

Regulation of effector-gene expression as concerted waves in *Leptosphaeria maculans*: a two-players game

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Leptosphaeria maculans is a phytopathogenic fungus causing stem canker on oilseed rape (*Brassica napus*), on which it displays a complex lifecycle. As for other host-interacting fungi, effector-genes (encoding sets of molecules involved in manipulation of the host immune defense system allowing for spreading into the host) are specifically expressed during infection. So far, all effector-genes cloned in *L. maculans* are located within repeat-rich regions of its genome. We have previously shown that i) effector-genes are associated with the repressive histone modification H3K9me3 during axenic culture, ii) a local removal of H3K9me3 was a pre-requisite for their specific induction *in planta*, yet iii) removal of H3K9me3, through inactivation of the lysine methyltransferase *KMT1*, does not induce expression of effector-genes at the same level as observed during infection of oilseed rape. Our hypothesis is that, although the control of H3K9me3 localization is key to regulate expression of effector-genes, a second layer of regulation, such as action of a transcription factor (TF), is involved in the tight coordination of effector-gene expression. We investigated the involvement of Pf2, a TF belonging to the fungal specific Zn2Cys6-family, in the control of effector-gene expression, and showed its concerted role in the regulation of pathogenicity, together with *KMT1*. Notably, transcriptomic analyses revealed an enhanced effect of the over-expression of *LmPf2* on effector-gene expression in a $\Delta kmt1$ background. Thus, *LmPf2* and *KMT1* would have an antagonistic role: *KMT1* inhibiting and *LmPf2* activating the expression of effector-genes. Our work unraveled a dual layer control of pathogenesis and effector-gene expression, involving a chromatin-based control and a TF-regulation.

Keywords: plant-pathogen interaction, effector, regulation, epigenetic, RNA-seq, transcription factor, CRISPR-Cas9