Helicobacter pylori infection in neonatal mice prevents allergic asthma

In this preclinical study, the authors hypothesised that neonatal infection with an immunomodulatory pathogen such as Helicobacter pylori provides protection from allergic airway inflammation and hyper-responsiveness seen in allergic asthma.

C57BL/6 mice were orally infected with H. pylori at 6 days (neonatal) and 6 weeks (adults) after birth. Infected and non-infected mice underwent ovalbumin sensitisation followed by ovalbumin challenge. Infected mice showed significant reduction in airway hyperresponsiveness to methacholine challenge. This reduced inflammatory response was indicated by low eosinophils and interleukin 5 in bronchoalveolar lavage fluid and reduced inflammation of Th2 and Th17 cells. These changes were absent in adult infected mice indicating that only early life exposure to H. pylori infection confers protection against asthma in mouse models.

The authors explained the immunological process by carrying out further tests, which showed that H. pylori-mediated asthma protection in neonatally infected mice is due to the suppressive activity of CD4+FoxP3+ Tregs and the presence of semimature dendritic cells, both of which accumulate in the lungs during the inflammatory process. Based on the results of the mouse model, it is possible that allergic asthma is associated with the loss of indigenous microbial flora in the neonatal period; however, extrapolating this evidence to a human population will require more direct evidence from human studies.


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Published Online First 22 September 2012

Thorax 2012;67:301. doi:10.1136/thoraxjnl-2011-201061