MECHANISMS OF ACTION OF LYPOPENE

Abstract

Tomatoes are the fourth most commonly eaten fresh vegetable and the most commonly eaten canned vegetable in the United States. Dietary intakes of tomatoes and tomato products containing lycopene have shown to be associated with the decreased risks of chronic diseases including cancer and cardiovascular diseases in numerous studies. Lycopene functions as a very potent antioxidant, and this is clearly a major mechanism of lycopene action. However, evidence is accumulating for other mechanisms as well. Lycopene at physiological concentrations can inhibit human cancer cell growth by interfering with growth factor receptor signaling and cell cycle progression specifically in prostate cancer cells without evidence of toxic effects or apoptosis of cells. Studies using human and animal cells have identified a gene, connexin 43, whose expression is upregulated by lycopene and which allows direct intercellular gap junctional communication (GJC). GJC is deficient in many human tumors and its restoration or upregulation is associated with decreased proliferation. The combination of low concentrations of lycopene with 1,25-dihydroxyvitamin D3 exhibits a synergistic effect on cell proliferation and differentiation and an additive effect on cell cycle progression in the HL-60 promyelocytic leukemia cell line, suggesting some interaction at a nuclear or subcellular level.

The combination of lycopene and lutein synergistically interact as antioxidants, and this may relate to specific positioning of different carotenoids in membranes. There is a growing body of evidence that carotenoids have unexpected biological effects in experimental systems, some of which may contribute to their cancer preventive properties in models of carcinogenesis.