Format: Abstract

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Taurochenodeoxycholic acid ameliorates and ursodeoxycholic acid exacerbates small intestinal inflammation.

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Abstract

Intraluminal bacteria, food intake, and bile play important roles in indomethacin-induced small intestinal inflammation in rats. Tauroursodeoxycholic acid (TUDCA) and ursodeoxycholic acid (UDCA) inhibit hydrophobic bile acid-induced damage in various types of cells. We investigated the effects of these bile acids along with the possible influence of other bile acids on this model of inflammation. Clinical and intestinal inflammatory parameters and bile secretion were assessed after 7-day dietary bile acid pretreatments and subsequent indomethacin injections. UDCA significantly enhanced indomethacin-associated reductions in food intake and body weight, increases in gross inflammatory scores and myeloperoxidase activity, and the shortening of small intestinal length. Taurochenodeoxycholic acid (TCDCA) significantly normalized the clinical inflammatory parameters, prevented indomethacin-induced increases in the biliary contents of secondary bile acids and hydrophobicity index, and tended to attenuate the intestinal inflammation. Although elevated biliary levels of muricholic acids and a decreased hydrophobicity index were evident before indomethacin injection in the TCDCA case, these alterations could not explain the TCDCA-mediated protection. Dietary TCDCA attenuates whereas UDCA exacerbates intestinal inflammation in this model. Alterations in the bile composition (increases in UDCA and chenodeoxycholic acid) may explain the observed modification effects.

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