Highlights from this issue

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IT NOT YOUR GENES BUT HOW YOU WEAR THEM IN IPF THAT'S IMPORTANT

The genes you own are determined at birth but their expression, and the proteins that they make, can change over time. These epigenetic changes are thought to be responsible for a number of diseases associated with aging. In this issue of Thorax Korfei and colleagues describe how changes in key molecules associated with gene expression, the HDAC family, lead to cellular features associated with fibrosis in both epithelial cells and fibroblasts, providing evidence to pursue investigation targeting this family of molecules as a potential therapy for IPF (***see page 1022***). May be it really is time to try a new style of genes?

AFTER ALL THAT, LUNG CANCER REALLY IS ALL IN THE GENES?

30 years ago Professor Geoffrey Rose famously wrote in his essay *Sick Individuals and Sick Populations* that 'if everybody smoked 20 cigarettes a day then lung cancer would be a genetic disease'. It turns out he was both right and wrong. In a new epidemiological study using US registry data of over 5500 lung cancer cases assessing the genetic contribution to lung cancer Carr and colleagues found that patients who don't smoke have the strongest evidence for a genetic contribution to lung cancer, which was also observed in relatives of patients with smoking related lung cancer suggesting predisposition genes in smokers confer the highest risk (***see page 1033***). So Professor Rose was half-right, lung cancer is a genetic disease but even if you don't smoke.

AND SO ARE COPD EXACERBATIONS?

It's been known for a while that alpha 1 anti-trypsin deficiency is associated with increased exacerbation of COPD and further evidence for this is provided in the study by Ingebrigtsen an co-workers (***see page 1014***). However they showed no association between genotype and the observed levels of acute phase markers fibrinogen and alpha1 anti-trypsin in serum. Clearly the interaction between acute phase proteins and genotype is somewhat more complicated than many may have thought and determining what is leading to increased 'risk' of exacerbations may still be a little way off.

IT'S THE ENVIRONMENT STUPID!

In Tom Lehrer's Periodic Table song Indium comes between Proctactinium and Gallium, and like Gallium it can be radiolabelled for use in nuclear medicine studies. It is generally considered non-toxic, with the EU not considering it a chemical of "high concern" and in the latest version of the Periodic Table song Indium is associated with Tin-cans. Its use has increased dramatically in recent years through TV monitor production. The study by Amata and colleagues carefully assesses the lung function, radiology and serum levels of indium and other biomarkers of alveolar injury in a cohort of exposed workers (***see page 1040***, Editors' choice). They conclude that indium inhalation may promote emphysema and reducing exposure can reduce interstitial injury. Maybe its time to re-write the Periodic table song once more to include the detrimental health effects of 'cute' elements.

NO MORE NEED TO ENJOY THE SILENCE?

Hearing loss is a major side effect of aminoglycoside antibiotics especially when prescribed long term for conditions such as multidrug-resistant TB. Dr Kranzer and colleagues performed a systemic review which identified three studies of co-administration of N-Acetylcysteine (NAC) with long-term aminoglycosides which appears to be protective whilst studies of long term NAC on its own has an increased risk of gastrointestinal disturbance (***see page 1070***). The authors conclude that a prospective a trial should be undertaken to ensure a policy of truth in preventing aminoglycoside toxicity.

YOU JUST CAN'T KEEP A GOOD EDITOR DOWN

Only weeks after handing over the reigns of Thorax to a new mob Professor Bush is busy writing his own stuff (***see page 1078***). The chILD-EU protocols for diagnosis and initial treatment of Interstitial Lung Disease in children will be a valuable resource for paediatricians around the world.

AND FINALLY WE MUST GO TO SLEEP…

The diagnosis of obstructive sleep apnoea (OSA) can be made with limited respiratory polygraphy using portable monitoring, and full polysomnography (PSG) with electroencephalograhy (EEG) viewed as the gold standard. Each of these approaches has their clinical, and technical, advantages and limitations. Vat *et al* have provided a scoring system using portable monitoring, measuring 3% oxygen desaturation index and pulse wave amplitude, and compared with PSG with EEG (***see page 1047***). We can now simplify the approach to the diagnosis of mild, moderate and severe OSA without the need for EEG recordings. Once we have made the diagnosis of OSA, is it important to know if OSA occurs during rapid eye movement (REM) sleep or not. According to the data from Wisconsin Sleep Cohort, if OSA occurs during REM then there is a higher risk systolic non-dipping and diastolic non-dipping blood pressure during OSA and this could have important clinical implications in terms to cardiovascular risk stratification (***see page 1062***). And finally, how do we get the patients to adhere to continuous positive airway pressure (CPAP) treatment. Does the use of telemedicine have a role? Is this better than face-to-face consultation? The Spanish Sleep Network have shown telemedicine for the follow-up of patients on CPAP was as effective, in terms of CPAP adherence and symptom control, and at a lower cost (***see page 1054***). Indeed, the Spanish Sleep Network may really be the Skynet Network as the machines are to start to take over the world…don't worry we'll be back!