Cancer chemoprevention by tea polyphenols through mitotic signal blockade, a commentary.

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Abstract

Tea is a popular beverage. The consumption of green tea is associated with a lower risk of several types of cancer, including stomach, esophagus, and lung. The cancer chemopreventive effect of tea has been attributed to its major phytopolyphenols. The tea polyphenols comprise about one-third of the weight of the dried leaf, and they show profound biochemical and pharmacological activities including antioxidant activities, modulation of carcinogen metabolism, inhibition of cell proliferation, induction of cell apoptosis, and cell cycle arrest. They intervene in the biochemical and molecular processes of multistep carcinogenesis, comprising tumor initiation, promotion, and progression. Several studies demonstrate that most tea polyphenols exert their scavenging effects against reactive oxygen species (ROS); excessive production of ROS has been implicated for the development of cardiovascular diseases, neurodegenerative disorders, and cancer. Recently, we have found that the major tea polyphenol (-)-epigallocatechin-3-gallate (EGCG) suppresses extracellular signals and cell proliferation through epidermal growth factor receptor binding in human A431 epidermoid carcinoma cells; EGCG also blocks the induction of nitric oxide synthase by down-regulating lipopolysaccharide-induced activity of the transcription factor NFkB in macrophages. Furthermore, EGCG blocks the cell cycle at the G1 phase in MCF-7 cells. We have demonstrated that EGCG inhibits the activities of cyclin-dependent kinases 2 and 4; meanwhile, EGCG induces the expression of the Cdk inhibitors p21 and p27. These results suggest that tumor promotion can be enhanced by ROS and oxidative mitotic signal transduction, and this enhancement can be suppressed by EGCG or other tea polyphenols. BIOCHEM PHARMACOL 58;6:911–915, 1999.