Urea and creatinine clearances in the trimesters of pregnancy
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The aim of this study was to determine the glomerular filtration rate (GFR) in the three trimesters of pregnancy, using creatinine and urea clearances. A total of 108 healthy subjects between the ages of 18 and 37 years were divided into non-pregnant (28 ± 5 years, n = 30), 1st trimester of pregnancy (28 ± 5 years, n = 18), 2nd trimester of pregnancy (28 ± 4 years, n = 30), and 3rd trimester of pregnancy (30 ± 4 years, n = 30) women. Serum and urine samples were collected in mid-trimester period. Creatinine and urea concentrations in serum and urine were determined using Jaffe’s method (Bosnes et al. 1945) for creatinine, and the urease method (Martinek, 1964) for urea.

There was a significant increase (mean ± SD in creatinine clearance, but a significant fall (p < 0.001) in blood creatinine concentration in the three trimesters of pregnancy when compared with the control value. The increases of 43 ± 26 ml/min (non-pregnant vs. 1st trimester), of 39 ± 21 ml/min (1st vs. 2nd trimesters) and of 42 ± 25 ml/min (2nd vs. 3rd trimesters) of were similar. Similarly, there was a significant increase in the Urea clearances in the 1st, 2nd and 3rd trimesters of pregnancy, and remained elevated during pregnancy. In contrast, blood urea concentration increased significantly (p<0.01) in the three trimesters, despite an increase in the 12 hour urine volume output.

The results in Table 1 show that Ccr and Cur increase and peak during the first trimester of pregnancy. Although there was a reduction in Ccr in the 3rd trimester compared to the 2nd trimester, the clearance remained significantly higher (p<0.05) than in the non-pregnant women. This study agrees with other workers (Logoglu et al. 1990; Susan & Donna, 1992), who showed similar increases of GFR during pregnancy.

It is concluded that GFR increases and peaks during the first trimester of pregnancy and remains relatively stable till term.

<table>
<thead>
<tr>
<th></th>
<th>Non-pregnant, ml/min</th>
<th>Pregnant, ml/min</th>
<th>1st Trimester</th>
<th>2nd Trimester</th>
<th>3rd Trimester</th>
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</thead>
<tbody>
<tr>
<td>Blood creatinine conc., mg/100ml</td>
<td>0.84 ± 0.21</td>
<td>0.52 ± 0.15</td>
<td>0.40 ± 0.12</td>
<td>0.50 ± 0.14</td>
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<tr>
<td>Creatinine clearance, Ccr, ml/min</td>
<td>97 ± 20</td>
<td>127 ± 18</td>
<td>162 ± 28</td>
<td>130 ± 28</td>
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<tr>
<td>Blood urea conc., mg/100ml</td>
<td>12.0 ± 4.0</td>
<td>16.1 ± 4.6</td>
<td>19.4 ± 4.8</td>
<td>16.7 ± 5.2</td>
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<tr>
<td>Urea Clearance, Cur, ml/min</td>
<td>62.2 ± 21</td>
<td>87 ± 10</td>
<td>96 ± 18</td>
<td>96 ± 12</td>
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</table>

with adjustment for age, sex and lean mass. tCys was the strongest plasma variable associated with fat mass, stronger than and independent of plasma lipids. Women in the highest tCys quintile had fat mass 6 kg greater than that of women in the lowest quintile (95% CI: 5, 7 kg), with adjustment for plasma lipids, physical activity, and dietary fat, protein, and total energy intakes. Corresponding values for men were 4 kg (95% CI: 3, 5 kg; P<0.001 for ANOVA across quintiles in both genders). A higher baseline tCys and a rise in tCys over 6 y were both associated with greater fat mass at follow-up (P<0.001 by linear regression), with no effect on lean mass.

Discussion

Literature evidence points to tCys as a powerful but ignored determinant of fat mass. Homocystinurics with genetic deficiency of CBS enzyme (and hence decreased cysteine synthesis) are thin and underweight [3], a feature not reported for other types of homocystinuria, in which cysteine synthesis is normal. In contrast, Down syndrome patients, having triple copies of the CBS gene and elevated tCys, are overweight. Dietary cysteine supplements enhance weight gain in cachectic AIDS and cancer patients [4]: an effect generally attributed to improved lean mass, but do we know? Dietary restriction of the cysteine-precursor methionine reduces visceral fat mass in rats [5]. Several early studies on rat adipocytes demonstrate potent antilipolytic and lipogenic actions of cysteine.

Conclusions

Overall, our data and literature evidence suggest that cysteine could be an important modulator of body fat mass in humans, and if so, provides an attractive anti-obesity target.


We would like to thank Professor AD Smith for his valuable insight and review of the study.

Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.
transcriptional disease mechanism for altering muscle phenotype in human type 2 diabetes


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C13 and PC23

Increased energy intake in children with an obesity-associated FTO gene variant

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C14 and PC24

Illness, body mass index and leptin

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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.
Extremely short duration high intensity training substantially improves insulin action in young healthy males

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Introduction: The prevalence of type 2 diabetes is increasing in western societies resulting in increased healthcare/economic costs. However, the risk of developing T2D is positively modified by regular physical activity. Despite this, there is a lack of consensus on the ideal strategies for ensuring adult participation in exercise. Current recommendations involve performing moderate intensity exercise for several hours per week; however, the general population fails to adhere to such regimes typically stating time as a major barrier. Recently an extremely low volume high-intensity training paradigm (HIT), ~7.5 minutes of exercise per week, has been proposed as a novel, time-efficient exercise regime for inducing aerobic adaptations. This has challenged our understanding of aerobic adaptation in humans. Aerobic training has been demonstrated to improve insulin action in humans and we sought to establish if HIT can enhance insulin sensitivity in sedentary younger male subjects.

Methods: 25 young men (21±5 y) were randomly assigned to control (n=9) or HIT (n=16) groups. Subjects underwent an oral glucose tolerance test (OGTT), a VO2peak test and 2x250kJ cycling time trials. The control group adhered to their normal routine for 2 weeks prior to a second OGTT. HIT comprised of 15 min exercise over 2 weeks, consisting of 4-6 x 30-second cycle-sprints per session. At 48 or 72 hr post training, subjects underwent a second OGTT followed by a third time trial 24h later.

Results: Following 2 weeks of HIT, the area under the plasma glucose, insulin and NEFA concentration-time curves were all reduced (12%, 37%, 26% respectively, all P<0.001). Insulin sensitivity (Cederholm index) was markedly improved following 2 weeks of HIT (23%, P<0.001). Fasting plasma NEFA concentration was also reduced (pre: 350 ± 36 μmol.l⁻¹, P<0.05) while fasting plasma insulin and glucose concentrations remained unchanged. There were no changes in the control group.

Conclusions: These results demonstrate the remarkable efficacy of HIT to improve insulin action in young healthy men. Therefore, there is a need to reappraise the current trends in exercise prescription for increasing energy expenditure, to focus on strategies that are both preventative and more likely to be adhered to by young and middle-aged people.