Comparative QTL mapping for fire blight resistance

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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 × 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 × 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 × robusta 5 and E. amylovora (Burrill) Winslow et al..