Highlights from this issue

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Small is NOT beautiful in 2014: a candidate biomarker of the year? Having just awarded the peripheral blood eosinophil count *Thorax*'s biomarker of the year for 2013 we are now presented with another often ignored peripheral blood cell component as a potential candidate for the 2014 award. Michelle Harrison and colleagues (***see page 609***) show in a retrospective analysis of 1343 patients hospitalised with COPD lung attacks that a raised blood platelet count is an independent predictor of mortality over the following year. This might be expected to be due to increased vascular deaths, or secondary to the known association between ventilatory failure and thrombocytosis. However, careful analysis suggested a more complex and interesting link between thrombocytosis and mortality, perhaps involving chronic inflammation. What is particularly noteworthy about this study is that patients taking anti-platelet therapy with aspirin or clopidogrel had reduced mortality suggesting that the platelet count might be a biomarker of risk that can be reduced – and any biomarker which does not lead to clinical action and benefit cannot enter our competition. The authors are appropriately cautious about their findings and call for prospective randomised controlled trials. Don Sin agrees (***see page 603***) but also emphasises the biological plausibility of the reported findings and suggested mechanism.

Oh I do like to be beside the seaside! Not to be outdone by platelets and eosinophils, the neutrophil also gets an outing this month. Charlotte Summers and colleagues (***see page 623***, **Editors' choice; Editorial,** ***see page 606***) use radio-labelled peripheral blood neutrophils and γ-scintigraphy to show that the healthy human lung de-primes activated neutrophils and that failure of this process in ARDS might explain neutrophil-mediated damage at remote sites. We are delighted to have such an elegant demonstration of normal homeostasis and disease pathogenesis in the pages of *Thorax*. We were particularly thrilled with the following quote from Andrewes (*Lancet* 1910;175:1737–800), used by the authors in their response to a reviewer's comment: "it may be that if the bone marrow is the birthplace of these cells and the spleen their ultimate tomb, while the blood is their means of transit, the lung may serve as a weekend at the seaside, where they may recuperate their energies. I cannot otherwise explain the accumulation of polynuclear cells in the pulmonary tissue." If your paper is not on PubMed it is normally more airbrushed out of history than any Stalinist purge victim, so congratulations to the authors for actually reading such a paper and confirming Andrewes' suspicions after more than 100 years. Good luck in the search for the mechanism and the usual unappetising *Thorax* special prize to anyone who can quote a relevant paper older than this one.

The dark side of the macrophage Black carbon particles in airway macrophages are known to be a *bad thing* and associated with impaired spirometry in children (*N Engl J Med* 2006;355:21–30). Now Jonathan Grigg's group report that black carbon particles are *scarcer* in the alveolar macrophages of children with severe compared with mild asthma and normal controls (***see page 654***, **see cover).** This does not mean that all the severe asthmatics came from the unpolluted countryside, but it's another piece of evidence that macrophage function is impaired in severe asthma. They went on to show that there was an inverse correlation between black carbon and urinary metabolites of prostaglandin (PG) D_2 and E_2, and that PGE_2 suppressed carbon particle phagocytosis by human and rat alveolar macrophages *in vitro*. Is alveolar macrophage dysfunction cause or consequence of asthma? Either way, the empire, or rather the macrophage, strikes back against the peripheral blood biomarkers so lauded above; could failure of phagocytosis lead to epithelial injury and a subsequent portal of entry for allergens?

'A paradox, a paradox, a most ingenious paradox' So sang the Pirate King to young Frederick in the *Pirates of Penzance*, and rather more discordantly, Professor Pavord to anyone who would listen at the Bullingdon club. A paradox so ingenious that it continues to defy an explanation is the very low prevalence of atopic disease in the offspring of Friesian Dairy farmers. Being born in a barn may have an attraction after all. But does it work through aeroallergen exposure, drinking unpasteurised milk (today's *Thorax* Trivial Pursuit fact: there are more bacteria in pasteurised milk than unpasteurised), exposure to a diverse microbiological flora or what? Robbe *et al* (***see page 630***, **Hot topic**) prepared dust extracts from cattle and pig farms and bulb and onion industries (what a great task to be allocated!). They then exposed the mice to combinations of the extracts and house dust mite. They reported on airway inflammation and T-cell polarisation, seeking confirmation of the latter in human farm workers. In the mice, the dust extracts induced interleukin (IL)-17, IL-1β and IL-6, with a corresponding pulmonary neutrophilia. The extracts however protected against Th2 responses and methacholine bronchial hyper-responsiveness. Occupationally exposed humans had more peripheral blood IL-17 and interferon gamma expressing cells than controls. So dust induces a Th/Tc-17 (yes, another acronym for the immunologically intellectually challenged to master!) inflammatory response. The good news: this may potentially protect against atopic disease. The bad news: it could increase susceptibility to other respiratory diseases. The worst news: no women included in the study – so their Athena Swan award is downgraded to cardboard.

Searching in vein? This a not nightmare vision of something in Son of Arachnophobia or a piece of knitting that went badly wrong, but a reconstruction from a chest CT of a 56 year-old women with a pelvic tumour. What is it and what to do? When you haven't worked it out, turn to *Images in Thorax*, (***see page 689***).