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HISTONE MODIFICATIONS: THE DOUBLE EDGED SWORD IN GYNAECOLOGICAL CANCERS

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Abstract

Gynaecological cancers are one among the fatal cancers that affects women worldwide. It includes endometrial, ovarian, cervical, vaginal and vulvar cancers. Epigenetic aberrations play a crucial role in the development and progression of such cancers, which includes global genomic hypomethylation, Cytosine-Guanine (CpG) island promoter hypermethylation, changes in histone modifications and changes in chromatin-modifying enzymes. This review aims at elaborating the significance of epigenetic changes specifically the histone modifications, at H3K9, H3K27, H3K4 and others involved in gynaecological tumourigenesis. They can independently or synergistically act along with DNA methylation for repression of the tumour suppressor genes and possibly for the activation of various oncogenes like CLDN3, CLDN4, GATA4, etc. in gynaecological cancers. These modifications may pave the way in the future for the identification of biomarkers in early diagnosis and prognosis with an opportunity for targeted drug delivery. A systematic review was done using Internet based scientific databases. Relevant clues about the role of Histone modifications like acetylation, deacetylation, mono/di/trimethylation, demethylation of the histone tails (H3/H4) involved in tumour development and progression were reviewed. Keywords- Gynaecological Cancer, Histone modification, Acetylation, Deacetylation, Demethylation

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A LITERATURE REVIEW ON TRANSLATIONAL GENOMICS OF THE MALIGNANT RHABDOID TUMOURS AND ITS CURRENT PERSPECTIVES

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Abstract

Background: Malignant rhabdoid tumor is a rare childhood tumor that commonly starts in the kidneys but also can occur in other soft tissues or in the brain. Malignant rhabdoid tumor occurs most commonly in infants and toddlers; the average age of diagnosis is 15 months old .In rhabdoid tumours, nearly all cases are thought to develop from a single copying (translation) error of one gene, the SMARCB1 (INI1) gene. Each cell of the body contains two SMARCB1 genes. Both of these need to be damaged or altered to result in the development of a rhabdoid tumour

Objectives: To provide a overview about translational genomics of the rhabdoid tumours and its current perspectives method of study: By reviewing

scientific and research publications-journals

Results and Conclusion: Once localized, tumors can be biopsied to assess risk and select therapy. Deployment of screening technologies in the clinical setting will depend on their ability to improve clinical outcomes in an efficient and cost-effective manner. Importantly, not only will such advances benefit patients and families affected by rhabdoid and related tumors, the results of such investigations are likely to be generalizable to a wide array of *SMARCB1*-dependent cancers and the epigenetic control of neoplasia in general.

Keywords: rhabdoid, *SMARCB1*, Translational genomics

