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News Release

Chronically stressed intestines

A new mechanism has been discovered in the development of inflammatory bowel disease

Inflammatory bowel disease, which includes Crohn's disease and ulcerative colitis, is a lifestyle disease and on the rise. Even though the underlying cause of this condition remains unclear, scientists are one step closer to understanding its development. Researchers at the Technische Universität München (TUM) have discovered that the interaction between an immune system signaling protein and a stress protein is out of kilter.

The intestine is a fascinating organ. In adults it is around eight meters long with a surface area of 300 square meters, comparable to the floor space an apartment house. This is the result of the unique structure of the intestinal walls, the insides of which are covered with finger-like protrusions and fine hairs. These are in turn coated with a razor-thin, single-cell layer of goblet cells, the intestinal epithelium. It is this membrane that ultimately controls which nutrients are absorbed by the body. Toxins and pathogens are recognized and prevented from passing into the blood system. In their function as guardians over "good and evil", epithelial cells form an integral part of the immune system.

In patients with chronic intestinal inflammation, like Crohn's disease and ulcerative colitis, this immune response does not function properly. The epithelial cells no longer discern which substances the body can tolerate, and which should kept out of the body's metabolic cycle. Researchers at the TUM have now deciphered an important mechanism in the development of this ailment. Prof. Dirk Haller and his colleagues from the Nutrition and Food Research Center at the Center for Life and Food Sciences Weihenstephan discovered that the cells in the thin boundary layer are relatively stress intolerant.

If these epithelial cells are not supplied with adequate levels oxygen and energy they will die off, sooner or later. In persons with chronic intestinal inflammation the TUM researchers discovered surplus production of the stress hormone grp-87. At the same time the anti-inflammatory immune system signaling protein interleukin-10 was missing. The disrupted interaction of these two actors seems to carry at least part of the blame for the occurrence of inflammatory bowel disease. With too much grp-87 and too little interleukin-10, the plethora of bacteria in "normal" intestinal flora is classified as dangerous, causing the intestinal system to lose control of inflammation processes. Inflammatory bowel disease inhibits nutrient absorption and increases the risk of contracting colon cancer.

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Professor Haller from the Chair for Biofunctionality of Food is using these fundamental findings on the processes leading to inflammatory bowel disease in his search for new treatment options. The research project is supported by the European Union. Over the next three years, Heller, together with his colleague Prof. Michael Schemann from the Chair for Human Biology, will receive research funding of EUR 650,000. In collaboration with research associates from England, Ireland and Belgium, the two TUM researchers will use the funds to explore new strategies in the treatment of inflammatory bowel disease.

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Photo proposals:
Darm-unter-Mikroskop.jpg: Unlike healthy intestinal mucous tissue (left), inflamed intestinal epithelial cells (right) lose their important immune response functionality.
(Picture: TUM/D. Haller)
ZIEL.jpg: The TUM Nutrition and Food Research Center makes interdisciplinary cooperation possible. (Picture: TUM/U. Benz)

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