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Extending the gut-brain axis: The curious case of *Helicobacter pylori*

There is increasing recognition that localized microbe-host interactions in humans can influence the physiology and function of distal tissue and organ systems in a manner that can have profound impact on human health and disease. For example, human gut microbiota communicates with the central nervous system (CNS) via the neural, endocrine and/or immune systems, thereby influencing brain function. Cytokines from the peripheral immune system interact with the central nervous system (CNS) via active transport across the blood brain barrier as well as direct entry via the circumventricular organs. Here, we evaluated the hypothesis that chronic gastric infection with the human pathogenic bacterium *Helicobacter pylori* (*Hp*) is causal for elevated systemic inflammation and neuro-inflammation. Chronic *Hp* infection is characterized by sustained inflammation of the gastric mucosa, which is highly associated with progression to gastric ulcer disease or gastric cancer in approximately 10% and 2% of infected individuals, respectively. Our studies employing a Sprague-Dawley rat model for chronic *Hp* infection revealed sustained gastric inflammation and tissue damage. What is more, *Hp*-infected rats demonstrated elevated inflammation with the bloodstream and CNS, with activated microglia and elevated TNF- α detected in the brain of the infected animals. The causal relationship between gastric *Hp* infection and brain sequelae was supported by the loss of elevated systemic and CNS inflammation upon clearance of *Hp* infections following antibiotic administration. Because approximately half the world's population is infected with *Hp*, these results have implications for the impact of microbes on cognition and behavior during human brain development.

Biography

Professor Blanke joined the Department of Microbiology at the University of Illinois as an Associate Professor in May 2005. He is also a member of the Host Microbe Systems Research Theme in the Institute for Genomic Biology. Professor Blanke received his B.S. degree from Virginia Tech University and a Ph.D. in Biochemistry from the University of Illinois at Urbana-Champaign in 1989. He subsequently was a National Institutes of Health Postdoctoral Fellow at Harvard University. Prior to joining the University of Illinois, Professor Blanke was a faculty member in the Department of Biology & Biochemistry at the University of Houston. Professor Blanke's research explores the fundamental molecular and cellular mechanisms used by pathogenic bacteria to establish infection and persist within a host.

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