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Divergent Biological Activity of Conjugated Linoleic Acid (CLA) Isomers

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The growth of human cancer cells (NCI-N87, gastric; Hep3B, liver; Capan-2, pancreas; and NCI-H522, lung) were greatly inhibited by t,t conjugated linoleic acid (t,t CLA), relative to c9,t11 CLA and t10,c12 CLA. The t,t CLA inhibited the growth of all cancer cell lines; 75% NCI-N87, 44% Hep3B, 26% Capan-2, and 41% NCI-H522 at 50 μ M concentration for a 6-day incubation. The c9,t11 CLA and t10,c12 CLA inhibited the growth of NCI-N87 and Hep3B cells, but they stimulated Capan-2 cells. In the case of NCI-H522 cells, the growth was inhibited by t10,c12 CLA, but stimulated by c9,t11 CLA. Linoleic acid inhibited the growth of Hep3B and NCI-H522 cells, and stimulated Capan-2 and NCI-N87 cells. The reduction rate of 5-bromo-2'-deoxyuridine (BrdU) incorporation into NCI-N87 cells by t,t CLAs was 77.3%, relative to 89.8, 84.5, and 93.6%, respectively, by c9,t11 CLA, t10,c12 CLA and linoleic acid. In addition, t,t CLA inhibited benzo[a]pyrene (BP)-induced mouse forestomach neoplasia, relative to other CLA isomers, due to, in part, the induction of apoptosis. These results indicate that the t,t CLA consistently inhibited the growth of cancer cell lines tested, whereas c9,t11 CLA, t10,c12 CLA or linoleic acid affected positively or negatively, relevant to kinds of cancer cell lines and mouse forestomach carcinogenesis regimen.