airwaves

Wisia Wedzicha, *Editor in Chief*

LUNG FUNCTION DECLINE IN ASTHMA

Although it is recognised that asthmatic subjects have an accelerated decline in lung function, factors affecting the long term outcome of asthma are still relatively unknown. In this issue of *Thorax* we report two studies and publish an editorial on this topic. Lange and colleagues describe the 10 year follow up of two groups of asthmatic subjects, one of which was treated with inhaled corticosteroids (ICS). After adjustment for other variables, treatment with ICS was associated with a significant reduction in the decline in forced expiratory volume in 1 second (FEV_1), with a decline of 51 ml/year in patients not receiving treatment with ICS and 25 ml/year in the ICS group. In his editorial Ernst discusses the implications and limitations of this study and suggests that, although the effects of ICS on disease progression were evident in the group as a whole, the benefit may vary substantially between asthmatic individuals. Dijkstra and colleagues report an interesting 23 year follow up study of asthmatic subjects in which they found that ICS reduced disease progression in men who smoked <5 pack years, but this effect was not seen in men who smoked more heavily or, indeed, in women. Cigarette smoking therefore interferes with the long term action of ICS in asthmatics. The authors also discuss some of the reasons for the different response in women—an issue for further study.

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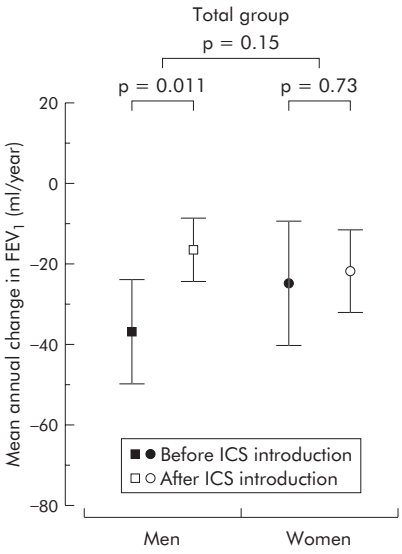

IMPROVED NATURAL HISTORY OF CF

Lung function decline is an important predictor of death in cystic fibrosis (CF), and the decline in forced expiratory volume in 1 second (FEV_1) is a commonly used outcome measure. However, with recent advances in treatment, one would expect the natural history of the disease to have improved. In this month's *Thorax* Que and colleagues report on the decline in FEV_1 in five successive birth cohorts of CF patients followed at the Royal Brompton Hospital, London, UK. The results show that the annual rate of decline in lung function among current patients with CF is lower than that in previous generations of adults with CF. The authors suggest that the decline in FEV_1 may now be too insensitive for use in shorter clinical trials in CF.

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SMOKING CESSATION AND AIRWAY INFLAMMATION

Patients with COPD who stop smoking have fewer respiratory symptoms and reduced disease progression, although previous studies have shown that airway inflammation persists despite smoking cessation. In this issue of *Thorax* Lapperre and colleagues describe a study of smoking cessation and its duration on airway inflammation in patients with COPD at GOLD stages 2 and 3. They show that short term ex-smokers have more CD3+, CD4+, and plasma cells than current smokers, while long term ex-smokers have fewer CD8+ cells and more plasma cells. In the accompanying editorial Hogg discusses the importance of this study which was conducted in patients with more severe COPD in whom there is currently little information on smoking cessation. He also concludes that the mechanisms of persistence of the adaptive immune response after smoking cessation in severe COPD require further study.

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FARM DUST, ALLERGY AND ASTHMA

The "hygiene hypothesis" suggests that the recent increases observed in allergic diseases are associated with the microbial contact of children in early life. Children living on a farm have been shown to have fewer asthma and allergic disorders. In this issue of *Thorax* Peters and colleagues report an interesting study in which stable dust was collected from 30 farms in rural areas of the Alps and formed into an extract that was then administered to a mouse model of allergic asthma. Inhalation of the extract inhibited airway eosinophilia, hyperresponsiveness, cytokine responses, and antigen specific IgG_1 and IgE. The authors conclude that farm dust contains immune modulating agents that can interfere with both cellular and humoral immunity in a mouse model of asthma. They suggest that further work is needed to find out the nature and mode of action of the active factors.

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RISK IN PiMZ

Severe α_1-antitrypsin (AAT) deficiency is due to homozygosity for the protease inhibitor (Pi) Z allele (PiZZ), but low AAT levels are also found in subjects heterozygous for the Z allele (PiMZ). However, the relation between PiMZ and the development of COPD is not clear. Malerba and colleagues report on a study of induced sputum in PiMZ subjects with normal pulmonary function. The authors found that this PiMZ group had increased neutrophilic inflammation similar to that found in patients with stable COPD. The results suggest that PiMZ subjects are indeed at an increased risk of obstructive lung disease.

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