Highlights from this issue

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Andrew Bush, Ian Pavord, *Editors*

WE THROW DOWN TWO CHALLENGES TO THE USA

This month's *Thorax*, coinciding with the American Thoracic Society meeting, has a distinctly USA flavour, not least in the cover picture and the spelling of at least one title (***see page 511***). Americans, your articles are welcome in *Thorax*, which is only outranked by the Blue Journal amongst respiratory homes; we challenge you to send us your excellent papers. And secondly, writing in a Prozac-resistant depression after England's dismal world cup performance, anyone for cricket? (But you are not allowed to spoil the fun by practicing!)

BOLUS OR BOGUS?

Vitamin D again. A big randomised controlled trial of vitamin D supplementation in adult asthmatics showed that at baseline, Vitamin D deficiency was very common, but supplementation with big doses of Vitamin D made no difference to outcomes, and even a 'trend' could not be invoked (***see page 451;*** **Hot Topic**). Even those with the lowest Vitamin D levels at baseline, the group most likely to benefit, did not do any better. The dosing regimen used in the study could be criticised—maybe daily supplementation at lower dose would have been better—but the authors did achieve a significant improvement in Vitamin D levels in the active group. Furthermore, their regime allowed directly observed therapy, and thus almost uniquely for an asthma study (and indeed, clinical practice) adherence was assured. Despite the plethora of immunological papers, does D now stand for Dud?

A MISS MUFFET MOMENT?

Who you will remember, sat on a tuffet, eating her curds and whey. Resolution of inflammation is a biologically active process—the inflammatory process does not lose interest and disappear, but is actively switched off. WAP!! Not a Marvel comic super-hero in action, but the whey acidic protein family, characterised by one or more WAP domains (which were a novelty to your Editors]. Elafin and secretory leucocyte protease inhibitor are members of this family. Turns out WAP2 (also known as WFDC12—have we lost you yet?) may also be a significant player in the control of inflammation. Arlene Glasgow and colleagues (***see page 426***) show that WAP2/WFDC12 has anti-inflammatory and anti-protease activity *in vitro*, but more impressively, that levels of this protein are elevated in bronchoalveolar lavage fluid in patients with acute lung injury, and normals who were sufficiently foolish as to allow themselves to be challenged with lipopolysaccharide, compared to normal controls. Is this a WAP in the pan, or a really important new pathway to explore? Send more data to Thorax please, and the usual special prize to whoever draws the best WAP-man (or woman) superhero to fight inflammation in the lung.

DIAL M FOR MACROLIDE?

Perhaps the biggest advance in the treatment of airways disease since your editors have been in practice has been the use of long-term low dose macrolide antibiotics. Stories of patients putting their wheelchair on eBay and returning their oxygen are not unknown. But who is the right patient and how does treatment work? Two papers and a linked editorial from Guy Brusselle (***see page 401***) tackle these important questions. The evidence suggests that treatment is often curative in patients with diffuse panbronchiolitis, works well in many patients with cystic fibrosis and also bronchiectasis, quite well in patients with COPD and a bit in patients with non-eosinophilic asthma. Paul Corris and colleagues (***see page 442***) add lung transplant associated obliterative bronchiolitis to this list and suggest that the benefits of treatment include reversal of accelerated loss of lung function. Phil Hansbro and colleagues (***see page 458***; **editors' choice**) show in a murine model that the benefits are not solely driven by an effect on airway bacteria but also involve a macrolide-specific effect on airway immunity. We are very interested in hearing more unravelling this important topic in the form of original articles or research letters. M is definitely not for Muppet in this story!

VAGOTOMY FOR THE LUNG

Readers who are handy with the bronchoscope will be interested in Dirk-Jan Slebos and colleagues preliminary findings with targeted lung denervation (TLD) (***see page 411***). TLD therapy is delivered via a dual-cooled radiofrequency catheter which targets tissue heating to produce a narrow band of ablation around the main bronchi without damage to the inner surface of the airway. The authors suggest that is a novel bronchoscopic therapy ablates the parasympathetic innervation of the lungs and may therefore have a similar effect as anticholinergic drugs. The findings of this first in man proof of concept study provide some support for this view, showing a significant, durable and tiotropium-like beneficial effect on FEV_1, quality of life and exercise capacity. Promising findings, as with other bronchoscopic techniques for COPD, but we now need sham controlled trials large enough to clarify the risks and benefits and identify responsive subgroups. These studies would be most welcome in *Thorax*.

A BONE OF CONTENTION?

This is not the Chancellor of the Exchequer, who in the long and glorious tradition of his kind from all sides of the political spectrum and all over the world has gone from Puma to Pussycat as the election draws near, and promises riches beyond compare for all deserving causes (i.e. vote-winners). This is a lung biopsy from a 45 year old man. Work it out before you turn to *Images in Thorax*, (***see page 512***)