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Transcriptional and chromatin-based control of effector gene expression in the plant pathogenic fungus *Leptosphaeria maculans*

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Plant pathogens secrete an arsenal of small secreted proteins (SSPs) acting as effectors that modulate host immunity to facilitate infection. In fungi, SSP-encoding genes are often located in particular genomic environments and show waves of concerted expression during plant infection. To date, little is known about the regulation of their expression. *Leptosphaeria maculans*, a fungus responsible for stem canker of oilseed rape, has a bipartite genome structure alternating gene-rich and transposable element (TE)-rich regions. TE-rich regions, which encompass one third of the genome, are enriched in putative effector genes that present the same expression pattern (no or a low expression level during *in vitro* growth and a strong over-expression *in planta*). On these bases, we investigated the determinism of the concerted effector gene expression testing two hypotheses: (i) are one or several common regulators involved in the control of the concerted expression of effector genes? and / or (ii) are TE-rich regions targets of reversible chromatin modifications that affect the regulation of effector gene expression?

To identify putative regulators of effector gene expression, we established the repertoire of TFs of *L. maculans* and identified TFs only found in *L. maculans* genome or specifically induced during infection. We performed functional analyses on several TFs and showed that StuA plays a major role in infection and expression of effector genes in *L. maculans*. We also investigated the involvement of one histone modification, histone H3 lysine 9 tri-methylation (H3K9me3) in chromatin-based regulation of concerted effector gene expression. For this purpose, we silenced expression of two key players in heterochromatin assembly and maintenance, *HP1* and *Kmt1*, by RNAi. Whole genome oligoarrays performed on silenced-*HP1* and silenced-*Kmt1* transformants revealed an over-expression of SSP-encoding genes in TE-rich regions during *in vitro* growth. That increase of expression was associated with a reduction of H3K9 tri-methylation at two SSP-encoding gene loci. Our data strongly suggest that a chromatin-based control, mediated by HP1 and KMT1, represses the expression of at least part of the effector genes located in TE-rich regions during growth in axenic culture. We are now investigating the involvement of another key player in heterochromatin assembly, KMT6, involved in the heterochromatic-associated histone modification H3K27me3. Our hypothesis is that changes of lifestyle and a switch toward pathogenesis lift chromatin-mediated repression, allowing a concerted expression of effector genes mediated by one / or several TF(s).