



doi:10.1136/thoraxjnl-2015-207744

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A CHANGE IS IN THE AIR ... WAVES

Ian Pavord and Andy Bush brought you an “Airwaves” column which was by turns incisive, raucous and scurrilous. The Hart, Jenkins and Smyth version will start with a more sober and scientific tone but I expect we will soon warm up and in a few months the libellous invective of our predecessors will be restored. Here is our pick of highlights from the October issue.

VITAMIN D – NOT A PANACEA?

Champions of Vitamin D therapy have suggested it may prevent, cancer, fractures, relapses of multiple sclerosis and death in the elderly. A dozen Cochrane reviews have been published evaluating the proposed benefits of this panacea, with mainly negative results. In this issue of *Thorax*, Adrian Martineau and colleagues describe a cluster randomised trial of two monthly bolus doses of vitamin D to prevent respiratory infection in elderly people, living in sheltered housing (*see page 953*). Older readers – don’t rush to the chemist to buy your 2.4 mg doses of vitamin D3. The authors found that vitamin D supplements, given every 2 months, did not prevent pneumonia in their population. In fact, elderly residents receiving the supplements were more likely to get upper respiratory infections and, when they did, these lasted longer. So an effective treatment for the burgeoning supplement industry might be a regular dose of scepticism, washed down with some good evidence!

HELICOBACTER AND LUNG DISEASE – REALLY?

Barry Marshall and Robin Warren were awarded the Nobel prize for their discovery that *Helicobacter pylori* causes gastritis and peptic ulcers. Barry Marshall went to the length of infecting himself with *H. pylori* and undergoing gastric biopsy before and after, in order to prove Koch’s postulates applied to this curvaceous gram negative bacillus. Marc Sze and colleague didn’t go that far but they do present data on *H. pylori* from a large group of people with COPD (*see page 923*). In this issue of *Thorax*, they show that being

seropositive for *H. pylori* is associated with lower FEV₁ and height. However the association disappears when FEV₁ is corrected for height. In an accompanying editorial, Andrew Fogarty points out that *H. pylori* status does not determine rate of decline of lung function and so it is unlikely to be a modifiable risk factor (*see page 918*). Both *H. pylori* status and lung function may be related to environment in early childhood – emphasising yet again the childhood origins of much adult lung disease!

OCCUPATIONAL HAZARDS

In contrast to most asthma patients, individuals with occupational asthma may have attacks triggered by a specific allergen. In this issue of *Thorax*, a team from Belgium studied workers who were sensitised to latex and flour (*see page 967*). Even after allergen avoidance, persistent occupational asthma was diagnosed in two thirds of these patients, based on symptoms and histamine challenge. In this study, people with persistent asthma had myeloid dendritic cells which upregulated programmed death-ligand 2 (in response to allergen) and increased Th2-type cytokine production by CD4⁺ T cells. The authors speculate it may be possible to target this pathway to prevent asthma persistence after allergen avoidance.

THE RISE AND RISE OF “VAPERS”

Over the last few years e-cigarettes have become ubiquitous, filling hotel lobbies and hospital entrances with the incongruous aromas of bubblegum and menthol. This rise has put both the smoking cessation community and the tobacco barons on the back foot. But what of old fashioned (licensed) nicotine replacement – patches and gum? Emma Beard and colleagues present data derived from monthly surveys of over 14,000 English cigarette smokers and document a rise in “vaping” from 2% of smokers in 2011 to 21% in 2013 – after which the prevalence has plateaued (*see page 974*). The use of licensed nicotine products has declined since 2013, but at a time when e-cigarette use remained static, suggesting that users

had not switched from patch to vape. The authors blame “social contagion” for the rise in e-cigarette use.

SO WHAT EXACTLY DOES PROPHYLAXIS DO?

COPD is set to become the third leading cause of death worldwide. COPD exacerbations drive a decrease in quality of life and an increase in mortality. The Cochrane library tells us that the use of prophylactic antibiotics means that fewer patients experience exacerbations. But how does prophylaxis work and what price do we pay for long term antibiotic use? A trial from the Royal Free Hospital attempted to answer these questions by randomising patients to moxifloxacin, erythromycin or azithromycin *vs.* placebo (*see page 930*, Editors’ choice). The authors found no significant reduction in bacterial numbers by either conventional culture or 16S qPCR but increased bacterial resistance to all three antibiotics. So we conclude that bacteria are resilient little blighters and commend these authors for putting antibiotic resistance centre stage in a clinical trial. Antibiotic stewardship anyone?

LOST AND FOUND?

Images in Thorax brings you a pulmonary embolism with a twist (*see page 1007*). Make sure you take a full history or apply the anamnesis test!

