Poster

Chromatin regulation of virulence gene clusters in Ustilago maydis



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Keywords: Gene silencing; pathogenesis; regulation

ABSTRACT

Pathogenic fungi possess chromatin modification mechanisms that are involved in their virulence and interaction with the host 1. This modification usually occurs as a consequence of the presence or absence of certain molecular marks on histones, such as methylations or acetylations. It allows the transition between their two conformational states, euchromatin and heterochromatin, associated with gene transcriptional activation and gene silencing, respectively 2,3. The maize fungus Ustilago maydis has become a model organism for the study of plant pathogenesis due to its ease of handling and the multiple available genetic and cellular tools 4. This pathogen uses a large number of factors and pathways that regulate gene expression at each stage of the infective process, but little is known about the role of chromatin in that 1. Many genes involved in that process are clustered in gene regions and are activated at specific times 4,5. Therefore, we should think of an active role of chromatin in this regulation. However, the absence of typical heterochromatin-associated marks and regulators in U. maydis has so far been observed 1. In this laboratory, we have sought other likely regulators of this process and found that the histone methyltransferase Ash1 affects the virulence activity through the silencing of gene clusters involved in this process. In this project we try to find out if this is the only regulator of heterochromatin in U. maydis. For this purpose, different strains with antibiotic resistance markers in silenced regions controlled by Ash1 were developed, allowing antibiotic resistance when these regions are derepressed. Spontaneous resistant mutants were obtained from these strains and Ash1 was consequently sequenced to determine if the silencing was affected by a mutation in Ash1. Surprisingly, the methyltransferase gene was not mutated, suggesting the presence of other regulators. In addition, we performed a ChIP-qPCR to see if the epigenetic mark associated with Ash1 was still present. We observed a decrease in this mark in one of the spontaneous mutants in different virulence regions, which also suggests the presence of other regulators, such as some demethylases. Subsequently, in order to discover whether the decrease in this mark is maintained over time, we have studied its stability in successive generations. We noticed antibiotic-resistant colonies trended towards a decrease which might imply a reacquisition of the epigenetic mark.

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