Cytosine methylation: a versatile epigenetic mark for phenotypic adaptation

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Eukaryotic species use cytosine methylation to facilitate phenotypic adaptation to their environments, which can include both the modulation of developmental and adaptive gene expression programs. Variations in the complement of cytosine methyltransferase enzymes have been interpreted to reflect multiple versions of a toolkit for phenotypic adaptation. During evolution, specific parts of this toolkit could have been contracted or expanded to facilitate specific requirements for genome regulation. We are investigating this hypothesis in several independent models. For example, the methylation landscapes of human and mouse genomes, are characterized by high developmental plasticity, but substantial stability towards environmental changes, consistent with the highly canalized phenotypes of mammals. Other organisms (honeybees, locusts, crayfish), however, show a substantially higher degree of phenotypic plasticity. We are using whole-genome sequencing technologies to establish genome methylation maps of various model systems at single-base resolution and an overview of the results will be presented. Furthermore, many parasites have lost canonical DNA methyltransferase genes, and utilize the Dnmt2 enzyme for tRNA methylation. Our previous studies have established RNA methylation as a novel epigenetic mechanism that provides specific advantages in the short-term adaptation to environmental changes. The significance of this mechanism for epigenetic regulation in parasites will be discussed.

Key words: DNA methylation, Schistosoma, evolution