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Leptosphaeria maculans, a hemibiotrophic fungus responsible of stem canker, colonises oilseed rape in two stages: an early stage of cotyledon or leaf colonisation, and a late colonisation stage during which the fungus colonises systemically without visible symptom the plant before stem canker appears. *L. maculans* presents 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-encoding genes that present the same expression pattern (no or a low expression level during *in vitro* growth and a strong over-expression during early infection in cotyledons and leaves; ‘early’ effectors). In contrast, gene-rich regions were recently reported to contain putative effector-encoding genes specifically expressed during the late stages of stem infection (‘late’ effectors). We have previously investigated the involvement of the chromatin structure of repeat-rich regions on the expression of ‘early’ effector genes: RNAi silencing of two genes encoding key players in heterochromatin assembly through histone modification H3K9me3, HP1 and KMT1, induced an over-expression of genes located in AT-isochores, particularly ‘early’ effector genes but no modification of ‘late’ effector genes expression. Here, we performed analysis of nucleosome positioning, chromatin structure and gene expression at the genome scale combining MAINE-seq, ChIP-seq and RNA-seq data during *in vitro* growth of *L. maculans*. We analysed *in vitro* ChIP-seq data targeting heterochromatin modifications, H3K9me3 and H3K27me3, and a euchromatin mark, H3K4me2 and found that gene-rich regions are associated with H3K4me2 and H3K27me3 while TE-rich regions are associated with H3K9me3. Effector genes are also associated with distinct heterochromatin marks according to their genomic location and expression kinetics: while ‘early’ effector genes located in TE-rich regions are associated with H3K9me3, ‘late’ effector genes located in gene-rich region are associated with H3K27me3. Genome-wide nuclesome positioning was analysed using MAINE-seq data, showing distinct nucleosome organization for genes located in TE-rich or gene-rich regions, and according to gene expression level. Finally, we recently investigated the role of another key player in heterochromatin assembly, KMT6, involved in the heterochromatic-associated histone modification H3K27me3, on the control of effector gene expression. RNAi silencing of *KMT6* leads to a deregulation of genes not only associated with H3K27me3 in the wild type strain, suggesting a relocation of different histone modifications.