



International Cancer Study & Therapy Conference

April 4-6, 2016 Baltimore, USA

N-terminal domain of FHIT can arrest HT1080 cells in G2 phase of cell cycle

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FHIT as a tumor suppressor executes a critical function in inhibiting p53 degradation by MDM2. Previous studies have revealed the interaction of FHIT with MDM2 or p53; however, there is no detailed study for interpreting the functional domains of FHIT involved in the tumor inhibition. In this study, we evaluated the various domains of FHIT owning the cytotoxic properties of the full length protein. Based on our previous *in silico* screening and MTT results, truncated forms of FHIT were selected and their cell cycle inhibiting properties were assessed in HT1080 human fibrosarcoma cell line. The functional analysis showed that these fragments can arrest cells in G2 phase of the cell cycle as indicated by flowcytometry.

These findings suggest FHIT functional domains as lead compounds for the discovery of future drug design and/or gene transfer for cancer therapy.

Keywords: Fragile histidine triad, HT1080, flowcytometry

Biography:

Dr. Ameneh Eslamparast is an assistant professor of pharmaceutical biotechnology at Ardabil University of Medical Sciences. She obtained her Pharm. D. from the Shahid Beheshti University of Medical Sciences in 2005. She obtained her PhD in Pharmaceutical Biotechnology from the Pasteur Institute of Iran in 2014, where she carried out studies about FHIT anticanceric truncates. She joined Ardabil University of Medical Sciences in 2014 as an Assistant Professor and is continuing her research studies about anti-cancers with emphasis on peptides and plant extracts. Dr. Ameneh Eslamparast is a scientific writer with research publications in peer-reviewed scientific journals. Dr. Ameneh Eslamparast has received awards including the Ministry of health of Iran scholarship, for PhD course and Pasteur Institute of Iran award for cancer research.