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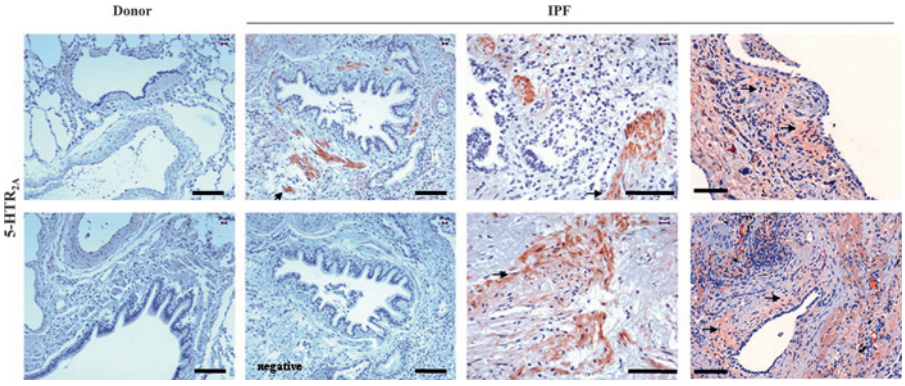
The naked emperor

One of the great religious divides in the world of cystic fibrosis (CF) is the use of serological tests to diagnose early infection with *Pseudomonas aeruginosa*. Two large Australasian groups have combined to deliver what should be the death blow to *Pseudomonas* serology. The AREST-CF cohort (annual bronchoscopy as a routine) was the discovery population, and the ACFBAL group (testing whether bronchoscopic guided treatment adds value to conventional therapy) was the validation set. Both commercial and in-house serological tests were used. The answer in both cohorts: serology has good negative predictive values, but is less good than flipping a coin when it comes to positive prediction of early infection. The tests are good for confirming the blindingly obvious chronic infected state. No doubt as in all debates, some will find a way of proving that green is red and that the data really show it is a terrifically useful test, but most will now give a decent burial to *Pseudomonas* serology. Congratulations to two great groups on combining to provide the nails for the coffin. ***See page 978.***

Serotonin and pulmonary fibrosis

The paper by Melanie Königshoff *et al* in this issue of *Thorax* (***see page 949***) is an example of first class bench to bedside research. The authors have carried out a logical series of studies on lung biopsy samples from well characterised patients, human lung fibroblasts in vitro and laboratory animals. Their findings provide a compelling case for a role for serotonin acting through the 5-HTR_{2A} and 5-HTR_{2B} receptors in the pathogenesis of pulmonary fibrosis. This receptor family is already known to be involved in heart valve fibrosis induced by the anorexic agent fenfluramine and sometimes seen in patients with carcinoid syndrome. Terguride, an antagonist of both receptors, is already available and used to treat ovulatory failure in patients with hyperprolactinaemia. This agent effectively inhibited collagen production and impaired lung function induced by bleomycin in a mouse model, probably by inhibiting transforming growth factor β1- or WNT3a-induced collagen production. In a linked editorial (***see page 946***) Fabre and Crestani ask whether the time has come for clinical trials. We say why not. Our patients say soon please, we're desperate.



Expression and localisation of the serotonin (5-HT) receptor 5HTR_{2A} in lung tissue from controls (transplant donors) and patients with idiopathic pulmonary fibrosis (IPF). ***See page 951***

COPD co-morbidity

The ability to link and pool computerised data from primary care databases and use it to test hypotheses in large well characterised populations has been an important recent advance. Databases from UK primary care practices have been particularly valuable since the populations are large and the data is thought to be of high quality. In this issue of *Thorax* (***see page 956***) Feary *et al* have used data from the 1 200 000 patients in the health improvement network (THIN) to investigate the relationship between COPD and cardiovascular disease, cerebrovascular disease and diabetes. These conditions were respectively 5, 3.3 and 2 times more common in patients with COPD. Young patients with COPD were particularly likely to have cardiovascular disease and the association was, to a large extent, independent of smoking history. The analysis was compromised by the absence of spirometric confirmation of COPD in 38% (we must do better than this). Even so, a staggering 40% of patients with a diagnosis of COPD had one of more of these co morbid factors. The findings emphasise the importance of an integrated, general medical approach to patients with COPD. They also point towards common pathogenic pathways, perhaps involving systemic inflammation and birth factors.