Highlights from this issue

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YOU SCHMOOZE, YOU DON'T LOSE (IF YOU ARE THE TOBACCO INDUSTRY)

In this issue, we co-publish an editorial (***see page 1090***) setting out an even more stringent policy with regard to tobacco funded research—namely, don't bother us with it under any circumstances; Goebbels has a better chance of publishing in the *Jewish Chronicle*. However, it's good to record that, at a time when Britain wants to leave the EU because they are out of step with our thinking on human rights and other issues, we are as one on tobacco control. So the EU parliament have courageously opted to phase out menthol cigarettes over 8 years not three; have heroically arm-wrestled the tobacco industry into agreeing to the industry's own proposal on the size of health warnings on cigarette packets; and rejected the idea that e-cigarettes should be subject to the same controls as nicotine patches. The EU Tobacco commissioner, speaking from his offices in Never Never Land, has hailed this as 'positive'. An announcement from the same source, that limiting wife beating to no more often than thrice daily, is also positive is imminently expected. Never has more than €1 million (reportedly spent by Philip Morris in lobbying European MPs) been better used. Trebles and lung cancer all round!

YOU'LL FIND THEM HERE, YOU'LL FIND THEM THERE

What have short ribs, progressive blindness, renal cysts and (to make it easy) infertility in common with the molar tooth sign (which is nothing to do with the dentist—ask your registrar!)? Answer: all are related to ciliopathy. Is pulmonary fibrosis also a ciliopathy? Primary cilia are not implicated in primary ciliary dyskinesia (sorry; not our fault), but are ubiquitous, usually one per cell, and are a powerhouse of chemosensory and signalling functions. These include Sonic Hedgehog and Hippo (how come developmental biologists choose the best names?). Would people care more about COPD if it was called Sonic Hedgehog syndrome? Be that as it may, Yang *et al* showed that expression of genes found in both primary and motile cilia were associated with increased honeycombing in Idiopathic pulmonary fibrosis (***see page 1114***); importantly this finding was validated in a second cohort. This is really interesting: cystic kidney and liver disease are also non-motile ciliopathies. Are primary cilia genes driving cyst formation in the lungs? Challengingly, dynein arm genes are implicated—what does that say? Bingle *et al* extend these observations in correspondence arising from this work and introduce another catchy name by showing that *BPIFB1/LPLUNC1* (which sounds like a good double bass player at a Jazz festival; much better that the rather more feeble alternative name *C20orf114*) is expressed in honeycombing in IPF, but not hypersensitivity pneumonitis (***see page 1167***). In an accompanying editorial (***see page 1088***), Sarah Wiscombe and colleagues review the basic science of ciliary signalling and challenge the IPF community to take this research to the real 'so what?' places and change outcomes. So, will IPF doctors rock around the clock to the (ciliary) beat of *LPLUNC*, or snooze and lose?

INTERSTITIAL LUNG DISEASE: A CANDIDATE FOR MICROBIAL COMMUNITY CARE?

Another interesting study looks at microbial communities in healthy controls and patients with a variety of interstitial lung diseases assessed using molecular methods (***see page 1150***, **Hot Topic**). There is some interest in this area, not least because of the striking findings with long-term treatment with cotrimoxazole in idiopathic pulmonary fibrosis reported in this journal. Microbial communities were assessed in the upper and lower airway, the latter using bronchoalveolar lavage. No obvious differences were seen. The study was limited to small numbers of patients studied during exacerbations but they do establish that this work can be done and provide a strong basis for bigger more definitive studies.

THE POWER AND THE LACK OF GLORY

We have previously highlighted the worrying increase in a number of respiratory infections in patients treated with high dose inhaled corticosteroids (ICS). Here we publish a report that ICS increases TB risk in a moderate TB prevalence region (***see page 1105***, **Editors' Choice**). The authors did a nice job calculating dose equivalence of the various inhalers prescribed and used the data to show a dose response effect for both asthma and COPD. Unfortunately they were not able to report comparative safety related to the topical potency of the ICS prescribed. These are really worrying data, and underscore the need to be really sure your patient needs potent ICS before you prescribe them; potentially they divert money from more useful medications in developing world contexts, as well as actually doing harm. Sabroe *et al* (***see page 1085***) nicely summarise the immunological issues, the pros and cons of the use of ICS and poke fun at the egregious clichés beloved of immunologists in their editorial. ICS should never be used as an 'airway tonic', what PG Wodehouse would describe as an airway Mulliner's Buck-U-Uppo, but only when there is a clear indication to treat, with an understanding that ICS can cause airway immunosuppression just as surely as systemic steroids cause systemic immunosuppression and as surely as European politicians will not take tough decisions on tobacco.

I'M DREAMING, OF A SAD CHRISTMAS

Which was reality for a Welsh (clue!) family. Was this bird the hero or zero? Who consulted which diagnostic website? See the front cover for a further clue, and the *Pulmonary Puzzle* for the answer (***see page 1175***).