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Enhanced anti-tumor effects of Fluorouracil against gastric cancer by 5-aza-2'-deoxycytidine and Trichostatin A

Geng Bian, Guodong Shen and Shilian Hu
Anhui South Medical University, Anhui Provincial Hospital, China

Gastric cancer is one of the most common malignant tumors in the world, and only China contributes to almost half of all global new cases each year. Our previous studies have shown that the promoter hypermethylation of RUNX3 and E-cadherin plays an important role in the occurrence and development of gastric cancer. In this study, we analyzed the gene expression and inhibitive effects of gastric cancer cell lines MKN-45 and SGC-7901 treated with Fluorouracil alone and in combination with 5-aza-2'-deoxycytidine (5-Aza-dC) or/and Trichostatin A (TSA). Gastric cancer cell lines were treated with Fluorouracil, with or without 5-aza-dC or/and TSA for different time and doses, and the inhibitive effects were measured by MTT kit to determine cell proliferation. The promoter methylation status and protein expression of RUNX3 and E-cadherin genes were analyzed by the methods of real time quantified methylation-specific polymerase chain reaction (RTQ-MSP) and western blot, respectively. Treatment with 5-aza-dC or TSA alone was found not to inhibit the proliferation of gastric cancer cells as much as Fluorouracil did, but the effect had a remarkable enhancement by Fluorouracil combined with 5-aza-dC and TSA. The results showed that 5-aza-dC or TSA could reduce methylation and increase expression of RUNX3 and E-cadherin genes, and the combination of 5-aza-dC and TSA increased more, but Fluorouracil did not affect their protein expression. Our findings imply that 5-aza-dC and TSA induce strong reactivation of RUNX3 and E-cadherin genes, and have potentials as therapeutic candidates alone or combination with other tumor-targeted drugs in gastric cancer therapy.