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Molecular mechanism of heterochromatin formation in the fission yeast, *Schizosaccharomyces pombe*

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The stable propagation of transcriptional states through numerous cell divisions is central to adaptability of all living organisms. This process, coined epigenetic inheritance, was first understood molecularly in lysogenic *E. coli* harboring a lambda prophage. Decades of research has revealed that all epigenetic pathways from bacteria to humans exploit the same molecular principles: specific and cooperative molecular interactions target specific regions of the chromosome, and create positive and negative feedback loops, respectively, which ultimately lock transcriptional states into a specific fate. The fission yeast heterochromatin has served as a faithful model for understanding the molecular mechanism of epigenetic inheritance in eukaryotes. Heterochromatin in *S. pombe* like other higher eukaryotes is demarcated by methylated lysine 9 of histone H3 (H3K9me). Several key silencing proteins, including heterochromatin protein 1 (HP1) bind to H3K9me and recruit repressive activities to heterochromatin. These repressive activities operate in cis by limiting RNA Pol II transcription and degradation of heterochromatic transcripts via the exosome- or RNAi-dependent mechanisms. In the fission yeast, a single histone methyltransferase, Clr4 belonging to the highly conserved Suv-39 family of proteins, methylates H3K9. Current models of epigenetic pathways posit that recruitment of Clr4 to heterochromatin is the initiating event for heterochromatin formation, tethering all repressive activities via H3K9me and downstream modifications. Here we show that several silencing proteins 'prime' chromatin for Clr4 recruitment, and are required for efficient H3K9 methylation. These data reveal a novel previously underappreciated step in heterochromatin formation, which precedes H3K9 methylation and regulates heterochromatin formation in eukaryotes.

Biography

Mo Motamedi recently started his lab at Massachusetts General Hospital Center for Cancer Research as an Assistant Professor of Medicine at Harvard Medical School. He is a recipient of the 2012 V Scholar Award. Prior to his faculty appointment, he was an NSERC and CIHR postdoctoral fellow in Cell Biology at Harvard Medical School. His lab is focused on the mechanisms by which chromatin and non-coding RNAs initiate, maintain and propagate epigenetic states in eukaryotes.

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