Highlights from this issue

doi:10.1136/thoraxjnl-2011-200110

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Cancer, cancer everywhere, and still tobacco is smoked (with apologies to the Ancient Mariner)

This is our first shot at a major theme issue of *Thorax*, in which we have grouped a number of papers on lung cancer. It includes a paper by Margaret Branthwaite based on her brilliant BTS summer meeting talk on assisted dying. Other themed issues will follow periodically, including long term consequences of childhood disease. Suggestions for more will be welcome.

Women on top?

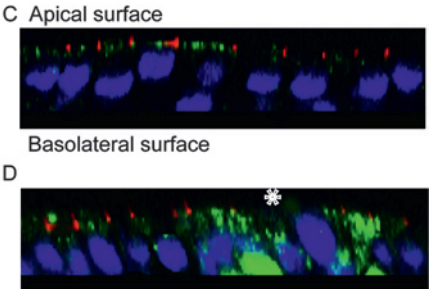
Lung cancer incidence is rising; in a huge database study Sagerup *et al* show that it is rising more than threefold more rapidly in women than men, but despite this, women do better. They are diagnosed with less advanced disease, and even after correction for stage, age at diagnosis, time period of diagnosis, and histological subtype, men have a greater risk of excess deaths. Anyone who has seen a man suffering the tortures of a trivial rhinovirus upper respiratory infection will not believe that early diagnosis is because women complain more than men. What are the mechanisms behind these data? Sex hormone related, or some other factor? Perhaps trying to understand them might open the way for new treatments? A threefold reduction in excess deaths in men would be a worthy goal for which to strive. ***See page 301***.

VOMIT syndrome

Victims of modern imaging technology (VOMIT) syndrome is epidemic. Spread initially from North America, the condition is now impacting on other westernised healthcare systems. Our typical patient is fit and healthy but frightened to their wits end by an incidental finding of a pulmonary nodule (an incidentaloma) on CT or MRI. We are seeing more and more patients because of increasing numbers of scans done to investigate suspected pulmonary embolus or coronary artery disease. Well person checks and lung cancer screening (***see page 308***) will exacerbate the problem. Almost all incidentally detected solitary pulmonary nodules turn out to be innocent. Our current approach to management, based on guidelines from the Fleischner Society, strikes us as being overly conservative, potentially unsafe, and unlikely to be feasible or affordable in sensible healthcare systems. Can we stream it down? Ashraf *et al* (***see page 315***) suggest that PET-CT and assessment of volume doubling time might help. Baldwin (***see page 275***) and Gould (***see page 277***) offer perspectives from both sides of the Atlantic. Both agree that nodules that are not growing and are not hot on PET scanning can be followed less intensively. This is a start but more work is urgently needed.

Threat or promise?

And finally for something completely different. One mechanism by which bacteria preserve their existence is the formation of biofilms, in which they lie dormant and protected from the effects of antibiotics. Biofilms are important in intravascular foreign bodies such as catheters, and also in the airways, in cystic fibrosis (CF). Chattoraj *et al* report that rhinovirus (RV) infection of CF airway cells in vitro leads to release of planktonic mucoid *Pseudomonas aeruginosa* from the biofilm, and an increased pro-inflammatory chemokine response from the airway epithelial cells. The authors speculate that this may be a mechanism by which RV causes CF lung attacks, and since an excessive inflammatory response in the CF airway is known to be deleterious under some circumstances, this may be an important mechanism to explore therapeutically. It is interesting to speculate that perhaps there may be a flip side to this: the liberated planktonic bacteria are likely more vulnerable to antibiotics than if they had kept below the parapet in their biofilm; could a new therapeutic strategy for mucoid *Pseudomonas* be programmed RV infection in conjunction with aggressive intravenous and nebulised anti-*Pseudomonal* antibiotics? We certainly need new approaches to an infection which is notoriously persistent in the airway. Perhaps one for the future! ***See page 333***.



Pseudomonas aeruginosa (green) transmigrating across epithelial cell layers in the presence of irradiated (C) and live (D) RV infection. The red stain is zona occluding-1.

A triple decker?

What was the cause of the mild interstitial inflammation seen on this transbronchial biopsy in this 19-year old asthmatic who developed pleuritic chest pain? ***See page 285***.