hOGG1 Codon 326 or XRCC1 Codon 399 Polymorphisms and the Risk of Prostate Cancer

Introduction and Objective: Prostatic carcinogenesis is a complex, multistep, multifactorial process. Like many malignancies, this cancer is the result of interactions between genetic factors of the host and environmental factors. One of the proposed mechanisms involves oxidative DNA damage caused by reactive oxygen species (ROS). Base excision repair (BER) is a very important mechanism for repairing oxidative DNA damage. Human oxoguanine glycosylase 1 (hOGG1) and X-ray repair cross-complementing 1 (XRCC1) are enzyme genes of BER.
Materials and Methods: We studied the effect of hOGG1 codon 326 and XRCC1 codon 399 on prostate cancer susceptibility in a case control study of 168 prostate cancer patients and 172 male controls, to determine whether this polymorphism is a biomarker for the risk of prostate cancer.
Results: No significant difference between prostate cancer and hOGG1 codon 326 (OR: 1.62, 95%CI: 0.97-2.73) or XRCC1 codon 399 polymorphisms (OR: 0.45, 95%CI: 0.18-1.14) was observed.
Conclusions: We found no association between hOGG1 codon 326 or XRCC1 codon 399 polymorphisms and occurrence of prostate cancer in Japanese populations.