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Effect of hypermethylation in ovarian cancer: computational approach

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Abstract

Ovarian cancer in women represents a mortality rate of more than four percent worldwide. In cancer, CpG islet hypermethylation of some gene promoters often leads to the inactivation of tumor suppressor genes. Ovarian cancer is characterized by a targeted hypermethylation, hence the need to study this hypermethylation. We have adopted the hypothesis that a very high mutation rate can cause the epimutation phenomenon, causing a 5-fold deamination methyl-cytosine in thymine, consequence of the mutation. To test this, we chose the P53, RASSF1A, WAF1, ARHI, CAV1, DOC-2, BRCA2 and BRCA1genes. The methylation status was first determined by the Chargaff coefficient, which represents a high percentage of about 50 percent for all the sequences chosen, secondly, the $CpG_{o/e}$ ratio was calculated. ANOVA test has been applied in order to be sure of the correlation between these two calculations and showed that the Chargaff coefficient is significant. Lastly, we adopted Hidden Markov Models (HMM). To indicate exactly the CGI region in the promoters of the studied genes. This model maximizes the mathematical probability of having a hypermethylated region.

Keys words: Ovarian cancer, suppresser tumor gene, hypermethylation, hidden Markov models.

Specifications Table

Subject area	Biology
More specific subject area	Breast cancer and bio-informatic
Type of data	Table, graph, figure
How data was acquired	Algorithm
Data format	analyzed,
Experimental factors	traitment of sequence
Experimental features	Calcul mathematic, traitment by algorithm, ANOVA test,
Data source location	world wide
Data accessibility	State if data is in public repository. www.ncbi.com