H. pylori modulates DC functions via T4SS/TNF α /p38-dependent SOCS3 expression

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Abstract

Helicobacter pylori is one of the most prevalent human pathogens worldwide, with over 4 billion people infected. Without antibiotics, the bacteria can persist in the GI tract of its host for life and cause gastritis, ulcers, and even gastric cancer. Although *H. pylori* induces a severe inflammatory response, it can't be cleared by its host's immune system. A recent study sought an explanation for why the immune response against *H. pylori* is not effective. Researchers examined the characteristics of human monocyte-derived DCs infected with *H. pylori*. They found that *H. pylori*-infected DCs had higher levels of a suppressor of cytokine signaling (SOCS) protein. SOCS3 expression was specifically increased by *H. pylori* infection. This change was mediated by a pathway relying on the type IV secretion system, TNF α , and p38. Silencing SOCS3 in DCs prior to infection resulted in increased cytokine signaling and downregulated T cell activation and proliferation. These results highlight the important role of SOCS3 in controlling the immune response during *H. pylori* infection and suggest that targeting this pathway may help to develop new therapies for *H. pylori* infection.