Contributors Steering Committee (initiated trial design, data collection and statistical analysis plan): Jesper Holst Pedersen (Chairman/Guarantor), Asger Dirksen (Guarantor), Karen Skjældstrup and John Brodersen, Paul Frost Clementsen, Martin Tønnesen, Haneen Hansen, Klaus Fuglsang Kofold, Klaus Richter Larsen, Jann Mortensen, Niels Seersholm, Birgit Guldbakke Skov, Hanne Thorsen, Philip Tønnesen, Haseem Ashraf (2007) and Zaigham Saghir (2009/Guarantor). HH and KSB oversaw overall design and findings from baseline screening. HH also performed (or supervised) data analysis. HS, JM, DB, NH, MT and KB performed data cleaning. ZH, AD, DB, KB, HT, NS, BGS, PT and AJ participated in the development of the COS and COS-LC questionnaires. ZS and HA collected, cleaned and analysed data. JK collected and analysed data. JR performed diagnostic workup. AD was responsible for event monitoring and following protocol. JB and HT performed reading of CT scans. AD, PFC, KRL, JM, NS, BGS, PT conducted diagnostic workup. AD was responsible for event monitoring and following protocol. JB and HT designed the COS and COS-LC questionnaires. ZS and HA collected, cleaned and analysed data. JR performed reading of CT scans. AD revised the paper. All revised the paper.

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REFERENCES


Journal club

**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 aerosolised ovalbumin challenge 4 weeks later. Infected mice as compared with non-infected mice showed significant reduction in airway hyper-responsiveness to methacholine challenge. This reduced inflammatory response was indicated by low eosinophils and interleukin 5 in bronchoalveolar lavage fluid and reduced infiltration 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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