

Six proteins upregulated years before ulcerative colitis diagnosis

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(HealthDay)—Six inflammatory proteins are upregulated in plasma

several years before diagnosis of ulcerative colitis, according to a report published in the November issue of *Gastroenterology*.

Daniel Bergemalm, M.D., Ph.D., from the Örebro University Hospital in Sweden, and colleagues characterized preclinical systemic inflammation in [ulcerative colitis](#) in plasma samples biobanked from 72 individuals who developed ulcerative colitis later in life and 140 matched healthy controls. Ninety-two proteins related to inflammation were measured. The biological relevance of findings was validated in an inception cohort of 101 ulcerative colitis patients and 50 healthy controls. The influence of genetic and [environmental factors](#) on these markers was examined in a cohort of 41 healthy twin siblings of patients with ulcerative colitis and 37 matched healthy controls.

The researchers found that six proteins were upregulated in preclinical ulcerative colitis versus controls in univariate and multivariate models (MMP10, CXCL9, CCL11, SLAMF1, CXCL11 and MCP-1). Several potential key regulators were identified, including interleukin-1 β , [tumor necrosis factor](#), interferon-gamma, oncostatin M, nuclear factor- κ B, interleukin-6, and interleukin-4. In the inception cohort, a multivariable model differentiated treatment-naïve patients with ulcerative colitis from controls with leave-one-out cross validation (area under the curve, 0.92). Among the healthy twin siblings, MMP10, CXCL9, CXCL11, and MCP1, but not CCL11 and SLAMF1, were significantly upregulated, although their relative abundances were higher in incident ulcerative colitis.

"Our findings provide novel data on the early sequence of inflammatory events that eventually cause ulcerative colitis because activation of several of the proteins was triggered by exposure to genetic and environmental risk factors," the authors write.

Several authors are employees of Olink Proteomics.

More information: [Abstract/Full Text](#)

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