THE ROLE OF SHORT CHAIN FATTY ACIDS IN THE PATHOGENESIS OF COPD

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Introduction: Significant amounts of SCFA are synthesized in the colon by microbacterial flora from non-absorbed carbohydrates and proteins. Due to colon absorption, the end products of bacterial metabolism are diffused into general circulation and get rid off with by expiration. Quantitative and qualitative changes of SCFA can impact the pathogenic mechanisms of COPD. Results: Total levels of SCFA in patients with COPD were decreased 1.6-fold compared with those in the normal in sputum $(0.15\pm0.01 \text{ mg/g})$, 4.3-fold compared with those in the normal serum $(0.05\pm0.01 \text{ mg/g})$, and 4.5-fold compared with those in the normal stool (2.34±1.12 mg/g). Conclusions: A decreasing of SCFA could be linked with the quantitative and qualitative changes in the colon microbacterial flora in COPD patients due to hypoxia in the colon mucosa, a change of pH and gas metabolism. Besides, an increase in respiratory pathogens due to COPD exacerbation can additionally change a local SCFA production in the lungs, which was confirmed by changing profiles of SCFA. An increased level of C2 could be linked with the activation of aerobic microflora. Increasing butyrate concentration can stimulate differentiation of airway epithelium, hyperplasia, and hypersecretion of goblet cells and mucus production. Absorbed C3 can directly activate bronchial smooth muscle contractile activity that aggravates bronchial obstruction and induces exacerbation of COPD.