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Genetic variation in PPARGC1A may affect the role of diet-induced inflammation in colorectal carcinogenesis

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As chronic inflammation plays an important role in colorectal carcinogenesis, inflammation related gene-diet interactions may affect colorectal cancer risk. Therefore, we investigated whether genetic susceptibility alters the effect of diet-induced inflammation on the risk of colorectal cancer. This study included 701 colorectal cancer patients and 1402 controls. We selected six polymorphisms in four genes (IL1B, TNF, PPARG and PPARGC1A) and calculated diet-induced inflammation using the dietary inflammatory index (DII). Multiple logistic regression models were applied to estimate odds ratios (ORs) and corresponding 95% confidence intervals (CIs) of the main effect of genetic variants and the DII as well as their interactions. Subgroup analyses were performed by anatomic site and other risk factors. Among the investigated polymorphisms, heterozygous carriers of rs3774921 in the PPARGC1A gene were at higher risk of colorectal cancer (OR=1.30; 95% CI, 1.05-1.62 for TC vs. TT). When the data were stratified by rs3774921 genetic variants, the role of a proinflammatory diet in colorectal carcinogenesis was more prominent among homozygous variant allele carriers (OR=4.35; 95% CI, 1.89-10.03 for high vs. low DII) (P for interaction=0.022). When stratified by anatomic site, this association was much stronger for rectal cancer patients (OR=7.57; 95% CI, 2.30-24.93 for high vs. low DII) (P for interaction=0.013). Additionally, this interaction was significant among those older than 55 years old, not exercising regularly and drinking alcoholic beverages. Conversely, the other investigated polymorphisms did not show any association or interaction with diet-induced inflammation in relation to colorectal cancer risk. This study suggests that a pro-inflammatory diet has a differential effect on colorectal cancer risk based on PPARGC1A genetic variation with differential associations according to anatomic location and other risk factors. Although the findings support the molecular link between metabolism and inflammation, future studies are required to confirm our results.

Biography

Jeongseon Kim is a Professor in the Department of Cancer Control and Policy in Graduate School of Cancer Science and Policy. She has completed her PhD in Nutritional Epidemiology from New York University, USA. She has been primarily involved in the investigation of dietary factors, using epidemiologic approaches, in the cause and prevention of chronic diseases especially cancer and its important conditions.

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