

Why does aspirin increase the susceptibility of Helicobacter pylori to antimicrobials?

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Resent studies reported that aspirin inhibited the growth of *H. pylori* in a dose-dependent manner and significantly affected the activity of virulence factors of *H. pylori*. In addition, aspirin increased the susceptibility of *H. pylori* to antimicribials including metronidazole, clarithromycin and amoxicillin. However, the mechanisms remained unknown.

A research team led by Prof. Wang from Peking University First Hospital of China addressed this issue and their results will be published on February 28, 2009 in the *World Journal of Gastroenterology*.

H. pylori reference strain 26695 and two metronidazole-resistant isolates of *H. pylori* were included in this study. The effect of aspirin on the permeability of the outer membrane of *H. pylori* was determined using [7-3H] tetracycline. The effects of aspirin on the expression of OMPs of *H. pylori* were also determined. Taqman-based real-time quantitative PCR was used to analyze the influence of aspirin on the expression of the related OMPs genes.

They found that the mutations in rdxA gene did not change in metronidazole resistant isolates treated with aspirin. The radioactivity of *H. pylori* increased when treated with aspirin, indicating that aspirin improved the permeability of the outer membrane of *H. pylori*. However, the expression of two OMP bands between 55 kDa and 72 kDa altered in the presence of aspirin. The expression of the mRNA of hopA, hopB, hopC, hopD, hopE and hefA, hefB, hefC of *H. pylori* did



not change when treated with aspirin.

Their results indicated that although aspirin increases the susceptibility of *H. pylori* to metronidazole, it has no effect on the mutations of rdxA gene of *H. pylori*. Aspirin increases endocellular concentrations of antimicrobials and probably by altering the expression of the outer membrane proteins (OMP) of *H. pylori*. Their study will help understand the mechanisms of the resistance of *H. pylori* to antibiotics more intensively and discover a more effective eradication regimen in clinical practice.

Source: World Journal of Gastroenterology

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