Nature Reviews Microbiology | Published online 27 Mar 2017; doi:10.1038/nrmicro.2017.32

BACTERIAL PHYSIOLOGY

When things turn sour for *Helicobacter*

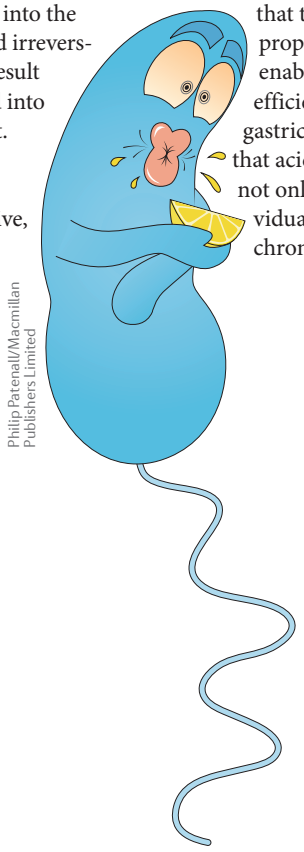
> different binding properties of BabA enable *H. pylori* to adapt efficiently to changes in gastric mucosal pH

Helicobacter pylori causes persistent infections of the human gastric mucosa and has been identified as the main risk factor in the development of gastric cancer. *H. pylori* colonizes the mucosal surface by forming tight interactions with epithelial cells through adhesins, such as BabA, which mediates high-affinity binding to Leb blood group antigens that are present on the surface of epithelial cells. However, mucus and epithelial cells are constantly shed into the acidic gastric lumen, and irreversible attachment would result in bacteria being carried into this hostile environment.

Now, a study reports that BabA-mediated adherence is acid-sensitive, with binding reduced at low pH and fully restored by increased pH. The authors propose a dynamic model whereby *H. pylori* detach from cellular debris that has been shed as they move towards the acidic lumen, which enables the bacteria to return to the epithelial surface and reattach. Moreover, the authors showed that acid sensitivity of the adhesin differs among clinical isolates and isolates from different gastric sites. The findings suggest that the different binding properties of BabA enable *H. pylori* to adapt efficiently to changes in gastric mucosal pH and that acid sensitivity evolves not only in different individuals but also during chronic infection and disease progression; for example, when chronic inflammation leads to a gradual shifts in gastric acidity. Finally, the authors report that acid-sensitive binding depends on the so-called pH-sensor region of BabA, which is located in its binding domain.

In summary, the results of this study suggest that the BabA protein sequence determines adaptation to the individual gastric environment, in which changes in acidity could result from the development of gastric disease and/or long-term use of acid-suppressing drugs.

Andrea Du Toit



Philip Patenall/Macmillan Publishers Limited

ORIGINAL ARTICLE Bugaytsova J. A. *et al.* *Helicobacter pylori* adapts to chronic infection and gastric disease via pH-responsive BabA-mediated adherence. *Cell Host Microbe* **21**, 376–389 (2017)