- 26 **Bullock GR**, Steyaert I, Bilbe G, *et al*. Distribution of type-1 and type-2 angiotensin receptors in the normal human lung and in lungs from patients with chronic obstructive pulmonary disease. *Histochem Cell Biol* 2001;**115**:117–24.
- 27 **Specks U**, Martin WJ, Rohrbach MS. Bronchoalveolar lavage fluid angiotensin-converting enzyme in interstitial lung diseases. *Am Rev Respir Dis* 1990;**141**:117–23.
- 28 **Grafe M**, Auch-Schwell W, Zakrzewicz A, *et al*. Angiotensin II-induced leukocyte adhesion on human coronary endothelial cells is mediated by E-selectin. *Circ Res* 1997;**81**:804–11.
- 29 **Ito H**, Takemori K, Suzuki T. Role of angiotensin II type 1 receptor in the leucocytes and endothelial cells of brain microvessels in the pathogenesis of hypertensive cerebral injury. *J Hypertens* 2001;**19**:591–7.
- 30 **Keogh KA**, Standing J, Kane GC, *et al*. Angiotensin II antagonism fails to ameliorate bleomycin-induced pulmonary fibrosis in mice. *Eur Respir J* 2005;**25**:708–14.
- 31 **Uhal BD**, Wang R, Laukka J, *et al*. Inhibition of amiodarone-induced lung fibrosis but not alveolitis by angiotensin system antagonists. *Pharmacol Toxicol* 2003;**92**:81–7.
- 32 **Renzone EA**, Abraham DJ, Howat S, *et al*. Gene expression reveals novel TGF β targets in adult lung fibroblasts. *Respir Res* 2004;**5**:24.
- 33 **Vancheri C**, Mastruzzo C, Sortino MA, *et al*. The lung as a privileged site for the beneficial actions of PGE₂. *Trends Immunol* 2004;**25**:40–6.
- 34 **Kohyama T**, Ertl RF, Valenti V, *et al*. Prostaglandin E(2) inhibits fibroblast chemotaxis. *Am J Physiol Lung Cell Mol Physiol* 2001;**281**:L1257–63.
- 35 **Fine A**, Poliks CF, Donahue LP, *et al*. The differential effect of prostaglandin E2 on transforming growth factor-beta and insulin-induced collagen formation in lung fibroblasts. *J Biol Chem* 1989;**264**:16988–91.
- 36 **Lama V**, Moore BB, Christensen P, *et al*. Prostaglandin E2 synthesis and suppression of fibroblast proliferation by alveolar epithelial cells is cyclooxygenase-2-dependent. *Am J Respir Cell Mol Biol* 2002;**27**:752–8.
- 37 **Cheng HF**, Wang JL, Zhang MZ, *et al*. Angiotensin II attenuates renal cortical cyclooxygenase-2 expression. *J Clin Invest* 1999;**103**:953–61.
- 38 **Bonner JC**, Rice AB, Ingram JL, *et al*. Susceptibility of cyclooxygenase-2-deficient mice to pulmonary fibrogenesis. *Am J Pathol* 2002;**161**:459–70.
- 39 **Hodges RJ**, Jenkis RG, Wheeler-Jones CP, *et al*. Severity of lung injury in cyclooxygenase-2-deficient mice is dependent on reduced prostaglandin E2 production. *Am J Pathol* 2004;**165**:1663–76.

LUNG ALERT

Infants too young to receive pneumococcal conjugate vaccine benefit from herd immunity

▲ Poehling KA, Talbot TR, Griffin MR, *et al*. Invasive pneumococcal disease among infants before and after introduction of pneumococcal conjugate vaccine. *JAMA* 2006;**295**:1668–74

The Netherlands and the UK have recently joined a relatively small number of European countries to recommend a heptavalent pneumococcal conjugate vaccine (PCV7) as part of universal childhood immunisation programmes. PCV7 was incorporated into the US childhood vaccination programme in June 2000 and is recommended for all children aged 2–23 months. Since its introduction, evidence has shown that the rate of invasive pneumococcal disease (IPD) among children younger than 2 years has reduced by at least 60%. This study investigated for the first time rates of IPD in children aged 0–90 days before and after the introduction of PCV7.

A prospective population based study was undertaken with active laboratory based surveillance for IPD in infants aged 0–90 days across eight US states before (July 1997–June 2000) and after (July 2001–June 2004) the introduction of PCV7. The mean rates of IPD for infants aged 0–90 days decreased 40% from 11.8 (95% CI 9.6 to 14.5) to 7.2 (95% CI 5.6 to 9.4; $p = 0.004$) per 100 000 live births following PCV7 introduction. Notably, there was a significant decrease of 42% from 7.3 (95% CI 5.6 to 9.5) to 4.2 (95% CI 3.0 to 5.9; $p = 0.01$) per 100 000 live births in rates of IPD among infants aged 0–60 days—that is, those too young to receive the vaccine. Furthermore, subgroup analysis by race showed that the previous disproportionately high incidence of IPD in black compared with white infants was eliminated after the introduction of PCV7.

The authors conclude that this is the first study to suggest that neonates and infants too young to receive PCV7 are benefiting from herd immunity. They emphasise the importance of continued surveillance of IPD to observe if the trend continues, and to determine if serotypes not included in PCV7 emerge as an important cause of IPD in neonates and young infants.

R J F Carter

Senior House Officer, Hull Royal Infirmary, Hull, UK; rjfcarter@hotmail.com