Effects of epidermal growth factor and its signal transduction inhibitors on apoptosis in human colorectal cancer cells

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Abstract

AIM: The study investigated if EGF signaling inhibitors, EGF antibody and tyrphostin 51 (a tyrosine kinase inhibitor), mediated the action of EGF on apoptosis and the expression of EGF receptors and p21 (a cyclin-dependent kinase inhibitor) of human colorectal cancer cells. METHODS: Human colorectal adenocarcinoma cells (SW480) were incubated with 0.6 mL/L dimethyl sulfoxide (DMSO, the control group), 225 ng/mL (37.5 nmol/L) EGF in 0.6 mL/L DMSO, 225 ng/mL EGF+2.5 microg/mL (17 nmol/L) EGF antibody in 0.6 mL/L DMSO, or 225 ng/mL EGF+215 ng/mL (0.8 micromol/L) tyrphostin 51 in 0.6 mL/L DMSO. RESULTS: After 48 h incubation, the levels of EGF in medium significantly increased ($P<0.05$) in the EGF-treated groups. The numbers of apoptotic cells were significantly fewer ($P<0.05$) in the EGF + EGF antibody and EGF + tyrphostin 51 groups than those in the control and EGF groups after 12 h treatments. The expression of phosphorylated EGF receptors in the EGF, EGF + EGF antibody, and EGF + tyrphostin 51 groups was 176.8%, 62.4%, and 138.1% of the control group, respectively. The expression of p21 protein in the EGF, EGF + EGF antibody, and EGF + tyrphostin 51 groups was 115.7%, 4.8%, and 61.5% of the control group, respectively. CONCLUSION: The data suggest that EGF antibody and tyrphostin 51 can inhibit the action of EGF on apoptosis in human colorectal cancer cells through down-regulation of EGF receptor and p21 expression.