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Colin Clairet[1], Jessica L. Soyer[1], Nicolas Lapalu[1], Adeline Simon[1], Françoise Blaise[1], Eva H. Stukenbrock[2] and Isabelle Fudal[1]

[1]UMR BIOGER, INRA, AgroParisTech, Université Paris-Saclay, 78850 Thiverval-Grignon, France
[2]Max Planck Institute for Evolutionary Biology, Plön, and Christian-Albrechts University of Kiel, Germany

Leptosphaeria maculans, a fungus causing stem canker, colonises oilseed rape in two stages: an early stage of leaf colonisation and a late stage of systemic stem colonisation without visible symptom before stem canker appears. *L. maculans* produces at least two waves of effectors, key elements of pathogenesis facilitating host invasion. *L. maculans* presents a bipartite genome structure alternating gene-rich and transposable element (TE)-rich regions. While TE-rich regions are enriched in putative effector genes strongly over-expressed during early infection, gene-rich regions contain putative effector genes specifically expressed during late infection. Here, we investigated influence of reversible histone modifications affecting genomic regions sheltering different sets of effector genes on their concerted expression. We analysed nucleosome positioning, location of histone modifications and gene expression at the genome scale combining MAINE-seq, ChIP-seq and RNA-seq data during axenic growth and performed functional analysis of two chromatin modifiers (KMT1 and KMT6). We analysed *in vitro* ChIP-seq data targeting two heterochromatin modifications, H3K9me3 and H3K27me3, and a euchromatin modification, H3K4me2, and found that gene-rich regions are associated with H3K4me2 and H3K27me3 while TE-rich regions are associated with H3K9me3. Analysis of *in vitro* MAINE-seq data showed distinct nucleosome organization for genes located in TE-rich or gene-rich regions. While RNAi silencing of *KMT1*, which encodes a protein involved in H3K9me3 deposition, induced an over-expression of genes located in TE-rich regions, particularly ‘early’ effector genes, silencing of *KMT6*, involved in H3K27me3 deposition, leads to a deregulation of genes not only associated with H3K27me3 in the wild type strain, suggesting a relocation of different histone modifications.