transcriptional disease mechanism for altering muscle phenotype in human type 2 diabetes
The clinical cohort study was supported by the Danish National Research Foundation Grant DG 02-512-555 (BKP). This study was supported by an Affymetrix Translational Medicine award (JT), the Swedish Diabetes Association (JT) and the Chief Scientists Office Scotland (JT). GH is a Wellcome Trust CD fellow. JR is supported by the SIROCCO FP6 program.

Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.

C13 and PC23
Increased energy intake in children with an obesity-associated FTO gene variant
P.W. Watt1, J.E. Cecil2, R. Tavendale3, M.M. Hetherington4 and C.N. Palmer3
1Sport and Exercise Science, University of Brighton, Eastbourne, UK; 2Bute Medical School, University of St Andrews, St. Andrews, UK; 3Biomedical Research Centre, University of Dundee, Dundee, UK and 4Psychology, Glasgow Caledonian University, Glasgow, UK

TITLE ONLY
Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.

C14 and PC24
Illness, body mass index and leptin
A. Taylor1, R. Baskerville2 and L.M. Castell1
1Nuffield Dept of Anaesthetics, University of Oxford, Oxford, UK and 227 Beaumont Street, Oxford OX1 2NR, UK

Introduction
There is anecdotal evidence that obese individuals suffer from poor wound healing and frequent minor illnesses such as upper respiratory tract infections (URTI) cf. lean individuals. The altered metabolic, psychological and endocrine status of obesity may lead to immunodepression. Our group has investigated URTI in fatigued athletes and military personnel (Castell, 2003); one study focused on the obesity hormone, leptin which regulates energy balance, and its role in immune function. Decreased circulating leptin can lead to hyperphagia in some genetically obese individuals (O’Rahilly, 2002). However, human obesity is usually characterized by excessive leptin, rather than deficiency. The leptin functional receptor is found in all immune response cell types: thus leptin may link nutritional status, energy balance and immune function. Sleep loss, partial or chronic, is linked to decreased immune function. Obesity is a factor in obstructive sleep apnoea, which significantly reduces sleep duration and quality.

Aims
This pilot study surveyed the incidence and severity of URTI, psychological and sleep profiles in sedentary participants of different ages, and body mass indices (BMI). The aim was to observe whether mild obesity (BMI 30-35) predisposed people to an increase in URTI cf. individuals with a lower BMI. The survey was a precursor to a more detailed study.

Methods
Eight lean, 2 overweight, 8 obese sex matched, sedentary participants were recruited for this ethically approved survey. Anthropometrics (ht, wt, waist, hips) were taken; a resting blood sample measured plasma leptin; daily questionnaires were given for 6 wks to monitor URTI; POMS (depression, stress, fatigue, motivation); Sleep (duration, quality); Dietary diaries. A multiple regression General Linear Model was used to explain the “illness score”.

Results
A higher BMI was significantly associated with higher symptoms of illness (p<0.01). Calorie consumption was associated with BMI (p<0.05). BMI correlated with plasma leptin concentration. Poor sleep quality predisposed individuals to an increase in URTI symptoms (p<0.032); in addition it was associated with higher levels of depression (p<0.005) and stress (p<0.005). Sleep duration did not have any effects.

There was an apparent link between smoking and increased URTI symptoms.

Discussion
The study showed links between increased BMI, poor sleep quality and the incidence of self-reported URTI. It is suggested that obesity is associated with an increase in URTI cf. normal or lean weight. BMI was linked with blood leptin concentration, and it is tempting to speculate that there might be a link between leptin and increased URTI.


We are grateful to the volunteers for their cheerful participation

Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.
The role of peripheral and central chemoreceptors in the hypermetabolism-induced increases in the ventilatory chemosensitivity to CO₂ in humans

C. Lykidis¹, P. Kumar² and G. Balanos¹

¹Sport and Exercise Sciences, University of Birmingham, Birmingham, UK and ²Department of Physiology, University of Birmingham, Birmingham, UK

The role of hypermetabolism in the mediation of exercise hyperpnoea is contentious. Recent animal data has shown that the carotid body may be directly involved in establishing the hyperpnoea via a metabolic rate (MR)-induced elevation in the ventilatory sensitivity to CO₂ (Bin-Jaliah et al., 2005). A previous human study had also evidenced augmented chemosensitivity to CO₂ in response to raised MR, yet no inferences were made on the potential role that peripheral chemoreception could have played on these observations (Zwillich et al., 1977). Therefore we aimed to appraise the importance of peripheral chemoreception in the hypermetabolism-induced increases in chemosensitivity in awake humans.

Fourteen healthy subjects took part in either one of two separate experiments. Local ethical approval was obtained and all subjects gave written informed consent. Each experiment involved a different methodology to assess the ventilatory response to euoxic (PetO₂ = 100mmHg) hypercapnia before and 210 minutes after the ingestion of a fixed amount of food; a five-minute ramp protocol (PetCO₂ = 0 to 8 mmHg above normal; n=7) in which peripheral chemoreception has been known to play an important role, and a step change protocol (PetCO₂ = 0, 2, 4, 6 and 8 mmHg above normal; each step lasting 3 minutes; n=7) in which the role of central chemoreceptors is predominant. MR was measured as oxygen consumption prior to each challenge of hypercapnia. Data were analysed by means of paired t-tests (SPSS 16.0) and significance was taken at p<0.05.

All data are expressed as mean±S.E.M. MR was significantly increased by food ingestion in both experiments (*Ramp* experiment: 0.22±0.02 to 0.30±0.02; *Step* experiment: 0.25±0.01 to 0.30±0.02, baseline vs 210 min, All P<0.03). The ventilatory sensitivity to CO₂ was increased significantly in the ramp protocol (1.13±0.11 to 1.78±0.15 L/min/mmHg, baseline vs 210 min, P=0.0001) but, although the mean was elevated, the sensitivity to CO₂ was not significantly increased in the step protocol (1.14±0.13 to 1.42±0.22 L/min/mmHg, baseline vs 210min, P=0.26).

Our results suggest that the hypermetabolism-induced increases in the ventilatory sensitivity to CO₂ might be primarily mediated through elevations in the peripheral chemoreceptor gain. Enhancement of this gain appeared to be facilitated even by mild increments of MR. The mechanism that couples metabolism to changes in chemosensitivity is not known.


Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.