

# New insight into inflammatory bowel disease

5 March 2015



The entire intestinal tract is lined with a single layer of [epithelial cells](#) which become inflamed and destroyed by IBD.

Together with Imtiyaz Thagia from Lancaster University and colleagues from Manchester, Dr Rigby investigated the role of a [protein](#) in the intestine called SOCS3 which is increased in IBD.

The research, funded by the Medical Research Council, is published in the *American Journal of Physiology*.

The protein SOCS3 limits [inflammation](#) of the intestine, but the researchers found that its increase in cases of IBD had negative effects on repair of the epithelial lining.

Dr Rigby said: "Our latest study shows that SOCS3 limits microbial-induced epithelial wound healing.

"These results provide further evidence to support the regulatory role of epithelial SOCS3 in intestinal health and suggest that the increased expression of SOCS3 observed in IBD may serve to perpetuate inflammation."

**More information:** "Intestinal epithelial suppressor of cytokine signaling 3 enhances microbial-induced inflammatory tumor necrosis factor- $\alpha$ , contributing to epithelial barrier dysfunction." *Am J Physiol Gastrointest Liver Physiol*. 2015 Jan 1;308(1):G25-31. [DOI: 10.1152/ajpgi.00214.2014](#). Epub 2014 Nov 6.

The development of inflammatory bowel disease (IBD) may be influenced through a protein in the gut leading to inflammation according to research.

As many as one in a hundred people in the UK will develop IBD in their lifetime, including Crohn's disease and Ulcerative Colitis which together affect 260,000 people.

The rate at which the lining of the [intestine](#) is repaired is dependent on interactions with bacteria in the gut.

Dr Rachael Rigby of Lancaster University said: "Potential reasons why IBD is on the increase in developed countries include changes in the microbes residing in the intestinal gut.

This influences the rate at which the lining of the intestine is repaired since this provides a first line of defence against infectious agents within the gut contents."

Provided by Lancaster University

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