Instability in the composition of gut bacterial communities (dysbiosis) has been linked to common human intestinal disorders, including inflammatory bowel disease and colorectal cancer; however, it is unclear if dysbiosis can instigate disease or if it is a consequence of the underlying disorder.

In this issue of the *Journal of Clinical Investigation*, researchers led by Mathias Chamaillard at the University Lille Nord de France in Lille, France, examined intestinal inflammation and tumorigenesis in a mouse model of dysbiosis. Dysbiosis enhanced intestinal inflammation and increased the risk for inflammation-associated colon cancer.

Treatment with antibiotics or transplantation of fecal material from normal mice reduced disease risk and instigated long-term, beneficial alterations in intestinal bacteria.

Conversely, transplantation of normal mice with dysbiotic fecal material increased intestinal inflammation and enhanced the risk of inflammation-associated colon cancer.

These results demonstrate that gut bacterial communities play an integral role in protecting against intestinal inflammation and associated tumorigenesis.

**More information:** NOD2-mediated dysbiosis predisposes mice to transmissible colitis and colorectal cancer, *Journal of Clinical*

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