Therapeutic effect of worm-derived proteins on experimental colitis

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Worms are important source of immunomodulatory proteins that could be used in the development of new drugs for the treatment of immune-mediated diseases such as inflammatory bowel disease (IBD). A research group in Belgium investigated the therapeutic effect of worm-derived proteins on experimental colitis in mice. Treatment with worm proteins ameliorated motility disturbances during murine experimental colitis. This suggests that worm proteins have great potential to be used as therapeutic agents in IBD.

Patients with inflammatory bowel disease (IBD) suffer from chronic inflammation of the gut leading to gastrointestinal motility alterations with symptoms such as abdominal pain, cramps and diarrhea that profoundly affect their quality of life. The lack of exposure to worm infections, as a result of improved living standards and medical conditions, might have contributed to the increased incidence of IBD in the Western world. Epidemiological, experimental and clinical data support the idea that worm infection provides protection against IBD. However, treatment of patients with living worms may have serious drawbacks such as infection and/or invasion of the parasite to other tissues. Therefore, therapy with worm-derived proteins might provide a more acceptable form of treatment.

A research article published on February 14, 2010 in the World Journal of Gastroenterology addresses this question. The research team led by Professor Pelckmans from the University of Antwerp used a mouse model of experimental colitis to study the beneficial therapeutic effect

of worm-derived proteins on inflammation and gastrointestinal motility disturbances. This paper further enlightens the therapeutic effect of worm proteins on colitis by investigating the effect on the inflammatory process and on the motility disturbances. The results agree with previous studies showing a beneficial effect of worm infection on intestinal inflammation and of worm proteins in experimental animal models of asthma and type 1 diabetes.

The induction of colitis in mice causes severe gastrointestinal motility alterations such as increased intestinal transit time and abrogation of colonic peristaltic activity, as seen in IBD patients. This paper describes that treatment with worm proteins causes normalization of intestinal transit time and amelioration of colonic peristaltic activity in mice with colitis. Treatment of control animals with worm proteins did not influence gastrointestinal motility. Furthermore, attenuation of inflammation and amelioration of motility disturbances after treatment with worm proteins both appear at the same time. This raises the question whether the beneficial effect of worm proteins on gastrointestinal motility is directly or indirectly related to amelioration of inflammation.

In addition, the paper also reports a shift in the balance between different T lymphocyte subsets (Th1, Th2, Th17 and Treg) after the induction of colitis and treatment with worm proteins. The therapeutic effect of worm proteins appears to be mediated by an immunological pathway involving Th1, Th17 and Treg cells. Unraveling the interaction between the immune system and the nervous system will expand our knowledge on how worm proteins affect gastrointestinal motility. More research on this topic is eagerly awaited.

This study shows that treatment with worm proteins attenuates intestinal inflammation and normalizes gastrointestinal motility disturbances in mice with colitis. These results demonstrate that worm proteins, by

influencing intestinal inflammation and the related symptoms during colitis, may provide an attractive option in the management of gastrointestinal inflammation in IBD patients.

More information: Ruyssers NE, De Winter BY, De Man JG, Ruyssers ND, Van Gils AJ, Loukas A, Pearson MS, Weinstock JV, Pelckmans PA, Moreels TG. Schistosoma mansoni proteins attenuate gastrointestinal motility disturbances during experimental colitis in mice. World J Gastroenterol 2010; 16(6): 703-712. www.wjgnet.com/1007-9327/16/703.asp

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