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5-Fluorouracil (5-FU) is used as a cytostatic agent for a variety of solid tumors, including gastrointestinal cancers. However, the underlying mechanisms of 5-FU-induced apoptosis have not been fully characterized. This study was aimed at understanding the cellular and molecular events that induce apoptosis in the small intestinal mucosa in response to 5-FU. In order to assess the role of 5-FU in the induction of apoptosis in vivo, a 5-FU-induced intestinal apoptosis mouse model was established. Apoptosis in the intestine was assessed by the terminal deoxyribonucleotidyl transferase mediated dUTP nick end labelling technique.

Apoptosis was also analysed at the molecular level by the measurement of caspase-3/7 activity and by the analysis of caspase-3 activation through proteolytic cleavage. The effect of 5-FU on the expression of apoptosis-related proteins was also studied by western blotting. Our data revealed that 5-FU induced apoptosis in the small intestine of mice. The amounts of cleaved PARP and caspase-3 were significantly increased in a time-dependent manner. In addition, 5-FU significantly increased the levels of cleaved caspase-3 and decreased the levels of p53, Bcl-2 and Rb. These findings suggest that the decrease in Bcl-2 and increase in p53 may facilitate apoptosis in the small intestinal mucosa of 3e33713323

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