# airwaves

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## INCREASING INHALED STEROIDS FOR ASTHMA EXACERBATIONS

The prevention of asthma exacerbations is a major goal in the management of asthma. Asthma guidelines have suggested that doubling the dose of inhaled steroids at the time of worsening asthma may treat or prevent the worsening of an asthma exacerbation. However, there is little evidence for this approach. In this issue of *Thorax* FitzGerald and colleagues report a randomised double blind controlled trial to determine whether doubling the dose of inhaled budesonide at the time of an asthmatic exacerbation or continuing with the usual maintenance dose has a beneficial effect on progression to a more severe exacerbation. The authors found that there was no difference in treatment failure (courses of systemic steroids, unscheduled physician visits) between the two groups. This suggests that doubling the maintenance dose of inhaled steroid does not change the pattern of the exacerbation. In the accompanying editorial, Busse and Lemanske discuss some of the reasons for these results. They suggest that, as respiratory viruses are a major cause of asthma exacerbations, this will lead to a lower airway neutrophilic inflammatory response. Evaluation of the mechanisms of virus induced airway inflammation is likely to lead to new treatments suitable for prevention of asthma exacerbations and also for COPD exacerbations.

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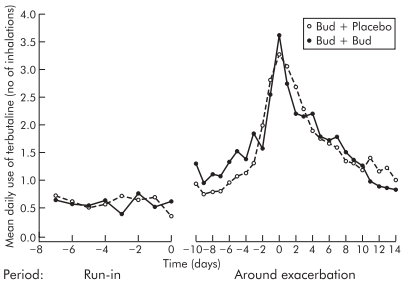


Pattern of short acting $\beta_2$ agonist use around the time of exacerbation. Bud = budesonide.

## TIMP-3 AND PIGEON LUNG

Various gene variants of matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) may influence susceptibility to a number of conditions including cancer, COPD, and cardiovascular disease. TIMPs have an important role in controlling the turnover of extracellular matrix in the lung, so variants which alter gene expression in TIMP-3 may affect susceptibility to lung fibrosis. Hill and colleagues describe the frequency of two promoter variants in the TIMP-3 gene in Mexican patients with idiopathic pulmonary fibrosis (IPF), pigeon breeders' disease (PBD), and healthy controls. The variants had no effect on susceptibility to IPF but the two promoter variants did affect susceptibility to PBD. The authors also found that the effect was not related to the fibrotic reaction but may relate to inflammatory processes that occur early in the disease.

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## SYSTEMIC INFLAMMATION IN COPD

There is now increasing interest in the role of systemic inflammation in COPD and its relation to weight loss and cardiovascular morbidity. A number of studies have shown a relationship between markers of systemic inflammation and lung function in stable COPD. In this issue of *Thorax* Gan and colleagues describe a systematic review of 14 of these studies and found that, compared with healthy controls, patients with COPD had increased systemic levels of C-reactive protein, fibrinogen, white blood cells, and tumour necrosis factor $\alpha$. They also point out that non-current smokers with airflow obstruction have increased systemic inflammation, which suggests that smoking cessation may not attenuate it. The challenge is now to evaluate how systemic inflammation develops in COPD and how it can be reduced.

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## BONE DISEASE AND SYSTEMIC INFLAMMATION IN CF

Adults with cystic fibrosis commonly have reduced bone mineral density (BMD) and it has been suggested that this reduction is due to the effects of systemic proinflammatory cytokines. In this month's *Thorax* Haworth and colleagues investigate the relationship between BMD and systemic inflammatory markers in adult patients with cystic fibrosis followed for 1 year. They found that BMD was related to the systemic levels of interleukin 6 (IL-6) and C-reactive protein (CRP), intravenous antibiotic use, and oral corticosteroid use. Analysis showed that IL-6 levels, colonisation with *Burkholderia cepacia*, and changes in body mass index were independent predictors of a change in BMD.

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## SMOKING CESSATION NEEDS OUR ATTENTION

In this issue of *Thorax* we publish two articles on smoking cessation. In his editorial Britton discusses the availability of smoking cessation clinics and states that, in England, all smokers should be able to access cessation services that can provide evidence based and cost effective interventions. However, the reality is that these services are not being used and half of UK hospitals are not providing a cessation counselling service for inpatients. The second article is a review of smoking cessation in developing countries by Abdullah and Husten, who remind us that by 2030 the developing world is expected to have annually 7 million smoking related deaths. They describe a number of obstacles to promoting smoking cessation in the developing world—including economic and political factors and deficiencies in healthcare systems.

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