# RESISTANCE TO *HETEROBASIDION ANNOSUM* S.L. IN *PICEA ABIES* IS ASSOCIATED WITH A MORE ACTIVE ALLELE OF PaLAR3, A LECUANTHOCYANIDIN REDUCTASE GENE

Title presented at workshop:
**DO ALLELE-SPECIFIC EXPRESSION PATTERNS CONTROL RESISTANCE TO *HETEROBASIDION ANNOSUM* IN *PICEA ABIES*?**

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Norway spruce [*Picea abies* (L.) Karst.] wood is economically important to the European forest industry and infections by the *Heterobasidion annosum* s.l. species complex, and particularly *Heterobasidion parviporum* the major forest pathogen on Norway spruce cause substantial losses. The economic losses to this pathogen amounts to approximately 200 0000 USD every day, only in Sweden. One major route for infection is carryover between rotations on already infected forest sites. Thus, replantation material with improved resistance could be a valuable resource in the management of *H. annosum* s.l. There is sufficient genetic variation in resistance against *H. annosum* s.l in Norway spruce for substantial progress to be made through genetic selection. The variation in resistance in Norway spruce is quantitative in its nature, and the genetic component explains 10–46 percent of the variation (Arnerup et al. 2010; Chen et al. 2018). No adverse correlations have been found between growth and resistance which makes resistance an attractive trait to implement in breeding programs (Chen et al. 2018). Resistance of *P. abies* to *H. annosum* s.l., is linked to a number quantitative trait loci (QTL) in the host (Lind et al. 2014).

We have examined one of the QTLs for variation in fungal growth in sapwood (FGS) reported by Lind et al. (2014) in 14 unrelated Norway spruce families from Northern Europe. The QTL includes *PaLAR3*, a *leucoanthocyanidin reductase* gene, (Nemesio-Gorriz et al. 2016). We found two conserved *PaLAR3* allelic lineages in Norway spruce and higher resistance to *H. parviporum* was associated with the minor allele *PaLAR3B* in all studied families. Trees carrying at least one copy of *PaLAR3B* showed a significant reduction in FGS after inoculation with *H. parviporum* compared to their half-siblings homozygous for the major allele. Progenies homozygous for *PaLAR3B* have significantly higher levels of (+)-catechin, the catalytic product of *PaLAR3*, in their bark than their siblings homozygous for *PaLAR3A*. Differences in transcriptional regulation between alleles is a likely explanation for the resistance associated with *PaLAR3B*. The two isoforms of the *PaLAR3* protein showed similar in vitro catalytic properties, but allele-specific transcript levels were significantly higher for *PaLAR3B* in the heterozygous progenies (Nemesio-Gorriz et al. 2016). Expression profiling suggests that *PaLAR3* is part of a wider transcriptional network responding to *H. annosum* s.l. infection in Norway spruce involving a number of transcription factors possibly interacting differently with promoters of the two *PaLAR3* allelic lineages in Norway spruce (Dalman et al. 2017, Nemesio-Gorriz et al. 2017).

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