hay fever and for raised IgE for common inhalation allergens at age 11. It would therefore be most informative if the authors could provide additional data on the differences between the neonatal anthropometric data of the children with and without caesarean section, and on the indications for caesarean sections themselves. This issue is of significant importance and of clinical relevance, because, if indeed a causal relationship exists between mode of delivery and development of asthma, this would certainly make an argument against elective caesarean section for non-medical reasons. It would seem that there is currently insufficient evidence to infer a causal relationship, but it certainly seems worthwhile sorting this out.

P Merkus
Correspondence to: Dr P Merkus, P O Box 9101, 6500 HB, The Netherlands; p.merkus@cukz.umcn.nl
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REFERENCES

Caesarean section and asthma: alternative explanations?
In their detailed analysis of almost 3000 children followed from birth until the age of 8 years, Roduit et al showed that children born by caesarean section have a higher risk of asthma than those born by vaginal delivery. Surprisingly, the authors offer only one explanation for this finding—namely, delayed microbial colonisation—whereas we believe other mechanisms cannot be excluded.

As an alternative hypothesis we propose to investigate the possibility of confounding by factors already present at/before birth. This hypothesis is supported by studies showing that immunological parameters in cord blood are different between children born by vaginal delivery and those born by caesarean section. One such factor could be head circumference which has been repeatedly found to be related to increased IgE and the development of asthma and related disorders,1–3 and babies born by caesarean section probably have relatively high values.

J C van der Wouden,1 R M D Bernsen2
1Department of General Practice, Erasmus MC, University Medical Center, Rotterdam, The Netherlands; 2Department of Community Medicine, United Arab Emirates University, Al Ain, United Arab Emirates
Correspondence to: Dr J C van der Wouden, P O Box 2040, Rotterdam 3000 CA, The Netherlands; j.vanderwouden@erasmusmc.nl
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Presence of MBL in airways: is it a disease severity marker or an additional host defence mechanism?
We welcome the paper by Figler and colleagues reporting the presence of mannose-binding lectin (MBL) in infected airways.1 MBL is an important acute phase protein with pro- and anti-inflammatory immunomodulatory functions.2 The collectin family comprises surfactant protein (SP)-A, SP-D and MBL, of which the latter is structurally similar to lung collectins and its presence at a physiological level in the lung. It is possible, however, that the presence of MBL in the bronchoalveolar lavage (BAL) fluid of infected children might just be a marker of lung infection or disease severity. The data of Figler et al clearly show a trend suggesting that MBL was more consistently detectable in acute than in chronic diseases; this may simply be a correlate of alveolar epithelial permeability. A similar study performed by our group on HIV-infected adults showed that the levels of MBL in BAL fluid were undetectable even when present in serum. The levels of SP-D in the same study were not significantly different in lung fluid from HIV-uninfected and HIV-infected individuals with a high CD4 count (>200), but were raised in HIV-infected individuals with a low CD4 count.4 We tested the hypothesis that levels of SP-D or MBL in HIV-infected individuals would be lower than in HIV-uninfected individuals, but this was not the case. The phenomenon that levels of defence factors are poorly associated with protection has also been shown with other defence factors such as antibodies.5

In conclusion, we totally agree with Figler et al that future studies should focus on measuring the functional aspect of collectins. Functional assays will help to determine whether the presence of MBL in the lung acts as an additional host defence or whether it is just a marker of disease severity.

K C Jambo, S B Gordon
Pulmonary Immunology Group, Liverpool School of Tropical Medicine, Liverpool, UK
Correspondence to: K C Jambo, Pulmonary Immunology Group, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5DA, UK; kjambo@liverpool.ac.uk
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REFERENCES

Thoracic ultrasound: an important skill for respiratory physicians
We read with interest the article by Qureshi and colleagues describing thoracic ultrasound (TUS) characteristics for the detection of malignant pleural effusions.1 This relatively simple bedside technique has been routinely performed by the respiratory physicians in our department in a busy general hospital for the last 4 years, resulting...
Presence of MBL in airways: is it a disease severity marker or an additional host defence mechanism?

K C Jambo and S B Gordon

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