Highlights from this issue

doi:10.1136/thoraxjnl-2012-202347

Andrew Bush and Ian Pavord, *Editors*

Out of the frying pan?

School age children spend a lot of time in the classroom, where one would hope they might escape adverse home environmental factors, such as smoking ('I never smoke in front of the children, Dr, so when I need a cigarette I send the kids outside to play') and aeroallergens ('Honey the kids are allergic to the cat; we had better get rid of the kids'). However, the classroom may not be the safe haven we thought. Cat allergic children in classes where more than 20% of their schoolmates own a cat develop an occupational asthma like picture (*Am J Respir Crit Care Med* 2001;**163**:694–8), with asthma worsening during the week and improving at the weekend and during holidays. Now Annesi-Maesano *et al* demonstrate that French primary school class rooms are not exactly havens of unpolluted air ***(see page 682)***. Elevated levels of major pollutants were common, and associated with prevalent asthma and rhinoconjunctivitis. In some classrooms, fine particle and NO_2 levels were above WHO proposed safety levels. Unsurprisingly, the effects were greater in allergic children. Unless French schools are uniquely polluted, which seems unlikely, should we not be considering how we can ensure children really do breathe clean air at school? Legislators, over to you!

Half-baked elastic: auto-antibodies in COPD

Airway inflammation and damage persists long after patients have stopped smoking implying that there is an on-going immune response independent of the original stimulus. The presence of oligoclonal CD4, CD8 and B-lymphocytes in tertiary lymphoid tissue has focused attention on autoimmuity. Elastin has been in the frame as a likely auto-antigen as it is relevant to the pathology and a preliminary but a high profile study published in Nature Medicine identified reactive T-cells against elastin peptides together with anti-elastin antibodies in patients with severe COPD (*Nat Med* 2007;**13**:567–9). Regrettably these findings could not be replicated by Rinaldi *et al* ***(see page 694)*** in what appears to us to have been a carefully conducted and well controlled study. The authors did find that T-cell immunity against anti-collagen-V was prevalent in patients with COPD and smoking controls. This is an intriguing and potentially important finding as collagen-V is a leading candidate as an auto-antigen in obliterative bronchiolitis and atherosclerosis.

It's my snoring, Doctor!

The captain of the legion of excuses for failing to lose weight is "But I don't eat anything, Dr!", a phrase which must be heard even more frequently that untruths from politicians. However, there may now be a new excuse on the block. In this issue of *Thorax*, Borel *et al* demonstrate a two way interaction between sleep and obesity ***(see page 735)***. Unsurprisingly, a life style intervention which led to weight reduction improved indices of sleep disordered breathing, but those with the most abnormalities actually improved less with the intervention. So should obese men not merely be screened for sleep disordered breathing, but treated for it aggressively as part of the intervention? And would such an approach lead to better weight loss, and ultimately enable respiratory support during sleep to be discontinued? Watch this space, but in the meantime expect a continued stream of the profoundly anorexic obese to fill the clinics.

Did the goalposts move for you (and not just in England penalty shoot-out)?

Is it time to abandon the acute bronchodilator response? Surprisingly little is known about its measurement characteristics even though it is widely used to identify patients with airflow obstruction who should and should not receive early treatment with corticosteroids. Two original articles ***(see pages 701 and 718)*** and an editorial ***(see page 667)*** address this important topic. The bottom line is that bronchodilator responsiveness varies considerably within patients, identifies different populations according to the criteria used to define change, does not define a population who behave differently in a clinically important way, and performs appallingly as a diagnostic test for self-reported asthma. The presence of a very large bronchodilator responses (i.e >400 ml improvement in FEV1) probably does indicate an unusually bronchodilator responsive individual but it cannot be assumed to be associated with corticosteroid responsive pathology. A raised exhaled nitric oxide or sputum eosinophil counts might be a better way to identify this characteristic. However, the inflammatory goal posts also move with time, at least in children ***(see page 675)***. Perhaps the real issue is that we often fail to consider the time domain in phenotyping—not least because making and understanding multiple longitudinal measurements is a whole heap more difficult than pontificating about single time-points.

A whiter shade of pale?

This patient's previous surgery is a clue to his present problem. The right hemithorax is a virtual whiteout, and is in fact full of fluid. The diagnosis was made before the chest was drained, thus ensuring the complexions of the medical team were not also a whiteout. Work out the diagnosis before turning to *Images in Thorax*, ***(see page 757)***