Highlights from this issue

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CLARITY IN THE MURKY WATERS OF STATINS AND COPD LUNG ATTACKS

The 5794 patient analysis of the Copenhagen lung study showing that statin use is associated with a reduced risk of COPD lung attacks (***see page 33***) is a much better paper as a result of an effective collaboration between the authors and reviewers (next stop, collaboration between the England and Scotland football teams). The manuscript arrived at *Thorax* at about the same time as the authors of the STRATCOPE study (*NEJM* 2014;370:2201–10) reported that simvastatin treatment had no effect on the number of COPD lung attacks in patients who had severe airways disease but no evidence of or risk factors for cardiovascular disease. The key remaining question, well-articulated in a *Hot off the Breath* (*Thorax* 2014;69:891–4), is whether this result is generalisable to the wider population of patients with COPD. The reviewers and authors saw an opportunity to answer this question so the latter rebooted their computer and got back to work. The revised analysis confirmed no benefit of statin treatment in the highly selected subgroup of patients with pure airways disease but a clear and clinically important beneficial effect in the rest. CRP looks like a promising biomarker for a statin response and might identify a subgroup of patients with 'systemic COPD' who should receive treatment outside current recommendations.

MORE MURKY WATERS—BUT NO POLITICAL CLARITY

The unborn baby floats blissfully in a bag of warm water, but how pure is its environment? We know that pollution is bad for children's lungs, so it is unsurprising and very worrying that exposure of the pregnant woman to environmental pollution has long term effects on her unborn child's lungs. Morales *et al* (***see page 64, Hot topic***) measured environmental benzene and nitrogen dioxide during pregnancy, and showed that levels of both in the second trimester of pregnancy were inversely related to spirometry at age 4.5 years in 620 children from their birth cohort. This means that the dead hand of antenatal pollution will affect these children for life. Peter Sly (***see page 3***) highlights the public health implications—we must have regulation and legislation to protect our unborn babies. Do we really need sources of pollution in residential areas? See that large pink mammal flying past the window—don't hold your breath for strong political action.

STRATIFYING IPF: 19TH OR 21ST CENTURY?

We have lagged behind our organ-based speciality cousins in new drug discovery. In our defence, the lung response to insults is perplexingly heterogeneous, not only between and within individuals, but also within the lung. As a result traditional physiological and histopathological-based classification systems fail to identify discrete, treatable mechanisms; and it is unsurprising that current 19th century approaches have yielded 19th century results. Why do we cling to this approach like an infant to a security blanket? Progress in airway diseases has required us to think differently about patterns of disease and embrace less traditional markers to stratify patients. A team led by Joseph Arron (also a pioneer in airways disease biomarker research) think this approach may be applicable in IPF (***see page 48***). They identify two groups of patients with IPF and a poor prognosis differing markedly in blood CXCL13 and MMP-3 levels. These systemic biomarkers were found to reflect the contribution of lymphoid aggregates (CXCL13) and bronchiolisation (MMP-3) across total lung tissue. Gisli Jenkins (***see page 9***) is enthusiatric about this approach but points out that the key question is whether these biomarkers have clinical utility as treatment targets and/or prognostic markers.

GOING UP IN THE WORLD (OR IN THE AIRWAY)

The united airway is not a concept that appeals to either of your editors, but in this edition we have at least risen above the carina. Exercise induced laryngeal obstruction (EILO – what?) is the subject of an original article, a lesson of the month and an editorial. Penny in the slot thinking is that noisy breathing on exercise is wheeze is due to exercise induced bronchoconstriction (EIB). Johansson *et al* (***see page 57***, ***Editor choice***) carried out a cross-sectional questionnaire survey of nearly 4000 adolescents, and standardised exercise tests in a selected sample of nearly 150, and showed that, in those breathless on exercise, 39.8% had EIB, 6% had EILO and 4.8% had both. Presumably the rest were just unfit? An editorial from Jim Hull (***see page 7***) highlights that inhalers were grossly over prescribed in this group, and also that a key feature of EILO is that it comes on *during*, not after exercise, in contrast to EIB. CLE means congenital lobar emphysema to the paediatrician but continuous laryngoscopy on exercise to the EILO-ologist, and the *Lesson of the Month* (***see page 95***) is the first description of EILO in two elite female rowers. Nought out of ten for enterprise however; the authors used a laboratory rowing machine, instead of rowing alongside the women during a race. So what do we learn? Most adolescents who are breathless on exercise do not need inhalers, think EILO as well as EIB, and Jim Hull wimps out of serious exercise.

OH WHAT A TANGLED WEB WE WEAVE…

Images in Thorax contributes part 2 of the upper airway theme. An 18 year old with a complex metabolic disorder became breathless. Measure arterial pH, look at the CT reconstruction or give up and turn to ***page 101***.