DIETARY FATTY ACIDS INTAKE AND COLORECTAL CANCER RISK: THE ROTTERDAM STUDY

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Introduction: Different dietary components have been associated with colorectal cancer (CRC). However, whether dietary fat intake is associated with CRC is unclear. Also, various components of diet and plasma may modify the effect of dietary fatty acids on CRC risk. We aimed to investigate whether dietary polyunsaturated (PUFA) and saturated (SFA) fatty acids intake is associated with CRC and whether this association could be modified by dietary fiber and serum lipid levels.

Methods: We analyzed data from 4902 participants of the first cohort of the Rotterdam Study, a prospective follow-up study among subjects aged 45 years and older. At baseline, diet was measured by a food frequency questionnaire. CRC events were classified according to the tenth edition of the International Classification of Disease. Multivariable adjusted hazard ratios (HRs) were calculated using Cox regression models.

Results: During a follow up of 24 years, we identified 218 incident CRC cases. There was no association between PUFA intake and CRC. A positive linear relationship was established between SFA intake and CRC (HR = 1.02, 95% CI = 1.01-1.03). In participants with high dietary fiber intake (>median), PUFA intake was associated with an increased risk of CRC (HR = 2.12, 95% CI = 1.04-4.29 for the 4th quartile vs. 1st quartile). Among subjects with low dietary fiber intake (<median), SFA intake was associated with higher CRC risk (HR = 1.52, 95% CI = 1.12-2.06 for the 4th quartile vs. 1st quartile). In contrast, among subjects with high dietary intake (> median), SFA intake was associated with higher CRC risk (HR = 0.97, 95% CI = 0.95-0.995). Higher PUFA intake was associated with an increased risk of CRC in subjects with low serum cholesterol (PInteraction = 0.01 for n-3 PUFA and fiber intake; PInteraction = 0.05 for n-6 PUFA and serum cholesterol).

Conclusion: The results from this large prospective cohort suggest that dietary fat intake may be involved in colorectal carcinogenesis, but a complex interaction with dietary fiber and serum lipids may be involved. Further studies are needed to evaluate the potential interaction between dietary and non-dietary factors and their effects on colorectal cancer risk.

References