airwaves

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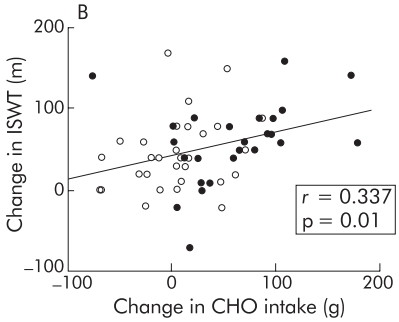
EXTRA CARBOHYDRATE IN COPD: WHO BENEFITS?

An important objective of treatment in COPD is to improve physical performance and daily activities. Physical training is an essential component of pulmonary rehabilitation although the response can be variable and strategies to enhance training are required. In this issue of *Thorax* Steiner and colleagues describe a randomised controlled trial of nutritional supplementation using a carbohydrate rich diet during pulmonary rehabilitation; the results are further discussed in the accompanying editorial by Wouters. Although patients in the supplement group gained weight and those in the placebo group lost weight, there was no overall effect of the supplement on exercise performance. However, in patients who were well nourished with a BMI of >19 kg/m^2, exercise performance was better in the supplemented group. The authors point out that physical activity imposes a high energy cost and this may limit optimal training. However, there may be a role for nutritional supplementation in selected patients with COPD.

See page 745



MUSCLE WEAKNESS INCREASES AT COPD EXACERBATION

COPD exacerbations are characterised by both increased airway and systemic inflammation, and patients with a history of frequent exacerbations have increased airway inflammatory markers. In this issue of *Thorax* Spruit and colleagues show that peripheral muscle weakness is increased during COPD exacerbations in hospitalised patients and recovers somewhat afterwards. Furthermore, they show that levels of systemic interleukin 8 (CXCL8) are inversely related to peripheral muscle force. This raises the intriguing hypothesis that systemic inflammation may affect peripheral muscle function at exacerbation. As Polkey points out in the accompanying editorial, strategies employed for reducing exacerbation frequency may have an important effect in maintaining exercise performance.

See pages 741 and 752

PROMISING ROLE FOR MMP-9 IN AIRWAY INFLAMMATION

Airway remodelling is a feature of chronic airway inflammatory conditions. Collagen degradation plays a major part in this process and is regulated by the balance of matrix degrading enzymes such as metalloproteinases (MMPs) and their inhibitors (TIMPs). MMP-9 has been shown to be involved in the pathogenesis of asthma, though little is known about its regulation. In this issue of *Thorax* Hetzel and colleagues evaluated the role of the transcription factor peroxisome proliferator activated receptor gamma (PPARγ). The study shows that PPARγ is expressed in airway epithelial cells and that PPARγ activators inhibit MMP-9 through an interaction with the transcription factor NF-κB. Further evaluation of this novel mechanism is required to determine any therapeutic potential. Also in this issue, Oshita and colleagues report that exacerbations of asthma are associated with increased circulating MMP-9 activity, suggesting a mechanism as to how asthma exacerbations may contribute to airway remodelling.

See pages 778 and 757

NO ROLE FOR GC HAPLOTYPES IN COPD

Cigarette smoking is a major risk factor for COPD. As only about 10–15% of smokers develop airflow obstruction, there has been considerable interest in genetic factors that may contribute to the pathogenesis of COPD. The Lung Health Study was designed to study the effect of early smoking cessation on the natural history of COPD, and this unique, well characterised cohort is very useful for genetic studies. In this month's issue of *Thorax* Kasuga and colleagues evaluate group specific component (GC) haplotypes in these patients, as there have been a number of earlier reports of associations of GC haplotypes with COPD. No relation was found in the GC genotype frequencies or in the GC haplotypes in low or high lung function groups. The same group has previously also shown that GC haplotypes have no effect on the decline in FEV_1 in COPD.

See page 790

COFFEE, BLOOD PRESSURE AND SLEEP APNOEA

Obstructive sleep apnoea (OSA) causes excessive daytime sleepiness that resolves with nasal CPAP therapy. There is some evidence that nasal CPAP also reduces blood pressure, but patients with OSA also drink more coffee, presumably to keep awake. Thus, the observed reduction in blood pressure with CPAP may be due to less coffee consumption once sleepiness has improved with treatment. Stradling and colleagues measured plasma caffeine levels in two groups of patients during a trial, one group treated with CPAP and the other with "subtherapeutic" CPAP. There was no difference in caffeine levels in the two groups, suggesting that the reduced coffee consumption is not the reason for the fall in blood pressure with CPAP.

See page 801