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EPIGENETIC DNA METHYLATION STUDY OF BIVALVE TUMORS

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Abstract: DNA methylation, the best studied epigenetic mechanism, is essential in regulating gene expression and gene silencing in the eukaryotic genome. Different cell types have specific and pre-established DNA methylation profiles. Alterations in those profiles may lead to cell malfunction, illness and, ultimately, tumorigenic events. DNA methylation is known to be abnormal in most cancer types, either being the cause behind these tumorigenic events or just a byproduct. Recent studies suggest for a crucial role of epigenetic marks on both bivalve early development and their adaptation to environmental stressors, but the involvement of DNA methylation in bivalve tumorigenesis is far from understood. In this work we tested out the global methylation profiles in healthy and neoplastic golden carpet shell, *Polititapes aureus*, and cockles, *Cerastoderma edule*, by means of a Methyl-Sensitive Amplification Polymorphism (MSAP) approach. This assay, based on differential cleavage reactivity of *HpaII* and *MspI* to cytosine methylation, allows assessment of differences in methylation profiles among experimental groups. Each genomic DNA sample was digested in parallel with *EcoRI* + *HpaII* and *EcoRI* + *MspI*. Following AFLP reading by capillary electrophoresis the resulting restriction profiles were scored for all samples and analysed. The results suggest that the largest contribution to the differences are due to genomic rearrangements, with a small contribution of methylation to the overall MSAP variation.

Key words: Disseminated neoplasia, Bivalves, Methylation

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