

Cytokine mediates obesity-related factors linked to colorectal cancer

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Credit: Mary Ann Liebert, Inc., publishers



A new study describes the mechanistic relationship between the cytokine interleukin-1ß, (IL-1ß) and obesity, showing that when IL-1ß levels are increased in obesity, IL-1 receptor signaling activates multiple pathways leading to colon cancer. The study shows that obesity is linked with systemic increases in IL-1ß, activation of Wnt, and proliferation of mouse colon cells, as reported in an article published in *Journal of Interferon & Cytokine Research* (JICR) from Mary Ann Liebert, Inc., publishers.

Joel Mason, Tufts University and Tufts University School of Medicine, Boston MA, and colleagues from Tufts University, coauthored the article entitled "Interleukin-1 Signaling Mediates Obesity-Promoted Elevations in Inflammatory Cytokines, Wnt Activation, and Epithelial Proliferation in the Mouse Colon."

The researchers set out to define the role of IL-1ß in mediating the events leading up to <u>obesity</u>-promoted colorectal cancer. They compared the role of IL-1ß in mice fed either a high-fat (obese) or low-fat (lean) diet. Among the changes they found were that obese mice had 30-80% greater concentrations of IL-1ß in the colonic mucosa, a significant increase in the Wnt signaling cascade, and a significant expansion in the proliferation zone of the colonic crypt.

"This study reveals the close linkage of obesity and the inflammatory response and reflects the broad actions of IL-1ß that define obesity as one of many inflammatory diseases," says *Journal of Interferon & Cytokine Research* Editor-in-Chief Michael Gale Jr., Department of Immunology, University of Washington, Center for Innate Immunity and Immune Disease.

More information: Anna C. Pfalzer et al, Interleukin-1 Signaling Mediates Obesity-Promoted Elevations in Inflammatory Cytokines, Wnt Activation, and Epithelial Proliferation in the Mouse Colon, *Journal of*



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Provided by Mary Ann Liebert, Inc

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