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Butyrate and the colonocyte. Production, absorption, metabolism, and therapeutic implications

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Abstract

Butyrate, a SCFA generated by microbial fermentation of dietary substrates, is produced in the colon of humans and may influence colonic disease. It is possible to manipulate the diet in order to enhance levels of butyrate in various regions of the large intestine. Butyrate is absorbed by colonocytes in the proximal colon via passive diffusion and by active transport mechanisms which are linked to various ion exchange transporters. In the distal colon, the main mechanism of absorption is passive diffusion of the lipid-soluble form. Butyrate and other SCFA are important for the absorption of electrolytes by the large intestine and may play a role in preventing certain types of diarrhea. The mechanism by which butyrate and other SCFA exerts control over fluid and electrolyte fluxes in the colon is not well delineated though it may occur through an energy generated fuel effect, the up-regulation of various electrolyte transport systems, as well as possible effects on neuroendocrine factors. Butyrate has been shown to have beneficial effects on some colonic pathologies. This SCFA may be protective against colorectal neoplasia. Butyrate regulates colonic motility, increases colonic blood flow and may enhance colonic anastomosis healing. Butyrate may reduce the symptoms from ulcerative colitis and diversion colitis and it may prevent the progression of colitis in general. Further investigations are needed to confirm these findings in controlled, randomized, double blinded clinical studies.

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