airwaves

Wisia Wedzicha, *Editor in Chief*

DOES CADMIUM CAUSE COPD?

Cigarette smokers have variable susceptibility to COPD and the reasons for these differences are being extensively investigated. Cadmium is found in cigarette smoke and has been linked to emphysema in workers exposed to it. In this month's *Thorax* Mannino and colleagues report some intriguing data from the Third National Health and Nutrition Examination Survey. They found that urinary cadmium levels were higher in current smokers than in former smokers or never smokers. The cadmium levels in current and former smokers were also associated with lower lung function. The authors conclude that urinary cadmium levels, which reflect the total body burden of cadmium, may be a cause of tobacco related lung disease. In the accompanying editorial, Hendrick explains how cadmium is metabolised in the body and also discusses the case for and against cadmium in tobacco smoke being a cause of emphysema. Differences in susceptibility to the development of COPD may be due to variation in metabolic pathways related to cadmium. Further longitudinal data are now required to assess the relation between lung cadmium levels and decline in lung function.

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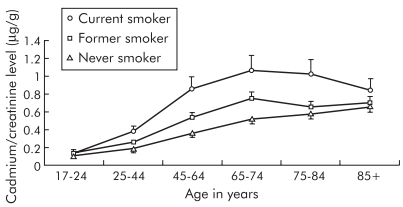

Geometric mean creatinine adjusted urine cadmium levels stratified by smoking status. From the Third National Health and Nutrition Examination Survey, 1988–94

NEW COPD GUIDELINES: NICE ONES

This issue of *Thorax* is accompanied by a supplement in which we publish the long awaited COPD guidelines developed by the National Institute for Clinical Excellence (NICE) Collaborating Centre for Chronic Conditions based at the Royal College of Physicians, London, UK. In the accompanying editorial Halpin, the Chair of the Guideline Development Group (GDG), describes the key priorities in the guidelines, new evidence that has been used to update the recommendations, and some controversial areas. An important feature of the NICE COPD guideline development was that extensive literature searching and professional systematic reviewing were used to determine the evidence base. In this issue I also describe the formal review process established by NICE which was followed during the development of the guideline. The COPD guidelines contain a number of important recommendations; now we all need to ensure that they are implemented into our clinical practice to optimise the care of patients with COPD.

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PATIENTS WITH CF DO BETTER IN HOSPITAL?

Infective exacerbations are a feature of cystic fibrosis and lead to decline in lung function. These episodes are treated with intravenous antibiotics which may be administered at home or in hospital. Thornton and colleagues describe the longer term outcome of antibiotic therapy in cystic fibrosis and show that, over a year of treatment, patients treated in hospital have a better outcome than those treated at home, as shown by spirometric testing. The authors discuss the reasons for these differences and suggest that it is likely that adherence to treatment is suboptimal at home. Although there is a need for more home therapies, this study concludes that closer supervision is required during home treatment for cystic fibrosis.

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HOPE FOR TREATMENT OF PULMONARY FIBROSIS

Treatment of pulmonary fibrosis is still difficult and the condition has a poor prognosis. There has been some recent interest in the role of heme oxygenase (HO) in pulmonary fibrosis, and the generation of free iron and carbon monoxide by HO may produce oxidant induced lung damage. In this issue of *Thorax* Atzori and colleagues describe a study of the effect of an HO inhibitor on the development of pulmonary fibrosis in the bleomycin mouse lung model. They found that use of the HO inhibitor resulted in a reduction in lung collagen, alveolar cytoprotection, and less fibrosis. Alteration of the oxidant/antioxidant balance in the lung may therefore be a promising advance in the treatment of pulmonary fibrosis.

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POLLUTION AND AIRWAY INFLAMMATION

Associations have been described between ambient pollution and excess morbidity and mortality from cardiovascular and respiratory disease, but the mechanisms involved are largely unknown. Airway inflammation caused by pollution may be the key factor which leads to systemic inflammation and thus vascular disease. In this month's *Thorax* Adamkiewicz and colleagues report an association between ambient pollution and airway inflammation as measured by the exhaled nitric oxide (NO) level in the elderly. This relationship was also stronger where there was a diagnosis of COPD. Exhaled NO may be a useful tool to evaluate further the relation between pollution, airway inflammation, and cardiorespiratory disease.

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