Airwaves

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Action not words, please!

Does Respiratory Syncytial Virus (RSV) cause asthma, or is merely a marker for an asthmatic tendency. The sceptics point to the impaired pre-morbid lung function and the immunological changes in cord blood preceding RSV infection, and the Tucson data showing that post-RSV symptoms gradually recede as time goes by. The true believers principally cite the meticulously conducted follow up studies led by Dr Nele Sigurs, showing a high prevalence of asthma in the years following severe RSV infection. We here feature another instalment of the Tucson versus Sweden controversy. The original Sigurs cohort has been restudied with almost 100% retention, an amazing feat. The previously reported high prevalence of allergic asthma was shown to persist to age 18. In an accompanying editorial, Stein and Martinez speculate that early RSV and later asthma are both manifestations of an underlying innate immune dysregulation. How much longer do we have to endure words not action? The only way to settle an issue of enormous public health importance is to randomise high risk infants to the anti-RSV monoclonal or placebo, and follow them up to see if (a) RSV infection is prevented, and (b) whether asthma is prevented. The study would be costly, but only a fraction of the price of the umpteen NHS reorganisations we have all endured. ***See pages 1033 and 1045***

Just when you thought it was safe not to vaccinate…

Were we all starting to think that TB is gone, if not forgotten; routine BCG vaccination has gone, and only those perceived to be at high risk were to be offered immunisation. Here we publish a stark reminder that the risks may not be as low as was thought. A white teacher ('only ethnic minorities get TB') was found to be an asymptomatic case of smear positive TB, after 9 months working in a Nursery. More than 250 children and adults were screened, 12 cases of active TB (9 in children) were identified, and 42 (30 children) latent TB were found. Some but not all the children had had BCG, and the calculated protective efficacy of the vaccine was 66%. A second paper and an editorial draw attention to the fact that BCG may do more than protect against infection, but may also be beneficial in increasing treatment success in established disease, as well as effects on other diseases. So the question posed by these data: should we routinely immunise *all* newborns with BCG? Given that TB can rear its ugly head anywhere; given that Eriksen *et al* have shown highly significant protection against TB infection in children (both clinically as well as statistically important); given that young children are most prone to the devastating effects of disseminated TB, especially tuberculous meningitis; how many parents would opt to have their newborn immunised given the choice? Is it not time for a re-think about BCG, including involving the public in this debate (not very fashionable in today's politics, we know)? ***See pages 1036, 1067 and 1072***

Suffer, unborn children

When do you get allergic? Probably not when you first meet allergens in the first months and years of life, important though that time is, but more likely the baby sets off on the "allergic crawl" even before birth. Previous work from Perth, Australia has suggested that a rapid weight gain in the first year of life is associated with a reduced gain in lung function and more wheeze. Now, the spotlight turns on antenatal events. Pike *et al* measured both ante- and postnatal growth, and showed that rapid growth trajectory in the 11th to 19th week of pregnancy followed by late gestation attenuation of growth was associated with post-natal atopy. Lower early foetal growth rate was associated with non-atopic wheeze. The implications are twofold. Firstly, the Perth observations may after all be accounted for by antenatal events. Secondly, this is yet more evidence of the importance of getting very early health right. The Children's Commissioner, Professor Sir Albert Aynsley-Green, is forever wasting his sweetness on the desert air with regard to the inadequacy of Children's Services in the UK. When will we start to invest properly in securing the long term lung health of our children? ***See page 1099***

Surrogates of surrogates

The prevailing view is that a high exhaled nitric oxide (FE_{NO}) reflects eosinophilic airway inflammation which is itself a surrogate marker of corticosteroid responsive disease. However, the cross sectional relationship between FE_{NO} and markers of eosinophilic airway inflammation is not close and interventional studies with specific inhibitors of eosinophilic airway inflammation show that they can be modified independently. A further concern, highlighted by Scheich *et al* is that the relationship between FE_{NO} and the induced sputum eosinophil count is modified by demographic characteristics, notably the intensity of inhaled corticosteroid treatment. The key question is does this matter? In a linked editorial Taylor & Cowan argue not. They suggest that we should move away from assessing a surrogate marker of a surrogate and towards a position where FE_{NO} is seen as a non-invasive marker of corticosteroid responsive disease. In this respect FE_{NO} performs much better than traditional indicators such as the pattern of symptoms, demographic variables and lung function abnormalities. The high negative predictive value of a low FE_{NO} is likely to be particularly valuable. We suggest that this additional information is particularly important at two points in the management of inflammatory airway disease: the decision to initiate what is likely to be long-term treatment with inhaled corticosteroids; and the decision to escalate treatment to high dose inhaled or oral corticosteroid therapy. ***See page 1039 and 1031***

Not very sweet?

A 70-year-old man presented with a 2-month history of cough and bloodstained sputum. This is the bronchoscopic appearance and endobronchial biopsy prior to treatment. What is going on, and what would you do? ***See Images in Thorax, page 1119***