Aliment Pharmacol Ther. 2016 Jan;43 Suppl 1:27-36. doi: 10.1111/apt.13436.

Review article: the potential mechanisms of action of rifaximin in the management of inflammatory bowel diseases.

Sartor RB[1].

Author information

1 Center for Gastrointestinal Biology and Disease, Department of Medicine, Microbiology and Immunology, University of North Carolina, Chapel Hill, NC, USA.

Abstract

BACKGROUND: Gut microbiota dysbiosis contributes to the pathogenesis of inflammatory bowel diseases (IBD). Although the microbiota's role in IBD pathogenesis, specifically Crohn's disease (CD), provides a rationale for antibiotic treatment, antibiotic use in CD remains controversial. Rifaximin, traditionally identified as a nonsystemic bactericidal antibiotic, may be therapeutically beneficial for inducing CD remission.

AIM: To examine the role of rifaximin in the management of IBD and its potential mechanisms of action.

METHODS: A literature search using the following strategy: ('inflammatory bowel disease' OR 'Crohn's' OR 'ulcerative'), 'rifaximin' AND ('barrier' OR 'translocation' OR 'adhesion' OR 'internalization' OR 'pregnane X'), AND 'pregnane X' AND ('Crohn's' OR 'ulcerative colitis' OR 'inflammatory bowel disease').

RESULTS: In vitro data suggest rifaximin mediates changes in epithelial cell physiology and reduces bacterial attachment and internalisation. In experimental colitis models, rifaximin antagonised the effects of tumour necrosis factor-α on intestinal epithelial cells by activating pregnane X receptor, which inhibits nuclear factor-κB-mediated proinflammatory mediators and induces detoxification genes (e.g. multidrug resistance 1 and cytochrome P450 3A4). Rifaximin also inhibits bacterial translocation into the mesenteric lymph nodes.

CONCLUSION: Accumulating evidence suggests that mechanisms of action of rifaximin in IBD may not be limited to direct bactericidal activity; therefore, rifaximin could potentially be redefined as a gut environment modulator.

PMID: 26618923 DOI: 10.1111/apt.13436