

## Highlights from this issue

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**TWO STRIKES AND YOU'RE OUT?**

The Southern Californian Childrens' Health Study (CHS) has made major contributions to our understanding of the effects of pollution on the lung health of children, and all but the most one-eyed adult physician have come to acknowledge the lifelong health effects of impaired lung growth in childhood. In this issue, the CHS suggests that both regional and near-roadway pollution affected spirometric indices in children (*see page 540, hot topic*). Of course these studies are complex and subject to differing interpretation, as an accompanying editorial points out (*see page 503*). In the ideal study, exposures would be measured on an individual rather than a community basis. However, a favourite dictum of the late, very great David Denison (*Obituary, see page 591*) was, 'if you cannot measure something very accurately, measure it a large number of times', and certainly the CHS is an impressively huge study. In the meantime, it is difficult to think of a mechanism whereby living close to a busy road is a GOOD THING. What price the rights of the child to breathe clean air as well as drink clean water? What chance anyone has the political will to tackle this?

**CATS IN THE ANTARCTIC?**

Asbestos is the Count Dracula of environmental toxins, responsible for that most feared associated condition, mesothelioma. Exposure in the work place accounts for most cases but could exposure elsewhere play a role? Lacourt and coworkers (*see page 532, editors' choice*) show in a high quality case-control study that occupational asbestos exposure is reported by 87% of male and only 64% of female cases of mesothelioma. What is responsible for the remaining cases? Is Count Dracula preying on asbestos virgins or are occult, non-occupational exposures important? Cat allergen gets everywhere, even to the South Pole, presumably on people's clothing. Could asbestos fibres be being spread the same way? Answers in future work please, but the implied belief that no asbestos exposure equals no mesothelioma is severely challenged by this manuscript.

**ANOTHER FROM THE DEPARTMENT OF NON-DIAGNOSTIC DIAGNOSES**

The old cliché that 'all that wheezes is not asthma' is gradually being replaced by 'all that is airway disease is not asthma' (or COPD for that matter), and we need to define the components of inflammation, bronchial responsiveness and fixed airflow obstruction rather than giving inhaled steroids to all as a sort of airway Mulliner's *Buck-U-Uppo* (if you are not a PG Wodehouse fan, shame on you, find one and read up on it). So is there more 'asthma' in children with sickle cell disease (SCD)? Chaudry *et al* (*see page 580*) looked for airway disease in a cohort of SCD children previously shown to have only mild pulmonary vascular disease. They showed that airflow obstruction was present in these children, but there was no increase in bronchial hyper-responsiveness or exhaled nitric oxide compared with ethnically matched controls, and there was no association with atopy. So what is this airway disease? Clearly not eosinophilic asthma, but could it be due to micro-infarcts in the bronchial wall circulation? And could early sickle complications relate to primarily a ventilatory not vascular disorder? Pure speculation, but more work needed. Meanwhile, hold the inhaled steroids unless the child has coincident genuine atopic asthma with SCD. Given recent work on the interactions between inhaled steroids and risk of infection, including tuberculosis, and given that infection can have major adverse consequences in SCD, judicious use of inhaled corticosteroids is particularly important in these children.

**REGULATE OR PERISH! OR AT LEAST GO ON WHEEZING**

Another reason for getting airway disease right is that we are entering a new treatment area. Hitherto, asthma therapy has involved painting over the cracks and playing infantile games of 'let's pretend' that it isn't there, rather than treating the underlying processes driving the disease and thus modifying natural history. Galling to those of us who are not allergists, immunotherapy is the only currently available disease modifying strategy for atopic conditions. Another potential

approach is described by John Campbell and colleagues (*see page 565*) who used a mouse model of ragweed induced allergic airways disease to show that intranasal CpG containing oligodeoxynucleotides (ODN) resulted in significant suppression of bronchoalveolar lavage eosinophilia and interleukin 4, 5 and 13 levels. After at least five treatments the effect was maintained for 13 weeks despite continued ragweed exposure. From previous work, the mechanism was thought likely to involve inhibition of allergic Th-2 immune responses mediated by stimulation of Th-1 responses via toll-like receptor 9. Interestingly, in the current manuscript, suppression of allergic airway disease was associated with induction of a regulatory T-cell response rather than signs of increased Th-1 immunity. As futuristic as time travel? Maybe, but there is evidence of efficacy of this sort of approach in patients (*see J Allergy Clin Immunol* 2013;131:866-74). Perhaps this group has achieved better regulation, a task well beyond the rarefied managerial and political echelons making a pig's ear of the silk purse that was once the NHS.

**KISS AND TELL?**

Did these 'tonsils' get lost in translation and end in the trachea? What (shown here) was caught red-handed in a 62 year old man? Work it out before turning to *Images in Thorax*, (*see page 600*).

