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Epigenetic Regulations are Combined in DNA Methylation and Transcriptomes

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INTRODUCTION

As sessile organisms, plants are exposed to constantly changing environments, often multiple stresses. Pathogen invasions such as viral, bacterial and fungal infections severely inhibit plant growth and development, lead to significant yield losses and threaten food/feed security worldwide. Through evolution, plants have adapted complex systems involving coordinated global gene expression networks to defend against pathogen attack. In recent years, growing evidence suggests that pathogen infection triggers local and global epigenetic changes that reprogram the transcription of plant defense genes and help plants fight pathogens. Here, we summarize plant defence pathways and epigenetic mechanisms and extensively review current knowledge on histone modifications and chromatin remodeling factors found in epigenetic regulation of plant responses to biological stress. It is hoped that epigenetic mechanisms can be investigated in developing tools to generate stress-tolerant plant cultivars.

DESCRIPTION

DNA methylation (DNAme) and post-translational histone modifications (PTMs) play important roles in transcriptional regulation. Although many reports have individually characterized the functions of such chromatin markers, recent genome-wide studies have revealed a surprisingly complex interplay between them. Here, we focus on the interaction between DNAm and methylation of specific lysine residues in histone H3 tails. We describe the effects of genetic disruption of related mouse methyltransferases on the chromatin marking landscape and transcriptome. In addition, we describe specific neurological growth syndromes and cancers that result from pathogenic mutations in human orthologues of these genes. The synthesis of these observations highlights the fundamental importance of crosstalk between DNA and histone H3 methylation in development and disease.

The role of histone modifications in transcription is not fully understood. Here we investigate the relationship between histone modifications and transcription using experimental perturbations combined with sensitive machine learning tools. Transcription predicted variations in complex chromatin states, such as active histone marks and bivalent promoters, to single nucleosome resolution, with an accuracy comparable to agreement between independent His ChIP-seq experiments. Blocking transcription rapidly removed two punctate markers, H3K4me3 and H3K27ac, from chromatin, indicating that transcription is required for active histone modifications. Consistent with the role of RNA in PRC2 recruitment, H3K27me3 maintenance also required transcription.

CONCLUSION

Recent developments in spatial omics methods have enabled single-cell profiling of the transcriptome and 3D genome organization at high spatial resolution. By expanding the repertoire of spatial-omics tools, spatially resolved single-cell epigenomics methods accelerate our understanding of the spatial regulation of cell and tissue function. Here we report a method for spatially resolved epigenome profiling of single cells using in situ tagmentation and transcription followed by multiplexed imaging. We demonstrated the ability to profile histone modifications that mark active promoters, putative enhancers, and silent promoters in single cells, and constructed a high-resolution spatial atlas of hundreds of active promoters and putative enhancers in embryonic and adult mouse brains. Our results suggested putative promoter-enhancer pairs and enhancer hubs that regulate developmentally important genes. We believe that this approach is generally applicable to spatial profiling of epigenetic modifications and DNA-binding proteins, thereby enhancing our understanding of how gene expression is spatially and temporally regulated by the epigenome.

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