

Evaluation of Caspase 9 Activity Level in Hela Cell Line Exposed to Cytotoxic Dose of Aspirin

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Abstract: *The research have shown that aspirin may have anticancer effects on several types of cancer cells. The aim of this study was to determine the effects of aspirin on apoptosis in cervical cancer (Hela) cells exposed to aspirin through evaluation of caspase-9 activity. In this experimental laboratory study, cervical cancer cells were purchased from Iran Cell Bank (Pasteur Institute, Tehran, IRAN). Cell lines were cultured in DMEM culture media. The cells were transferred to 6 well plate and incubated for 24 hours. After incubation, the cells were exposed to 10, 1, 0.1, 0.01, 0.001 and 0.0001 mg/ml of aspirin and cell viability was measured using MTT assay method. Caspase-9 activity in response to IC-50 dose of aspirin was evaluated using ELISA reader. The results indicated that the caspase-9 activity was significantly higher in cervical cancer cells exposed to IC50 dose of aspirin than control group ($P < 0.05$). Our findings demonstrated that IC50 dose of aspirin induces intrinsic apoptotic pathway in cervical cancer cells.*

Keywords: Aspirin, HELA cells, Caspase-9.

1. Introduction

Caspase-9 is a key player in the intrinsic or mitochondrial pathway which is involved in various stimuli including chemotherapies, stress agents and radiation. Failing to activate caspase-9 has profound physiological and pathophysiological outcomes, leading to degenerative and developmental disorders even cancer[1].

HeLa cells were named for Henrietta Lacks, who died in 1952 from an infection of a special type of cancer[2]. As a nearly ubiquitous inhabitant of laboratories using tissue culture techniques, its aggressive growth characteristics make it a problematic contaminant that can overgrow less robust cell lines [3].

Aspirin is integral to the secondary prevention of cardiovascular disease and acts to impair the development of platelet-mediated atherothrombotic events by irreversible inhibition of platelet cyclooxygenase-1 (COX-1) [4].

Previous studies have shown that NSAIDs play significant role in apoptosis and caspase enzymes activation in cancer cells including cervical cancer cells [5], [6]. The aim of this study was to evaluate the effects of IC50 dose of aspirin on caspase 9 activity level in cervical cancer cells.

2. Material and methods

In this experimental laboratory study, cervical cancer cells were purchased from Iran Cell Bank (Pasteur Institute, Tehran, IRAN). Cell lines were cultured in DMEM culture media. The cells were transferred to 6 well plate and incubated for 24 hours. After incubation, the cells were exposed to 10, 1, 0.1, 0.01, 0.001 and 0.0001

mg/ml of aspirin and cell viability was measured using MTT assay method. Caspase-9 activity in response to IC-50 dose of aspirin was evaluated using ELISA reader.

3. Results

The results indicated that the caspase-9 activity was significantly higher in cervical cancer cells exposed to IC50 dose of aspirin than control group ($P < 0.05$) (Figure I).

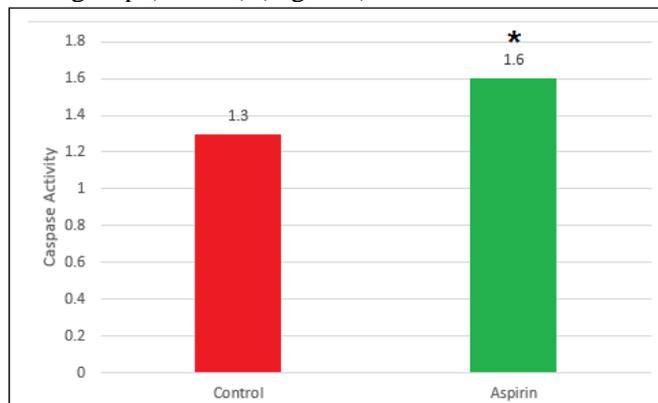


Fig. 1. Caspase 9 activity level in cervical cancer cells exposed to IC50 dose of aspirin compared with control group. * indicates significant difference at $p < 0.05$.

4. Discussion

Our findings indicated that aspirin induces apoptosis in cervical cancer cells through enhancing of caspase 9 activity level which is involved in intrinsic apoptosis pathway. In line with our finding recent data have suggested that regular aspirin use improves overall and cancer-specific survival in the subset of colorectal cancer (CRC) patients [7]. Aspirin is also a promising chemopreventive agent and exerts significant therapeutic potentials in pancreatic cancer [8]. NSAIDs and aspirin after but not before diagnosis were associated with improved breast cancer survival, including breast-cancer-specific mortality [9]. The research also have shown that aspirin may reduce the risk of endometrial cancer [10]. The change of caspase-9 expression in some cancer cells suggests that it may be involved in the carcinogenesis cancer cells. The overexpression of caspase-9 exhibits an inhibitory role in cancer growth and proliferation while promoting apoptosis [11]. It has been shown that aspirin can induce the antitumor effect mediated by caspase pathway in cervical cancer cells [12]. In a study it has been demonstrated that aspirin treatment caused changes in the mitochondrial membrane potential, release of cytochrome c from mitochondria, and activation of caspase-9 and -3 [13]. Caspase enzymes are also involved in aspirin-induced apoptosis in gastric cancer cells [14].

5. Conclusion

Our findings demonstrated that IC50 dose of aspirin induces intrinsic apoptotic pathway in cervical cancer cells.

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7. References

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