Gene-diet interaction on cancer risk in colorectal region.

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ABSTRACT

Genetic factors clearly play a role in carcinogenesis, but migrant studies provide unequivocal evidence that environmental factors are critical in defining cancer risk. Therefore, one may expect that the lower availability of substrate for biochemical reactions leads to more genetic changes in enzyme function. At an international level, colorectal cancer (CRC) is a major cause of morbidity and mortality. Diet plays a major etiologic role, and a range of putative dietary carcinogens have been identified. The probability with which these lead to mutations, and thereby cause cancer, is strongly impacted by variants in genes coding for xenobiotic metabolizing or DNA repair enzymes. Nutrient deficiencies also play a role, which will be exacerbated by variants in metabolic genes. Characteristics of gene and dietary factors are divided into four categories: one carbon metabolism-related gene polymorphisms and dietary factors including folate, vitamin B group and methionines; oxidative stress-related gene polymorphisms and antioxidant nutrients include vegetable and fruit intake; carcinogen-metabolizing gene polymorphisms and meat intake including heterocyclic amines and polycyclic aromatic hydrocarbon; and other gene-diet interactive effect on cancer. Some diet-regulated genes (and their normal, common variants) are susceptibility genes and likely to play a role in the onset, incidence, progression, and/or severity of chronic diseases.The degree to which diet influences the balance between healthy and disease states may depend on an individual's genetic makeup.

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