Sateesh Nallapalle et al., J Cancer Sci Ther 2014, 6:9
http://dx.doi.org/10.4172/1948-5956.S1.038

OMICS Group Conferences
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Global Cancer Conference & Medicare Summit
September 15-17, 2014 Hyderabad International Convention Centre, India

Promoter polymorphism of FASL confers protection against female specific cancers and those of FAS impact the cancers divergently

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Breast, ovarian, cervical and endometrial tissues, and the malignancies of these tissues are sensitive to estrogen/estradiol, age at menarche, child bearing and menopausal age. Moreover, hormonal therapy and anticancer drugs also have been shown to predispose these patients to cancer. In view of the commonalities among female specific tissues and cancers of these tissues, we hypothesized that similar mechanisms may be operating in the female specific cancers. To begin to test the hypothesis, it was investigated and compared the association of FAS (-1377 G>A and -670 A>G) and FASL (-844 T>C) promoter polymorphisms with breast, ovarian, cervical and endometrial cancers. The genotype AA of FAS -1377 G>A polymorphism was associated with an increased risk for breast cancer alone while the GA variant of the polymorphism enhanced the risk of both breast and cervical cancers (P≤0.005). In contrast, FAS -670 AG variant was significantly associated with lowered risk of breast cancer alone but not that of other cancers. On the other hand, the prevalence of CC variant of FASL-844 T>C was lower in cases and was found to be protective towards breast, ovarian, cervical and endometrial cancers (P≤ 0.01). We also report that the effect of combined genotypes of FAS and/or FASL SNPs differed from that of individual polymorphisms. Although, risk and protective haplotypes of FAS SNPs were observed across the cancer phenotypes, the association of the haplotypes was significant for breast cancer alone with a 3-fold enhanced risk. Our results indicate that FASL-844 T>C polymorphism (CC genotype) is protective against female specific cancers, whereas the FAS -670 AG variant lowers risk of breast cancer alone. In contrast, FAS-1377 G>A polymorphism was found to elevate risk of breast and cervical cancers, significantly. The protective effect of the FASL SNP seen in this study is in line with the proposed hypothesis and suggests similar bio-molecular mechanisms involving FASL and/or FAS may play a role in female specific cancers.

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