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engineeringRegulation of p38, ERK1/2,
and c-jun expression and apoptosis by
methylglyoxal in human colonic cells.
Methylglyoxal (MGO) induces
intracellular oxidative stress and

regulates the expression of proinflammatory and antiapoptotic genes in macrophages and monocytes. Mitogen-activated protein kinases (MAPK) and c-jun are proapoptotic genes. The purpose of this study was to determine whether MGO regulates proapoptotic genes and MAPK expression in human colonic cells. HT-29 and CCD-18Co cells were treated with 30 to 300 microM MGO for 24 hours, followed by lipopolysaccharide (LPS; 10 ng/ml) for 6 hours and analysis of apoptosis, gene expression, and protein activation. MGO induced MGO-specific glycooxidation (36.5 +/- 2.9 nmol MGO per nmol protein) and DNA damage (40.6 +/- 1.7 fmol MGO/10(5))

bases) in both cells. Furthermore, MGO induced cytochrome c release and caspase-3 activation in HT-29, but not in CCD-18Co cells. MGO decreased expression of c-jun, Bcl-2, Bax, and p-ERK1/2 in HT-29, but not in CCD-18Co cells. MGO induced cell death in HT-29 and CCD-18Co cells. MGO, 300 microM, resulted in a 10% decrease in cell viability in HT-29 and CCD-18Co cells. Furthermore, MGO, 300 microM, induced caspase-3 activation, cytochrome c release, and p-ERK1/2, but not c-jun cleavage. MGO, but not LPS, resulted in similar patterns of c-jun expression. MGO regulated Bcl-2 expression in both colonic cells. MGO

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