Richter², Magda-Viola Hanke¹, Cesare Gessler³, Giovanni Broggin³, Johannes Fahrenttrapp³, Tania Garcia-Libreros⁴, Andreas Peil¹, Henryk Flachowsky¹

¹Julius Kühn-Institut, Institute for Breeding Research on Horticultural and Fruit Crops

²Julius Kühn-Institut, Institute for Resistance Research and Stress Tolerance

³Plant Pathology, Institute of Integrative Biology, ETH Zurich, Zurich, Switzerland

⁴AIT Austrian Institute of Technology GmbH, Seibersdorf, Austria

thomas.woehner@jki.bund.de

Fire blight is one of the most necrotic diseases affecting pome fruits like pear and apple or other members of the *Rosaceae* family. It is caused by the gram negative bacteria *Erwinia amylovora* (Burrill) Winslow *et al.*. The massive economical losses of the pome fruit producing industry in the last decade as well as the lack of efficient control strategies and the susceptibility of common apple cultivars justify an interest in fire blight resistance breeding. An effective solution would be planting of resistant cultivars with high fruit quality which are comparable to market leading cultivars. In the past many efforts have been made to study the genetics of fire blight resistance. A first mapping for fire blight resistance in the cross population 'Idared' x *Malus* x *robusta* 5 (Mr5) using the *E. amylovora* strain Ea222 resulted in the detection of a major QTL on LG 3 of Mr5, assuming a major gene responsible for the resistance. In the present study we inoculate the cross population with a deletion mutant strain

(pZYRKD3-1) of the *avrRpt2_{EA}* avirulence gene of *E. amylovora*. To compare the results, we additionally inoculate the progenies of 'Idared' x *Malus* x *robusta* 5 with the wild type strain of the deletion mutant (Ea1189). After inoculation with the wild type strain Ea1189 the average necrosis shoot length of the progenies was 40 %. In contrast to the wild type, the deletion mutant strain caused a 37 % higher average necrosis length (total shoot necrosis of 77 %). In comparison to Ea222, we were able to confirm the QTL on LG 3 after inoculation with the wild type strain Ea1189. The deletion mutant strain (pZYRKD3-1) of *E. amylovora* caused a breakdown of the QTL. The results imply that the knock out of the avirulence gene *avrRpt2_{EA}* causes a higher virulence of the mutant strain and an overcoming of resistance of Mr5. The different host-pathogen interactions are a first evidence for a gene for gene relationship between *Malus* x *robusta* 5 and *E. amylovora* (Burrill) Winslow *et al.*.