

The resistance locus *Ren3* and its powdery mildew isolate-specificity

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The obligate biotrophic pathogen *Erysiphe necator* causing powdery mildew on grapevine which belongs to the order of *Ascomycetes* was first introduced to Europe from America around 1845. Since then it has spread and is now present in every winegrowing region worldwide. It is thought to be one of the most devastating fungal diseases known to *Vitis vinifera* L. and is responsible for a considerable annual yield loss if no counteractions such as the application of fungicides are taken.

To this date several natural genetic resistances are known to confer resistance to *E. necator*. Nearly all of them originate from wild American *Vitis* species which have obtained the aforementioned by their co-evolution with the pathogen. These genetic resources are sources for breeding resistant grapevine cultivars with high vine quality and therefore reducing the application of fungicides. The grapevine cultivar 'Regent' is one example of this approach trying to combine resistance with high vine quality. In 2004 Fischer et al. detected two resistance loci conferring resistance to downy and powdery mildew in 'Regent' by QTL-analysis in the cross population of 'Regent' x 'Lemberger'. The latter locus was named *Ren3* (resistance to *E. necator*) and was found to be located on chromosome 15.

To this date only few genes have been characterized to be responsible for resistance to *E. necator*. One of the best

described resistance gene analogs (RGA) is *MrRun1* which belongs to the family of TIR-NBS-LRR (Toll/Interleukin-Receptor-Nucleotide Binding Site-Leucin Rich Repeats) genes.

So far the resistance locus *Ren3* spans an approximate interval of 4 Mb and covers a total of three clusters of these so-called RGAs. Recently we observed a shift of the maximum QTL over these RGA clusters. Therefore we analyzed plants with recombination in *Ren3* to discriminate the involvement in resistance to *E. necator* of the different RGA clusters.

However, due to a lack of recombination in *Ren3* in the F1 progeny of the 'Regent' x 'Lemberger' cross population, we had to screen other *Ren3* carrying cultivars originated from the repository at the JKI Geilweilerhof for recombination. This approach identified two useful recombinants which delimit the identified RGA clusters.

By controlled infection of detached leaflets of the identified *Ren3* recombinant cultivars and the *Ren3* recombinant F1 progeny we were able to detect hypersensitive reactions (HR) associated with appresoria. Two of the RGA clusters seem to be able to trigger HR due to the fact that plants carrying either one of the two clusters showed the same HR response.

These results suggest the involvement of both RGA clusters in the recognition of the same or probably different *E. necator* isolates.