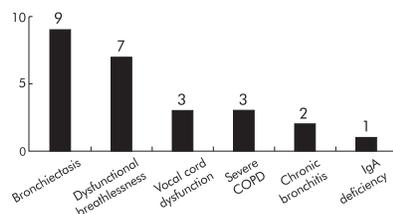


## HOW TO IDENTIFY DIFFICULT ASTHMA?

In this issue of *Thorax* Heaney and colleagues describe a detailed evaluation of patients with therapy resistant asthma (TRA). A cohort of asthma patients was systematically evaluated and a group identified with TRA. The objective was to identify factors that predicted the prognosis of patients with difficult asthma. Considerable co-morbidity such as oesophageal reflux and psychological problems were found in both groups of asthmatics, so TRA is not specifically related to co-morbidity. Factors predicting difficult asthma were a high dose of inhaled steroid at presentation, FEV<sub>1</sub> below 70% predicted, and previous specialist assessment. As Harrison points out in the accompanying editorial, it is hoped that the approach used in this paper will encourage others to become interested in this difficult patient group, to set up special clinics, and evaluate new therapeutic strategies. **See pages 555 and 561**



Additional diagnoses causing respiratory symptoms in 73 sequential referrals to a difficult asthma service. Twenty five of the 73 subjects (34%) had an additional diagnosis.

## MILK AND BROWN BREAD REDUCES ASTHMA RISK

We now know that the environment may play an important role in the development of susceptibility to asthma. In this month's *Thorax* Wijga and colleagues describe their results of an investigation of food consumption and asthma risk in pre-school children.

Recent asthma at age 3 was more common in those who had consumed daily full cream milk, milk products and butter at age 2 than those who did not. Eating brown bread daily was also associated with less asthma and wheeze in these children, but there were no relationships with fish, fruit or vegetables. Thus, a modern western lifestyle is associated with an increased risk of asthma and wheeze in pre-school children.

**See page 567**

## TB IN THE CANARY ISLANDS

In industrialised countries it is assumed that most cases of TB are due to reactivation of previous infection with *Mycobacterium tuberculosis*. In this issue of *Thorax* Pena and colleagues describe the results of molecular typing using restriction fragment length polymorphism of *M tuberculosis* strains of all patients diagnosed with TB over a 4 year period on the Spanish island of Gran Canaria which has a relatively closed population. An unexpected finding in this study was the high rate of clustering, together with some large clusters, thus providing evidence for a high rate of recent transmission of TB on the island. The authors conclude that increased control measures are required, especially in immigrant populations, and adequate adherence to treatment must be ensured, with legal controls if necessary.

**See page 618**

## ANTI-INFLAMMATORY EFFECTS OF PHOSPHODIESTERASE 4 INHIBITORS

COPD is associated with airway inflammation that is particularly difficult to treat. Neutrophils play an important role in the pathogenesis of the inflammation in COPD and treatments that modulate neutrophilic inflammation are eagerly awaited. In this issue of *Thorax* Profita and colleagues describe the effects of a selective phosphodiesterase 4 inhibitor (cilomilast) on the release of neutrophil chemoattractants from bronchial epithelial cells (BEC) and sputum cells from COPD patients. Cilomilast reduced the release of TNF $\alpha$  from BEC and GM-CSF from sputum cells, although there were no effects on IL-8 production. Neutrophil chemotaxis was also reduced by cilomilast. Few drugs have been found to inhibit neutrophil function; the results of this study make a case for further evaluation and offer some future hope for patients with COPD.

**See page 573**

## WHICH COMPONENTS OF DIESEL EXHAUST CAUSE PROBLEMS?

Exposure to ambient particulate matter has been shown to be associated with an increase in morbidity and mortality from respiratory disease. Diesel exhaust particles (DEP) are an important constituent of ambient particles and increase lung inflammation, although the actual components of DEP that affect inflammation are not known. In this month's *Thorax* Yanagisawa and colleagues show that the components of DEP that are important in enhancing bacterial endotoxin (lipopolysaccharide) induced lung injury are the residual carbonaceous nuclei rather than organic chemicals extracted from DEP. They also point out that the lung injury is mediated by inflammatory cytokines, chemokines, and Toll-like receptors.

**See page 605**