# *Helicobacter pylori* and relatives in the fight against oxidative stress

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In the summer of 1984, Australian scientist Neil Noakes prepared a mixture of bacterial cells and beef extract and gave it to a gastroenterologist - Barry Marschall - to drink it. Barry Marshall developed the first symptoms in the form of vomiting three days after consuming the liquid. This daring experiment started a research that confirmed, that stomach ulcers are not related to stress as previously thought, but to gastric infection caused by *Helicobacter pylori*. In 2005, Barry Marschall and Robin Warren received the Nobel Prize for their discovery and further research on *H. pylori*. Over the past thirty years, numerous experiments have been conducted to understand the mechanism of infection of this bacterium. We know that *H. pylori* has been co-evolving with humans since leaving Africa, which took place over 70,000 years ago. Over such an extended period, this species has developed various defence mechanisms against its host's immune system.

One of the mechanisms that the immune system created to fight microorganisms is the so-called respiratory burst - the rapid release of reactive oxygen species (ROS), such as superoxide anion ($^{\bullet}O_2^-$), hydrogen peroxide ($H_2O_2$), or the hydroxyl radical ($^{\bullet}OH$). ROS contribute to the destruction of lipids, proteins and nucleic acids of the cell, which ultimately leads to its death. However, in the course of evolution, bacteria have developed defence tactics to protect them from the harmful effects of these molecules, including by producing enzymes that lead to their neutralization.

In the world of bacteria, the synthesis of proteins (enzymes are proteins mainly) involved in the fight against the stress factor often is controlled through an intermediate protein - a specific regulator that receives the signal and then activates the synthesis of target proteins, directly involved in neutralizing the threat. Interestingly, despite many years of research, no regulator has been found in *H. pylori*, the activation of which would be dependent on oxidative stress.

Our research has identified a potential protein - HP1021, which may be a regulator of this type. Moreover, in our experiments, we try to determine the set of all genes regulated by HP1021, the so-called regulon. Also, in the future, we plan to understand the mechanism of action and characterize the regulators of similar proteins from bacteria related to *H. pylori*: *Arcobacter butzleri* and *Campylobacter jejuni*. *A. butzleri* belongs to emerging pathogens, i.e., pathogens causing

diseases whose incidence has increased recently over the past twenty years and could have significant consequences in the future. *C. jejuni*, on the other hand, is the factor responsible for the highest number of deaths from enteritis in the world.

Even though this is basic research, in the future, it may contribute to the development of new drugs that are substitutes for antibiotics, which are an increasingly weaker weapon in the fight against bacteria.

## Graphical abstract