Highlights from this issue

doi:10.1136/thoraxjnl-2013-204861

Andrew Bush, Ian Pavord, *Editors*

COMING IN FROM THE COLD

Lung transplantation represents the last hope for many patients with end-stage lung disease, but given the scarcity of organs and the worse results of re-do transplantation, the transplanted lung must be protected from damage as carefully as the reputations of our egregious politicians. Respiratory viruses are already causally implicated in the lung attacks which cause so much long term damage in asthma, COPD, cystic fibrosis (CF) and other diseases. In this issue, Bridevaux *et al* (Editors' choice; ***see page 32***) report a long-term prospective study of the prevalence of common respiratory viruses in lung transplant recipients. They found that viruses were common, almost invariably associated with symptoms, but they could not show any association with rejection. In an accompanying editorial, Glanville (***see page 1***) speculates that the picture may not be quite as reassuring as the properly cautious interpretation in the manuscript. Viruses cause epithelial damage and aberrant repair of recurrent insults is thought to prime the airway for downstream rejection. One could also add that viral infection causes neutrophilic inflammation, another hallmark of rejection. Remember traffic pollution, another cause of neutrophilic inflammation, has also been implicated in bad outcomes after transplantation (see *Thorax* 2011;66:748–54). These data highlight the need for precise diagnosis of respiratory symptoms in transplant recipients, so that viral infection is not treated as acute rejection, but perhaps also trials of nebulised interferon at the time of viral-induced respiratory symptoms to try to improve long-term outcome.

TROUBLE AT T'MILL?

More on CF lung attacks. The adverse effects of these on lung function and prognosis have been well rehearsed in *Thorax*, as has as the lack of diagnostic precision. Can we predict trouble ahead by any biomarker? The lung clearance index (LCI) has been shown to be a sensitive marker of airway disease in CF, becoming abnormal before conventional parameters such as spirometry and lung volumes in longitudinal studies. The ease of performance has made it an attractive test across the whole age range. In this issue of the journal, Vermeulen *et al* show that LCI can be used in young CF patients to predict frequency of subsequent lung attacks, and also the time to next attack (***see page 39***). There is also a correlation with quality of life, which will interest those terminally addicted to the affectionate embracing of trees. These findings hold up even in those with a normal FEV_1. Some caution is needed; the study, although well-conducted, is not large, and it would be wrong to extrapolate the findings to other suppurative lung diseases, where the relationship of LCI to other parameters may not be so clear. This work adds weight to the validity of LCI in clinical trials, but hopefully will stimulate clinical use to define a high risk group of patients in whom interventions could be deployed, perhaps regular preventive intravenous antibiotics.

OMALIZUMAB: HOW NOT TO IDENTIFY YOUR TARGET POPULATION

Confining omalizumab treatment to those with allergy and a narrow range of IgE levels makes all kinds of sense theoretically. One problem: there is not a shred of evidence that the response to treatment is related to either serum IgE levels or the presence of allergen-specific IgE. Marek Lommatzsch and colleagues (***see page 94***) review the increasingly compelling evidence that patients with non-atopic asthma and low IgE respond just as well as those who meet the manufacturers' suggested criteria. It is now clear that simple, readily available biomarkers such as the blood eosinophil count (a leading candidate for Thorax's biomarker of the year, at least in adults) and exhaled NO identify very effectively patients who do well with omalizumab treatment and poorly without (see *Am J Respir Crit Care Med* 2013;187:804–11). Your editors were amongst the first to eke this information out of reluctant manufacturers (see *NEJM* 2011;364:2556–7). The key message is that careful scrutiny of hard clinical data is the only rational way to make decisions about who should receive treatment.

HOW IS ASTHMA LIKE EMMENTAL CHEESE?

Answer, both appear to be full of holes. Sarah Svenningsen *et al* (***see page 63***, **Hot topic)** used hyperpolarised helium to produce remarkable images in their report on focal ventilation defects in patients with asthma (see the cover). Subsegmental defects were present in two thirds of patients; they were shown to be associated with worse lung function, increased airway inflammation, airway hyperresponsiveness and greater airway wall thickening assessed using contemporaneous CT scans. These findings suggest that, like eczema, asthma is a patchy condition. Ventilatory defects are stable over time and change in the anticipated direction following induced bronchoconstriction and bronchodilation. Intriguingly very preliminary data suggests that the efficacy of bronchial thermoplasty might be mediated via improvements in focal airway wall abnormalities. Might it be possible to deliver this and other localised treatments in a targeted fashion? More work in this exciting area would be most welcome.

HALF A LOAF?

This was the surprising and unusual bronchoscopic appearances seen in a smoker with haemoptysis, whose chest radiograph showed volume loss and opacification in the right hemithorax. The answer is in the Pulmonary Puzzle, with a clue in the title of this annotation (***see page 93***).