Effects on sister chromatid exchange frequency of polymorphisms in DNA repair gene XRCC1 in smokers.

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摘要

Abstract

The association between metabolic polymorphisms and cigarette smoking-induced cancers has been documented. However, the role of DNA repair polymorphism in carcinogenesis is less clear. To investigate if the polymorphisms of metabolic traits and DNA repair modulate smoking-related DNA damage, we used sister chromatid exchange (SCE) as a marker of genetic damage to explore the relationship of microsomal epoxide hydrolase (mEH), glutathione S-transferase M1 (GSTM1), and X-ray cross-complementing group 1 (XRCC1) and cigarette smoking-induced SCE. Sixty-one workers without significant exposure to mutagens were recruited. Questionnaires were completed to obtain detailed occupational, smoking, and medical histories. SCE frequency in peripheral lymphocytes was determined using a standard cytogenetic assay and GSTM1, mEH (exons 3 and 4), XRCC1 (codon 399) genotypes were determined using polymerase chain reaction-restriction fragment length polymorphism (PCR/RFLP). Smokers had higher SCE frequency than non-smokers (8.4 versus 7.1, $P<0.05$). Among workers who had smoked equal to or greater than 10 cigarettes each day, those with XRCC1 Arg/Gln+Gln/Gln had higher SCE frequency than those with XRCC1 Arg/Arg after adjusting for potential confounders (9.0 versus 7.9, $P<0.05$). The interaction of XRCC1 and cigarettes smoked per day on SCE frequency was also observed ($P=0.02$). There was no significant interaction between cigarettes smoked per day with GSTM1 and mEH on SCE frequency. Our results support previous epidemiological studies that XRCC1 may play a role in cigarette smoking-induced lung cancer.