Highlights from this issue

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DIRTY DANCING?

Like a latter day George Orwell, David Strachan has taught us dirty is good, clean is bad, and so the hygiene hypothesis was born. So off to the barn not just for a surreptitious leg over, but also for the delivery of the baby to minimise the risk of atopic disease. However geographical differences which just do not fit with received wisdom have the potential to challenge, or, as Oliver Cromwell might have said, 'I beseech you, in the bowels of Christ, think it possible you may be mistaken' (don't try this one on when appealing an editorial decision; we never are!). From Ecuador, far from the maddening lab and in a really challenging environment in which to do research comes just such another 'doesn't fit' paper to make us think again. Cooper *et al* (***see page 232***) studied nearly 9000 urban and rural children and were unable to show the expected higher prevalence of atopic disease in the urban population. Indeed some farming exposures were associated with increased wheeze and rhinitis! What does this mean? The authors saw the expected relationship between birth order and atopic disease, a useful positive control. Congratulations to the authors, and more South American studies please —we may have globalisation, but individual differences still have a lot to teach us.

FRINGE BENEFITS?

Many years ago, life was so simple: positive sweat test cystic fibrosis (CF), negative=no CF. For most cases, the sweat test still reigns supreme. There have always been difficult cases bordering on CF, or CF-like disease but negative tests. Enter the geneticist! The initial hope was that genetic diagnosis would make all things clear and the days of the equivocal case would be gone. Not so—nearly 2000 cystic fibrosis transmembrane regulator mutations have been described, many of even less significance than the latest political promise to get tough on the tobacco industry. Through the focussed efforts of all connected with the CFTR-2 project (http://www.cftr2.org) the number of known disease causing mutations has raised from 23 to 122. For the genetically challenged, turn first to Basic Science for the Chest Physician, (***see page 295***). How has that helped? Ooi *et al* evaluated 202 patients with single organ disease compatible with CF (***see page 254***, ***Hot topic***). They found that only 17 (8.4%) were diagnosable even with this new extended gene panel. Nasal potential difference (nPD) diagnosed more patients (nearly one third). Measurements of CFTR function by sweat testing and nPD were frequently discordant in the same patient, so where does that leave us? At the end of a careful study, the authors rightly state that the diagnosis of CF may be elusive. Clearly genetics has a long way to go before we can pack up the other diagnostic tests or have done with CF diagnostic dilemmas. Back to the Jurassic ages of the Editors' student days, when clinical acumen and experience had to be used to reach a diagnosis; or better yet, never mind the diagnosis but for heavens' sake treat the patient.

IF CLOTTERS FIBROSE WHAT DO BLEEDERS DO?

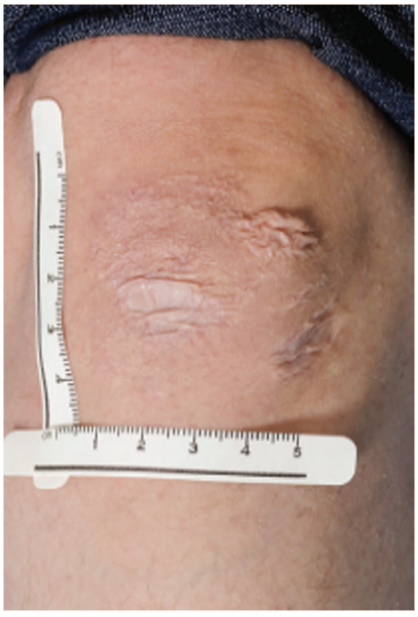
Evolution has ensured that numerous genes associated with a profibrotic state have survived through the generations. These genes might lower the risk of peripartum bleeding but they increase the risk of serious thromboembolic disease in later life, well after the selfish gene has replicated. A high quality case-control study in this issue of Thorax (***see page 207***, **Editors' choice)** suggests that an additional consequence might be a higher risk of idiopathic pulmonary fibrosis (IPF). The association between clotting abnormalities and IPF was large and it is biologically plausible, making it unlikely that Berkson's fallacy is at play. This association cannot establish chicken and egg and any potential mechanism is likely to be complex. It is notable that warfarin is not strikingly effective in IPF. However, as our editorialist rightly points out (***see page 203***), this is another piece to the puzzle of IPF. One immediate thought is if clotters fibrose, what do bleeders do? Inflame? Research in the bleeding obvious? Go into politics? The usual Thorax prize of immense prestige but less than zero monetary or aesthetic value (in this case a signed photograph of the editors) for the most entertaining answer.

GET IT OFF YOUR CHEST!

Patients often notice that colds no longer provoke asthma attacks after their asthma is controlled following the introduction of an inhaled corticosteroid. Anny Sykes and colleagues (***see page 240***, ***Hot topic***) provide some mechanistic insight by showing that the defective bronchial epithelial antiviral interferon response to viral infection previously demonstrated in asthma is not seen in patients whose asthma is well controlled. Defining control is not necessarily a straightforward matter in asthma, as we now know that control defined by symptoms and lung function is not the same as control of lower airway inflammation. The latter is more obviously associated with a reduced risk of asthma attacks suggesting a causal link between defective anti-viral immunity and airway inflammation. Or are they parallel processes, combining to make a bad situation worse like our multiplicities of NHS reforms?

KNEES UP, PROFESSOR PAVORD?

Is the appearance of this knee the result of an excess of Professorial devotions as penance for his Bullingdon club excesses? Why might there have been blood on the carpet? Work it out and then turn to the Pulmonary Puzzle (***see page 298***).



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Correction

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In the sentence 'Evolution has ensured that numerous genes associated with a profibrotic state have survived through the generations', the word 'profibrotic' should read 'prothrombotic'.

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