Public release date: 22-Aug-2008

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Going from ulcers to cancer

Appearing in JBC online Aug. 22

Researchers have uncovered a big clue as to why some of the bacteria that cause stomach ulcers pose a greater risk for serious problems like stomach cancer than others; it turns out these bacteria can exploit the surrounding stomach cells to protect them from the immune system.

Helicobacter pylori is a bacterial strain that can infect the human stomach and induce inflammation, ulcers, and potentially even stomach cancer. However, only a small fraction of H. pylori infections ultimately lead to cancer, leading researchers to figure out what biological events will trigger this path.

One type of H. pylori strain that seems to increase disease risk is the cag+ strain, which contains a set of proteins that allows it to inject bacterial proteins into cells following attachment to the stomach lining; this interaction between bacteria and gastric cells may be a key contributor to chronic damage.

Richard Peek and colleagues investigated a cag+ strain in mouse models of H. pylori infection and found that a protein called CagE could induce gastric cells to turn on a receptor called Decay-accelerating factor (DAF); DAF acts to remove nearby immune proteins that can kill cells to prevent unwanted immune damage.

In essence, the bacteria use the DAF receptor on the host cell they're attached to like a bodyguard to protect them from the immune system. Peek and colleagues also note that by continually inducing DAF expression, H. pylori creates an environment of persistent inflammation that can reduce the threshold required for more serious diseases to develop.

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From the *JBC* article: "Regulation of the Helicobacter pylori cellular receptor Decay-accelerating Factor" by Daniel O'Brien, Judith Romero-Gallo, Barbara G. Schneider, Rupesh Chaturvedi, Alberto Delgado, Elizabeth J. Harris, Uma Krishna, Seth R. Ogden, Dawn A. Israel, Keith T. Wilson, and Richard M. Peek Jr

Article Link: http://www.jbc.org/cgi/content/abstract/M801144200v1

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