Highlights from this issue

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Dial D for danger?

Many of us are following the vitamin D papers with the same apprehension as we contemplate our erratic teenage sons—huge potential for future good, but how much damage may be done to the family home and the parental psyche in the meantime? In this issue of *Thorax*, we feature two contributions to the unfolding Vitamin D story. In *Basic Science for the Chest Physician*, Pfeffer and Hawrylowicz steer the non-immunologists through the complexities of the actions of 1,25(OH) D3, which is now known to be generated in far more organs than just the kidney, the nuclear Vitamin D receptor, and the end-results on the expression of genes concerned in the adaptive and immune responses (***see page 1017***). Although Vitamin D deficiency seems to have numerous adverse effects in a number of respiratory diseases, is it true that D is good but is more better? Not so, say the Southampton group, at least in pregnancy (***see page 950***); there was no relationship between maternal serum Vitamin D at 34 weeks gestation and any wheeze outcome, atopic status or lung function through to age 6 in a large group of mother-child pairs. So D would appear to be for Don't do it—Don't give vitamin D supplements to pregnant women in the hope that asthma will go away.

The natural history of COPD lung attacks. A 'must have' slide for your slide collection

All readers with an interest in COPD will want to add a copy of figure 3 in Samy Suissa and colleagues classic paper on the natural history of COPD lung attacks to their slide collection (***see page 957 and cover***). The figure shows, compellingly, an acceleration in the frequency and severity of COPD lung attacks requiring hospital admission, most obviously after the second episode. The findings are more in keeping with a step wise decline in COPD than the smooth progression often assumed. Wedzicha and Donaldson (***see page 935***) emphasise that patients presenting with recurrent COPD lung attacks require careful attention. Reassuringly outcomes seemed to be better in patients entering the cohort after 2000, perhaps reflecting an improvement in COPD management. The large effect of long-term low dose macrolide antibiotics on attack frequency (bizarrely described as 'not recommended' by GOLD—dare we suggest this because these drugs are out of patent) and the emergence of specific anti-eosinophil therapies suggest that this progress will continue although understanding the phenotype specificity of these treatments will be important in order to realise their full potential. Individualised therapy of complex airway disease is a key theme for the future (although not apparently on GOLD's radar—never mind the indication, give them everything expensive). We will be returning to this theme.

Make mine a DECAF (skinny or otherwise)

Another area where outcomes might be improved is the management of the COPD lung attack. Better risk stratification might allow clinicians to focus their efforts more effectively. Existing tools are either not designed for the purpose or are limited by their reliance on subjective measures. John Steer and colleagues (***see page 970***) have come up with a better alternative: the DECAF score, which, judging by its good performance in a large population and its catchy title should succeed. Readers may be surprised that E stands for a peripheral blood eosinophil count $<0.05\times10^9$/l, which featured as a poor prognostic marker, probably because eosinopaenia is associated with significant sepsis. There may be a wider importance in measuring the blood eosinophil count as Mona Bafadhel and colleagues (see AJRCCM 2012;186:48–55) have shown that the benefit of oral corticosteroids in the treatment of COPD lung attacks are confined to patients with an eosinophil count $>0.2\times10^9$/l and that the net risk is greatest in the rest.

Said Captain Beaky, 'Follow me!' And ran head first into a tree

Which adapted quotation should go into the consent form for randomised controlled trials of treatment in idiopathic pulmonary fibrosis, at least if our North American friends' views prevail. The alternative is to say that 'we want to give you this new treatment and see if you die more or less quickly' (true but not necessarily encouraging). In this month's *Hot off the Breath*, Wells *et al* go into battle;if a medication kills patients, few would deny it is unsafe, but progression of the disease is an acceptable surrogate for efficacy, and you should not wait for death before reaching a conclusion (***see page 938***). Admittedly you cannot argue with death, taxes or politicians (these last because of profound 8th cranial nerve shut-down), but how many efficacious treatments currently employed in many fatal disease would fail the mortality reduction test? And also, dare we say it, are *both* warring factions at fault for failing to ask the patients for their views? It is after all they who have to suffer the ravages of the disease, and in some cases (see the Panther trial) the ravages of treatment. Anyway, read all about it and respond—and the editorial PCs have been placed in explosion proof bunkers in anticipation of a vigorous correspondence.

Not the knobbliest knees competition…

which was won by an anonymous editor in chief from Leicester, but the knobbliest trachea. What caused the cough? Have a look at the figure, and then turn to (***see page 1022***). And the knees—maybe in a special Halloween issue?