Researchers find ulcer bacteria *H. pylori* to be present in blood vessels

**BETHESDA, Md.** – A research group from the Uniformed Services University of the Health Sciences (USU), in collaboration with European researchers, demonstrated that the peptic ulcer bacterium *Helicobacter pylori* (*H. pylori*) can reach the blood circulation. Thus, the bacterium can disseminate in the body and may cause other chronic inflammatory conditions of unknown origin, such as atherosclerosis and rheumatoid arthritis.

Andre Dubois, M.D., Ph.D., professor of medicine, surgery, and emerging infectious diseases, and chief, Laboratory of Gastrointestinal and Liver Studies at USU, and members of his research group, Drs. Cristina Semino-Mora and Hui Liu, co-authored the article with European researchers led by Dr. Thomas Borén. Dr. Borén is professor of biochemistry and biophysics at Umeå University, Umeå, Sweden.

Dr. Dubois’ latest observations elaborate on the 1980s landmark studies by the 2005 Nobel laureates Barry Marshall and Robin Warren. These Australian investigators demonstrated that *H. pylori* is the cause of gastritis, peptic ulcers and stomach cancer, one of the most common types of cancer worldwide. Dr. Dubois’ research group subsequently showed that *H. pylori* could also invade normal and cancerous gastric epithelial cells. The latest research of the USU team now demonstrates that, in addition, *H. pylori* can enter the stomach’s blood vessels and attach to red blood cells.

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The mechanism of this attachment relies on proteins (adhesins) that are located on the bacterial surface and bind to carbohydrates located both on the stomach cell surfaces and on red blood cells. This firm attachment allows survival and persistence of the microbe in the acidic and turbulent stomach environment, and its role in the blood circulation may be clarified by the present observations.

Importantly, *H. pylori* attachment to red blood cells is mediated by the SabA adhesin, which has a different composition among different *H. pylori* strains from different patients and the host carbohydrates to which SabA attaches also varies among individuals. As a result, the SabA adhesin may determine the inherent feature of adaptation to cell surface characteristics of the individual host. Because *H. pylori* can adjust its adherence properties both for the individual gastric mucosa of the particular host, it also can adapt to the defenses constructed by the host in response to infection during chronic inflammation. Thus, the outcome of the *H. pylori* infection will depend on how the bacterium can adapt to the host defenses and vice-versa.

The research paper can be viewed online at: [http://pathogens.plosjournals.org/XXX](http://pathogens.plosjournals.org/XXX) and will be published in the October issue of the *PLoS Pathogens Journal*.

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