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Curcumin and its synthetic analogue dimethoxycurcumin differentially modulates antioxidant status of normal human peripheral blood mononuclear cells

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Abstract

Curcumin is a polyphenol derived from the herb Curcuma longa, which has been extensively studied in terms of its antitumour, antioxidant, and chemopreventive activity as well as various other effects. In the present work we compared curcumin with its synthetic analogue dimethoxycurcumin (dimc) in terms of its antioxidant enzyme-modulating effects in human peripheral blood mononuclear cells (PBMC). We found that these compounds modulate antioxidant enzymes differentially. Both curcumin and dimethoxycurcumin effected a decrease in lipid peroxidation status in PBMC, however, curcumin had better activity in this regard. An increase in the activity of catalase was seen in the case of curcumin-treated PBMC, whereas dimc increased catalase activity significantly to almost twofold level. Real time-polymerase chain reaction (RT-PCR) analysis revealed significant up-regulation of catalase at mRNA level post treatment with curcumin as well as dimc, however, dimc had better activity in this regard. Glutathione reductase (GR) activity and reduced glutathione levels increased in the case of peripheral blood mononuclear cells (PBMC) treated with curcumin, however, the trend was reversed with dimethoxycurcumin where, both glutathione reductase activity and reduced glutathione levels were significantly reduced. RT-PCR analysis of glutathione reductase mRNA levels showed decrease in mRNA levels post treatment with dimethoxycurcumin (dimc) further corroborating GR enzyme assay results, however, we could not obtain significant result post curcumin treatment. NFkB reporter assay and western blot analysis of nuclear as well as cytosolic fractions of NFkB revealed that curcumin inhibits NFkB activation whereas inhibition was much less with dimc. It has been reported that curcumin and dimc exerts differential cytotoxicity in normal and tumour cells and the reason for this had been attributed to the differential uptake of these compounds by normal cells and tumour cells. Based on our results we propose that differential modulation of antioxidant enzymes via NFkB pathway could be the reason behind differential cytotoxicity of dimc as well as curcumin in normal cells and tumour cells in addition to differential uptake of these compounds as reported previously.