

Resistances to *Erysiphe necator* *Ren3* and *Ren9* are encoded on chromosome 15 from 'Regent' in close vicinity

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The obligate biotrophic pathogen *Erysiphe necator* (Ascomycete) causing powdery mildew (PM) on grapevine was first introduced to Europe from America around 1845. Since then it has spread and is now present in every winegrowing region worldwide. PM is 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.

Breeding programs nowadays aim at combining different resistances against a certain pathogen (pyramiding). This strategy pursues the goal that pathogens will not easily overcome resistances of newly bred cultivars. For this approach of resistance breeding a combination of different resistance mechanisms is highly beneficial. For most of the loci conferring resistance to *E. necator* a hypersensitive response (HR) upon infection is described but for only one of them it is known which gene mediates this reaction. Molecular characterization of these loci is therefore a crucial step to combine different resistance mediating genes.

For the resistance locus *Ren3* only the location on chromosome 15 was published in 2004 and confirmed over the following years. This locus was found in

a cross population of 'Regent' x 'Lemberger' and was recently confirmed in 'Regent' x 'Cabernet Sauvignon'. Detailed analysis of this resistance locus involving fine mapping of chromosome 15 and analysis of *Ren3* recombinant genotypes we could narrow the locus down to an interval of around 200kb. The identified region includes four genes of the NBS-LRR (nucleotide binding site – leucine rich repeat) type which resemble typical resistance mediating genes known from *Run1* (from *Vitis rotundifolia*) and *Ren1* (from *Vitis vinifera* 'Kismisch Vatkana'). Microscopy showed that *Ren3* carrying cultivars react with HR upon infection with *E. necator* and therefore can restrict the growth of the pathogen.

During the characterization of *Ren3* we observed a shift of the QTL for resistance to *E. necator* to the anterior part of chromosome 15 early in the epidemic. Analysis of further recombinant plants delimited the two QTL regions and indicated resistance response of the HR type for both loci. The new locus was named *Ren9* and could be delimited to around 1,8 Mb. Screening of the 'Regent' BAC-library is currently done to obtain the sequence information for this locus which will allow a search for possible candidate genes.