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MECHANISM OF CAPSAICIN-INDUCED APOPTOTIC CELL DEATH IN STOMACH CANCER CELL

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Capsaicin, a major pungent ingredient in red hot pepper, has long been used in food additives and drugs. We have previously reported that capsaicin induces apoptosis in Korean stomach cancer cell line, SNU-1. In the present study, the mechanism of capsaicin-induced apoptotic cell death was investigated in SNU-1. Treatment of capsaicin to SNU-1 produced dose-dependent increase of apoptotic cell death and $[Ca^{2+}]_i$ concentrations. Because disruption of the mitochondrial transmembrane potential($\Delta\Psi m$) is a common metabolic alteration in all apoptotic processes, we evaluated the role of mitochondrial permeability transition. Using a cytofluorimetric approach, we have determined that DNA nuclear loss induced by capsaicin is proceeded by an increase of the production of reactive oxygen species(ROS) and by a subsequent $\Delta\Psi m$ dissipation in mitochondrial membrane of SNU-1. We investigated the expression of apoptosis-related proteins, p53, bcl-2 and cytochrome c. As our results, apoptosis-related proteins, p53, bcl-2 and cytochrome c expressed dose - and time- dependent manner. We also present data for the implication of a possible vanilloid receptor in capsaicin-induced apoptosis. Collectively, these results suggest that capsaicin may be involved in the mechanism of physiological protection from the genesis of stomach cancer. In addition, apoptosis induction by capsaicin may be a potential tool for the therapy-related studies of human stomach cancer.