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A high fat diet may increase colon cancer risk through the action of deoxycholate, a carcinogenic secondary bile acid

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Dietary fat causes bile acid secretion into the gastrointestinal tract. Among individuals in the United States, a relatively high fat diet (based on increased levels of milk fat and beef fat) doubles the level of bile acids in the colon. High fat Western diets increase the risk of colon cancer.

The bile acid deoxycholic acid (DOC) is likely important in colon cancer etiology. Exposure of colon cells to DOC induces reactive oxygen and nitrogen species, and DNA damage. Long-term exposure to DOC causes selection of apoptosis resistant colonic epithelial cells. We showed that DOC causes aneuploidy and micronuclei formation, indicators of genomic instability, in colon epithelial cells.

We tested DOC as a potential colon carcinogen in mice. Adding 0.2% DOC to the diet of wild-type mice resulted in fecal DOC of about 4.6 mg/g dry weight, comparable to DOC in feces of humans on a high fat diet of about 6.4 mg/g dry weight. Feeding a DOC-supplemented diet to 18 wild-type mice for 8 to 10 months caused colonic tumors in 17 mice, where 10 of the mice developed at least one colon cancer while 7 of the 8 remaining mice developed one or more serrated sessile adenomas.

Addition of the antioxidant chlorogenic acid at 0.007% to the DOC-supplemented diet significantly reduced tumor formation in the mice. These results suggest that a high fat diet in humans increase the risk of colon cancer through the mediation of bile acids, and that some dietary anti-oxidants may ameliorate this carcinogenicity.

Biography

Carol Bernstein, PhD, is a faculty member in the Dept. of Cell Biology and Immunology and has published more than 100 refereed papers, of which 45 have been in the areas of bile acids, DNA damage and cancer. Her other areas of interest are in the central importance of DNA damage and DNA repair both in aging and in the selective advantage of sex (why sex exists).