arthritis complicating CD. Most likely, CD patients with severe and recurrent axial arthritis should be treated with infliximab without a therapeutical attempt with azathioprine or methotrexate alone. Prospective studies are necessary to confirm this result.

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EFFICACY OF RECTAL BECLOMETHASONE DIPROPIONATE VS. 5-AMINOSALICYLIC ACID IN MILD TO MODERATE DISTAL ULCERATIVE COLITIS: A META-ANALYTICAL APPROACH

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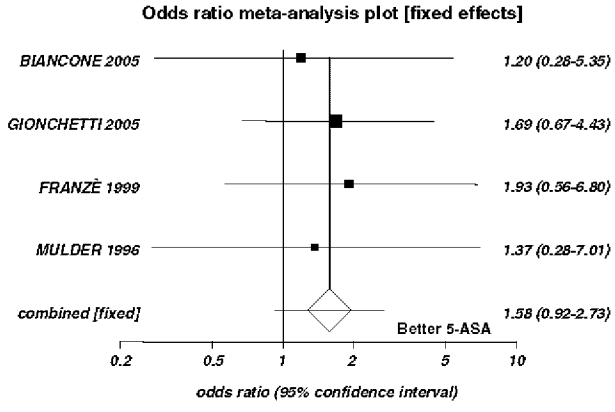
Background and aim: A meta-analysis of seven trials examined critically the role of conventional rectal steroids or 5-ASA in the management of active distal ulcerative colitis (UC): 5-ASA was significantly better for inducing remission of symptoms, endoscopy, and histology [1]. Beclomethasone dipropionate (BDP) is a new steroid with topical effects and a minimal systemic activity. Major criticisms about BDP in UC treatment have been raised for the lack of data regarding tapering, long-term effect of treatment, and the advantage against higher doses of 5-ASA [2]. Our purpose was to define with a meta-analytical approach the clinical efficacy in left-sided mild to moderate UC after treatment with enema/foam of BDP or 5-ASA.
Material and methods: We used appropriate key words to perform a literature search in MEDLINE or in databases of ISI Web of Knowledge TM, as well as manual searches to identify abstracts from relevant international meetings with data not included in extenso publications. Odds ratio meta-analysis was done with the Mantel-Haenszel model that provides a pooled odds ratio across the strata of fourfold tables.
Results: The efficacy of rectal 5-ASA vs. BDP has been addressed in 4 clinical trials [3–6] in which a total of 185 UC patients were treated with 5-ASA (1-4 g o.d.) and 189 with BDP (3 mg o.d.). The treatment time ranged from 4 to 8 weeks. 5-ASA induced improvement or remission of UC in 157 (84.9%) patients while BDP in 148 (78.3%). Mantel-Haenszel pooled estimate of odds ratio was 1.58 (95% Confidence Interval (CI) = 0.92-2.73) (p = 0.13).



Conclusions: Rectal 5-ASA showed a tendency of superior efficacy vs. BDP in inducing clinical improvement or remission of mild to moderate left-sided UC, even though the CI may hypothesize equal effects. Despite the lack of convincing evidence to date in UC for the rectal BDP treatment, further controlled clinical trials will be necessary to delineate the exact role and to explore the potentiality of this steroid.

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INFLUENCE OF CARNITINE ON BUTYRATE METABOLISM OF COLONOCYTES IN A RAT MODEL OF EXPERIMENTAL COLITIS

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Background and aim: It has been postulated that colitis may be the result of inability to oxidize butyrate, the main source of energy for colonocytes. Since carnitine plays a pivotal role in fatty acid metabolism, we evaluated carnitine transporter expression and carnitine uptake as well as butyrate metabolism in colonocytes isolated from TNBS-treated and control rats. Butyrate metabolism was analysed in cells before and after supplementation of liposome-encapsuled carnitine.
Material and methods: Total, free, and acyl-carnitine fractions were determined by HPLC and tandem-mass-spectrometry. Butyrate metabolism was evaluated in isolated colonocytes incubated by 1-14C-labelled butyrate. Expression of carnitine transporters was analyzed by RT-PCR, real time RT-PCR and in-situ hybridization. Functional properties of carnitine transporters were investigated on the basis of substrate specificity. The inhibitory effect of several compaunds on L-carnitine transport was also determined.
Results: The functioning of high-affinity (Octn2) and low-affinity carnitine transporters (Atb0+) was demonstrated in rat colonocytes. Expression of Octn2 and Atb0 was also confirmed by real-time RT-PCR and in-situ hybridization. Experimental colitis induced a decrease in colonocyte carnitine uptake and a significant reduction of Octn2 and Atb0+ transcripts. The concomitant decrease in carnitine cell content paralleled with a substantial decrease in butyrate utilization by colonocytes. Carnitine supplementation in colonocytes by liposomes proved capable of restoring butyrate metabolism, while secondary carnitine deficiency in vivo increased colitis severity.
Conclusions: In this study, we present evidence for the complex interactions between carnitine, carnitine transporters and butyrate metabolism. The altered colonocyte carnitine uptake in colitis leads to impaired butyrate oxidation and this, in turn, results in energy failure. Thus, it is not a decreased butyrate uptake but rather an altered utilization of this SCFA that produce toxic effects on the target cell. This may explain the contradictory therapeutic efficacy of butyrate in inflammatory bowel diseases, as proved by the last decades' clinical trials. Our results suggest that colitis is characterised by an energy deficiency, in which a lack of carnitine leads to cell death and chronic inflammation by perturbing butyrate metabolism.

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EFFICACY OF INFLIXIMAB IN ULCERATIVE COLITIS IN CLINICAL PRACTICE

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Background and aim: Efficacy of infliximab in severe steroid-