The Effects of Cortisol on Colon Cancer (HCT) Cells Viability in Cell Culture

Amin Sarkhoh
Department of Biology, Faculty of Basic Sciences, Hamedan Branch, Islamic Azad University, Hamedan, Iran
(Email: aminsarkhosh21573@gmail.com).

Abstract

Background and Aim: Several studies have reported anticancer effects of corticosteroids on various cancers including cancers of digestive system. We exerted the present study to determine the cytotoxic effects of cortisol on colorectal cancer (HT29) cells in vitro.

Methods: In this laboratory – experimental study colon cells were divided to control (untreated) group and group exposed to different concentration of cortisol. Viability of cancer cells were measured using MTT assay method. The data were analyzed using one-way analysis of variance (ANOVA) method.

Results: The results of this study indicated that treatment of HT29 cells with 0.0001, 0.001, 0.01, 0.1, 1 and 10 mg/ml of cortisol did not significantly change the cell viability in HT29 cells compared to control group.

Conclusion: Despite reports indicating that corticosteroids have anticancer effects on colon cancer cells, we have shown that cortisol has not cytotoxic effects on colorectal cancer (Ht29) cells in vitro.

Keywords: Cortisol, HT29 cell line, Viability.

Introduction

Cortisol is a steroid hormone located in the glucocorticoid hormones category. When used as a medicine, it is called hydrocortisone. This hormone is produced from the adrenal cortex or within the adrenal gland. It responds to stress and low levels of blood glucose, which acts to increase blood glucose through gluconeogenesis to suppress the immune system and contribute to the metabolism of fat, protein, and carbohydrates, as well as to reduce bone formation [1]. Colorectal cancer, also known as colon cancer and colon cancer, is the development of cancer from the large intestine or rectum (the large intestine). Signs and symptoms include the appearance of blood in the stool, changes in the gut motion, weight loss and persistent fatigue. Most colorectal cancers are due to peripheral and lifestyle factors, and in some cases due to genetic disorders. Some peripheral factors, including poor diet, obesity, smoking and physical inactivity, as well as nutritional factors that increase the risk, include red and processed meat (sausage, salami) and alcohol consumption [2]. More colon cancer patients are expected to fully recover as a result of improvement of general healthcare, optimization of cancer treatment and increased earlier detection of colorectal cancer through screening programs [3]. New treatments for colorectal cancer have emerged; However, these new treatment options have had limited impact on cure rates and long-term survival [4].

Studies show that there is a relationship between steroid hormones and cancers [5]. Research has shown that steroid hormones and gastrointestinal cancer are interrelated [6]. The results also show...
that steroid hormones affect on colon cancer cells [7]. Association of cortisol has been found with cancers [8], including gastrointestinal cancers [9]. According to studies, there is a significant correlation between cortisol and colon cancer [10]. The aim of this study was to investigate the cytotoxic effects of cortisol on colon cancer in vitro.

Materials and Methods
Colon Cancr (HT29) cell line was obtained from National Cell bank of Iran (NCBI) (Pasteur Institute of Iran (Pasteur Institute, Tehran, Iran). Cells were maintained in a humidified water-jacked incubator with 5% CO2 at 37°C in RPMI-1640 supplemented with 10% fetal bovine serum (FBS), penicillin (100 U/mL), and streptomycin (100 μg/mL) (all purchased from Sigma, Germany). Cytotoxicity of the cortisol was evaluated using the MTT assay, which is based on the ability of viable cells to metabolize yellow tetrazolium salt MTT to purple formazan crystals by mitochondrial dehydrogenases. Briefly, cells were seeded at a density of 15000 per well in 96-well plates; subsequently, after 24 h incubation, they were treated with various concentrations of cortisol for 24 h. The untreated well was considered as a negative control. Afterward, the suspended medium was thrown away and 20 μL of 5 mg/mL MTT solution was added to each well and further incubated for 4 h at 37°C. Subsequently, the whole suspended medium was discarded from each well before adding 200 μL DMSO and 50 μL Sorenson buffer. In order to complete dissolution, the plate was incubated for 30 min with gentle shaking for 5 min. The cytotoxic effects of cortisol were monitored by measuring the absorbance of each well at 570 nm.

Results
The results of this study indicated that treatment of HT29 cells with 0.0001, 0.001, 0.01, 0.1, 1 and 10 mg/ml of cortisol did not significantly change the cell viability in HT29 cells compared to control group, hence, did not have significant cytotoxic effects (Figure 1).

![Fig 1. Viability of HT29 cells treated with different concentration of cortisol.](image-url)
Discussion
Colorectal cancer is the leading cause of death from cancer in the Western world. The third most common cancer in the United States and in men and women is estimated to be between 103 and 170 in 2012, with a recent estimate of new cases of colorectal cancer. Despite the fact that several options for treating this cancer are available through surgery, chemotherapy, radiotherapy, immunotherapy and dietary supplements, but the success rate is not very encouraging [12]. Epigenetic changes in colon cancer have been more than genetic changes (mutations) [13]. The previous studies have shown that steroid hormones may affect on colon cancer cells proliferation [7]. Studies also have found an association of cortisol with a variety of cancers [8, 9]. Antitumor effects of corticosteroids have been reported in in vivo and in vitro studies, as well [14]. However, we have shown that cortisol has not significant cytotoxic effects on colorectal cancer (HT29) cells in vitro.

Conclusion
Despite reports indicating that corticosteroids have anticancer effects on colon cancer cells, we have shown that cortisol has not cytotoxic effects on colorectal cancer (Ht29) cells in vitro.

Acknowledgements
We appreciate Javid Biotechnology Lab staff for their help.

References


