عنوان مقاله:

The Small Molecule Enoxacin Suppresses The Growth and Invasiveness of Esophageal Cancer Cells

محل انتشار:

بیست و یکمین کنگره پزشکی تولید مثل و شانزدهمین کنگره زیست شناسی و فناوری سلول های بنیادی (سال: 1399)

تعداد صفحات اصل مقاله: 1

نویسندگان:

P Torabi - *Department of Stem Cells and Developmental Biology, Cell Science Research Center, , Royan Institute for Stem Cell Biology and Technology, ACECR, Tehran, Iran,Department of Developmental Biology, Faculty of Basic Sciences and Advanced Technologies in Biolo*

R Miri - *Cancer Research Center, Cancer Institute of Iran, Tehran University of Medical Sciences, Tehran, Iran*

SH Moradi - *Department of Stem Cells and Developmental Biology, Cell Science Research Center, , Royan Institute for Stem Cell Biology and Technology, ACECR, Tehran, Iran*

خلاصه مقاله:

Objective: Esophageal cancer (EC) is one of the deadliest can-cers worldwide. The global down-regulation of microRNAs, i.e. non-coding RNAs involved in post-transcriptional gene regulation, is observed in many cancer types including EC. We therefore hypothesized that the microRNA-enhancing com-pound enoxacin might impair the growth of EC cells.Materials and Methods: EC cells were cultured in RPMI medium containing ۱۰% FBS. The cells were analyzed three days after treatment with enoxacin (۱۲۴ μM). To minimize the unwanted increase in pro-tumor microRNAs by enoxacin, one major pro-EC miRNA, i.e. miR-۱۰۶a, was inhibited using antagomiRs. Viability assays were performed using MTS and Live/Dead staining kits. Cell cycle analysis was done using flow cytometry. The anti-miR-۱۰۶a was delivered using Dhar-maFECT۱. Human EC tumor tissues were obtained from Imam Khomeini Hospital and exposed to enoxacin for ۱۰ days.Results: We found that enoxacin inhibited the viability and cell cycling of EC cells (KYSE-۳۰ and YM-۱ cell lines). It also sup-pressed the migration and colony-formation ability of EC cells. Notably, blocking miR-۱۰۶a activity significantly reduced the viability, cell cycling, migration and colony formation of EC cells. Moreover, the combination of enoxacin and anti-miR-۱۰۶a induced a decrease in the growth of EC cells that was much more prominent than either of them alone. Finally, we treated tumor samples from EC patients with enoxacin ex vivo and found that the tumor tissues were negatively affected by exposure to enoxacin (with or without anti-miR-۱۰۶a).Conclusion: Enoxacin, alone or in combination with miR-۱۰۶a inhibitor, inhibits the growth and migration of EC cells both in vitro and ex vivo.

کلمات کلیدی:

microRNA, Cancer Cells, Enoxacin, Tumorigenesis, miR-۱۰۶a

لینک ثابت مقاله در پایگاه سیویلیکا: