

ever diet is thought to play a major role. One dietary factor that has attracted interest in the past two decades is tea. Animal and cell culture experiments have shown tea to have potential anti-carcinogenic properties, which could relate to CRC. Conversely, epidemiological studies have shown mixed results.

Objective: This aim of this study was to investigate the effect of tea consumption on the risk of colorectal cancer using data from a case-control study of Western Australian adults.

Methods: The Western Australian Bowel Health Study examined the association between tea consumption ten years ago, and incident colorectal cancer. This study included 371 cases and 525 frequency matched controls, aged between 41-80 years, from across Western Australia. Lifestyle and dietary information was collected via self-administered questionnaires. The data were analysed using multivariable logistic regression. The exposure variables were hot green, black and white tea (i.e. tea with milk). Incident colorectal cancer cases were identified through the Western Australian Cancer Registry and confirmed microscopically for the period 1st June 2005 to 30th May 2007.

Results: Tea was not significantly associated with colorectal cancer for any exposure category. There was a non-significant decrease in CRC risk following low-level green tea consumption (adjusted OR=0.69, 95%CI=0.38-1.25).

Conclusion: There was no observed effect on the risk of CRC following consumption of tea. It is possible that higher exposures to tea, especially green tea, may be significantly associated with a decrease in CRC risk.

11q13), an amino acid transition has been reported at codon 105 (Ile105Val), leading to expression of an active but functionally different protein. The aim of this study was to investigate the frequencies of Ile105Val polymorphism in the exon 5 of GSTP1 gene and its effect on the risk of developing breast cancer in Mersin sample of Turkish population. In addition, we investigated whether an association exists between breast cancer and other risk factors including the age, at menarche, age at menopause, smoking, BMI, and family history.

Materials and Methods: Our study group consisted of 167 individuals of whom 99 healthy women controls and 68 breast cancer cases. The experimental group was comprised of women who had been diagnosed with breast cancer at the Department of Medical Oncology, Mersin University, Turkey. Controls were selected by taking age and sex variable into consideration. Genomic DNA from breast cancer patients and control subjects was analyzed by PCR-RFLP.

Results: For the exon 5 of GSTP1 gene, the distribution of AA and AG genotypes in Mersin sample of Turkish population were 64% and 4% in control group whereas this genotype distribution were 58% and 7% in patients, respectively. Putative risk factors including age, body mass index or family history were found to be correlated with the developing breast cancer. However; It was determined that smoking, menarch age and menopause status weren't associated with breast cancer risk.

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THE INVESTIGATION OF RELATIONSHIP BETWEEN THE POLYMORPHISM IN EXON 5 OF GLUTATHIONE S-TRANSFERASE P1 (GSTP1) GENE AND BREAST CANCER

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Introduction: Beside environmental factors, genetic factors have an important place in the etiology of breast cancer which is one of the most common and highest mortality cancers among women in the worldwide. Breast cancer is associated with different types of somatic genetic alterations such as mutations in oncogenes and tumor suppressor genes. Glutathione S-transferases (GSTs) are a superfamily of enzymes that are potentially important in regulating susceptibility to cancer because of their ability to metabolize reactive electrophilic intermediates to usually less reactive and more water soluble glutathione conjugates. In GSTP1 (chromosome

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BIOMARKER IDENTIFICATION IN HORMONE RELATED CANCERS

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Tumor markers have positively impacted the detection, diagnosis and prognosis of hormone related cancers, specifically prostate and breast cancers. A nutritional based steroid hormone, vitamin D3 (VD3) is useful for inducing apoptosis of prostate cancer cells through specific gene targets (1). Human primary prostate cell lines were used to obtain cDNA microarray data under different culture conditions, in the

presence and the absence of VD3. Our study covered the signal transduction pathway of an animal model prepared by knocking out the VDR gene (2) and the searching of new prostatic genes starting from their cDNA in the Human Genome Data Base. Translational bioinformatics has been used widely. We validated the down-regulation of heat shock proteins (Hsp)-70 and bcl-2, and up-regulation of the Apaf1, Hsp-90, estrogen receptor- (ER), Her-2/neu, and paxillin genes at the protein level. Moreover, we found that VD3 is capable of inhibiting the expression of multiple anti-apoptotic proteins in