Taha A Kumosani, J Cancer Sci Ther 2015, 7:8
http://dx.doi.org/10.4172/1948-5956.C1.057

OMICS International
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5th World Congress on
Cancer Therapy

September 28-30, 2015 Atlanta, USA

Hypermethylation of P15, P16, and E-cadherin genes in ovarian cancer

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Both p16 and p15 proteins are inhibitors of cyclin-dependent kinases that prevent the cell going through the G1/S phase transaction. E-cadherin is a transmembrane glycoprotein that mediates calcium-dependent interactions between adjacent epithelial cells. Two groups of patients were selected: the first group suffered from epithelial serous ovarian tumors and the second group suffered from benign ovarian lesions; ovarian tissue samples from all the subjects (benign and malignant) were subjected to methylation-specific polymerase chain reaction for methylated and unmethylated alleles of the genes (E-cadherin, p15, and p16). Results obtained showed that aberrant methylation of p15 and p16 genes were detected in 64.29 and 50% of ovarian cancer patients, while E-cadherin hypermethylation was detected in 78.57% of ovarian cancer patients. Methy- lation of E-cadherin was significantly correlated with different stage of disease ($p < 0.05$). It was found that the risk of E-cadherin hypermethylation was 1.347-fold, while risk of p15 hypermethylation was 1.543-fold and p16 was 1.2-fold among patients with ovarian cancer than that among patients with benign ovarian lesions. In con- clusion, Dysfunction of the cell cycle and/or the cell–cell adhesion molecule plays a role in the pathogenesis of ovarian cancer and that the analysis of the methylation of p15 and E-cadherin genes can provide clinically important evidence on which to base the treatment.

Biography

Taha A Kumosani has studied Biochemistry and Biochemistry of Cancer for 25+ years, during which time he has authored more than 150 research articles. He has served on the editorial boards for many Scientific Journals. including his current membership with many Scientific Society.

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