# airwaves

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## NEUTROPHILS, AIRWAY SMOOTH MUSCLE AND COPD

The nature of the airway inflammation in COPD and its relation to function is an important topic for research. The importance of infiltration of airway smooth muscle in asthmatics with mast cells has recently been demonstrated and, in this issue of *Thorax*, Baraldo and colleagues describe the nature of the inflammatory cells in airway smooth muscle in patients with COPD. They show that smokers with COPD had increased neutrophils and CD8+ cells (characteristically found in COPD) in the airway smooth muscle compared with non-smokers, while smokers with normal lung function had less smooth muscle infiltration with neutrophils. There were no differences between the various groups in the number of mast cells in the smooth muscle. The authors also show that there was a relation between lung function and the number of neutrophils in the airway smooth muscle. This paper suggests that there may be an interaction between airway inflammatory cells and smooth muscle and this relationship deserves further study. We are now getting a step closer to evaluating the complex nature of airway inflammation in COPD, so that novel therapies can be planned.

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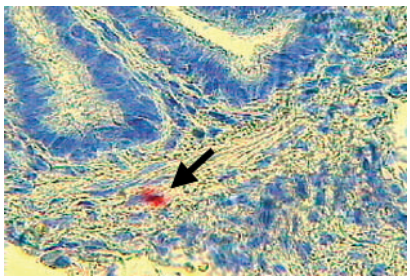

Micrograph showing neutrophils (arrow) within airway smooth muscle of peripheral airways of smokers with COPD

## MOLECULAR EPIDEMIOLOGY FOR TB

In this month's *Thorax* we publish two papers that show the value of using modern molecular techniques to investigate tuberculosis (TB) transmission. Ruddy and colleagues describe a major outbreak of isoniazid resistant TB in north London, UK that has still not reached its peak. It is of concern that prison detention was one of the factors predisposing to TB, and this is also the first documented outbreak in a UK prison. In the second paper Hernández-Garduño and colleagues used molecular epidemiology to study transmission of TB from smear negative cases, and show that these smear negative patients account for at least one sixth of culture positive episodes of TB transmission. In the accompanying editorial, Davies points out that the more we look for TB, the more we find it, and that TB is being transmitted with surprising efficiency.

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## SEX DIFFERENCES, PARENTAL SMOKING AND ADULT LUNG DISEASE

It is now accepted that there is an increased risk of wheeze and asthma in children whose parents smoke. However, there are few data on the longer term consequences into adulthood of parental smoking. In this issue we publish a paper by Svanes and colleagues using data from the European Community Respiratory Health Survey, with an accompanying editorial by Upton. Svanes *et al* show that both intrauterine and environmental exposure to parental smoking had an effect on lung function in adulthood. The study also evaluated sex differences for the effects and showed that maternal smoking in pregnancy increased the risk of obstructive disease in women and possibly also in men, while exposure to cigarette smoke in childhood was related to more respiratory symptoms in men but not in women. Smoking in early life therefore produces permanent effects on lung health.

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## SUDDEN INFANT DEATH IS NOT RELATED TO SLEEP APNOEA

It has been suggested that one of the causes of sudden infant death syndrome (SIDS) is obstructive apnoea in infants. In addition, some early data showed that there is an increased frequency of SIDS in family members of sleep apnoea patients. Vennelle and colleagues conducted an interesting study in which they performed polysomnography and ventilatory control measurements on parents of documented SIDS cases and controls. No relationship was found between SIDS and sleep apnoea. Minor changes in oxygenation during sleep and resistive loading were seen, but the significance of these findings is not known.

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## LINKS BETWEEN MMP-9, IL-17 AND NEUTROPHILS

Matrix metalloproteinases (MMPs) are thought to play an important part in airway remodelling and there has recently been considerable interest in their regulation. One of the group, MMP-9, has been shown to be increased in the airways of patients with asthma and COPD and, although it has a number of sources, airway neutrophils are probably the most important. In this issue of *Thorax* Prause and colleagues report that the T lymphocyte derived cytokine IL-17, which causes airway neutrophil accumulation, increases the load of MMP-9 in mice. The levels of MMP-9 were related to the neutrophil load, suggesting that IL-17 increases neutrophils which then lead to more MMP-9 production. IL-17 may therefore be a target in airways diseases such as COPD in which airway neutrophil accumulation and remodelling occur.

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