Leaf necrosis induced by the insecticide carbaryl in *Vitis rupestris* 'B38'

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Carbaryl is an acetylcholine esterase inhibitor-type insecticide used for pest control on grapevine. We repeatedly observed the occurrence of interveinal leaf necrosis following carbaryl spray application in a *Vitis rupestris* x *Vitis riparia* F1 hybrid progeny vineyard. Spray applications induced necrosis in this progeny under both Missouri and New York field conditions an approximate one-to-one sensitive-to-insensitive segregation ratio and with 42% concordance. Results of subsequent *in vitro* experiments established causality between carbaryl treatment and leaf necrosis and confirmed the pattern of segregation observed in the field. We consistently map this phenotype to a major QTL on chromosome 16 of the female parent *V. rupestris* 'B38' regardless of whether we used field or *in vitro*-generated phenotype data. The PN40024 12x.v1 genome sequence under the QTL peak is a gene-rich region encoding several receptor-like kinases and nucleotide-binding leucine-rich repeat receptors. RNA-seq and qPCR analyses of the carbaryl-induced transcriptome demonstrated the up-regulation of genes encoding the immune response regulator EDS1, pathogenesis-related proteins and stilbene synthases in sensitive, but not in insensitive progeny plants. While the development of leaf necrosis involved certain components of pathogen-triggered cell death regulatory pathway, other molecular events did not agree with the "misguided immune response" paradigm. An extensive screen of native North American grapevine accessions suggested that carbaryl sensitivity is rare in *Vitis*, and possibly unique to the *V. rupestris* 'B38' genotype, though members of *Parthenocissus*, another Vitaceae genus, are damaged by carbaryl.

Keywords: Insecticide damage, Vitis rupestris 'B38', leaf necrosis, immune response, quantitative trait locus