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Should the diet of colon cancer patients be personalized based on gene mutation profiles of tumor cells

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Colon cancer is a major cause of cancer related deaths and is known to be strongly associated with diet. Both carcinogenic and chemopreventive effects of diet have been described. In colon cancer different molecular profiles determine the carcinogenic progression, malignancy and ultimately the response to radio and chemotherapy. Mutations in particular genes such as KRAS, BRAF and P53 are major players in the abnormal proliferation of cancer cells and constitute molecular targets for cancer therapy. Recent evidence shows that colon cancer cells with different mutation profiles respond differently to compounds present in traditional foods and other plant sources that may even potentiate the effects of therapeutic drugs. Activating mutations in the MAP Kinase pathway (KRAS or BRAF) or of the PI3K pathway both contribute to increased cell proliferation. However, anticancer responses are observed for rosmarinic acid in KRAS mutated cells whereas only cells with PI3K activation respond to ursolic acid, both compounds present aromatic herbs and other food plants. Importantly, compounds present in the diet may also potentiate the response to chemotherapeutic drugs, such as 5-FU, quercetin increasing p53 dependent cell death in response to 5FU and ursolic acid increasing p53 independent cell death. The implication is that not all tumors are sensitive to the same dietary constituents and that personalizing the diet according to the molecular profile of the patient's tumor may be desirable to maximize the beneficial effects of diet.

Biography

Cristina Pereira-Wilson has completed her PhD from the University of Tondheim, Norway and is currently a Professor at the University of Minho, Braga, Portugal. Her scientific interests are in the areas of Physiology, Biochemistry, Nutrition and Pharmacology. She has published more than 40 papers in reputed journals.

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