

عنوان مقاله:

Improvement of 5-fluorouracil chemosensitivity in colorectal cancer cells by siRNA-mediated silencing of STAT6 oncogene

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نویسندگان:

Omid Rahbar Farzam - *Department of Medical Biotechnology, School of Medicine, Kermanshah University of Medical Sciences, Kermanshah, Iran*

Behzad Baradaran - *Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran*

Bahman Akbari - *Department of Medical Biotechnology, School of Medicine, Kermanshah University of Medical Sciences, Kermanshah, Iran*

Soozan Najafi - *Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran*

Mohammad Amini - *Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran*

AmirHossein Yari - *Department of Biology, Tabriz Branch, Islamic Azad University, Tabriz, Iran*

Reza Dabbaghipour - *Medical School, Shiraz University of Medical Sciences, Shiraz, Iran*

Vahid Pourabdollah Kaleybar Pourabdollah Kaleybar - *Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran*

Shiva Ahdi Khosroshahi - *Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran*

خلاصه مقاله:

Objective(s): Colorectal cancer (CRC) remains a major health concern worldwide due to its high incidence, mortality rate, and resistance to conventional treatments. The discovery of new targets for cancer therapy is essential to improve the survival of CRC patients. Here, this study aims to present a finding that identifies the STAT6 oncogene as a potent therapeutic target for CRC. **Materials and Methods:** HT-29 CRC cells were transfected with STAT6 siRNA and treated with 5-fluorouracil (5-FU) alone and combined. Then, to evaluate cellular proliferation and apoptosis percentage, MTT assay and annexin V/PI staining were carried out, respectively. Moreover, the migration ability of HT-29 cells was followed using a wound-healing assay, and a colony formation assay was performed to explore cell stemness features. Gene expression was quantified via qRT-PCR. Afterward, functional enrichment analysis was used to learn in-depth about the STAT6 co-expressed genes and the pathways to which they belong. **Results:** Our study shows that silencing STAT6 with small interfering RNA (siRNA) enhances the chemosensitivity of CRC cells to 5-FU, a commonly used chemotherapy drug, by inducing apoptosis, reducing proliferation, and inhibiting metastasis. These results suggest that combining 5-FU with STAT6-siRNA could provide a promising strategy for CRC

treatment. Conclusion: Our study sheds light on the potential of STAT δ as a druggable target for CRC cancers, the findings offer hope for more effective treatments for CRC patients, especially those with advanced stages that are resistant to conventional therapies.

کلمات کلیدی:

fluorouracil, Chemosensitivity, Colorectal cancer, siRNA, STAT δ -5

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