Abstract


Vitamin A exerts its antiinflammatory activities in colitis through preservation of mitochondrial activity.

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OBJECTIVES: The aim of this study was to assess the protective effects of vitamin A in a rat model of colitis to elucidate a possible mechanism of action.

METHODS: Male rats were fed for 21 d with either a normal diet or high vitamin A diet (5000 IU/d). On day 22, colitis was induced with 2,4,6-trinitrobenzenesulfonic acid (TNBS). Rats were sacrificed after 24 h and colonic tissue was removed for evaluation.

RESULTS: Morphologically, in the supplemented group preservation of tissue architecture, no vasculitis or necroses were detected. Biochemically, decreased myeloperoxidase activity and protection of the mitochondria as evaluated by preserving tissue oxygen consumption, mitochondrial DNA, and expression of cytochrome c, was observed. Vitamin A supplementation also increased the levels of nuclear respiratory factor (NFR)-1 and mitochondrial transcription factor-A (TFAM) in normal colon tissue and in colon tissue under inflammatory conditions.

CONCLUSION: The results indicate that tissue damage in colitis is accompanied by the arrest of mitochondrial respiration, loss of mitochondrial DNA, and the expression of mitochondrial proteins. Vitamin A effectively protects colon mitochondria by upregulation of mitochondrial transcription factors, NFR-1 and TFAM, and prevents inflammatory and necrotic changes in colitis. Vitamin A is therefore a potential therapeutic agent in inflammatory bowel disease.

PMID: 26429662