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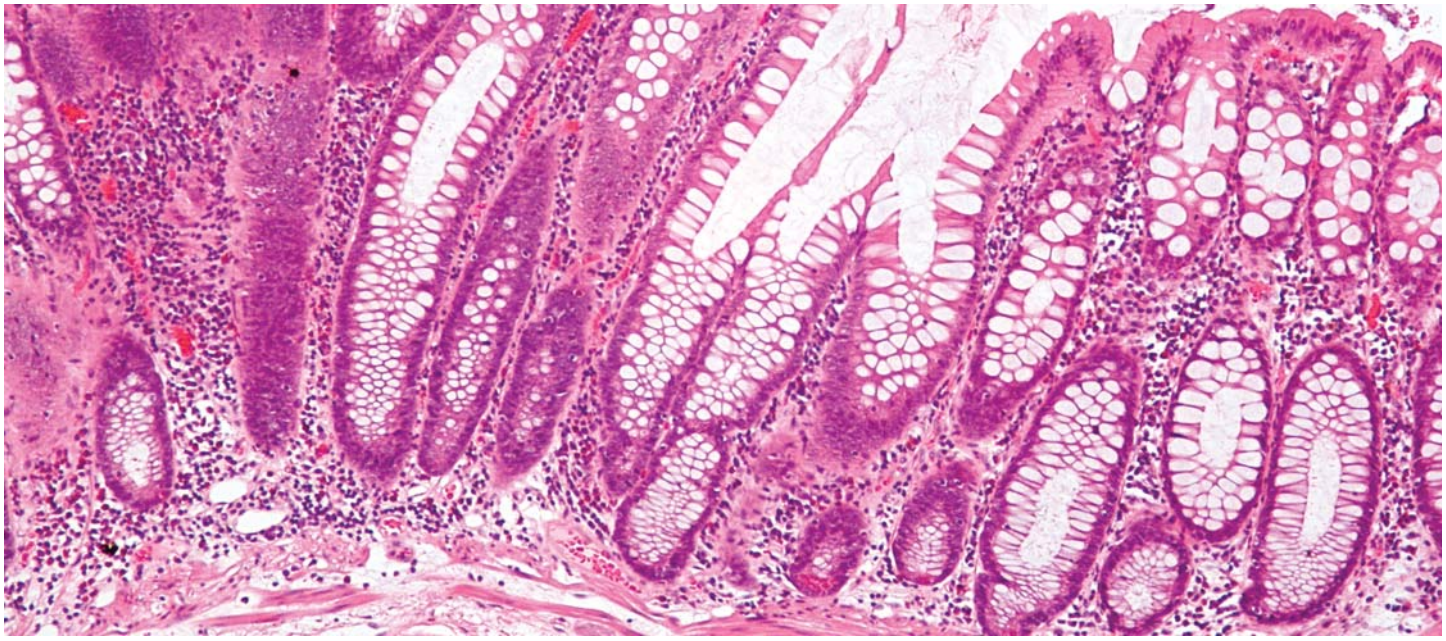

Research Highlight

BENEFICIAL EFFECTS OF BUTYRATE IN THE MAINTENANCE OF COLONIC EPITHELIUM

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Key words:
Butyrate | bacterial fermentation | colonic mucosal growth | ulcerative colitis | heat shock proteins | immune activation | colonic epithelial cells | heat stress

Butyrate is a four-carbon (4C) short-chain fatty acid which is normally present in the colon. Butyrate is produced by the bacterial fermentation of carbohydrates and it plays a significant role in the maintenance of colonic epithelium.

Moreover, it also regulates colonic mucosal growth, barrier function as well as epithelial proliferation[1].

Butyrate also exhibits anti-inflammatory properties and act as modulators of chemotaxis and adhesion of immune cells[2]. Butyrate affects immune cell migration, adhesion, and cellular functions such as proliferation and apoptosis. It is reported that a low concentration of butyrate increases cell proliferation and a high concentration of butyrate plays a key role in the induction of apoptosis[3].

In addition, it is reported that butyrate enemas have been employed to lessen the colonic mucosal inflammation in inflammatory conditions including Ulcerative Colitis (UC) as well as radiation colitis[4].

On the other hand, the heat shock proteins (Hsp) that are also known as stress proteins are imperative regarding cellular response in case of stress as well as in cellular homeostatic functions like manufacturing of protein and its transportation across membranes.

However, besides these fundamental functions, these stress proteins play an important role in the activation of the immune system as well as its recognition. Therefore, they can be cytotoxic[5]. Several studies also support a role of Hsp in the inflammatory response which demonstrates that Hsp participate in cytokine signal transduction as well as in the control of cytokine gene expression[6].

Accordingly, scientists planned to carry out a new study in order to investigate the hypothesis that cytoprotection of colonic epithelial cells by butyrate can be arbitrated by means of modulation of heat shock protein expression. For this purpose, HT29 intestinal epithelial cell monolayers were exposed to two forms of non-lethal stress; heat or chemical with or without butyrate.

Afterward, cells were radiolabeled with ^{35}S-methionine and incubated at 37°C for a duration of two hours. Cells were then lysed, subjected to 1D SDS-PAGE, followed by autoradiography. Moreover, the impact of initial exposure to sub-lethal heat on consequent exposure to lethal heat was also analyzed[7].

During this study, non-lethal stress-induced Hsp70 expression in HT29 cells was restrained by butyrate. However, this activity of butyrate

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was observed only at a lower amount. Inhibition of Hsp70 synthesis was also noticed by the research team because of the capability of the cells to bear a second lethal heat stress.

Butyrate was found to amplify colonic epithelial cell survival by considerably decreasing Hsp70 expression. Conclusively, this research points out the useful effect of butyrate on the survival of colonic epithelial cells. This beneficial aspect can occupy clinical significance in various conditions involving the colonic mucosa.

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