C15

Pain and changes in peripheral resistance at high vascular transmural pressure in the human forearm

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Overt forearm pain can be experienced by the crews of high performance military aircraft during manoeuvring. The pain could be associated with a hydrostatic rise in forearm vascular transmural pressure and an increase in forearm blood flow, which has been observed following exposure to vascular transmural pressures of 150mmHg and above (Greenfield & Patterson, 1954). To test the hypothesis that high-G arm pain is related to a rise in forearm blood flow, the relationship between vascular transmural pressure, forearm vascular resistance and pain was examined.

Following approval by the RAF Experimental Medicine Ethics Committee, eight healthy male volunteers (mean ± S.D.; age 33.3 ± 9.3 years) were studied in accordance with local guidelines and the Declaration of Helsinki. Subjects placed one arm through a port in a hypobaric chamber, and forearm vascular transmural pressure was elevated by a series of 1 minute hypobaric exposures, at incrementing differential pressures of 40, 80, 120, 140, 160 and 200mmHg with 2 min at 0mmHg between exposures. The sequence was repeated after a 30 min rest interval. Forearm venous pressure (FVP) was measured by indwelling catheter in the median antecubital vein, and blood velocity in the proximal axillary artery was monitored by 2D and Doppler ultrasound imaging. Forearm vascular resistance was assessed by Doppler resistive index (RI), which is defined as $(S\text{-}D)/S$, where S is the systolic velocity waveform peak, and D is the end-diastolic trough. Pain rating was recorded by visual analog scale. Data are presented as means ± S.E.M., and compared using the Wilcoxon signed rank test (for pain scores) and paired t tests (for vascular transmural pressure).

A linear relationship between FVP and pain was observed (r^2=0.8, n=35). In all subjects, a change from the normal high resistance type flow pattern of the axillary artery occurred at high FVP (176mmHg ± 7.7mmHg). The flow pattern changed to that of a low resistance vascular circuit, with flow in diastole, such that RI fell to a value of less than 1. This was associated with an increase in pain rating (P<0.001) from 5.4 ± 0.7 in the exposure preceding the change to 8.4 ± 0.5. In the second series of exposures, there was a fall in the transmural pressure at which this phenomenon was observed (P<0.01), to a mean FVP of 135.4mmHg ± 11.2mmHg.

The observed fall in RI suggests a profound reduction in vascular resistance occurs at high vascular transmural pressure in the forearm, which is associated with the development of severe pain. Repeated exposure reduces the transmural pressure at which this effect occurs.

Greenfield ADM & Patterson GC (1954). *J Physiol* **125**, 508-524.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C16

Prophylactic *L*-ascorbate infusion stimulates local release of vascular endothelial growth factor during ischaemia-reperfusion surgery in humans; evidence for redox-regulation

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The present study examined changes in reactive oxygen species (ROS) and vascular endothelial growth factor (VEGF) protein during ischaemia-reperfusion surgery. Twenty two patients scheduled for either elective abdominal aortic aneurysm (AAA) repair or infra-inguinal bypass (IIB) surgery were recruited. Patients were assigned double-blind to either a vitamin C (AAA: n = 6, IIB: n = 4) or placebo (AAA: n = 7, IIB: n = 5) group. Following anaesthesia, each patient received an intravenous infusion of either 20ml saline (placebo) or 20ml saline containing 2g of *L*-ascorbate (vitamin C). Blood was obtained from local arterial and venous lines proximal to the site of repair and peripheral antecubital vein at the end of cross-clamp application (ischaemia) and within 10 min after clamp release (reperfusion). Samples were assayed for ascorbate, lipid hydroperoxides (LH), transition metal ions and α-phenyl-*tert*-butylnitrone (PBN)-adducts as previously described (Bailey et al., 2004) and total VEGF protein concentration via radioimmunoassay.

Vitamin C infusion increased the venoarterial concentration difference ($v\text{-}a_{diff}$) for ascorbate during ischaemia whereas a decrease was observed following reperfusion (Table 1). Vitamin C also increased LH and VEGF$v\text{-}a_{diff}$ during ischaemia. Electron paramagnetic resonance (EPR) spectroscopy provided direct evidence for an increase in the concentration of PBN-adducts and ascorbate radical ($A^{\cdot-}$) in peripheral venous blood during reperfusion in the vitamin C group (PBN-adducts: ischaemia = 4258 ± 2125 vs. reperfusion = 5984 ± 2361 arbitrary units (AU), $P < 0.05$; $A^{\cdot-}$: ischaemia = 1188 ± 284 vs. reperfusion = 1415 ± 202 AU, $P < 0.05$). Nuclear hyperfine splittings were consistent with the trapping of secondary oxygen (alkoxyl or hydroxyl) and carbon (alkyl)-centred species comparable to those generated during metal-catalysed reductive decomposition of LH. Manipulation of the EPR signal intensity of $A^{\cdot-}$ following *in vitro* addition of desferrioxamine mesylate and Fe(III)-EDTA to vitamin C-supplemented serum obtained during reperfusion confirmed that iron was available for oxidative catalysis.

The present findings suggest that high-dose ascorbate may increase local ROS generation during ischaemia through initiation of a Fenton reaction. Local release of reduced free iron during reperfusion may prove the catalyst for the secondary radicals detected in the peripheral circulation. The 'downstream' increase in VEGF protein may prove an adaptive response to 'salvage' vascular homeostasis.

Table 1. Proximal and distal metabolic responses to intravenous vitamin C

	Condition:	Ischaemia		Reperfusion	
Parameter	Site:	Local v-a_{diff}	Antecubital	Local v-a_{diff}	Antecubital
Ascorbate (µmol/L)	Vitamin C	4.1 ± 27.1*	117.9 ± 51.0†	-10.0 ± 24.8	89.4 ± 47.1†
	Placebo	1.7 ± 3.0	6.9 ± 4.6	-0.1 ± 3.4	6.1 ± 7.6
LH (µmol/L)	Vitamin C	0.40 ±0.78†	0.96 ± 0.29	0.04 ± 0.38	0.98 ± 0.64
	Placebo	0.00 ± 0.31	1.23 ± 0.49	-0.05 ± 0.51	0.85 ± 0.22
VEGF (pg/mL)	Vitamin C	0.7 ± 0.6†	5.1 ± 1.1	0.4 ± 1.1	6.1 ± 2.0†
	Placebo	-0.3 ± 1.4	5.0 ± 0.9	0.3 ± 0.7	5.3 ± 2.6

Values are means ± SD; *main effect for condition ($P < 0.05$ two-way repeated measures ANOVA); †different between groups for given condition and site ($P < 0.05$, Mann-Whitney U test).

Bailey, D.M., Young, I.S., McEneny, J., Lawrenson, L., Kim, J., Barden, J. & Richardson, R.S. (2004). *Am J Physiol Heart Circu Physiol.* 287, H1689-1699.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C17

An indication of a parabolic relationship between lower body negative pressure tolerance and maximum oxygen uptake in humans

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The relationship between orthostatic tolerance and aerobic fitness has yet to be accurately defined. Positive, negative and an absence of a relationship have all been reported in the scientific literature. This study determined tolerance to Lower Body Negative Pressure (LBNP, a measure of orthostatic tolerance), maximum oxygen uptake and blood volume in subjects in the trained and detrained state and in control subjects to examine whether a loss of aerobic fitness alters orthostatic tolerance.

Measurements were made on seven athletic subjects (age 27.9 ± 3.3 years) before and after 17 weeks of training or detraining. A control group of seven normal fit subjects (age 28.7 ± 3.9 years) had the same measures taken before and after a control period of a similar duration.

Data are given as means ± S.D. Between and within group differences were examined using Wilcoxon comparisons. For the test group the detrained state was associated with significantly lower ($p < 0.01$) aerobic fitness (maximum oxygen uptake reduced from 63.5 ± 9.9 to 53.6 ± 5.9 ml $kg^{-1}min^{-1}$) and blood volume (5.7 ± 1.0 to 5.3 ± 0.9 l) than the trained state. A significant increase ($p < 0.01$) in tolerance to LBNP as described by the cumulative stress index (product of duration and negative pressure endured before pre-syncope) was also found (increased from 785.7 ± 206.7 to 966.1 ± 337.9 mmHg min^{-1}). LBNP tolerance in the detrained state proved to be significantly greater ($p < 0.05$) than that of the control group (747 ± 185.7 and 738 ± 195.0 mmHg min^{-1} pre and post control period respectively). No changes in these variables occurred in the control group ($p > 0.05$) (maximum oxygen uptake 45.5 ± 8.7 and 44.3 ± 8.9 ml $kg^{-1}min^{-1}$ and blood volume 5.28 ± 0.8 and 5.27 ± 0.8 l).

Previous studies attempting to ascertain the relationship between aerobic fitness and orthostatic tolerance have rarely included control groups. The absence of a control group in the present study would have lead to the conclusion that the highly fit (trained) state is associated with low tolerance to orthostatic stress, and that detraining returns tolerance towards greater, normal fit values, a finding often reported in the open literature. The interesting finding that there was no difference between normal fit (control) and high fit LBNP tolerance and that a reduction of maximum oxygen uptake was in fact associated with tolerance to LBNP above that of normal fit subjects, supports the hypothesis that there could be a parabolic relationship between orthostatic tolerance and aerobic fitness under these specific circumstances.

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Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C18

Scaling Properties of the Human R-R Interval at Rest and when Cycling at Different Intensities

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Detrended fluctuation analysis (DFA), a statistical technique that probes the fractal or scaling properties of time series, has been applied successfully to a variety of data (Goldberger et al. 2000; Peng et al. 1995). In relation to the human cardiac rhythm, R-R records during exercise have been reported to exhibit scaling properties qualitatively different from those obtained at rest (Karasik et al. 2002). Specifically, exercise records demonstrated anticorrelation at short timescales and correlation at intermediate scales, while with resting records the opposite crossover pattern was observed.

The present study further investigated this question. Male human subjects (n=4) were fitted S810 Heart Rate Monitors (Polar Electro Oy, Professorintie 5, FIN-90440 Kempele). R-R intervals were recorded at rest, and when exercising at 50, 100 and 150W using a cycle ergometer at constant pedal velocity (50 rpm). Recording intervals varied from 20 min at rest to 10 min at 150W, and were chosen so as to yield usable records of approximately 1000 intervals, after discarding the first two minutes of each record. The DFA algorithm was obtained as a text file (Goldberger et al. 2000) and compiled using C++.

Log-log plots were constructed of the first-order DFA fluctuation function against segment length. The gradient of these plots reflects the correlation properties of the R-R time series over different timescales. The plots showed no abrupt discontinuities of gradient either at rest or during exercise. Two subjects showed some indication of decreasing gradient as segment length increased, both at rest and during exercise, but for the remainder the plots were essentially linear. Correlation coefficients for all plots exceeded 0.95. Linear regressions were fitted and their parameters compared using repeated measures ANOVA. There was a significant effect of exercise level on the gradient of the plots ($p<0.025$). At 150W, the average gradient was 19% higher

than at rest (1.089 v 0.914). There was also a significant effect of exercise level on intercept ($p<0.005$).
The results of this study therefore do not support the contention of a qualitative difference in scaling behaviour between resting and exercise conditions. Correlation properties, as evidenced by the gradients of the plots both at rest and during exercise, were either unaffected by segment length, or demonstrated a modest decline at increasing timescales. Exercise, particularly at 150W, did however increase the correlation properties of the R-R time series, over all time scales, compared with the resting condition.

Goldberger AL et al. (2000). Circulation 101, e215-220 [Circulation Electronic Pages; http://circ.ahajournals.org/cgi/content/full/101/23/e215]
Karasik, R et al. (2002). Phys Rev E 66 062902
Peng C-K et al. (1995) Chaos 5, 82-87.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C19

Non-invasive assessment of intracranial arterial and respiratory pressure waves via the trans aural route in man.

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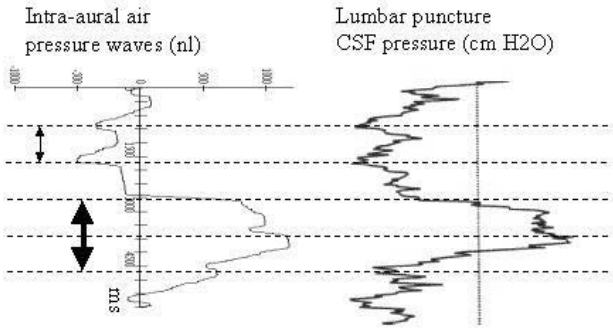
The clinical measurement of intracranial pressure usually requires invasive access to the cerebrospinal fluid compartment which is painful, hazardous in the unconscious patient and impractical for long-term follow-up studies.
The trans-aural route of intracranial pressure monitoring has been previously described (Marchbanks 1987, Samuel et al 1998). Ethical permission was obtained for systematic study of trans-aural intracranial pressure measurements in children. A 16 year old girl with longstanding headache, papilloedema and congenital heart disease underwent general anaesthesia and mechanical ventilation for replacement of her cardiac pacemaker and an opportunistic lumbar puncture was performed.
The continuous cerebrospinal fluid (CSF) pressure is shown (fig. 1)with the arterial and mechanical ventilation waveforms indicated by thin & thick arrows respectively (top trace: pressure in cmH2O). The trans-aural (TMD) air volume changes (bottom trace: volumes of air displaced in nanolitres) clearly match the CSF arterial and ventilation pulse wave changes. The arterial interpulse intervals range from 900-1000ms. Ventilated CSF respiratory pressure waves of 3.6cmH20 produced TMD volume displacements of 1684nl (peak-peak). This gives a CSF/TMD calibration factor of 0.021 mm saline / nl and the TMD will resolve displacements to less than 5nl or 0.10 mm saline. The CSF & TMD arterial pulse & mechanical ventilation amplitudes diminished with removal of CSF to reduce intranial pressure.
This represents the first description of accurate intracranial respiratory and cardiovascular pulse waves via the trans-aural route in humans & may contribute to the management of acute & chronic ehcephalopathy in children and adults.

Figure 1. Simulatneous intra-cranial CSF pressures (cmH20) & intra-aural air volume displacements (nl).



Marchbanks, R.J., Reid, A., Martin, A.M., et al (1987) The effect of raised intracranial pressure on intracochlear fluid pressure: three case studies. British Journal of Audiology, 21, 127-130.
Samuel, M., Marchbanks, R.J. and Burge, D.M. (1998) Tympanic membrane displacement test in regular assessment of intracranial pressure in eight children with shunted hydrocephalus. J Neurosurg 88:983-995.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C20

Cardiac limitations to systemic O_2 delivery and uptake during maximal exercise in humans

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Reductions in systemic and locomotive skeletal muscle blood flow and O_2 delivery limit maximal aerobic capacity during constant maximal exercise in humans (González-Alonso & Calbet 2003; González-Alonso *et al.* 2004). This study examined whether systemic O_2 delivery limits maximal aerobic power (VO_{2max}) during incremental exercise to exhaustion. We measured systemic haemodynamics, O_2 transport and O_2 uptake during incremental (20, 40, 60, 80, 90, 95, 100% of peak power output) and constant (372±11 W ∽ 85% of peak power output; 6.87±0.50 min; mean±SEM) cycle ergometer exercise to exhaustion in 8 trained male subjects (29±1 years, 4.85±0.1 l min^{-1}). Data were analysed by repeated measures one-way ANOVA and Tukey's honestly significant difference (HSD) post hoc procedure. Statistical significance was accepted at $P < 0.05$.
During incremental exercise, cardiac output and systemic O_2 delivery increased linearly up to 80% of peak power (both r^2=0.998, $P <0.001$), peaked at ~92% of peak power and thereafter plateaued or decreased in parallel to a decline in stroke volume ($P <0.05$) and increases in central venous and mean arterial pressures. In contrast, heart rate and pulmonary VO_2 increased linearly until exhaustion (r^2≥0.993; $P <0.001$) accompanying a continuous rise in systemic O_2 extraction to 84±2%. During constant load exercise, cardiac output, stroke volume and systemic O_2 delivery and uptake increased during the first ~5 min and dropped before exhaustion (all $P <0.05$) despite increasing or constant central venous and mean arterial pressures. The

fall in systemic O_2 delivery in both maximal tests was solely owing to the decline in cardiac output because arterial O_2 content increased until exhaustion.
In conclusion, these results in healthy trained humans indicate that maximal aerobic power is largely limited by the inability of the heart to sustain a linear increase in cardiac output and O_2 delivery. Furthermore, the similar impairment in stroke volume and systemic O_2 delivery during incremental and constant maximal exercise strongly supports a preponderant cardiac limitation to maximal aerobic power and capacity in humans.
This work was supported by the Gatorade Sports Science Institute.
All procedures accord with current local guidelines and the Declaration of Helsinki.

González-Alonso J & Calbet J (2003). Circulation **107**, 824-830.

González-Alonso J, Dalsgaard MK, Osada T, Volianitis S, Dawson EA, Yoshiga CC & Secher NH (2004). J Physiol **557**, 331-342.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C21

Computer estimation of the cortical silent period following transcranial magnetic stimulation using the cumulative sum technique and comparison with other methods in humans

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The contralateral electromyographic (EMG) silent period (CSP) following transcranial magnetic stimulation (TMS) has been used as a measure of intracortical inhibition (ICI) and is particularly useful when the conditioning-test method is impractical. We have designed an automated method based on the cumulative sum (Cusum) statistic (Davey et al., 1986) to estimate the CSP duration. This was evaluated against the conventional manual method performed by two independent experts (MM1 and MM2) and against two automated methods based on statistical difference (AM1) and mean cumulative difference (AM2) (Nilsson et al., 1997; Garvey et al., 2001).
With local ethics approval and consent, six adult volunteers were recruited. Surface EMGs were recorded from the right thenar during 20% maximum voluntary contraction. Trials of 20 magnetic stimuli were delivered using a Magstim 200 stimulator connected to a 9cm circular coil centred over the vertex. TMS intensity was selected by varying the TMS output in 5% steps so that a clear CSP could be identified at the lowest stimulus intensity. Analysis was performed offline using Signal Software (Cambridge Electronic Design).

The mean CSP durations were: 23.3ms (± 6.4) by Cusum, 22.0ms (± 6.4) by MM1, 19.3ms (± 6.4) by MM2, 18.4ms (± 6.8) by AM1 and 15.3ms (± 6.7) by AM2. The intraclass correlation coefficient (ICC) between MM1 and MM2 was 0.88 ($P<0.001$). Of the 3 automated methods, the Cusum showed the strongest correlation with the MM1 (ICC 0.98, $P<0.001$) and MM2 (ICC 0.87, $P<0.001$). CSP durations were significantly different (repeated measures ANOVA with Bonferroni adjustment; $P < 0.05$) between Cusum and both expert raters (MM1; MM2). When the stimulus intensity was increased by 5% of maximum stimulator output (MSO), the ICC between all methods increased to 0.72 compared to 0.45 at the test intensity. At 5% MSO below test intensity, ICC between all methods decreased to 0.33 and there was no longer a significant correlation between MM1 and MM2.

In conclusion, at higher intensity, both human raters and automated methods become more reliable while at lower intensity, when the ICI is barely detectable, all methods become less reliable. Overall, of the automated methods, the Cusum is best correlated with expert human raters and is a simple, graphical method of detecting CSP that can be easily automated.

Davey NJ, Ellaway PH, Stein RB (1986) Journal of Neuroscience Methods 17:153-166.

Garvey MA, Ziemann U, Becker DA, Barker CA, Bartko JJ (2001) Clinical Neurophysiology 112:1451-1460.

Nilsson J, Panizza M, Arieti P (1997) Journal of Clinical Neurophysiology 14:136-143.

International Spinal Research Trust

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C22

Corticospinal tract projection to erector spinae muscles is related to handedness in human beings.

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Moving the arm away from the body alters the centre of gravity and calls for truncal muscle activity to maintain stability. Corticospinal drive to the erector spinae (ES) muscles increases during contralateral arm abduction and led to speculation that activation of an arm and the opposite back muscle could originate from the same cortical hemisphere (Davey et al., 2002). Individuals use one arm in preference to the other for single handed activities and we now ask whether symmetry of cortical drive to ES muscles is related to handedness.
With local ethical approval six right handed and four left handed healthy individuals were recruited. Motor evoked potentials (MEPs) to transcranial magnetic stimulation (TMS) of the motor cortex were recorded bilaterally from the ES muscles at L4 spinal level. TMS was applied using a MagStim 200 stimulator connected to a double cone coil with its cross-over located over the vertex. The subjects were seated and asked to maintain 90° arm abduction against weak variable resistances of 2N, 4N, 6N and 8N applied at the wrist. Threshold (T) TMS intensity for MEPs in ES muscles was determined at 2N abduction. Six stimuli at 1.1T were delivered at each of the four force levels when the right arm was abducted. This procedure was repeated with the left arm abducted. The area of the averaged rectified MEPs in each trial was measured. In order to assess any bias to one or other ES muscle while excluding the effects of muscle bulk, a ratio was calculated in each trial; the MEP area in the ES muscle during contralateral arm abduction (Carm) was divided by the MEP area during ipsilateral arm abduction (Iarm).
In right handed individuals, the mean (± SEM) Carm/Iarm ratio was larger (Student's paired t-test; $P < 0.05$) in the left ES (2.55

± 0.45) than in the right ES (1.58 ± 0.15). Conversely, the left handed individuals showed a larger (but insignificantly so; $P > 0.05$) Carm/Iarm ratio in the right ES (1.60 ± 0.37) than in the left ES (1.00 ± 0.11). Furthermore, the mean Carm/Iarm ratio in the left ES was significantly larger ($P < 0.05$) in right handed subjects than in left handed individuals. However, the mean Carm/Iarm ratio in the right ES was no different ($P > 0.05$) between right and left handed subjects.
We conclude that the drive from the motor cortex to the ES muscles involved in stabilising the body during arm abduction is stronger during movement of the dominant arm. This raises the possibility that continued use of one arm in preference to the other could lead to asymmetry in the activation of paraspinal muscles which is a known link with low back pain.

Davey NJ, Lisle RM, Loxton-Edwards B, Nowicky AV, McGregor AH (2002) Spine 27:1355-1360.

International Spinal Research Trust

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C23

A cortico-cortical mechanism mediating object-driven grasp in humans

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Recent animal studies have shown strong facilitation from ventral premotor cortex (area F5) on late I-wave components evoked by test stimuli applied to motor cortex. (Shimazu et al., 2004). To investigate a possible grasping-related modulation of the late I-waves in humans we used a TMS paired pulse paradigm (Ziemann et al., 1998) that selectively facilitates I-wave components of the MEP.
Volunteers (thirty right-handed subjects, aged 19-32) were randomly presented with one of two different objects: a disc or a vertically oriented handle. The subjects were instructed to grasp the object or remain at rest in response to an auditory cue. Surface EMG activity recorded from the right 1DI and ADM muscles during the grasp showed high specificity of individual muscle activity for the grasped object (ANOVA $p=0.0001$). Single or paired (first stimulus suprathreshold and second subthreshold) TMS pulses were delivered to the left motor cortex through a figure-of-eight coil at interstimulus intervals (ISI) of 1.3, 2.1, 2.5, 3.3 and 4.1 ms, during object presentation with the hand at rest. We found a clear and highly significant modulation of the MEP obtained with the 2.5 ms ISI that showed a significant interaction between object and muscle (ANOVA $p=0.001$). This enhancement preceded the grasping movement by at least 600 ms and was correlated significantly with the subsequent EMG activity for hand shaping prior to object contact (t-test, ADM $p<0.05$; 1DI $p<0.05$).
Control experiments showed no modulation was found during object presentation alone, during mental imagery of the grasp action or during the performance of non-object driven movements which employ a similar pattern of muscle activity.
Our study has pinpointed significant facilitation at an ISI of 2.5 ms, which is likely to reflect augmented I2 corticospinal activity, in the period preceding a planned grasp. This approach allows us to investigate a specific cortico-cortical mechanism that probably subserves the transformation of cortical representation of the geometrical properties of an object to grasp-specific outputs from motor cortex that shape the hand.

Shimazu H et al 2004 J Neurosci 4;24:1200-11

Ziemann U et al 1998 J Physiol 511;1:181-90

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Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C24

How good is spatial working memory of arm position in man?

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Everyday experience suggests that individuals are adept at returning their limb to a remembered target position, for example, to pick up a glass of water. The remembered limb position is involved in establishing the motor programme for the next reach and does not modify the reach once it has started (Heath et al., 2004). This study has investigated the relative importance of vision, proprioception and efference copy of the motor command in laying down this memory.
With local ethical approval, 20 healthy subjects (aged 21-23) were recruited and seated 27 cm from a target board printed on A2 paper; a start marker was placed 20 cm in front of the subject. The target board consisted of 5 randomly positioned numbered red circular targets 2 cm in diameter and with a black centre. Each trial consisted of 25 reach tasks carried out in random order. Each reach task was carried out in three stages cued by three tones 3 seconds apart. With the index finger of the dominant hand on the start marker the hand moved to the selected target on the first tone, on the second tone the experimenter passively moved the hand back to the start marker, on the third tone the subject actively moved their hand back to the target. Absolute error was measured as distance from the centre of the target. Subjects carried out the tasks both with and without blindfolds and with either active or passive initial placement of the hand on the target. In the blindfolded active trials the target position was established by the initial active reach. Blindfolded active trials were also carried out in the non-dominant hand.
With vision, there was no difference (repeated measures ANOVA; $P > 0.05$) between the mean (±SEM) errors in active (1.4 ± 0.1 mm) and passive (1.3 ± 0.1 mm) trials although they were each very much more accurate ($P < 0.05$) than their corresponding blindfolded trial. When blindfolded, the active trials had a smaller ($P < 0.05$) error (26.9 ± 1.6 mm) than the passive trials (31.7 ± 1.6 mm). Furthermore, the blindfolded active trials performed

using the dominant hand had a lower ($P < 0.05$) error (26.9 ± 1.6 mm) compared with the non-dominant hand (31.7 ± 2.1 mm). In this experimental scenario, limb position is clearly predominantly driven by vision. However, when blindfolded, actively positioning the hand produces a more accurate memory of target position than passively positioning the hand; this suggests that an efferent copy of the motor command may be held in memory. Memory for trials performed with the dominant hand produces a lower error than with the non-dominant hand; this could be due to a relative paucity of sensorimotor control of the non-dominant hand.

Heath M, Westwood DA and Binsted G (2004) Motor Control 8:76-106.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C25

Selective influence of muscle mechanoreceptors on cardiac vagal activity humans

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We have previously shown that activation of muscle receptors by passive stretch (PS) increases heart rate (HR) with little change in blood pressure (BP) (Gladwell and Coote, 2002). We propose that the muscle mechanoreceptors inhibit cardiac vagal activity and therefore alter the baroreceptor-HR reflex. In this set of studies we attempted to test this by performing PS during alterations in vagal tone. A large decrease in vagal tone was caused both pharmacologically and by using mild rhythmic exercise. Further, milder alterations in vagal tone were achieved by altering carotid baroreceptor input: using neck pressure (NP) and neck suction (NS) to cause a small decrease and increase, respectively. Following local health authority ethical approval, fourteen (mean± SD, 21.9±2 years) healthy human volunteers participated in four different studies. In all experiments subjects lay supine allowing PS of the triceps surae. BP (FINAPRES), ECG, and respiration were recorded. Paired t-tests were performed between the 2 conditions within each study ($P \leq 0.05$). PS alone caused a significant decrease ($P \leq 0.05$) in RR interval 962±76ms at baseline compared to 846±151ms with PS, with a reduction in heart rate variability but without a significant change in BP. In study 1, (n=3), glycopyrrolate was infused i.v. (5μg/kg/h) following a bolus dose of 300 μg/kg. In study 2, (n=5) PS was performed with and without rhythmical handgrip (10% MVC). The decrease in RR interval with PS was significantly decreased by reducing vagal output using glycopyrrolate during PS (−8.1±4.5ms) compared to PS alone (−54±11ms) and by applying PS during handgrip (10±10ms) compared to PS alone (−74±15ms; $P \leq 0.05$). In study 3 and 4 (n=8), PS was applied with NP and NS. Interestingly, reducing baroreceptor input with NP resulted in a small but insignificant further decrease in R-R interval in response to PS (−107±17ms) compared to PS alone (−96±13 ms; $P > 0.05$). Conversely, increasing baroreceptor input with NS during PS there was a significantly smaller decrease in R-R interval (−39±5.5 ms) compared to PS alone (−86±17ms) ($P \leq 0.05$). BP was not significantly different in any of the four studies ($P > 0.05$). These data support the idea that muscle mechanoreceptors alter the excitation of cardiac vagal neurones. Further, it is likely that in this experimental model the carotid baroreceptors and the muscle mechanoreceptors interact at the level of the cardiac vagal neurones rather than earlier in the baroreceptor pathway at the nucleus tractus solitarii.

Gladwell, V.F. and Coote, J.H. (2002). J.Physiol. 540.3.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C26

Dynamic Characteristics of Elderly Human Muscle: Adaptations to Resistance Training

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The decline in dynamic strength with age is closely associated with a deterioration in functional performance (for review see Macaluso & De Vito 2004). Whilst resistive training in older adults has been shown to increase strength (e.g., Fiatarone, *et al.* 1990), little is known on the training-induced adaptations in the torque-velocity (T-V) relation. Hence, the present study investigated the effect of strength training on the T-V relation in elderly humans and the origin of any changes.

After receiving ethics committee approval, older adults were assigned to training (mean±SD age: 74.3±3.5 years; *n*=9) and non-training control (age 67.1±2 years; *n*=9) groups. Knee-extension and leg-press exercises (2 series of ~10 repetitions at 80% of the 5-repetition maximum) were performed 3 times/week for 14 weeks. Maximal isokinetic knee extension torque was assessed during concentric and eccentric contractions at angular velocities of 0.87, 1.75, 2.62 and 3.49 rad s^{-1}. Agonist-antagonist muscle activation was assessed using electromyography. Vastus lateralis (VL) muscle architecture (fascicle length and pennation angle) was examined *in vivo* using ultrasound imaging. VL muscle fascicle force-velocity (F-V) relation was estimated using a muscle-model taking into account muscle architecture, the contribution of the VL to knee extension force, patella tendon moment arm length and antagonist coactivation. Data were analysed using factorial analysis of variance, with a Sheffé post-hoc test applied where necessary.

Training increased concentric torque by 22-37% across the four angular velocities ($P<0.01$), but failed to modify eccentric torque ($P>0.05$; Fig.1). Fascicle force increased by 28, 26, 46 and 50% at shortening velocities corresponding to 0.87, 1.75, 2.62 and 3.49 rad s^{-1}, respectively ($P<0.01$).

Increased agonist muscle activation, increased fascicle lengths and greater elastic energy recovery from tendinous structures may explain the gain in concentric torque, whilst the lack of changes in eccentric torque and fascicle force likely reflects the preservation of eccentric force with ageing (Hortobagyi, *et al.* 1995). The present findings are expected to positively influence dynamic muscle performance in old age, such as stairs negotiation.

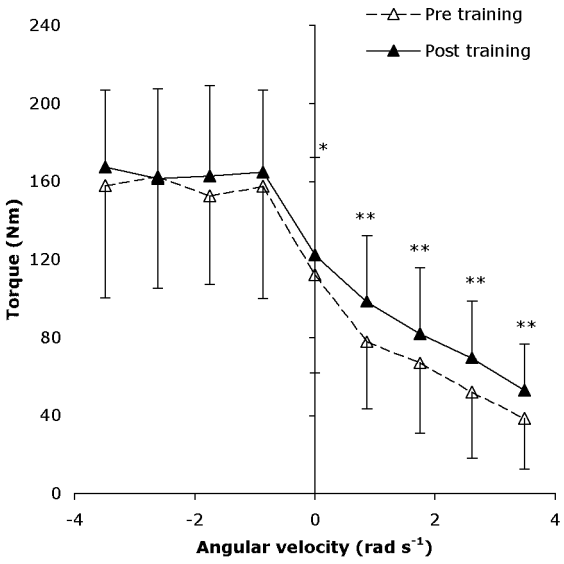

Figure 1. Knee extensor torque-velocity relation for the training group. Values are means and SD, significantly increased torque (*P < 0.05 and **P < 0.01) post training.

Fiatarone MA *et al.* (1990). *JAMA* **263**, 3029-3034.

Hortobagyi T *et al.* (1995). *J Gerontol A Biol Sci Med Sci* **50**, B399-406.

Macaluso A, De Vito G (2004). *Eur J Appl Physiol* **91**, 450-472.

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Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C27

Physiological cross sectional area and specific force are reduced in the gastrocnemius of elderly men

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Sarcopenia and muscle weakness are well known consequences of ageing. The aim of the present study was to ascertain whether the age associated muscle weakness was also due to a decrease in intrinsic muscle force.

In vivo physiological cross sectional area (PCSA) and specific force of the lateral head of the gastrocnemius muscle (GL) were assessed in elderly males (EM, aged 73.8 ± 3.5 years, height 173.4 ± 4.4 cm, weight 78.4 ± 8.3 kg, n=19, mean ± SD) and in young males (YM, aged 25.3 ± 4.4 years, height 176.4 ± 7.7 cm, weight 79.1 ± 11.9 kg, n=12). Procedures were approved by the Manchester Metropolitan University Ethics Committee. GL muscle volume (VOL) and Achilles' tendon moment arm length were evaluated using magnetic resonance imaging. Pennation angle (θ), and fibre fascicle length (Lf) were measured using B-mode ultrasonography during isometric maximum voluntary contraction of the plantarflexors (MVC). PCSA was estimated as VOL/Lf. GL fascicle force was calculated from the tendon force component (=tendon force/cos θ) after accounting for agonist and antagonist activation level (assessed by twitch-interpolation and EMG recordings), Achilles' tendon moment arm length and the relative PCSA of the GL within the plantarflexors group. GL specific force was calculated as fascicle force/PCSA.

Voluntary activation of the GL muscle was lower in the EM than in the YM (86 ± 7 % vs. 98 ± 2 %, respectively, P<0.05: one-way ANOVA). Compared to the YM, plantarflexor MVC torque and fascicle force of the EM were lower by 47 % and 40 %, respectively (P<0.01). Both VOL and PCSA were smaller in the EM by 28 % (P<0.01) and 16 % (P<0.05), respectively. Pennation angle was 12 % smaller in the EM, whereas there was no significant difference in Lf between the YM and EM. Remarkably, after accounting for differences in agonists and antagonists activation, the net specific force of the EM was 30 % lower than that of the YM (P<0.01). Thus demonstrating that the loss of muscle strength with ageing may not only be explained by a reduced voluntary drive to the muscle but mostly by a decrease in intrinsic muscle force. This phenomenon is likely due a reduction in single fibre specific tension; however the role of age-induced changes in tendon stiffness cannot be excluded.

Supported by EU Framework V funding ('Better-Ageing' Project, No. QLRT-2001-00323).

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C28

Measurement of postural stability during walking

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Assessment of gait and postural stability is often qualitative; gait laboratory access is rare (Toro, Nester, & Farren 2003). Common quantitative measures (standing sway and spatiotemporal walking parameters) have limited relevance to postural stability during activity (Moe-Nilssen 1998a); accelerometers have been suggested for this purpose (Moe-Nilssen 1998b). This preliminary study investigated walking stability in older people using accelerometry (King's College REC approved).

Eight subjects were assessed; four healthy older adults (aged 71±4yrs, BMI 24.7±1.2) and four with a history of either a fall or a trip (aged 71±4.5yrs, BMI 29.6±6.1). Accelerometers recorded vertical and mediolateral (ML) acceleration at the ankle and L5 (near the centre of mass, COM) during self-selected 'slow','comfortable' and 'fast' walking wearing shoes. Postural stability was quantified using standard deviation (S.D.) of acceleration (normalised for walking speed) for each of 5 strides per condition. Results were analysed using SPSS and MathCad. ANOVA tests were used for between group comparisons. Significance was set at P<0.05.

Average walking speed was significantly slower for the fallers (mean±S.D.) 0.84±0.37 m/s compared to control older subjects

1.41±0.41 m/s. Walking speed and gait cycle segment acceleration SD's are presented in Table 1. After normalising for walking speed, vertical and ML acceleration SD's at the ankle were significantly higher for the fallers for all walking types. Vertical spinal acceleration SD's were only significantly increased for the fallers during slow walking. ML spinal measures were significantly increased for the fallers for slow and comfortable speed walking trials.

Nine out of twenty-four measures of acceleration recorded at either the ankle or near the COM were able to significantly distinguish fallers or subjects who had experienced a trip from age matched non-fallers. The increased deviation of gait cycle acceleration near the COM during walking in the group of fallers indicate poor dynamic postural stability while measures at the ankle may also reflect unsteadiness in intended movement.

Accelerometers appear to allow sensitive, inexpensive and portable analysis of dynamic postural stability in elderly people.

			Single stride acceleration SD (m/s/s)			
		Walking speed	Ankle V	Ankle ML	L5 V	L5 ML
Walk & Gp	n	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)
Slow EC	20	0.96(0.16)	3.23(0.88)	1.82(0.48)	1.24(0.33)	0.73(0.15)
EF	13	0.74(0.33)	3.07(0.96)	2.02(1.20)	1.03(0.50)	0.77(0.20)
Self pace EC	20	1.38(0.14)	5.19(1.26)	2.98(0.73)	2.22(1.03)	1.10(0.21)
EF	16	0.91(0.33)	3.80(1.15)	2.69(1.57)	1.48(0.65)	0.90(0.24)
Fast EC	20	1.89(0.15)	6.69(1.27)	4.20(0.74)	3.22(1.08)	1.76(0.38)
EF	8	1.45(0.14)	5.93(0.36)	4.92(0.91)	2.60(0.64)	1.39(0.30)

Table 1. Walking speed and SD (within stride standard deviation) and s.d (deviation between strides/subjects) of ankle and spinal (L5), vertical (V) and ML acceleration over one gait cycle during slow, self pace and fast walking completed by 4 elderly control subjects (EC) and 4 elderly fallers (EF). n denotes total number of gait cycles analysed per group.

Moe-Nilssen, R. 1998a, "A new method for evaluating motor control in gait under real-life environmental conditions. Part 1: The instrument", Clinical Biomechanics, vol. 13, no. 4-5, pp. 320-327.

Moe-Nilssen, R. 1998b, "A new method for evaluating motor control in gait under real-life environmental conditions. Part 2: Gait Analysis", Clinical Biomechanics, vol. 13, pp. 328-335.

Toro, B., Nester, C. J., & Farren, P. C. 2003, "The status of gait assessment among physiotherapists in the United Kingdom", Archives of Physical Medicine and Rehabilitation, vol. 84, no. 12, pp. 1878-1884.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C29

Limit of endurance time in a non-exercising arm is shortened by exercising the other arm in healthy men

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Stimulation of the motor cortex using transcranial magnetic stimulation (TMS) and electromyographic (EMG) recordings has shown corticospinal excitability to be depressed following exercise (Brasil-Neto et al., 1994). When the exercise is exhaustive, depression of motor evoked potentials (MEPs) can also be seen in homonymous non-exercising muscles but appears to have little measurable effect on performance (Humphry et al., 2004). We have now induced central fatigue in non-exercising muscles and measured its influence on limit of endurance time (LOET).

With local ethical approval and informed consent, ten healthy male volunteers (right-handed, aged 20-24 years) visited the laboratory for two sessions one week apart and bilateral surface EMG recordings taken from biceps brachii (BB) muscles. In session 1, a 4kg weight was strapped to the left wrist and subjects performed left-armed biceps curls to exhaustion, in time with a tone repeating at 1.5 seconds interval. Time to exhaustion (without prior right arm exercise) was measured as the LOET. In session 2, a 5kg weight was strapped to the right wrist and subjects performed right-armed curls to exhaustion. Subjects were given five minutes rest before the 4kg weight was attached to the left wrist and left-arm curls were again performed to exhaustion and a post-exercise LOET was recorded. TMS was applied using a MagStim 200 stimulator connected to a 9-cm circular coil centred over the vertex and MEPs were monitored bilaterally during both sessions. In session 1, mean (±SEM) MEP areas were significantly ($P < 0.05$; Student's paired t-test) reduced to 31 ± 5% of baseline in the left (exercising) BB and to 55 ± 5% of baseline in the right (non-exercising) BB; the mean LOET in the left arm was 925 ± 123 seconds. In session 2, after exercising the right arm, mean MEP areas were significantly reduced to 29 ± 5% (right BB) and 79 ± 6% (left BB) of baseline values. The mean LOET in the left arm was significantly reduced to 565 ± 64 seconds (65 ± 4% of that measured before right arm exercise). The reduction in MEP area in the right arm did not correlate with the LOET (Pearson's correlation, $P > 0.05$). This study has confirmed that, following exhaustive levels of exercise, depression of MEP responses occurs in the non-exercising as well as the exercising BB. The LOET fell in the non-exercising BB. We conclude that reduced tonic corticospinal excitability may result in a reduced LOET.

Brasil-Neto JP, Pascual-Leone A, Valls-Sole J, Cammarota A, Cohen LG, Hallett M (1993) Experimental Brain Research 93:181-184.

Humphry AT, Lloyd-Davies EJ, Teare RJ, Williams KE, Strutton PH, Davey NJ (2004) European Journal of Applied Physiology 92:211-218

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C30

Continuous core temperature responses of man to mass participation distance running in heat

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Distance running in heat and humidity elevates body core temperature (Tc), limits heat dissipation, and increases the risk of heat illness (Armstrong et al. 1996). Post-race rectal temperature has traditionally quantified Tc rise during outdoor running (Cheuvront & Haymes, 2001). The invasive, obtrusive, and impractical nature of rectal thermometry has restricted continuous measurement of Tc during outdoor running to a few case-studies (e.g. Maron et al. 1977). The present study aimed to continuously measure Tc using ingestible telemetric

temperature-sensing pill technology in a sample of runners competing in a 21-km race in environmental conditions representing a high-risk of heat illness. Volunteers were 18 heat acclimatized male soldiers (aged 20-23 years) from the Singapore Armed Forces participating in the 2003 Singapore Army Half-Marathon. Each volunteer ingested a telemetric temperature-sensing pill (8-10 hours before running) and wore an ambulatory data recorder and heart rate (HR) monitor for the continuous measurement of Tc and HR, respectively. Pre to post-race changes in nude body mass quantified fluid balance. Environmental wet bulb globe temperature was 26.0°C at race start (0600 h) and 28.3°C at the time of the last finishing volunteer (0826 h). All volunteers finished the race asymptomatic in a mean ± SEM (range) time of 118 ± 3 (105-146) min. During the initial 30-min of running Tc rate of rise was 3.1 ± 0.2 (1.3-4.0)°C·hr. Ten volunteers achieved peak Tc during the race whereas eight volunteers achieved peak Tc at race finish. Peak Tc was 40.1 ± 0.2 (39.3-41.7)°C at 86 ± 8 (13-130) min and final Tc was 39.9 ± 0.2 (38.3-41.7)°C. All volunteers demonstrated Tc > 39°C; 56% (n=10) Tc > 40°C; and 11% (n=2) Tc > 41°C. Three general patterns of Tc response demonstrating large inter-subject variability were observed: 1) increase followed by steady state (n=7); 2) continual increase until race finish (n=5); and 3) increase followed by marked decline (n=6). The rate and magnitude of Tc response was unrelated (Pearson correlation coefficient, $P > 0.05$) to average running velocity for 21-km or any fluid balance variable (e.g. % dehydration, % sweat loss replaced). Peak Tc was significantly and positively related to peak ($r = .72$, $P < 0.01$) and mean ($r = .57$, $P < 0.05$) HR. Telemetric temperature pill technology demonstrated significant practical utility for continuous Tc measurement in unrestricted ambulatory subjects. Temperature responses demonstrated large inter-subject variability in magnitude and temporal nature, which were unrelated to fluid balance and race time.

Armstrong LE et al. (1996.) Med. Sci. Sports Exerc. 28, I-X.

Cheuvront SN & Haymes EM (2001) Sports Med. 31, 743-762.

Maron MB et al. (1977). J. Appl. Physiol. 42, 909-914.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C31

The Effects of Face-Cooling on the Perception of Exertion and Neuroendocrine Response to Hyperthermic Exercise

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Compared to cool conditions, exercise undertaken in the heat is associated with higher ratings of perceived exertion (RPE) and a greater neuroendocrine response, as evidenced by levels of circulating prolactin (PRL). For the same core temperature, exercise in the cool environment has lower RPE and PRL values (Bridge et al, 2003) suggesting that skin temperature may modulate the response to raised core temperature and that there may be a common mechanism linking the RPE and PRL responses. The face may be particularly effective in mediating the PRL response to skin cooling (Brisson et al, 1991) and we have investigated whether face cooling alone has similar effects on RPE and PRL responses to hyperthermic exercise. Following Local Ethics Committee approval and obtaining informed consent, ten, non heat-acclimatised volunteers (8 male, 19-26years; VO2max: 55±2 ml·kg·min-1) exercised for 40 min on a cycle ergometer at 65% maximum power output, at an ambient temperature of 35°C (relative humidity: 30%) with (FC) and without face-cooling (CON), in a randomised cross-over design. Forehead temperature was maintained ~6°C lower with FC whilst mean skin temperature for other parts of the body was ~0.5°C higher. A relative bradycardia was observed with heart rate during FC being approximately 5 bpm lower than CON. Blood glucose did not differ between trials but lactate was lower with face-cooling (FC 5.1±0.6; Con 6.5±0.7 mmol/l). Rectal temperature increased by 1.5±0.1°C with the same time course in both conditions. Levels of prolactin were maintained at ~150 miU/L for FC while values for CON increased to ~350miU/L so that towards the end of the exercise, for the same rectal temperature, PRL was significantly lower in the FC condition (Fig. 1; $p<0.05$; repeated measures ANOVA). No differences were observed between trials for global RPE although a separate rating for ventilation was reduced with FC.

We confirm that the neuroendocrine response to exercise in the heat is sensitive to face cooling but find that the temperature of the face may be only one component of the overall perception of exertion when exercising in hot conditions.

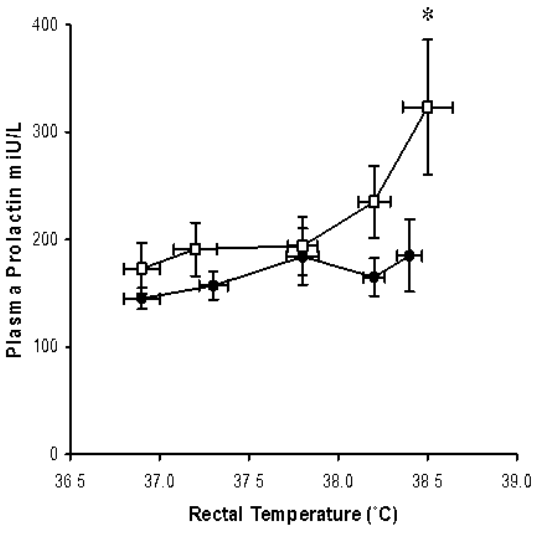

Fig. 1 Plasma prolactin concentrations in relation to rectal temperature during exercise at 35°C. ■CON; ●dFC. Data are mean ± S.E.M. *Indicates value significantly higher than FC, $p<0.05$.

Bridge et al. (2003). Exp. Physiol., 88, 627-635.

Brisson et al. (1991). J. Appl. Physiol. 70, 1351-1355.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C32

Metabolic cost of walking in elderly humans: effect of exercise training

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The elevated metabolic cost of walking (C_W) in elderly adults has been hypothesised to be a consequence of the declines in muscle mass, strength and stability (Malatesta et al, 2003). Exercise training partially reverses these declines; does it influence C_W?

Community dwelling elderly adults were assigned to a training (TRA, mean ± SD age = 73 ± 3, n = 22) or a non-training control group (CON, age = 73 ± 4, n = 12). The intervention lasted 12 months. TRA performed 1 hour supervised exercise sessions twice per week. Leg press, leg extension, calf raise, chest press, and seated row exercises were performed on resistance machines (2-3 sets of 8-10 repetitions at 80-100% of 8 repetition maximum) in addition to aerobic, balance, and flexibility exercises. In addition to supervised sessions, home based sessions utilising resistance bands and light aerobic exercises were performed once per week. Before and after the intervention, to assess C_W, the net (gross – standing) oxygen required to walk unit distance was measured using an automated analyser (Cosmed K4b^2) whilst participants walked on a motor driven treadmill at 0.83, 1.11 and 1.39 m/s. Isometric knee extensor muscle strength (torque at 90 deg), single leg balance time, and 6 min walk distance were also measured to assess changes in traditional measures of functional capacity. Interactions between group and time were assessed using ANOVA. The study had local ethical committee approval.

At baseline, mean C_W of the elderly adults in this study was 34% higher than in a convenience sample of young adults (age = 27 ± 3, n = 23). However, the intervention had no effect on C_W in either TRA or CON. This was despite improvements in mean knee extensor muscle strength, balance time, and 6 min walk distance in TRA (+20, +12 and +6%, respectively) that were greater than changes in CON (+5, -28 and 0%) ($F_{group*time}$, $P < 0.05$).

In conclusion, training-induced improvements in muscle strength and balance performance were not accompanied by improvements in walking economy. These findings do not support the hypothesis that the decline in muscle mass and strength in healthy elderly adults are responsible for the elevation in C_W. Task specific training may be required to reduce the elevated metabolic cost of movement.

Malatesta D *et al.* (2003). *J Appl Physiol* **95**, 2248-2256.

Supported by European Commission Framework V funding (Better-Ageing Project, No. QLRT-2001-00323)

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C33

Effect of maternal melatonin suppression during gestation on capuchin monkey (*Cebus apella*) fetal adrenal gland function

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Chronic maternal melatonin suppression by exposure to constant light in the last third of gestation, increases plasma cortisol concentration in the capuchin monkey early newborn (1). Melatonin inhibits ACTH-induced 3B-HSD expression and cortisol production by fetal adrenals in culture (1). To asses whether the increased cortisol in the newborn was due to effects on the fetal adrenal we studied the effects of chronic maternal melatonin suppression on: fetal adrenal weight and mRNA levels of 3B-HSD and the steroidogenic enzymes StAR, 11B-HSD1 and CYP21 and on *in vitro* steroid production (cortisol, progesterone and cortisone). Six pregnant female were maintained in constant light during the last third of gestation; 3 received a placebo (LL) and 3 received 500 ug/kg of melatonin daily (LL+Mel). 3 pregnant females in Light:Dark (14:10) were used as control. At 90% of gestation the females were sedated with ketamine (10 mg/kg) and anaesthetized with 1% halothane in oxygen. The fetuses were delivered by hysterotomy and immediately killed by an i.p. overdose of sodium thiopentone (100 mg/kg) (the remaining tissues from fetuses were frozen and stored at -80°C for later use). Fetal adrenals were dissected, weighed and cut in small explants (15 mg). Three explants were used to measure mRNA levels of 3B-SHD and steroidogenic enzymes (RT-PCR). The remaining explants were incubated 48-h in basal condition and plus 100 nM ACTH to measure steroids production in the supernatants (RIA). Approval was obtained from the local Ethics Committee.

Results: Maternal melatonin supression (Table1) decreased fetal adrenal weight and increased 3B-HSD mRNA levels. Basal and stimulated, progesterone and cortisone production were increased, consistent with an increased 3B-HSD activity. These effects were reversed by maternal melatonin replacement. The treatments did not affect fetal weight, mRNA levels of steroidogenic enzymes or *in vitro* basal and stimulated cortisol production.

Our results show a role of maternal melatonin in the regulation of the primate fetal adrenal gland. We speculate that maternal melatonin during gestation may have long term effects setting adrenal function for postnatal life.

Table1: Effects of maternal melatonin suppression (means ± SE)

	Control	LL	LL + Mel
Fetal adrenal weight (mg)	486±19	390±26*	522±38
3B-HSD (mRNA levels relative 18S-rRNA)	1,1±0,4	7,9±3,3*	2,3±0,1
Basal progesterone(ng/mg tissue)x 48-h	0,23±0,04	0,9±0,1*	0,20±0,06
Basal cortisone(ng/mg tissue) x 48-h	4,8±0,3	10,2±0,4*	6,8±0,2

* vs other treatments, p<0.05 ANOVA

Torres-Farfan C *et.al.* (2004). JPhysiol **554**, 841*P*.

Supported by Fondecyt 2010140, SBMC.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C34

Effect of inspiratory muscle work on exercise-induced quadriceps muscle fatigue in healthy humans

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During maximal exercise, the high demand for respiratory muscle blood flow compromises blood flow to working limb locomotor muscles because of sympathetically mediated vasoconstriction (Harms *et al.*, 1997). We hypothesised that partially unloading the inspiratory muscles would ameliorate exercise-induced quadriceps muscle fatigue and, conversely, that inspiratory muscle loading would exacerbate quadriceps fatigue.
Following local ethics committee approval and written informed consent, 8 male cyclists (mean±S.E.M. peak oxygen uptake [VO_{2peak}] 57.8±3.5 ml kg^{-1} min^{-1}) exercised at ≥90% VO_{2peak} to exhaustion (CTRL). At a separate visit, subjects exercised for the same time as during CTRL (13.2±0.9 min) but the work of breathing (WOB) was reduced using a proportional assist ventilator (PAV), which delivered positive pressure during inspiration in proportion to airflow and volume. Subjects also exercised to exhaustion (9.2±1.4 min; $P<0.05$ *vs.* CTRL) on two separate occasions while WOB was increased via inspiratory resistive loads (IRL). Two additional exercise tests were performed whereby subjects exercised for the same time as that achieved during IRL but with breathing unimpeded (IRL-CTRL). Quadriceps twitch force, in response to supramaximal paired magnetic stimuli of the femoral nerve (1-100 Hz), was assessed pre- and up to 70 min post-exercise.

The flow-resistive WOB, assessed as the integrated area of the oesophageal pressure-tidal volume loop, was 56±6% lower with PAV and 79±12% higher with IRL compared with control isotime values. The post-exercise decrease in quadriceps twitch force was attenuated with PAV (-20±5% for PAV *vs.* -28±5% for CTRL; $P<0.05$, paired-samples *t*-test). Repeated measures ANOVA (visit×perturbation) identified a significant within-group effect of IRL upon quadriceps twitch force (-20±7% for IRL *vs.* -13±8% for IRL-CTRL; $P<0.05$). The blood lactate response to exercise was not different ($P>0.05$) between either IRL and IRL-CTRL (9.0±0.7 *vs.* 9.2±0.7 mM) or PAV and CTRL (9.2±0.6 *vs.* 10.0±1.1 mM). Perceptual ratings of dyspnoea and limb discomfort (Borg CR10) were reduced with PAV and increased with IRL ($P<0.05$). The present results indicate the importance of WOB on locomotor muscle fatigue. The increase and decrease in quadriceps fatigue with inspiratory muscle loading and unloading, respectively, may have been due to a sympathetically mediated redistribution of blood flow in response to changes in the WOB (Harms *et al.*, 1997). The effect of WOB on muscle fatigue may also explain our previous finding that WOB affects exercise performance (Harms *et al.*, 2000).

Harms CA *et al.* (1997) *J Appl Physiol* **82**, 1573-1583.

Harms CA *et al.* (2000) *J Appl Physiol* **89**, 131-138.

Study funded by NHLBI.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C35

Clock gene expression in adult and fetal suprachiasmatic nucleus (SCN) and adrenal gland in the capuchin monkey

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There is limited information about the development of the primate circadian system. In adult mammals, circadian oscillation is driven by a transcription-transduction feedback loop of the clock genes Bmal1-2, Per1-3, Clock, Cry1-2. We investigated in adult and fetal capuchin monkey 1) expression of Bmal1, Per2, Clock and Cry2 in SCN and adrenal gland, 2) oscillatory expression of Bmal1 and Per2 in SCN and adrenal gland *in vivo* and 3) whether, Bmal1 and Per2 show an oscillatory expression in adrenal gland *in vitro*.
We obtained SCN and adrenal glands from 8 adult male capuchin monkeys sedated with ketamine (10 mg/kg) and euthanized with i.v. sodium thiopentone (100mg/kg) at 0200, 0800, 1400 (n=3) and 2000 hours (n=3) and from 7 late gestation fetuses delivered by histerotomies at 1400 (n=4) and 2000 hours (n=3), euthanized by injection of sodium thiopentone (100mg/kg) in the umbilical vein. mRNA levels were measured by RT-PCR using 18S-rRNA (18S)as housekeeping gene. Bmal1, Per2, Clock, Cry2 were measured in tissue obtained at necropsy and Bmal1 and Per2 in adrenal explants incubated and collected in sets, each 6 or 4 hours for 48-h from from 4 adults and 3 fetuses. Approval was obtained from the local Ethics Committee.
Bmal1, Clock, Per2 and Cry2 were expressed in adult and fetal SCN and adrenal gland. Bmal1 expression was higher in the adult adrenal gland (56.1±15.8 ng relative to 300 bp DNA standard) than in the SCN (9.9±3.3 ng), $P<0.05$, unpaired Student's t test. Per2 and 18S levels were similar. Bmal1/18S in the SCN was higher at 2000-0200 than at 0800-1400 ($P=0.06$, unpaired Student's t test), whereas in the adrenal values at 1400 and 2000 were higher than the two values obtained at 0200-0800. In the fetus, the expression of 18S and clock genes in the SCN and adrenal gland was lower than in the respective adult tissues and we did not detect differences in mRNA levels of Bmal1 or Per2 between 1400 and 2000 hours. In culture, both adult and fetal adrenals showed gene oscillation. In adult adrenal cultures Bmal1 mRNA showed a peak at 1400-2000. In fetal adrenal cultures, Per2 mRNA level showed a peak at 2400 ($P<0.05$, ANOVA).
Conclusion: Clock genes are expressed in adult and fetal capuchin monkey SCN and adrenal. We found oscillatory Bmal1 expression in adult SCN. The expression of clock genes in the adult and fetal adrenal gland and their oscillation in culture suggests that these glands are peripheral circadian oscillators.

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Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C36

The effect of exercise intensity on oxygen uptake kinetics in healthy young women

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The study of oxygen uptake kinetics (tau values) using self-paced walking as the mode of exercise may provide a convenient indirect measure of aerobic fitness. However, one concern is the effect of inadvertent differences in exercise intensity between tests upon derived tau values. Previous studies report conflicting results. The aim of this study was to examine the effect of exercise intensity on tau values derived from treadmill walking.

Six untrained healthy young (median age 22.5 years (21-24 years)) women underwent VO_2max treadmill testing (visit 1) to permit calculation of sub-maximal work-rates of 50%, 65% and 80% VO_2max. A familiarization bout (6 min at 65% VO_2max) was performed at the start of the 2nd and 3rd visits and excluded from further analysis. Subjects then performed 9 bouts of exercise (3 bouts of each intensity) over the 2nd and 3rd visits. Each 6 min exercise bout was followed by ≥20 min of seated rest. Breath-by-breath data (with spurious breaths removed) were interpolated on a second-by-second basis and time aligned to exercise onset. Ensemble averaged datasets were fitted with a monoexponential model to derive a tau value for each intensity.

Repeated measures ANOVA showed no significant linear trend ($F(1,5) = 0.24$, $p=0.883$). The within subjects coefficient of variation was 11.3% (Table 1).

In this sample of young women of comparable age and fitness, variations in tau between these exercise intensities were not physiological significant. On the basis of these findings in young individuals, any unavoidable variations in exercise intensity between tests would be unlikely to mask intervention related effects on tau values.

Table 1. Tau values (s) corresponding to each exercise intensity (50%, 65% and 80% VO_2max)

	50%	65%	80%
1	36.4	35.9	28.8
2	39.3	35.9	37.7
3	35.5	36.0	43.2
4	31.5	33.1	37.7
5	30.6	29.8	28.5
6	36.9	33.7	32.1
Median	36.0	34.8	34.9

The support of the Royal Society of Edinburgh/ Lloyds TSB and the University of Edinburgh is gratefully acknowledged.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C37

Contribution of exercise hyperpnoea to the blood lactate concentration of heavy endurance exercise

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Respiratory muscle work is not generally thought to make a significant contribution to blood lactate concentration ($[lac^-]_B$) during exercise. When minute ventilation (V_E) in maximal exercise is reproduced voluntarily at rest there is either no change in $[lac^-]_B$ (Babcock et al. 1995) or a modest 0.5 mmol·l^{-1} increase (Martin et al. 1984). However, at rest lactate production by respiratory muscles may not be reflected by an increased $[lac^-]_B$ due to clearance by other tissues. At maximal lactate steady state (MLSS), however, there is no "spare capacity" for lactate clearance, thus further lactate efflux from respiratory muscles should accumulate in the blood. We thus examined the effects of volitional hyperpnoea on $[lac^-]_B$ during exercise at MLSS.

With local ethics committee approval and written informed consent, 7 healthy males (aged 25.6 ± 1.8 yr) were studied. A lactate minimum test (Tegtbur et al. 1993) was used to estimate MLSS cycling power, which was then resolved using at least two separate 30 min constant-load tests. Thereafter, a 30 min control trial (CT) at MLSS was performed. On a further occasion (experimental trial, ET) CT was mimicked except that from 20-28 min maximal volitional hyperpnoea was performed. Isocapnia was maintained by adding CO_2 to the inspirate to maintain a constant end-tidal CO_2, which was monitored breath-by-breath. PCO_2 of arterialised venous blood ($PaCO_2$) was determined and corrected for rectal temperature changes. Data were analysed using factorial ANOVA and expressed as mean ± S.E.M. Statistical significance was set at $P < 0.05$.

MLSS power was 207 ± 8 W. Changes in V_E and $[lac^-]_B$ for the first 20 min of exercise were not different between CT and ET (Fig. 1). From 20-28 min V_E during CT and ET was 87.3 ± 2.4 and 168.3 ± 7.0 l·min^{-1}, respectively ($P < 0.01$), the latter being comparable to that achieved in the maximal phase of the lactate minimum test (171.9 ± 6.8 l·min^{-1}). From 20-30 min $[lac^-]_B$ during ET increased significantly ($P < 0.05$) by 0.9 ± 0.2 mmol·l^{-1} (+24.9%). There was also a slight 2.2 ± 0.8 mmHg increase in $PaCO_2$ from 20-30 min in ET, which may have attenuated the increase in $[lac^-]_B$.

This study shows that the work of breathing during heavy exercise influences $[lac^-]_B$, probably because of significant lactate production and release from respiratory muscles. However, respiratory muscle metaboreflex activation may have also compromised blood flow to, and thus lactate uptake by, locomotor muscles.

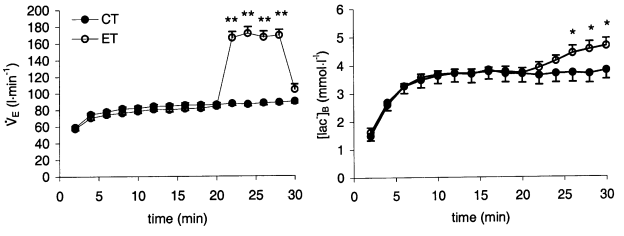

Figure 1. Changes in minute ventilation and blood lactate concentration during CT and ET. Significant difference between trials: *P < 0.05, **P < 0.01.

Babcock MA *et al.* (1995). *J Appl Physiol* **78**, 1710-1719.
Martin BJ *et al.* (1984). *Med Sci Sports Exerc* **16**, 82-86.
Tegtbur U *et al.* (1993). *Med Sci Sports Exerc* **25**, 620-627.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C38

THE EFFECT OF BODY POSITION ON ENERGY METABOLISM DURING CONSTANT LOAD CYCLING IN HUMANS

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Different body positions alter hydrostatic and colloid-osmotic pressure in the limbs. The effects of the latter on exercise metabolism have not been extensively researched. We investigated the metabolic response during constant load exercise at given absolute and relative intensities, with respect to the seated (UP) or supine body position (SUP). Following University of Essex ethics approval, eight healthy individuals [mean (SD) age 19.3 (1.05) years, height 1.75 (0.08) m and mass 70.8 (5.9) kg] performed two cycling incremental load tests for the determination of peak power (PP) and the corresponding blood lactate concentration (BLC) in UP and SUP. The incremental load tests started at a workload of 50 W and increased by 25 W every 3 min until exhaustion. Subsequently three constant load tests were randomly performed lasting 20 min or until premature termination due to exhaustion. Power output was equivalent to 65% of the PP attained in the corresponding body position incremental test: A) UP-65% peak UP test; B) SUP-65% peak SUP test; C) SUP-65% peak UP test. Aerobic constant workload conditions were identified using a generally accepted definition of a steady state of the BLC (Beneke, 2003). In the incremental load test, using paired t tests, PP and BLC were significantly higher ($p < 0.001$) in UP (187.5 (29.8) W and 8.88 (2.23) mmol l-1) than in SUP (164 (15.3) W and 6.27 (1.6) mmol l-1), respectively. In the constant load tests, A (130 (21.3) W) and B (108 (21.8) W) showed a steady state of BLC, irrespective of pedalling rate. However, four subjects did not show a steady state of the BLC with condition C (130 (21.3) W), with 3 of these tests terminated after the 8th, 10th and 11th minute, respectively. Using one-factor ANOVA and post-hoc t-tests, the BLC at test termination was not significantly different between A (3.0 (0.16) mmol l-1) and B (3.02 (0.21) mmol l-1), however, at C a higher ($p < 0.001$) level of BLC (4.8 (0.53) mmol l-1) than at A and B occurred. These results indicate that at a specific power output, the body position may shift the behaviour of the BLC from a steady-state to a non-steady state, changing the working condition from essentially aerobic to partly anaerobic. Consequently this needs to be considered for exercise testing where specific body positions are required such as in many clinical settings.

Beneke, R. (2003). Eur J Appl Physiol. 89

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C39

Effect of starvation and refeeding on insulin sensitivity and pyruvate dehydrogenase kinase expression in human skeletal muscle

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Muscle pyruvate dehydrogenase kinase (PDK) plays an important role in the development of insulin resistance in metabolic states (eg starvation) that are characterised by elevated plasma free fatty acid (FFA) levels (Wu *et al.* 1999). However, the mechanism linking insulin resistance and altered PDK expression in skeletal muscle is not clear. The aim of this study was to investigate the effect of fasting and refeeding on insulin sensitivity and gene expression of skeletal muscle PDK isoforms, peroxisome proliferator-activated receptors (PPAR) α and δ (key regulators of fatty acid oxidation) and forkhead transcription factor FOXO1 (transcriptional activator of PDK4).

Ten healthy male volunteers (age 26 ± 1 yr, BMI 25.5 ± 1.2 kg.m^2, mean $\pm$ SEM) participated in this study, which was approved by the local Ethics Committee. Subjects fasted for 48h (water, electrolytes and non-sugared beverages without caffeine were allowed only) and then consumed a high CHO diet (75% CHO, 10% fat and 15% protein) for 24h. Before and after fasting, and after refeeding, subjects underwent insulin tolerance tests (ITT) to quantify whole body insulin sensitivity. Muscle biopsies (*vastus lateralis*) were obtained in the fed state, after 24 and 48h of fasting, and after refeeding for the determination of PDK1-4, PPARα, PPARδ and FOXO1 mRNA by quantitative real-time PCR using Taqman probes and normalised to α-actin.

Whole body insulin sensitivity (calculated from the rate constant for blood glucose disappearance during ITT) decreased by 42 ± 5% during fasting ($P<0.01$, 1-way ANOVA, relevant assumptions were verified) and recovered by 21 ± 7% (of initial value) upon refeeding. Fasting decreased blood glucose ($P<0.01$) and insulin ($P<0.05$) concentrations and increased plasma FFA levels ($P<0.01$) and muscle PDK4 mRNA content by 2.4-fold after 24h and 4.3-fold after 48h ($P<0.05$). Refeeding completely reversed those responses. There was no effect of fasting/refeeding on gene expression of PDK1-3 and FOXO1. In contrast, PPARα and PPARδ mRNA content decreased by ~2-fold after 48h of fasting ($P<0.05$) and returned to basal levels upon refeeding.

In conclusion, starvation-induced insulin resistance was accompanied by a marked increase in PDK4 mRNA expression in human skeletal muscle, a response which did not involve changes in mRNA abundance of FOXO1 (as its expression remained unchanged during fasting/refeeding) or PPARα and PPARδ, suggesting that, in contrast to findings from animal studies, post-translational modulations in key regulators of substrate oxidation mediate the shift in substrate utilisation from CHO to fat in fasted humans.

Wu P et al. (1999). *Diabetes* 48, 1593-1599.

This work was supported by BBSRC.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C40

The effect of short-term cinnamon ingestion on *in vivo* glucose tolerance in humans

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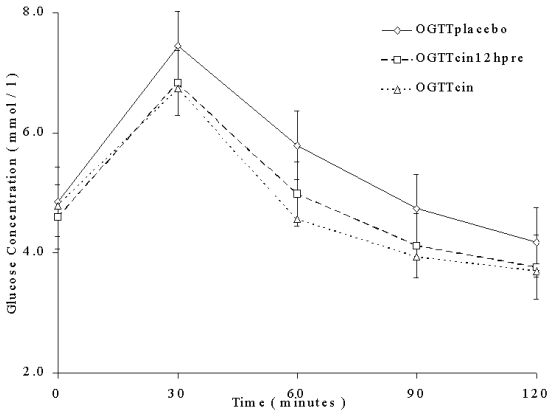
Recent epidemiological evidence shows an alarming increase in the prevalence of Type 2 Diabetes (Mokdad *et al.* 2001). Various natural compounds have been shown to be effective in improving glucose tolerance. For example, it has been shown that cinnamon may improve fasting glucose and triglyceride concentrations in type 2 diabetics (Khan *et al.* 2003), and *in vitro* work illustrates its potential as an insulin mimetic (Jarvill-Taylor *et al.*2001). The aim of the present study was to investigate the time-course of the effect cinnamon may have on glucose tolerance in normoglycaemic humans. Following local ethical approval seven sedentary males aged 26 ± 1 years with a BMI of 24.5 ± 0.3 kg m^{-2} (mean ± S.E.M.), underwent three oral glucose tolerance tests ($OGTT_{cin12hpre}$, $OGTT_{cin}$, $OGTT_{placebo}$) in a double-blind crossover design. The OGTT's followed the standard protocol of 75g D-glucose in 300ml water following an overnight fast. In the $OGTT_{cin12hpre}$ trial subjects consumed 5g of capsulated cinnamon 12 hours prior to the OGTT and 5g of placebo capsules with the OGTT; in the $OGTT_{cin}$ trial the order was reversed so that the cinnamon was consumed with the OGTT; and for the $OGTT_{placebo}$ subjects consumed 5g of placebo capsules 12 hours before and with the OGTT. Venous blood samples were taken at 0, 30, 60, 90 and 120 mins during the OGTT's. Plasma glucose and insulin concentrations were measured using colourimetric (ABX Diagnostics, COBAS) and ELISA (DRG International) assays respectively. All subjects kept a 24-hour food diary prior to the first trial, which was replicated prior to each subsequent test. Subjects also abstained from exercise, caffeine, alcohol and any cinnamon products for 24 hours prior to each trial. Data are reported as means ± S.E.M. and were analysed using repeated measures ANOVA (two-way for raw data, one-way for area under curve) and Tukey *post-hoc* tests were used to compare cinnamon and placebo trials. Both glucose (Fig.1) and insulin responses to the oral glucose bolus ingestion showed significant changes from baseline in all trials at t = 30 minutes ($P<0.01$). There was also an effect of trial on plasma glucose responses in both cinnamon trials compared to $OGTT_{placebo}$ ($P<0.01$, Fig.1). Area under the curve for plasma glucose was significantly decreased ($P<0.05$) in both the $OGTT_{cin12hpre}$ (604 ± 36 mmol·l^{-1}·2hours) and $OGTT_{cin}$ (584 ± 37) trials compared to $OGTT_{placebo}$ (675 ± 39). Area under the curve for insulin was not statistically different for the three trials. These findings illustrate that cinnamon can improve glycaemic control for at least 12 hours in humans.

Figure 1 - Plasma glucose concentration profile following OGTT in each trial. Main effect of time; 30 mins significantly higher than all other time points ($P<0.01$). Main effect of trial; both cinnamon trials significantly lower than placebo trial ($P<0.01$)



Mokdad et al. (2001) *JAMA*. 289: 76-79
Khan et al. (2003) *Diabetes Care* 26: 3215-18
Jarvill-Taylor et al. (2001) *J.Am.Coll.Nutr* 20(4): 327-36

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C41

Insulin stimulates L-carnitine accumulation in human skeletal muscle

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L-carnitine (LC) resides mainly in skeletal muscle and its Na^+ dependent transport occurs via the organic cation transporter OCTN2 (Tamai et al. 1998). Investigation of the effect of increasing muscle LC content on energy metabolism is warranted based on evidence that free LC availability may limit muscle fat oxidation during incremental exercise (van Loon et al. 2001). A limited amount of research has however addressed whether an increase in human muscle LC content is achieved by LC administration. This study aimed to determine whether insulin, which is known to increase Na^+/K^+ pump activity (Clausen, 2003), could augment LC accumulation in human muscle.

Eight healthy men (age 22.4 ± 0.4 years, B.M.I. 22.8 ± 0.5 kg m^{-2}) volunteered for the present study, which was approved by the local ethics committee. On two randomised occasions, separated by 14 days, subjects underwent a 6 h euglycaemic insulin clamp (5 and 105 mU m^{-2} min^{-1}) aimed at maintaining fasting or physiologically high plasma insulin concentrations. After 1 h, each insulin clamp was accompanied by intravenous infusion of LC (15 mg kg^{-1} bolus followed by 10 mg kg^{-1} h^{-1}) for 5 h. Arterialised-venous blood samples were obtained every hour and needle biopsy samples were obtained from the vastus lateralis immediately before and after each insulin clamp. Statistical analysis was performed using repeated measures one- and two-way ANOVA. Data are expressed as means ± S.E.M.

The 5 and 105 mU m^{-2} min^{-1} insulin clamp produced a steady state serum insulin concentration of 7.1 ± 0.4 and 149.2 ± 6.9 mU l^{-1}, respectively. Plasma LC concentration remained above 450 µmol l^{-1} throughout LC infusion, but was lower after 4 and 5 h of infusion during the 105 mU m^{-2} min^{-1} insulin clamp (P<0.01). Muscle total LC did not change when insulin was maintained at a fasting concentration (pre 22.4 ± 1.0 vs post 22.7 ± 1.1 mmol kg^{-1} dry weight), but increased from 22.0 ± 0.9 to 24.6 ± 1.4 mmol kg^{-1} during the 105 mU m^{-2} min^{-1} insulin clamp (P<0.05), which was associated with a 2.3 ± 0.3-fold increase in OCTN2 mRNA expression (n = 5; P<0.01).

The present findings demonstrate that maintaining a supra-physiological plasma LC concentration for 5 hrs has no effect on muscle total LC concentration or OCTN2 mRNA expression. However, increasing plasma LC availability in the presence of elevated serum insulin increases muscle LC concentration and OCTN2 transcription. We believe this is a result of insulin increasing sarcolemmal Na^+/K^+ pump activity and thereby Na^+-coupled transport of LC via OCTN2.

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Van Loon LJ, Greenhaff PL, Constantin-Teodosiu D, et al. (2001). J Physiol **536**, 295-304.

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Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C42

Effect of L-Carnitine supplementation on exercise metabolism at different workloads and following a high fat preload in man.

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The present study, which had ethics committee approval, examined the effect of 14 d supplementation of L-Carnitine L-tartrate (LCLT) on the metabolic response to exercise at different intensities and whether this was altered by the composition of prior dietary intake. Fifteen healthy, endurance trained males undertook this double-blind, placebo-controlled trial and were pair-matched on the basis of power output achieved at VO_{2max}, age and body composition before random allocation to placebo (P, n=7) or treatment (LCLT, n=8). Treatment consisted of 2 capsules b.i.d. (3g LCLT/d) for 15 d. Subjects attended the laboratory on 3 occasions (baseline, day 14 and day 15), 3 h after a standardised meal. For 2 d prior to baseline and day 14, subjects consumed a prescribed diet (3158 ± 335 kCal/d 59% carbohydrate (CHO), 25% fat, 15% protein) and were asked to undertake the same exercise each time. Between days 14 and 15, subjects were provided with an isoenergetic high fat (HF) diet (3182 ± 215 kCal/d 15% CHO, 70% fat, 15% protein). Exercise trials consisted of an 80 min cycle ergometer ride with 20 min at each of 20, 40, 60 and 80% of VO_{2max} incorporating indirect calorimetry and venous blood sampling. No difference in any blood metabolites or the estimated rate of CHO or fat oxidation was observed by RM ANOVA at any workload between groups at baseline (mean ± SD for first 60 min exercise: total CHO oxidation 120 ± 23 and 111 ± 30 g, total fat oxidation 26 ± 12 and 26 ± 14 g) or at day 14 (122 ± 30 and 103 ± 25 g, 21 ± 7 and 29 ± 11 g) for P and LCLT groups, respectively. The HF diet resulted in elevated resting plasma FFA (day 14 to HF trial: 0.11 ± 0.08 to 0.46 ± 0.12 and 0.35 ± 0.20 to 0.57 ± 0.30 mmol/L for P and LCLT, p<0.05); and an elevated plasma FFA and blood glycerol concentration, and increased fat and reduced CHO oxidation (fat: 47 ± 6 and 46 ± 8 g; CHO: 80 ± 12 and 69 ± 20 g for P and LCLT) during exercise. However, a greater change in the rate of fat oxidation was observed between days 14 and 15 with P than LCLT; significant at 20 and 40% workloads (p<0.02; Figure 1). The results indicate that 14 d supplementation with 3g LCLT/d does not alter the metabolic response to 20 min periods of exercise at 20, 40, 60 and 80% of VO_{2max}. The underlying mechanism for the suppressed shift towards increased fat oxidation at low exercise intensities in the LCLT group following the HF diet may be due to differences in circulating FFA between the two groups.

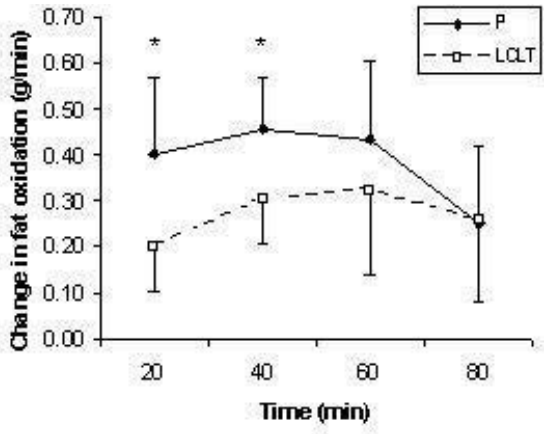

Figure 1. Change in fat oxidation rate during exercise between day 14 and day 15 trials (mean +/- SD). * Significant difference between P and LCLT, p<0.02 (t test).

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

C43

Bed-rest induced bone loss in the lower leg continues after re-ambulation in humans

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Bone loss from the lower extremities during bed rest is thought to be due to unloading, which stimulates remodelling activity and thus increases bone resorption, with the bone formation rate unchanged. One would expect, thus, that upon re-loading bone resorption is suppressed and that bone losses would stop. However, in the long term bed rest study (Watanabe, 2004), the dis-

tal tibia bone mineral content (BMC) was lower after 14 days recovery than at the end of 90 days bed rest.

The Berlin BedRest (BBR) study has shown that resistive vibration exercise (RVE) is an effective countermeasure to preserve bone during 56 days of strict bed rest (Rittweger, 2004). Twenty young healthy males between 24 and 43 years were randomized into either the control (Ctrl) or the exercise group (RVE), the latter performing 4 sets of ~80 s RVE for 6 min twice a day (except on Sundays).

BMC of the distal tibia epiphysis (4% of its length) was assessed by peripheral quantitative computed tomography (pQCT) with an XCT2000 (Stratec, Germany) on the 55th day of bed rest (BR55), and on days 4, 14, 28 and 90 of the recovery period (R+4, R+14 etc).

Mean values (±SD) of the BMC changes from baseline are given in the table.

A repeated measures ANOVA (Greenhouse-Geisser method) yielded a significant time effect ($p = 0.01$), but no time*group interaction ($p = 0.16$). Post-hoc testing with the paired-samples t-test (Bonferroni correction) showed no difference between changes on R+4, R+14, R+28 and R+90, but significant differences between the changes on BR55 and on R+4.

To interprete these results, the time delay between osteoid laydown and matrix mineralistation has to be considered. As the pQCT technique is x-ray based, it assesses only bone mineral, but not un-mineralized tissue. It probably takes 14 days for the first phase of mineralisaton (Martin, 1998). As a consequence, the ongoing loss of BMC after re-ambulation must be due to a still increased bone resorption. Thus, it seems as if active osteoclasts were more or less insensitive to increased loading. Possibly, their activity might be pre-programmed already when they are recruited.

	BR55	R+4	R+14	R+28	R+90
Ctrl	-2.3 % ± 2.0 %	-3.1 % ± 1.7 %	-3.5 % ± 1.8 %	-3.7 % ± 2.0 %	-2.7 % ± 1.3 %
RVE	-0.4 % ± 0.6 %	-0.8 % ± 0.4 %	-0.6 % ± 0.8 %	-0.7 % ± 0.5 %	-0.5 % ± 0.8 %

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Rittweger J, Felsenberg D (2004). J Bone Miner Res 19(Suppl 1) S37.

Watanabe Y et al. (2004). J Bone Miner Res 19(11), 1771-1778.

The BBR Study was supported by: European Space Agency (14431/02/NL/SH2), Charite - University Medicine Berlin (Campus Benjamin Franklin), DLR (German AeroSpace), MSD Sharp & Dohme, Lilly Germany, Servier Germany, Hoffmann-LaRoche, Siemens, Novartis, Danone, Seca

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC2

A comparison between haemodynamic responses to cold pressor test and mental arithmetic in healthy human volunteers.

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Cold stimuli and cortical arousal both increase mean systemic arterial blood pressure (MBP), but detailed comparative studies of the underlying cardiovascular changes in response to the stimuli in the same subjects have not been described.
With local Ethical Committee approval, we compared cardiovascular responses to immersion of a hand in ice-cold water (cold pressor test (CPT)) with those to mental arithmetic (MA) in healthy normal volunteers (21 males and 20 females, aged 20-23 years). The tests were applied on a single occasion with MA being applied for 3 min followed by CPT for 2 min, with a 15 min intervening period of rest. Measurements of heart rate (HR), MBP, stroke volume (SV), cardiac output (CO) and total peripheral resistance (TPR) were made using the Finometer (e.g., Leonetti et al., 2004) in supine subjects. Statistical comparisons were made using the Mann Whitney (between-groups) and Friedman (within-group) tests.
At rest, there were no significant differences between baseline cardiovascular variables (mean ± s.e.) in males and females (heart rate 72 ± 2, 77 ± 2 beats min^{-1}; MBP 96 ± 2, 94 ± 2 mmHg; SV 81 ± 3, 69 ± 3 ml $beat^{-1}$; CO 5.9 ± 0.3, 5.4 ± 0.3 l min^{-1}; TPR 10.3 ± 0.6, 11.1 ± 0.6 units, respectively). In males, MA and CPT both caused rises in HR and MBP (at 60 sec, +22 ± 3 and ++11 ± 2 beats min^{-1}, +14 ± 2 and +21 ± 3 mmHg, respectively), but MA caused no significant change in SV (+5 ± 3 %,) and hence a rise in CO (+14 ± 2% $P \leq 0.05$) and fall in TPR (−11 ± 3 % $P \leq 0.05$), whereas CPT caused a fall in SV (−12 ± 3 % $P \leq 0.05$) with no significant change in CO (+3 ± 5%) and a rise in TPR (+22 ± 7 % $P \leq 0.05$). The responses in females showed a similar pattern. Thus, in females, changes in cardiovascular variables at 60 sec during MA were: HR + 23 ± 2 beats min^{-1} $P \leq 0.05$, MBP +14 ± 2 mmHg $P \leq 0.05$, SV +1 ± 3 %, CO +27 ± 5% $P \leq 0.05$, TPR −9 ± 3% $P \leq 0.05$, and the corresponding changes during CPT were: HR +14 ± 2 beats min^{-1} $P \leq 0.05$, MBP +19 ± 3 mmHg $P \leq 0.05$, SV −13 ± 2% $P \leq 0.05$, CO +4 ± 3%, TPR +17 ± 4% $P \leq 0.05$.
The results show differential cardiac and vascular involvement in the pressor responses to CPT and MA, but with no apparent gender differences.

Leonetti P et al. (2004). Clin Auton Res 14, 176-181.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC4

The effect of 14 days supplementation with vitamin C on plasma interleukin-6 and hormonal responses to prolonged cycling in man

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Supplementation with antioxidants (vitamins C and E) for 28 days can reduce post-exercise increases in circulating interleukin (IL)-6 and cortisol concentrations (Fischer *et.al.*, 2004). These authors demonstrated that the smaller increase in plasma IL-6 concentration resulted from reduced release from contracting skeletal muscle, and this may be a major factor contributing to a blunted cortisol response. However, it has been suggested that there may also be a direct inhibitory effect of antioxidant supplementation on adrenal cortisol production and/or release (Peters *et.al.*, 2001). The aim of the present study was to determine the effect of 14 days of supplementation with a high dose of vitamin C alone on the systemic IL-6, adrenocorticotrophic hormone (ACTH) and cortisol responses to prolonged exercise.
With local ethics committee approval, 9 healthy male cyclists (age 26 ± 2 years, body mass 71.8 ± 2.2 kg, VO_2max 61.6 ± 2.4 $ml.kg^{-1}.min^{-1}$; mean ± S.E.M.) completed two main trials (2.5 hours cycling at 60% VO_2max), after 14 days of placebo (PLA) or vitamin C (VC; 1000 $mg.day^{-1}$) supplementation, in a single blind, counterbalanced-crossover design. This ensured a minimum of 14 days wash-out between trials. Venous blood samples were taken before exercise (Rest), immediately post-exercise (Post-Ex), and 1 h post-exercise (1 h Post-Ex). Plasma concentrations of IL-6, ACTH and cortisol were determined using ELISA kits. Plasma VC concentration was determined using an enzymatic spectrophotometric assay. Temporal changes were analysed for each trial separately using 1-way repeated measures ANOVA. To compare the two trials a 2-way repeated measures ANOVA was used. Paired t-test post hoc analysis was used where appropriate with the Holm-Bonferonni correction.
The results (Table 1) suggest that there may be a direct inhibitory effect of VC on adrenal cortisol secretion since there was no effect of VC supplementation on IL-6 or ACTH yet plasma cortisol concentration was significantly lower post-exercise in the VC trial. However, the cortisol response was not completely blunted, as observed in the Fischer *et.al.* (2004) study, which suggests that there is an important role for IL-6. Therefore, a longer period of supplementation and/or additional lipid soluble antioxidants such as vitamin E may be required for greater reductions in exercise-induced IL-6 and cortisol release to occur.

Table 1: Changes in plasma variables following exercise.

	Rest	Post-Ex	1h-Post-Ex	Main effect of Trial	Interaction
Vitamin C (mM)				YES	NO
PLA	47.1 (4.4)	57.3 (4.3) #	51.7 (4.1)	($P < 0.001$)	($P = 0.928$)
VC	91.7 (6.7) ¶¶	100.7 (5.9) ¶¶	96.5 (5.8) ¶¶		
ACTH (pg.ml^{-1})				NO	NO
PLA	24. (2.)	185. (49) #	44 (11)	($P = 0.303$)	($P = 0.269$)
VC	28 (2)	117 (28) #	30 (5)		
Cortisol (nM)				YES	YES
PLA	326 (34)	712 (77) ##	622 (90) #	($P < 0.001$)	($P = 0.038$)
VC	337 (42)	564 (70) ¶#	430 (51) ¶		
IL-6 (pg.ml^{-1})				NO	NO
PLA	0.6 (0.1)	8.0 (1.5) ##	5.9 (1.2) ##	($P = 0.882$)	($P = 0.875$)
VC	0.6 (0.2)	7.8 (1.7) ##	5.7 (1.1) ##		

Values are means (± S.E.M.). Significantly different from Rest (#$P < 0.05$; ## $P < 0.01$), Significantly different from PLA (¶ $P < 0.05$; ¶¶ $P < 0.01$).

Fischer CP *et.al.* (2004). J Physiol. 558, 633-45.

Peters EM *et.al.* (2001). Int J Sports Med. 22, 120-126.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC5

Long lasting effects of caffeine from cereal bars

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The psychostimulant effects of caffeine are well established and have been described in numerous reviews and publications. A threshold level of circulating caffeine (c.a. 2 mg/l although highly individual dependent) seems to be needed to observe stimulation, whereas at higher levels (> 10-12 mg/l) side effects such as excitation or gastric discomfort may appear. In order to safely prolong the stimulating effects of caffeine one way to maintain blood caffeine levels above a given threshold and to decrease peak values is to slow down caffeine absorption. Recently, a cereal bar was developed that is based on oat bran concentrate (OBC). OBC is rich in the soluble fibre beta-glucan, which, by developing a high viscosity in the intestine, slows down the absorption of glucose. It was hypothesized that beta-glucan could also slow down caffeine absorption by the same mechanism.

The aim of the present study was primarily to determine the pharmacokinetics of caffeine when incorporated as guarana extracts in OBC-based bars in healthy volunteers. Blood glucose levels were also measured and glycaemic indices determined because caffeine may have an effect on glucose metabolism.

A cereal bar containing ca. 6g of beta-glucan and 200 mg of caffeine as guarana extract was studied in 8 male volunteers (mean age: 35.3 (32-44) years, mean weight: 72.6kg (64-93), mean BMI: 22.7 (20-26)) after approval by the Nestle Ethical Committee. As reference treatments instant coffee (IC), white bread (WB) and IC + WB were also consumed. Because it is difficult to collect blood samples during long periods of time, plasma samples were collected during 3 h and saliva samples during 8 hours for caffeine alone. Plasma concentrations of caffeine and glucose were determined to obtain the pharmacokinetic variables of caffeine (one open compartment model with 1st order absorption according to Bateman function, Siphar/Simed Ltd software), the effects on glycaemia and eventual interaction of caffeine. Comparisons were analysed by either a paired t test or a repeated measures ANOVA test.

Surprisingly, ingesting white bread together with caffeine from IC did not affect caffeine kinetics as compared with IC only. Absorption rate of caffeine from the cereal bar was 4 times lower than from IC and from 4 h onwards after consumption caffeine levels from the cereal bar were significantly above those from IC (Fig. 1). The glycaemic response and index of the cereal bar vs. WB confirmed what had been observed in previous studies (GI = 49, Fig. 2) and no effect of caffeine on the glycaemic response was observed.

Incorporating caffeine from a natural source (guarana) in OBC-rich cereal bar did slow down its absorption (0.096 1/min. vs. 0.022 1/min, $p<0.001$), thus decreasing peak values (3.38 mmol/l vs. 2.71, $p<0.01$) and providing sustained levels presumably leading to prolonged stimulation.

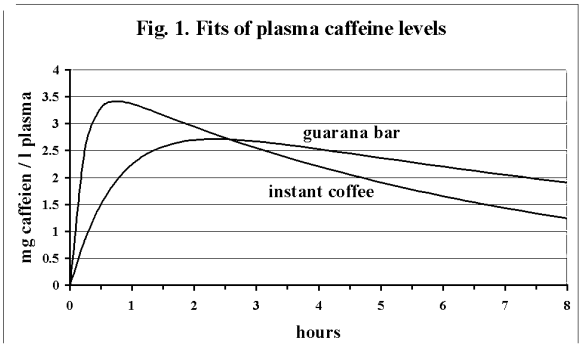

Fig. 1. Mean of plasma caffeine levels for all subjects after consumption of instant coffee or a guarana bar, each containing 200 mg of caffeine. Fits according to an open one compartment model with 1st order absorption.

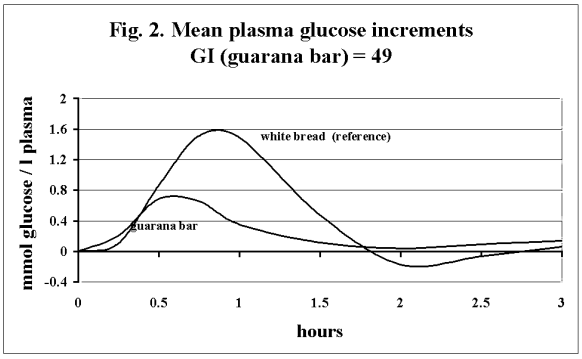

Fig. 2. Mean plasma glucose increments for all subjects after consumption of a guarana bar or white bread (reference), each containing 35 g of available CHO.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC6

Modality of exercise and rate of rise of rectal temperature during exercise in the heat

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Heat acclimation for athletes often occurs on cycle ergometers due to the limited size of environmental chambers and the convenience of being able to acclimatise several members of a team simultaneously. It has been established that a high deep body temperature is a key stimulus in the rate and extent of adaptations that occur in the body (Nielsen et al., 1997). Thus the aim of the present study was to test the hypothesis that deep body temperature will rise at a faster rate during steady rate running than cycling in the heat (30°C, 50% RH). After University ethical approval had been obtained, seven recreationally active males volunteered for the study. The VO_2 max for the treadmill was 55.7 ± 2.6 ml.kg^{-1}.min^{-1} and VO_2 max for the cycle ergometer was 46.5 ± 1.6 ml.kg^{-1}.min^{-1}. The volunteers completed a speed-lactate test for determination of lactate threshold (LT) and max test on a treadmill (Powerjog) and cycle ergometer (Monark). The running speeds and power outputs at 10%Δ(Δ being the difference between LT and max) were determined (Carter et al., 2000). The %Δ has previously been shown to result in a similar exercise stress between cycling and running as represented by changes in blood lactate and % maximum heart rates during exercise in moderate conditions. Subjects ran and cycled to exhaustion, or for a maximum time for 90 min in the heat (30°C, 50% RH) at the power output or speed equivalent to 10%Δ, 1 week apart in a random order. Data were analysed using a paired t-test or two-way ANOVA with repeated measures over time. Data are presented as mean ± SE.

Exercise time to exhaustion was greater during cycling (87.1 ± 2.9 min) than running (36.9 ± 6.1 min; P<0.001). Rectal temperature was higher and increased faster during treadmill running than cycling (main effect trial P<0.01; Fig. 1). Oxygen uptake was higher in the treadmill trial as an absolute value (CE: 2.2 ± 0.1 vs Tr: 3.2 ± 0.1 l min^{-1}; P<0.01), but was not significantly different as a percentage of mode specific VO_{2max} (CE: 63 ± 2 vs Tr: 76 ± 2%). Rate of perceived exertion, thermal comfort, heart rate as a percentage of mode specific maximum were also higher in the treadmill trial (heart rate, CE: 77 ± 8 vs Tr: 90 ± 4%; main effect trial P<0.01). Blood lactate and blood glucose did not differ during exercise but were significantly greater at the end of exercise in the running trial (lactate end, CE: 1.6 ± 0.3 vs Tr: 4.9 ± 0.9 mmol l^{-1}; glucose end CE: 3.9 ± 0.2 vs Tr: 5.0 ± 0.4 mmol l^{-1}; interaction trial x time P<0.05, post-hoc P<0.01). There was a greater sweat rate during the running trial (P<0.05), but there was no difference in body mass loss or fluid replacement. Therefore treadmill running causes a much faster rise in rectal temperature and a higher sweat rate than cycling and thus may be a better stimulus for acclimation.

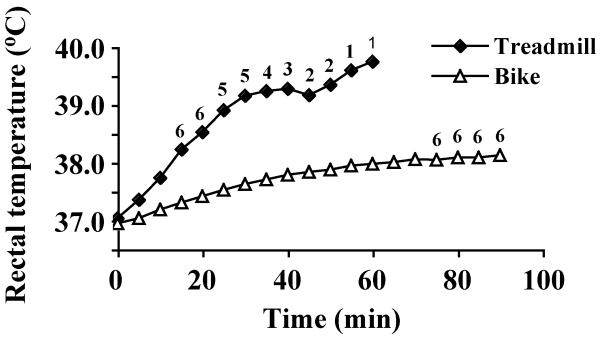

Figure 1. Rectal temperature during the cycling and running trials. Numbers are given at each time point. Main effect trial P<0.01.

Number of subjects are given above data points when less than 7.

Carter H et al. (2000) J Appl Physiol 89, 899-907.

Nielsen B et al. (1997). Pflügers Archive, 434, 49-56.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC7

Human gustatory-parotid salivary reflex responses to mixtures of sucrose with sodium chloride or monosodium glutamate

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It is generally accepted that there are five basic gustatory qualities, i.e. sour, salt, sweet, bitter and umami and there is a dose-response reflex parotid salivary secretion that exists for each of these stimuli. When these stimuli are mixed together interactions may occur in a suppressive, additive or synergistic manner. The simplest of these are binary interactions. The interactions between mixtures of two different stimuli using the gustatory-parotid salivary reflex have only been examined in a few studies. The aim of this study is to examine the effect of mixtures of sucrose with monosodium glutamate (MSG) or sodium chloride (NaCl) on reflex parotid salivary secretion in man.

With local ethics committee approval, salivary flows were recorded using Lashley cups and cannulae connected to an instantaneous flow meter. Gustatory stimuli used were solution of MSG (0.01 and 0.03M), sucrose (0.1 and 0.5M), NaCl (0.1 and 0.3M) alone and as mixtures of sucrose-MSG or sucrose-NaCl. The mixtures contained the same molar concentrations as those presented singularly. Stimuli were applied for 30s, and repeated following washout with water and when salivary flows had returned to resting levels. The stimuli were first presented as single solutions, followed by the mixtures. Each group of stimuli was repeated twice.

The salivary flows to the single stimuli were added together to give a calculated additive flow for the mixture. Large variation

in the salivary flows of individual subjects meant that salivary flows were normalised as a percentage of the calculated flow. The normalised results for the mean salivary flow for the sucrose-MSG mixtures compared to the calculated flows for these mixtures was significantly different (mean ± s.e., 81.2 % ± 3.4, $P<0.001$; paired t-test, n=8). The normalised results for the mean salivary flow for the sucrose-NaCl mixtures compared to the calculated flows for these mixtures was not significantly different (mean ± s.e., 95.5 % ± 7.5, $P>0.1$; paired t-test, n=7).

The observation that the flow produced by the sucrose-MSG mixture did not reach the additive response level suggests that some degree of suppression is occurring within the gustatory-salivary reflex pathways with these basic stimuli. Furthermore the salivary responses to mixtures of sucrose-NaCl were not significantly different from the calculated flow and this would suggest that there is an additive response within the gustatory-salivary reflex with these basic stimuli.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC8

Vasoconstriction in response to venous distension in the legs of women and men

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In the lower limbs, vasoconstriction triggered by venous distension and a local neural veni-arteriolar response is important for counteracting increases in capillary hydrostatic pressure during upright posture or limb dependency (Henriksen, 1991). In women, vasoconstriction in foot skin during leg dependency was attenuated in the luteal compared to follicular menstrual cycle phase, in association with increased fluid filtration which could contribute to premenstrual oedema (Hassan et al. 1990). Since leg dependency increases both arterial and venous hydrostatic pressures, it is not clear whether the smaller luteal vasoconstriction in women is due to a change in arterial myogenic constriction or the veni-arteriolar response.

With local ethical committee approval, we studied vasoconstrictor responses to venous distension (thigh cuff inflation to 50 mmHg for 5 min) in 13 young women (age 24±4 years, height 161±2 cm, mean±S.D), 5 not taking (nonOC) and 8 taking (OC) oral contraceptives during follicular (days 10-13) and luteal (days 18-27) phases of the menstrual cycle, and in 7 young men (age 25±3 years, height 178±7 cm). After 5 min of venous distension, Laser Doppler skin perfusion in the foot dorsum decreased similarly from resting during both test phases in nonOC women (56±7% follicular, mean±S.E.M., 68±6% luteal, NS Wilcoxon signed ranks test) and OC women (51±3% follicular, 55±3% luteal, NS). Maximum vasoconstriction in men (46±9% after 5 min) was no different from either female group in either phase (one-way ANOVA), but its onset was delayed to the 2nd minute of distension whereas in all women, it occurred within the 1st minute. These data do not support a role for female hormone effects on the magnitude of the veni-arteriolar response. The gender difference in response latency may be related to disparate superficial venous filling times in men versus women.

Veni-arteriolar vasoconstriction was also studied in the same groups in the calf, where venous distension occurs primarily in muscular rather than superficial vessels. Calf blood flow was measured using strain gauge plethysmography by brief thigh cuff inflations to 80-90 mmHg during venous distension (cuff at 50 mmHg for 5 min). After 1 min, blood flow decreased similarly in nonOC women (54±5% follicular, 54±8% luteal), OC women (54±5% follicular, 54±5% luteal) and men (52±3%). However, after 5 min, vasoconstriction in men had waned (16±5%) whereas in all women it had increased further (70%). This may be due to venous creep and relief of distension in men, relating to their greater calf venous compliance than women (Monahan & Ray, 2004). Locally mediated vasoconstriction in women may reduce their vasoconstrictor reserve and contribute to orthostatic intolerance.

Henriksen O (1991) Acta Physiol Scand 143 (Suppl. 603), 33-39

Hassan AK et. al. (1990) Clin Sci 78, 39-47

Monahan KD & Ray CA (2004) Am J Physiol 286, H895-H901

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC9

Functional and haemodynamic improvements after intermittent chronic electrical stimulation in patients with peripheral vascular disease

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Peripheral vascular disease (PVD) of the lower limbs is most apparent during ambulation, when ischaemic pain (intermittent claudication) elicits early exercise cessation. Functional capacity in claudicants was enhanced by a 4 week period of low frequency electrical stimulation of calf muscles, evidenced by increases in treadmill claudication (CD) and maximal walking (WD) distances, but improvements were not maintained when stimulation ceased (Tsang et al. 1994, Anderson et al., 2004). For stimulation to be considered an effective treatment for PVD, improved functional capacity must be sustainable. We therefore investigated whether repeating stimulation after a rest period would achieve this.

Following habituation with the assessment protocols, 8 stable intermittent claudication patients, aged 70±2 years (mean±S.E.M.) were randomised into active low frequency (8Hz) stimulation or control (TENS) treatment groups. Treatment lasted for a fixed period of 20mins, 3 times per day for 4 weeks (Bout1), with a repeat regimen (Bout2) applied after a 2 week rest period (total study duration 14 weeks). Functional capacity was assessed using treadmill measures, with lower limb blood flow estimated by venous congestion plethysmography. Approval for the study was obtained from the South Birmingham Research Ethics Committee, and University Hospital Birmingham, NHS Trust.

Four weeks active stimulation (Bout1) led to an 18% increase in pain-free walking distance from baseline (310±59 vs. 260±71

m, P⟨0.05, paired t test), with a similar improvement in WD. Following a period of 2 weeks rest, improvements in CD and WD had waned by approximately 15 m. A repeat regimen of stimulation (Bout2) reversed the decline in CD and WD after the rest period, and reinstated values back to those seen after the first stimulation period. Lower limb blood flow increased from baseline after Bout1 (3.5±0.6 vs. 2.1±0.3ml.min^{-1} (100ml)$^{-1}$, P⟨0.05) and did not decline over 2 weeks rest. Flow increased further after Bout2 (5.7±1.0ml.min^{-1} (100ml)$^{-1}$), and remained 92% above pre-treatment values at the end of the study. TENS did not induce improvements on any measure.

Chronic low frequency electrical stimulation of ischaemic calf muscles is therefore an effective stimulus for transient improvements in functional capacity in PVD. Applied intermittently, it enables improved functional capacity to be sustainable. We also show that stimulation provides amelioration of the haemodynamic compromise experienced by claudication patients

Tsang GMK et al. (1994). Eur J Vasc Surg 8, 419-422.

Anderson SI et al. (2004). Eur J Vasc Endovasc Surg 27, 201-209.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC10

Reliability of maximal muscle force and voluntary activation as a marker of exercise-induced muscle damage

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The loss of the ability of skeletal muscle to generate force is one of the most appropriate and valid means by which to quantify muscle damage. However, measurements of maximal muscle force may include many potential sources of error, the most common of which is the lack of central drive to the muscles. The aim of the present study was to therefore determine the reliability of maximal isometric quadriceps muscle force and voluntary activation over a time-scale that is typically employed to examine the aetiology of exercise-induced muscle damage. We also characterised the reliability of several twitch interpolation variables and determined the effects of different calculation methods on estimates of voluntary activation, specifically the central activation ratio (CAR) and interpolated twitch ratio as calculated from both unpotentiated (ITTUNPOT) and potentiated twitches (ITTPOT).

Eight healthy active males, mean (SD): age 21.4 (0.9) years; mass 83.6 (8.2) kg; height 179.8 (2.4) cm performed repeated maximal voluntary isometric contractions (MVC) of the quadriceps over a 7 day period (baseline and 2h, 6h, 24h, 48h, 72h, 7 days post). Differences across the 7 sessions were assessed using repeated measurements ANOVA. Ninety-five percent repeatability coefficients were also calculated according to Bland and Altman (1999). There was a significant difference (P<0.05) in estimates of voluntary activation between calculation methods with values of 98.9, 91.2 and 94.6% calculated for the CAR, ITTUNPOT and ITTPOT methods, respectively. The ITTPOT was considered the most valid estimate of voluntary activation. There was no evidence of any systematic changes over time in maximal muscle force, voluntary activation (ITTPOT), interpolated twitch, unpotentiated twitch or potentiated twitch (P>0.05) where 95% repeatability coefficients of 76.03 N, 4.42%, 8.44 N, 25.92 N and 43.58 N were observed, respectively.

These data indicate that young healthy well-familiarised subjects can reproduce their perceived maximal efforts both within and between days where activation levels of >90% are routinely achieved. Providing activation remains within these limits in the 7-days following an acute bout of exercise, the researcher may be 95% certain that exercise-induced muscle damage is present in individual subjects (taken from similar subject populations) if MVC force falls outside these limits.

Bland J.M. & Altman D.G. (1999). Stat Methods Med Res 8, 135-160.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC11

Responses to the perturbation of the leg during walking on a treadmill in elderly women fallers and non-fallers

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We have previously described two recovery strategies after perturbations of gait during treadmill walking. Fit older subjects more frequently used the strategy that appeared less stable than did the young (Bruce et al, 2000). We have now compared recovery strategies in elderly subjects who had recently fallen with those who had not.

Subjects were 24 women, mean age 74.2 (sd 3.4) years. All had fallen at least once in the last four years and had participated in exercise tailored to improve balance and gait. The "fallers" group was those 16 subjects who had fallen one or more times in the previous year. The study had RNOHT ethical committee approval; subjects gave written informed consent following Helsinki guidelines.

Subjects wore a safety harness and walked on a PowerJog GX100 treadmill at their own comfortable walking pace, which was the same for each group. Kinematics were recorded by the CODA mpx30 motion analysis system using 16 LED markers. At the beginning of swing phase during randomly selected strides CODA triggered the perturbations, applied by cords attached to each foot. The perturbations were not large enough to cause loss of balance.

We obtained 44 gait perturbations (each the average of 6 trials) from the fallers and 19 from the non-fallers. Each subject adopted a consistent strategy in response to perturbation (Bruce et al, 2000). The timing of lifting of the un-tripped leg immediately after the perturbation was delayed in a gretaer proportion of the fallers (14/44 fallers vs 1/19 non-fallers, Chi squared, p=0.023). Fallers took longer to return to their normal pattern

of foot placement after perturbation ($p<0.05$, unpaired t-test). Both groups showed increased flexion of the trunk immediately following the perturbation (by approximately 2 degrees). This lasted longer (approx. 0.25 sec) in the fallers than in the non-fallers and they then tended to overcompensate ($p<0.01$, unpaired t-test).

After a minor gait perturbation older people who fall persistently have a less stable response and are slower to recover.

Bruce SA, Birtles DB, Gentles H, Rosenberg M and Woledge RC. (2000) J. Physiol., 525P, 46P

We thank the Dunhill Medical Trust and The Tana Trust for their support

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC12

Effects of carbohydrate supplementation during the second of two prolonged cycling bouts on blood neutrophil responses

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It has been demonstrated that carbohydrate ingestion, compared with placebo, during a 3-h recovery interval between two bouts of prolonged exercise had only a limited effect on perturbation of blood neutrophil numbers and function during the second exercise bout although some attenuation of hypothalamic-pituitary-adrenal (HPA) axis activation occurred (Li and Gleeson, 2004). The aim of the present study was to determine the effect of carbohydrate supplementation during the second of two prolonged cycling bouts on neutrophil responses.

With Loughborough University Ethics Committee approval, nine males (age 28.7 ± 1.6 years, body mass 74.4 ± 3.2 kg, VO_2max 50.3 ± 2.4 mL.kg^{-1}.min^{-1}; means ± SEM) performed two bouts of 90 min cycling (EX1 started at 09:00 and EX2 started at 13:30) at 60% VO_2max after an overnight fast on two occasions, separated by at least 4 days in a randomised order. Subjects consumed 500 mL of a carbohydrate (10% w/v glucose; CHO) or placebo (PLA) beverage at 5-min pre-exercise and 250 mL every 20 min during the second exercise bout. Water ingestion was allowed *ad libitum* during the first exercise bout and recovery interval. Venous blood samples were collected at pre-exercise and post-exercise for both bouts. Haematological analysis was performed using an automated cell counter. Plasma hormone concentrations were determined using ELISA kits. Phorbol myristate acetate induced oxidative burst activity was measured using a chemiluminescence (CL) assay (Knight Scientific Limited, Plymouth) and bacterially-stimulated neutrophil degranulation was measured as described by Robson *et al.* (1999). Results were analysed using a two-factor (trial × time) repeated measures ANOVA with *post hoc* Tukey tests and paired *t* tests applied where appropriate.

The ingestion of CHO compared with PLA during the second exercise bout had no effect on blood neutrophil count, degranulation and oxidative burst responses although the plasma glucose concentration was better maintained ($P<0.01$) and plasma concentrations of adrenaline ($P<0.05$) and cortisol ($P<0.01$) were blunted (Table 1). These findings suggest CHO feeding during the second of two bouts of 90 min cycling at 60% VO_2max maintains better CHO availability, blunts HPA activation, but does not affect blood neutrophil count or function at immediate cessation of exercise.

Table 1. Plasma variables and blood neutrophil responses immediately before and after the second exercise bout (EX2).

	Pre-EX2		Post-EX2	
	CHO	PLA	CHO	PLA
Neutrophil count (10^9·L^{-1})	5.61 (0.69)	5.43 (0.58)	6.53 (0.61)*	7.27 (0.74)*
Elastase release per neutrophil (fg·cell^{-1})	215 (34)	217 (26)	191 (15)	182 (26)
CL per neutrophil (fold of Pre-EX1)	0.81 (0.06)	0.77 (0.04)	0.74 (0.04)	0.63 (0.02)*
Glucose (mM)	4.78 (0.13)	4.79 (0.06)	5.66 (0.17)	3.75 (0.22) ¶¶
Adrenaline (pM)	338 (46)	375 (29)	604 (76)	1417 (352)** ¶
Cortisol (nM)	234 (32)	209 (31)	438 (49)**	661 (55)** ¶¶

Values are mean (± SEM, n=9). Significantly different from pre-EX2 (*$P<0.05$, **$P<0.01$); significantly different from CHO (¶ $P<0.05$, ¶¶ $P<0.01$)

Li T-L & Gleeson M (2004). Medicina Sportiva, 8, 65-75.

Robson PJ *et al.* (1999). Int. J. Sports Med. 20, 128-135.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC13

The influence of serial feeding of beverages at different temperatures on thermoregulatory responses during prolonged exercise in man

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The effects of the temperature of ingested beverages on physiological responses during exercise have not been extensively studied. This study aimed to investigate the influence of beverage temperature on thermoregulatory responses during prolonged cycling exercise.

With ethics committee approval, 8 non-heat-acclimatised men exercised on a cycle ergometer on 3 occasions at 50 ± 3% (Mean ± SD) of their VO_2peak for 90 min in an ambient temperature of 25.3 ± 0.5°C with relative humidity of 60 ± 5%. A beverage volume of 400 ml at temperatures of 10 (C), 37 (W) or 50°C (H) was ingested within 2 min after 30, 45, 60 and 75 min of exercise. Immediately following exercise, subjects cycled at 95% peak to exhaustion. Rectal temperature (T_{re}) and weighted mean skin temperature (T_{sk}; Ramanathan 1964), from which total body heat content (TBHC) was derived, and heart rate (HR) were recorded at rest and during exercise. Expired air was collected to estimate substrate oxidation and metabolic heat production. Statistical differences between variables were assessed using ANOVA.

No significant differences were found in any variable prior to the ingestion of drinks. Therefore the 30th min of exercise was used

as the baseline throughout. The absolute rise in T_{re} at the end of exercise was 0.42 ± 0.20, 0.30 ± 0.22 and 0.46 ± 0.22?C for the C, W and H trials respectively (p=0.342). Mean T_{sk} appeared to be influenced by beverage temperature (34.49 ± 0.64, 34.53 ± 0.69 and 34.71 ± 0.48°C for trials C, W and H trials respectively; p=0.09). Significant differences in mean HR were observed (124 ± 9, 126 ± 8 and 129 ± 7 beats.min^{-1} for the C, W and H trials respectively; p<0.05; Figure 1). Endurance capacity was not different between trials (205 ± 88, 213 ± 74 and 215 ± 85 s for C, W and H trials respectively; p=0.963). Total body heat content increased by 314 ± 15, 289 ± 16 and 314 ± 17 kJ in trials C, W and H respectively. There was no difference in ΔTBHC between the C and H trials evaluated relative to trial W whereas the calculated difference in the heat required to warm/cool the respective beverages to body temperature amounted to 268kJ.
Therefore, the heat load/debt induced at 15 min intervals resulted in appropriate thermoregulatory reflexes during exercise, resulting in similar body temperatures and TBHC, as opposed to the body merely acting as a heat sink.

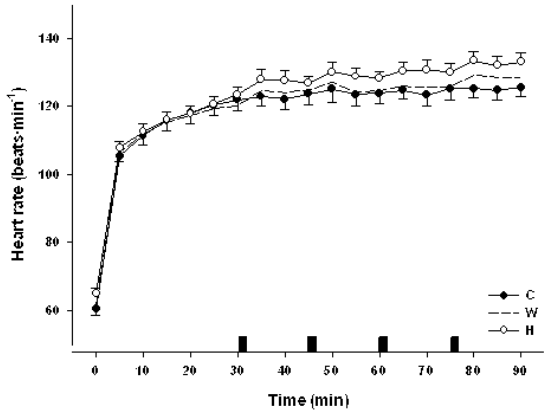

Figure 1. Heart rate (beats/min) during the 3 experimental trials. Mean values and S.E. are shown. Shaded blocks denote the ingestion of beverage.

Ramanathan, NL (1964). *J Appl Physiol* **19**, 531-3.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC14

Physiological adaptations to upper-limb aerobic exercise training that influence walking performance in patients with peripheral arterial disease.

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Upper-limb aerobic exercise is well tolerated and can improve walking performance in patients with peripheral arterial disease (PAD) (Zwierska et al. 2003). This study aimed to identify physiological adaptations to upper-limb aerobic exercise that could influence the improvement in walking performance.

Following approval from the North Sheffield Local Research Ethics Committee, 94 patients (median age 69 years, range 50-85 years) with PAD who were accustomed to the training and assessment protocols, were randomly allocated to arm-crank training (ACT), leg-crank training (LCT) or control groups. Training was performed twice weekly for 24 weeks at equivalent relative exercise intensities (75-85% peak oxygen uptake). Incremental leg-cranking assessments to maximum exercise tolerance were performed before and after the intervention and pulmonary gas exchange variables, blood lactate concentration, ratings of perceived exertion (Borg RPE) and leg pain (Borg CR-10) were recorded. Walking performance, defined as the claudicating distance (CD) and maximum walking distance (MWD) was also assessed using a shuttle-walk protocol (Walker et al. 2000). A mixed-design factorial ANOVA was used to compare groups, with statistical significance set at $P < 0.05$.
Peak oxygen uptake (mean ± S.E.M) for incremental leg-cranking improved after ACT (1.12 ± 0.07 versus 1.33 ± 0.09 l.min-1, P = 0.001) and LCT (1.12 ± 0.05 versus 1.31 ± 0.06 l.min-1, P < 0.001), and was associated with an increased blood lactate concentration in both training groups (P < 0.001), but not in the controls. The CD and MWD improved by 56 ± 9 % and 30 ± 4 % after ACT and by 65 ± 11 % and 35 ± 4 % after LCT, respectively (P at least 0.001). Improvements in MWD were accompanied by comparable increases in leg pain (6 ± 0.5 versus 7 ± 0.5 and 6 ± 0.4 versus 7 ± 0.5), RPE (14 ± 0.6 versus 15 ± 0.6 and 14 ± 0.5 versus 15 ± 0.5) and blood lactate concentration (1.95 ± 0.14 versus 2.40 ± 0.17 and 1.90 ± 0.11 versus 2.26 ± 0.14 mM) after ACT and LCT, respectively (P at least 0.05). The results suggest that ACT evoked similar responses to LCT, and upper-limb aerobic exercise training improved lower-limb exercise capacity via mechanisms that delayed the onset of claudication pain and elevated exercise pain tolerance.

Walker, R.D., Nawaz, S., Wilkinson, C.H., Saxton, J.M., Pockley, A.G. & Wood, R.F.M. (2000). J. Vasc. Surg. 31, 662-669.

Zwierska, I., Saxton, J.M., Male, J.S., Choksy, S., Pockley, A.G. & Wood, R.F.M. (2003). J. Physiol. 547P, C123.

We acknowledge support from the British Heart Foundation.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC15

Effect of static magnetic fields on human skeletal muscle performance

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The effects of static magnetic field (SMF) exposure on physiological factors affecting muscle performance were investigated during leg extension exercise at 2 different contraction intensities. 14 subjects participated in two double blind trials separated by one week and allocated by systematic rotation (sham or active arrangement 0.1 Tesla peak SMF intensity, 30 min pre-exposure), half (n=6, male and n=1, female) completed 10 sets

of 15 dynamic contractions at 50% maximum lifted weight (1RM) (L), and the remaining subjects (n=7, male) completed 21 sets of 4 contractions at 90% 1RM (H). Custom-designed devices (sham or active) were attached to the quadriceps of the subject. Biomechanical data were collected using in-line force transducer and electrogoniometry, and rectus femoris activation was assessed using surface electromyography (EMG). Data normalised against 1RM values were averaged across the repetitions (n=15, L; n=4, H) for each set (n=10, L; n=21, H) and shown as population means±standard error of the mean (SEM). Data were analysed by repeated measures ANOVA, and post hoc paired Student's t tests corrected for multiple comparisons using Holm- Šidák step-down procedure .

Root mean square (RMS) amplitude of muscle EMG activity increased over time during sham trials for both protocols (20±7, L; 1±4, H; %); SMF exposure modified this response (3±4, L, $P<0.001$; -6±5, H; %). The mean frequency of the EMG power spectrum decreased over time to a greater degree in sham (-7±1, L; -2±2, H; %) than active trials (-3±5, L, $P<0.05$; 0±2, H; %). Mean power output per contraction was higher throughout active compared to sham trials although this reached significance only during H trials (4±1, L, 16±1, $P<0.05$, H; %). Functional efficiency (power per unit RMS EMG amplitude) decreased to a greater degree during sham (-28±1, L; -11±9, H; %) than active (-6±9, L, $P<0.01$; 9±6, $P<0.05$, H; %) trials. The magnitude of the differences between sham and active trials increased over time, presumably as fatigue developed. The differences in the temporal pattern of fatigue development between low- and high intensity exercise protocols may account for the smaller SMF effect on muscle EMG parameters in H compared to L trials.

Muscle performance was preserved at both contraction intensities, by exposure to SMF. This effect appears to be mediated by alterations in the profile of fatigue development, as evidenced by the reduced change over time in muscle EMG parameters, power output and functional efficiency. It is possible that the magnetic field interacts with ionic membrane processes and muscle contractility. All procedures accord with current local guidelines and the Declaration of Helsinki.

This study was supported by The Wellcome Trust.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC16

Comparison of manual and mechanical vibratory massage following eccentric arm exercise

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Massage may have a favourable effect on reducing perceived muscle soreness following eccentric exercise through reducing muscle damage and lessening limb inflammation (Smith et al., 1994). The aim of this study was to compare the effect of mechanical vibratory massage with manual massage following a bout of eccentric exercise (EE) on perceived pain, serum creatine kinase (CK) activity as a marker of muscle damage and limb circumference (LC) as a marker of oedema (Kraemer et al., 2004).

30 male subjects (mean±SD: 25.1±3.9yrs, mass 75.6±6.8kg, 13.6±2.9% body fat, 1 biceps repetition max (1RM): 16.5±2.5kg) undertook an exercise bout of 3 sets at 1 min intervals of 10 eccentric bicep curls (non-dominant arm) at 80% of 1RM. On completion of the exercise bout (Perceived Exertion=19 (Extremely Hard)) subjects were then assigned to one of three recovery groups: Seated Rest (SR), Manual Massage (MM) of the upper arm (using both effleurage and petrissage), or Vibratory Massage (VM) (G5, Physiotherapie Generale France) for 8 min with follow up assessments at 4, 8, 24 and 48h post EE.

The highest perception of pain scores (0=No Pain; 6=Unbearably Painful) were immediately post EE (4.4±0.5) and was experienced in the mid forearm. VM was significantly more effective in reducing pain after 4h recovery (1.5±0.5, compared to 3.8±1.0 for MM and 2.9±1.0 for SR $p<0.05$, Repeated Measures ANOVA) to 48h recovery (0.5±0.4, for VM compared with 2.8±0.8 for MM and 3.4±0.7 for SR, $p<0.05$).

CK increased from 61.3±15.9 U . l-1 pre-EE to 163.3±47.9 following SR recovery ($p<0.05$) at 24h and to 305.4±69.4 ($p<0.05$) at 48h. Neither VM nor MM significantly affected CK levels.

Mean LC was calculated from measurements at the mid biceps, mid-forearm, distal humerus and proximal ulna. LC increased by 23.0±4.8mm post EE. The greatest changes in LC were observed in the mid forearm, the point at which the highest pain was experienced. Neither MM nor VM changed the recovery of LC, which remained at 6.5±3.9 and 5.5±3.2mm ($p<0.05$) respectively above pre exercise levels after 48hrs.

The results from this study support the contention that VM is more effective than MM or rest at reducing muscle pain induced by eccentric exercise, an effect that does not appear to be mediated through less muscle damage (as judged by CK release) or by decreased oedema (as judged by LC) but possibly by a greater mechanical stimulation of the skin by VM.

Smith et al. (1994). J Orthop Sports Phys Ther 19, 93-99.

Kreamer et al. (2004). ISMJ 5, 200-208.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC17

Serum prolactin responses to duathlon performance following pre-exercise dietary manipulation

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Serum prolactin (PRL) concentrations become elevated following a low carbohydrate diet as well as exercise which elicits significant rises is core temperature. The effect of pre-exercise dietary manipulation on PRL responses to exercise, which significantly elevates core temperature, has not been scrutinised.

Nine male subjects of mean (SD) age 28.6 (5.9) years, body mass 76.8 (8.5) kg, height 1.80 (0.7) m, and VO_2max 4.8 (0.6) $l.min^{-1}$ completed three laboratory simulated duathlon time trials consisting of a 5 km run, 30 km cycle, and a further 5 km run. Each

time trial was separated by at least 1 one week. Subjects fasted overnight before the duathlons and were randomly given isoenergetic meals (3998 (2.2) kJ) 3.5 h before the start of exercise. The meals consisted of predominantly low carbohydrate (LCHO) or high carbohydrate (HCHO) macronutrient components, or a fasting (F) condition, in which subjects ate nothing. Rectal temperature was used as an index of core temperature (Tc). Venous blood samples were taken immediately prior to and following the final running stage, in order to determine serum prolactin responses. The analysis of PRL was performed using enzyme-linked immunosorbent assay. A general linear model ANOVA, with repeated measures was used to determine significant differences ($P<0.05$). Means were compared using the Bonferroni confidence interval. Relationships between variables were made using Persons correlation coefficient.

PRL was unaffected prior to exercise by the meal strategies ($P>0.05$), but significant increases were observed following the completion of all the time trials ($P<0.05$). There were no significant differences in Tc as a result of the meal strategies, but the performance of the time trials caused significant increases in Tc ($P<0.05$). PRL was significantly correlated to Tc ($r=0.81$). Overall mean speed throughout the duathlons was used as an indicator of performance, but there were no significant differences as a result of the dietary conditions (6.6 (0.4) m s^{-1}, 6.5 (0.4) m s^{-1}, 6.4 (0.5) m s^{-1}) for F, LCHO and HCHO, respectively ($P>0.05$). These data suggest that following acute dietary manipulation, the PRL response to strenuous exercise is unaffected, and post exercise elevations are likely to be a consequence of the rise in Tc.

Table 1. Serum prolactin and core temperature responses

		Pre-Exercise	Post-Exercise
Serum PRL ($ng.ml^{-1}$)	F	5.71(2.5)	16.28 (4.1)
	LCHO	5.21 (1.8)	18.57 (8.7)
	HCHO	5.40 (2.1)	15.55 (6.2)
Tc (°C)	F	37.10 (0.3)	39.03(0.3)
	LCHO	37.16 (0.2)	38.99 (0.4)
	HCHO	37.26 (0.2)	38.83 (0.3)

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC18

Arterialised, venous and fingertip capillary blood glucose disposal following a single bout of exercise and carbohydrate ingestion

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The fingertip blood collection technique is a commonly used diagnostic tool for the monitoring of glycaemic control in both exercise and clinical settings as it is thought to provide a rapid, non-invasive analysis of circulating whole blood glucose concentrations (Ellison *et al*, 2002). The aim of this study was to compare the sensitivity of the fingertip collection technique and the efficacy of its use for the estimation of blood glucose disposal following a single bout of exercise and an oral glucose tolerance test (OGTT) in healthy male subjects.

Six male (age 22.2 ± 0.47 years; BMI 27.3 ± 0.9 kg/m^2; mean ± SE), glucose tolerant individuals (HbA1c 5.1 ± 0.1%; 2h OGTT venous glucose concentration 4.8 ± 0.2 mmol/l) participated in this investigation. Experimental procedures were approved by the East Sussex Local Research Ethics committee and conducted in accordance to the revised declaration of Helsinki. Subjects were studied in the fasted state and during two 75g Oral Glucose Tolerance Tests (OGTT). The first at rest and the second immediately following one hour of exercise at a power output corresponding to 90% of a pre-determined lactate threshold (LT). A cannula was inserted into a superficial dorsal hand vein and placed in a temperature regulated Perspex box maintaining the hand at ~60°C for collection of arterialised blood (Zello *et al*, 1990) and a second into the contralateral antecubital vein for venous blood sampling. Arterialised (A) (5ml), venous (V) (5ml) and fingertip (F) (~250μL) blood was sampled every 10 minutes. Whole blood samples were immediately analysed for glucose using the glucose oxidase method (YSI, Yellow Springs, CA). A repeated measure ANOVA was conducted to assess differences between sample sites and conditions.

There were no differences observed in glucose concentrations across the three sample sites following exercise or glucose ingestion. The rate constants ($k=min^{-1}$) calculated from the plot of log glucose versus time from peak glucose during each OGTT showed a significant difference ($P<0.01$) in fingertip glucose disposal during the rest and exercise trials (Table 1).

Our results suggest that the fingertip collection method offers a valid diagnostic tool for quantifying glucose disposal during exercise. However, at rest it may provide an invalid estimation of glucose disposal.

Table 1. Rate constant ($k=min^{-1}$) for glucose decay from peak glucose during OGTT at rest and after exercise

Sample Site	Rest	Exercise
F	$k=-3.38\pm0.01\times10^{-3}\,min^{-1}$	$k=-32.68\pm0.01\times10^{-3}\,min^{-1}$
A	$k=-25.9\pm0.01\times10^{-3}\,min^{-1}$	$k=-32.45\pm0.01\times10^{-3}\,min^{-1}$
V	$k=-35.1\pm0.01\times10^{-3}\,min^{-1}$	$k=-33.25\pm0.01\times10^{-3}\,min^{-1}$

Ellison JM *et al* (2002) *Diabetes Care.* 25, 961-964.

Zello GA *et al* (1990) *Ann Clin Biochem.* 27, 366-372.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC19

Does ventricular synchrony change with exercise in healthy men?

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Cardiac output is thought to be the main limiting factor of maximal aerobic exercise in healthy subjects (Coats, 2001). Alterations

in interventricular (interV) and intraventricular (intraV) synchrony of the heart produces change in cardiac output during exercise in heart failure patients (Nowark *et al.*1995). There is however no research into the effect of exercise on ventricular synchrony in healthy subjects where alterations in ventricular timings may represent more favourable haemodynamics for peak exercise.

Approval for this study was granted by the East Sussex Local Research Ethics Committee and conducted in accordance with the declaration of Helsinki. Five male subjects (mean±SEM age: 27.8±2.5yrs, mass: 80±1.6kg, BMI: 24.7±0.8 kg/m^2) took part in the study. Inclusion required a QRS duration <120ms indicative of normal electrical conduction, as determined by 12-lead ECG. Each subject completed an incremental exercise test on a recumbent exercise bicycle to volitional exhaustion. Echocardiographic readings were recorded at rest and peak exercise using a commercially available system. Images were analysed offline.

To assess interV synchrony (IVS), the difference between the LV and RV ejection times was analysed (Ghio *et al.*2004). Ejection time was defined as the time from q wave to flow assessed using pulsed wave Doppler in the right and left ventricular outflow tracts. An IVS>40ms was considered as dyssynchronus (Ghio *et al.*2004). To analyse IntraV synchrony pulsed wave tissue Doppler imaging (TDI) was performed using apical two and four chamber views at the level of the mitral annulus allowing analysis of longitudinal function of the anterior, inferior, septal and lateral walls of the left ventricle. All the possible differences between the peak contraction time (PCT) of the four basal segments were calculated. IntraV dyssynchrony was considered to be present if absolute differences between any two segments was greater than 1 standard deviation of the regional PCT (Ghio, 2004). Statistical analysis was achieved using paired t-tests.

All subjects displayed interV synchrony at rest and at peak exercise. The PCT for each of the LV segments at exercise were significantly greater when compared to rest (P<0.01).

No significant effect of exercise on interV synchrony was observed (P=0.638). A tendency was observed for the lateral/anterior (P=0.097) and the anterior/inferior (P=0.112) synchrony (Table 1) to reverse at peak exercise suggesting that further investigation may reveal an alteration in intraV timings at peak exercise.

Table 1. IntraV timings (ms) at rest and at peak exercise (mean±SEM)

	Rest	Peak exercise	P value
Lateral-septal	20.6±21.9	33.0±15.6	0.734
Lateral-anterior	-7.2±6.8	48.8±14.1	0.097
Lateral-inferior	35.1±31.1	27.6±20.3	0.835
Septal-anterior	-27.8±29.7	5.3±2.6	0.709
Septal-inferior	14.5±46.4	-5.4±12.9	0.677
Anterior-inferior	42.3±20.8	-2.3±7.7	0.112

Ghio S et al. (2004). Eur Heart J 25, 571-578

Coats A. (2001). Heart 86, 574-578

Nowak B et al. (1995). Am J Cardiol 75, 904-907

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC20

Reproducibility of haem oxygenase-1 protein induction following hydrogen peroxide treatment in human lymphocytes and monocytes

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The altered expression of the haem oxygenase-1 (HO-1) gene is used *in vitro* as a marker of oxidative stress (Tyrrell and Basu-Modak, 1994). This gene may also represent a useful marker of oxidative stress *in vivo*, although the reproducibility of the response to oxidant treatment first needs to be determined (Offord et al. 2000). The aim of the present investigation was to assess the reproducibility of the level of induction of HO-1 protein following hydrogen peroxide (H_2O_2) treatment in freshly harvested monocytes and lymphocytes.

After local ethics approval, ten male subjects (age 26 ± 4 y, height 1.77 ± 0.02 m, and body mass 80 ± 3 kg; mean ± S.D.) reported to the laboratory following an overnight fast on two occasions, separated by two weeks. Subjects rested in a supine position for ten min prior to a venous blood sample (25ml) being taken. Mononuclear cells were isolated from peripheral blood and exposed to 50 μM H_2O_2 for 30 min at 37°C. HO-1 protein was analysed by flow cytometry (FACScan, Becton Dickinson, USA) at 4, 6, 24 and 48 h after treatment. HO-1 protein was expressed as the fold change in the median fluorescence intensities of the treated *vs.* sham-treated controls. Peak HO-1 induction values were analysed for reproducibility between trials using the % change in the mean and the standard error of the measurement (SEM) as a % coefficient of variation (CV) as described by Bland and Altman (1996). Retest correlation for each pair of observations was also determined. Statistical significance was accepted at $P \leq 0.05$.

The % change in the mean ± SEM as a % CV for peak HO-1 induction was −2 ± 14 % and 13 ± 18 % in lymphocytes and monocytes respectively (figure 1). There was no systematic bias at any timepoint, indicating no systematic difference in the response between trials. Retest correlation was significant for peak HO-1 induction in both lymphocytes ($r = 0.92$, $P \leq 0.01$) and monocytes ($r = 0.67$, $P \leq 0.05$).

These findings demonstrate that the level of peak HO-1 protein induction in human lymphocytes and monocytes is a reproducible response to H_2O_2 treatment.

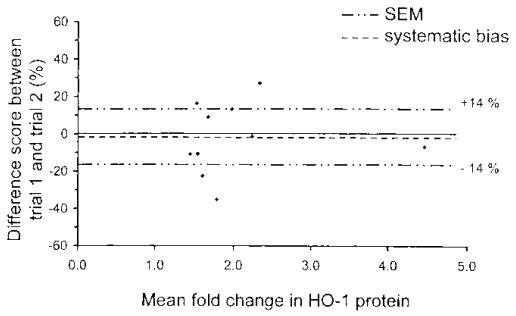

Figure 1: Reproducibility of peak HO-1 induction in lymphocytes. Data represent the differences (%) between trials 1 and 2, expressed relative to the mean fold change of the two measurements in HO-1 protein for each subject.

Bland JM & Altman DG (1996). *Br. Med. J.* **312,** 1654.

Offord E van Poppel G & Tyrrell RM (2000). *Free Rad. Res.* **33,** 5-19.

Tyrrell RM & Basu-Modak S (1994). *Methods Enzymol.* **234,** 224-235.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC21

Sliding and straightening of the ulnar nerve during limb movements studied in vivo in man using ultrasound imaging

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Peripheral nerves slide and stretch in order to accommodate changes in nerve bed length that occur during joint movements. It has been suggested that peripheral nerves can become slack in joint positions that reduce their bed length (Sunderland, 1978). This has been demonstrated in vivo for the median nerve (Dilley *et al.*, 2003). The present study uses an in vivo method to examine sliding and straightening of the ulnar nerve in response to upper limb movements.

High frequency ultrasound images were used to measure nerve trunk bowing in the upper arm in a series of positions from the arm by the side with the elbow and wrist straight to full elbow flexion, 90° shoulder abduction and wrist extension, a position designed to stretch the ulnar nerve. Ulnar nerve sliding was measured in the forearm and upper arm during single joint movements using frame-by-frame analysis of image sequences (Dilley *et al.*, 2003).

During 40° wrist extension (limb position = 90° shoulder abduction and elbow straight) (n = 7 subjects) the ulnar nerve moved distally by 1.1 ± 0.4 mm (mean ± S.E.M.) in the proximal forearm and by 2.0 ± 0.4 mm in the distal forearm. Neither reducing nor increasing the bed length by altering arm position (arm by side and 90° elbow flexion respectively) affected the amount of nerve excursion during wrist extension (n = 4). Nerve movement in the forearm during 90° elbow flexion averaged only 0.7 ± 0.2 mm (n = 4) and in the upper arm 0.2 ± 0.1 mm (n = 1) despite a large increase in ulnar nerve bed length. Shoulder abduction produced 0.3 ± 0.3 mm of nerve movement in the proximal forearm (n = 2), whereas a similar limb movement for the median nerve produced 3.4mm (Dilley *et al.*, 2003).

Images of the ulnar nerve showed considerable curvature when the elbow was straight with the arm by the side, and when the elbow was flexed the nerve straightened (fig 1). Across the 26mm image the length of nerve changed by an average of 0.27 ± 0.05 mm (n=4 subjects), accounting for a 1% length change. These findings are consistent with observations on cadavers using MRI (Patel *et al.*, 1998).

In summary, it would seem that the ulnar nerve, like the median, accommodates much of the change in bed length during joint movements by straightening rather than stretching.

All procedures accord with current local guidelines and the Declaration of Helsinki.

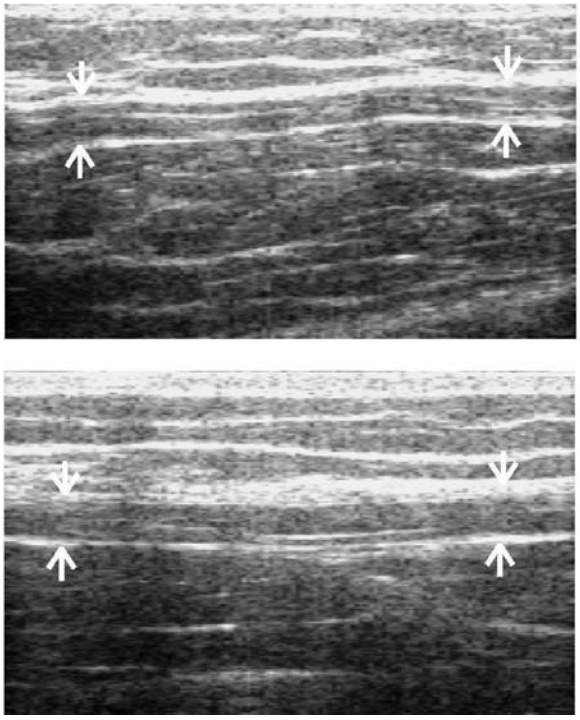

Figure 1. (upper) Nerve trunk bowing with arm by side and (lower) straightening with combined elbow flexion, shoulder abduction and wrist extension, a position designed to tension the ulnar nerve.

Dilley A et al. (2003). *Clin Biomech* **18**, 899-907

Patel V et al. (1998). *J Shoulder Elbow Surg* **7**, 368-374

Sunderland S. (1978). Nerve and Nerve injuries, second ed. Churchill Livingstone, Edinburgh

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC22

Influence of cold water immersion on indices of muscle damage following prolonged intermittent shuttle-running exercise

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The objective of this investigation was to elucidate the proposed ameliorative role of cryotherapy on indices of muscle damage following a bout of prolonged intermittent shuttle-running exercise. Following University Ethical Committee approval, twenty active males (age 22.7 ± 3.3 years, height 1.80 ± 0.05 m, body mass 83.7 ± 11.9 kg, $\dot{V}O_{2max}$ 52.5 ± 4.9 ml.kg^{-1}.min^{-1}; mean ± S.D.) performed a 90 minute intermittent shuttle-run previously shown to result in marked muscle damage and soreness (Thompson *et al.* 1999). Immediately following exercise subjects were randomly assigned to either cryotherapy treatment (n=10) or a control group (n=10). Cryotherapy consisted of 10 minutes lower limb immersion in cold water (10 ± 2°C). Ratings of perceived soreness (visual analogue scale), isometric maximal voluntary contraction (MVC) of the leg extensors and flexors and efflux of intracellular proteins were monitored prior to exercise, treatment and then at regular intervals up to 7 days post-exercise. Data were analysed using a mixed-design factorial ANOVA.

Exercise resulted in severe muscle soreness, temporary reduction in muscular function and elevated serum markers of muscle damage, all peaking within 48 hours post-exercise. Cryotherapy reduced muscle soreness at 1, 24 and 48 hours compared to the control group ($p<0.05$) (Fig. 1).

Decrements in isometric maximal voluntary contraction of leg flexors were reduced following cryotherapy treatment at 24 (12 ± 4%) and 48 hours (3 ± 3%) compared to the control group (21 ± 5% and 14 ± 5%) ($p<0.05$). Exercise induced increases in serum creatine kinase activity peaking 24 hours (1302 ± 92 U.l^{-1}, n=20) post-exercise ($p<0.05$), which was unaffected by cryotherapy. Serum myoglobin concentration was also elevated, peaking 1 hour post-exercise in the control group (45.9 ± 5.4 nmol.l^{-1}) but this response was ameliorated with cryotherapy treatment (29.6 ± 4.8 nmol.l^{-1})($p<0.05$).

These findings suggest cold water immersion immediately following prolonged intermittent shuttle-running reduces some indices of exercise-induced muscle damage.

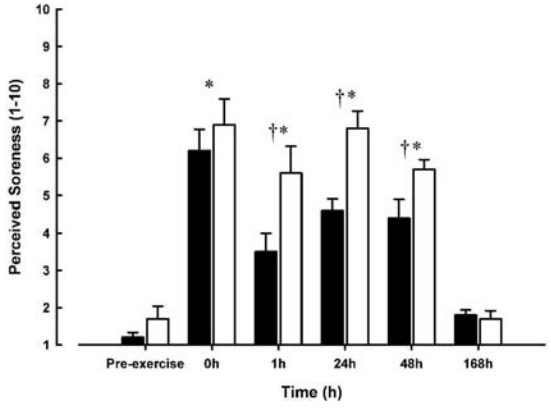

Figure 1: Perceived muscle soreness following exercise for cryotherapy (solid bars) and control (clear bars) groups. Values are mean ± S.E.M. * $p<0.05$ different from pre-exercise for both groups, $p<0.05$ different between groups.

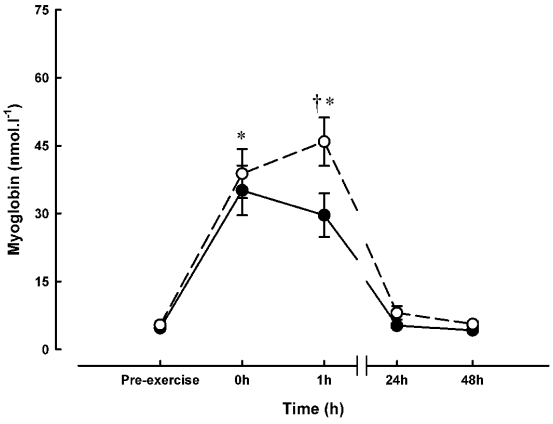

Figure 2: Serum myoglobin concentration following exercise for cryotherapy (solid line) and control (broken line) groups. Values are mean ± S.E.M. * $p<0.05$ different from pre-exercise for both groups, $p<0.05$ different between groups.

Thompson D *et al.* (1999) *J Sports Sci* **17**, 387-395.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC23

The effect of caffeine ingestion on human neutrophil oxidative burst responses following prolonged exercise.

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Strenuous exercise is associated with transient alterations in neutrophil number and function (Smith, 1997). Caffeine is a non-selective adenosine receptor antagonist consumed by athletes for its ergogenic properties (Graham, 2001). Neutrophils express both A_1 and A_2a adenosine receptors with their occupancy producing differing responses (Thibault *et al.*, 2002) yet little is known about the effect of caffeine on neutrophil responses to exercise. Therefore, the aim of this study was to investigate the effect of caffeine ingestion on neutrophil counts and the formyl-methionyl-leucyl-phenylalanine (fMLP)-induced oxidative burst response following prolonged cycling.

Following local ethics committee approval, 8 endurance trained males (mean ± SEM: age 24 ± 1 years; height 1.79 ± 0.03 m; body mass 72.8 ± 2.8 kg; VO_2max 65.6 ± 1.9 ml.kg^{-1}.min^{-1}) cycled for 90 min on a stationary ergometer at 72.3 ± 0.7% VO_2max. On two occasions, separated by 1 week, participants arrived at the laboratory following an overnight fast and 60 h abstention from caffeine containing products and were randomly assigned to ingest either 6 mg.kg^{-1} body mass of caffeine (CAF) or dextrose powder (PLA) 60 min before exercise. Participants consumed water only

(2 ml.kg^{-1} body mass) every 15 min during exercise. Venous blood samples were collected at rest, pre-exercise, immediately post-exercise and 1 h post-exercise. Neutrophil counts were performed using an automated cell counter. The *in vitro* neutrophil oxidative burst response to fMLP was assessed using a chemiluminescence (CL) assay (Knight Scientific Limited, Plymouth, UK). Serum caffeine and plasma adrenaline were determined using a spectrophotometric assay (Dade Behring, Milton Keynes, UK) and high-performance liquid chromatography, respectively. Results were analysed using a two-factor repeated measures ANOVA. *Post hoc t* tests with Holm-Bonferroni adjustment were applied where appropriate. Statistical significance was accepted at $P<0.05$.
Immediately post-exercise, serum caffeine and plasma adrenaline were significantly higher on CAF than PLA ($P<0.01$). At this time, blood neutrophil counts were ~125% greater than at rest (main effect of time, $P<0.01$) however, peak fMLP-induced neutrophil CL had fallen significantly on PLA only (CAF: rest, 11.3 ± 1.3 relative light units (RLU), post-exercise, 6.2 ± 0.7 RLU ($P>0.05$); PLA: rest, 13.5 ± 2.1 RLU, post-exercise, 4.9 ± 0.7 RLU, $P<0.01$). These findings suggest that in addition to its known ergogenic effects, caffeine may attenuate the exercise-induced fall in the neutrophil oxidative burst response to fMLP.

Graham TE (2001) *Sports Med* **31**, 785-807

Smith JA (1997) *Int J Sports Med* **18**, S46-S55

Thibault N *et al.* (2002) *J Leukoc Biol* **71**, 367-377

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC24

Leg muscle strength, power and symmetry of elderly fallers and non-fallers

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The ageing process is accompanied by a loss of skeletal muscle bulk and mechanical slowing and also an increased risk of falling. Falls in the elderly cause substantial health and personal problems. Many of them occur for medically unexplained reasons. Establishing the mechanisms for these falls should lead to the development of effective intervention programmes to decrease the incidence of injurious falls. Decreased muscle strength, power output and also increased asymmetry of strength and power in the lower limbs have been suggested as possible causes of unexplained falls. However evidence for the role of asymmetry is limited (Skelton et al. 2002) and conflicting for strength (Lord et al. 1999; Schwender et al. 1977). Forty four healthy older people (76.1 ± 0.8 years (mean ± sem), 29 women) and 35 with a history of unexplained falls (75.9 ± 0.6 years, 30 women) participated in the study which had local ethical approval. They were all living independently in the community. Bilateral measurements of peak leg extensor power (Nottingham Power Rig) and the maximal voluntary contraction (MVC) isometric strength of the quadriceps and ankle plantar flexors (isokinetic dynamometry) were measured. A univariate general linear model (SPSS) with sex as a co-factor was used for analysis. Post-hoc Dunnett tests were used where appropriate (alpha = 0.01).
In the fallers and non-fallers respectively the percentage difference between limbs was similar for the MVC of the quadriceps (11.4 ± 1.2 and 12.8 ± 2.0) and plantar flexors (19.9 ± 2.7 and 19.1 ± 3.3) and also for leg extension power (18.2 ± 3.1 and 13.9 ± 1.5). In view of the lack of differences between groups for limb symmetry, force data for the stronger limb are presented. Isometric strength was lower in the fallers than non-fallers for both quadriceps (335 ± 27 and 395 ± 21 N respectively, $P<0.006$) and plantar flexors (502 ± 49 and 643 ± 36 N, $P<0.001$). This was also the case for power output during leg extension (150 ± 12 and 180 ± 10 W, $P<0.001$).
Isometric strength and power showed a strong association with falling, confirming recent findings. However, asymmetry does not appear to relate to falls risk.

Lord, S.R. et al.(1999). J. Am. Geriatr. Soc. 47, 1077-81.

Schwendner, K.I. et al. (1997). J. Gerontol. Series A. 52, M155-M160.

Skelton, D.A. et al. (2002). Age & Ageing 31, 119-25.

We are grateful for funding from the GKT Charitable Foundation and the

European Commission Better Ageing Project

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC25

Gross and work efficiency of club level rowers on a Concept II rowing ergometer

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To ensure the reliability and consistency of testing in rowing, biomechanical and physiological analyses are usually performed in a laboratory. The British Olympic Association has standardised the stroke frequency (SF) during each stage of the lactate threshold test in order to ensure consistency, although rowers are still allowed to 'free pace' in trials. This study investigated the gross and work efficiency of ergometer rowing at varying intensities and stroke frequencies.
Ten experienced male rowers of mean (SD) age 23.5 (2.36) years, height 183.5 (7.63) cm, body mass 81.5 (4.0) kg, VO_{2MAX} 5.1 (0.21) l.min^{-1}, and lactate threshold 86.9% (2.8) of VO_{2MAX} participated. Rowers completed an incremental step test to volitional failure. On subsequent days rowers performed 10 minutes of ergometer rowing at SFs of 24, 28, and 32 strokes per minute at intensities of; 'no resistance' (NR), 200W, 230W, 260W and 290W. Heart rate, VO_2 and VCO_2 were measured every 10 seconds, and post-exercise lactate concentrations were assayed from finger prick blood samples. Gross (work accomplished/energy expended) and work (work accomplished/energy expended above that in rowing without a load) efficiencies were calculated as described by Gaesser & Brooks (1975). Statistical analysis was performed using a 2-way ANOVA ($P<0.05$). There were significant differences ($P<0.001$) in VO_2 between the three SFs during the NR trial and in plasma lactate ($P<0.05$) between the SFs of 24 and 32, but not between the heart rates. Oxygen consumption at the three SFs at power outputs of 200W, 230W, 260W and 290W were significantly different ($P<0.001$). A signi-

ficant difference in gross efficiency (P<0.01) between SFs of 24 and 32 (P<0.01), and SFs of 28 and 32 (P<0.05) were identified. Significant differences in work efficiency between SFs of 24 and 32 (P<0.01), and between 24 and 28 (P<0.001) were also identified (Table 1). Significant differences (P<0.05) in heart rate between SFs of 24 and 32 were observed. There were no significant differences (P>0.05) in plasma lactate levels for power output or SF. Oxygen consumption, gross and work efficiency are affected by SF. To ensure that rowing ergometer tests are reliable, SF must remain consistent for all physiological experiments.

Table 1. Mean (±SEM) VO_2, gross and mechanical efficiency during 10 min simulated rowing at SFs of 24, 28 and 32, with and without resistance (*significant difference between 24spm and 32spm, ** 28spm and 32spm, <#\160>24spm and 28spm)

	Power	24spm	28spm	32spm
VO_2 (l min^{-1})	NR	1.37* (0.06)	1.60** (0.05)	1.80 (0.05)
	Resistance	4.13* (0.06)	4.16** (0.06)	4.29 (0.07)
Gross Efficiency (%)	Resistance	15.8* (0.26)	15.7** (0.21)	15.2 (0.20)
Work Efficiency (%)	Resistance	22.1* (0.45)	25.1 † (0.48)	24.3 (0.71)

Gaesser, G and Brooks, G. (1975). J Appl Physiol, 38:1132-1139.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC26

Fluid Provision and Metabolic Responses to Soccer-Specific Exercise

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During a competitive soccer match there is a net reduction in muscle glycogen, and the exercise intensity is high enough to induce appreciable heat load, causing players to lose up to 3 litres of sweat. The aim of this study was to manipulate the administration of sports drinks during soccer-specific exercise and to investigate the effect on metabolic responses.

After providing written informed consent and undergoing familiarisation, 12 male soccer players of mean (±S.D.) age: 24 ±4 years; height: 1.80±0.1 m; body mass: 76.5±9) kg; VO_{2max}: 61.08±4 $ml.kg^{-1}.min^{-1}$ performed a soccer-specific protocol, incorporating static periods, walking, jogging, cruising and sprinting on a motorised treadmill on three occasions. On two occasions either 7 $ml.kg^{-1}$ BM of carbohydrate-electrolyte (CHOv) or placebo (PLA) solution was ingested 0 and 45 min (538±66 ml; total 1075±132 ml). On a third occasion the same volume of carbohydrate-electrolyte solution was consumed (CHOf) but in smaller volumes at 0, 15, 30, 45, 60, 75 min (179 ± 22 ml). Blood samples were collected at 0, 45 and 90 min and analysed for glucose, glycerol and insulin. Respiratory analyses were undertaken throughout to determine the rate of carbohydrate oxidation (Frayn, 1983). Trials were performed in a double-blind counter-balanced manner. Repeated measures ANOVAs were used to identify differences and significance was accepted at P<0.05. Glucose and insulin concentration (Table 1), and carbohydrate oxidation (PLA: 3.02 ±0.7 $g.min^{-1}$; CHOv: 3.60 ± 0.8 $g.min^{-1}$; CHOf: 3.50 ± 0.6 $g.min^{-1}$) were higher (P<0.05) during CHOv and CHOf compared with PLA and there were no differences between CHOv and CHOf. Glycerol (Table 1) was higher (P<0.05) during PLA compared with CHOv and CHOf.

Ingesting carbohydrate-electrolyte solution significantly affected plasma metabolites and increased carbohydrate oxidation. The timing and volume of ingestion did not significantly affect metabolism.

Table 1: Glucose, glycerol and insulin concentration (means ± S.D.)

	0 min	45 min	90 min
Plasma glucose (mM)			
PLA	5.08 ± 1	5.23 ± 1	4.94 ± 1
CHOv	5.14 ± 1	5.76 ± 1	5.64 ± 1
CHOf	5.16 ± 1	6.52 ± 1	6.16 ± 1
Plasma glycerol (uM)			
PLA	67.5 ± 27	112.4 ± 44	181.4 ± 75
CHOv	63.7 ± 32	82.1 ± 30	114.3 ± 32
CHOf	65.2 ± 30	83.8 ± 32	131.5 ± 50
Serum insulin (mIU/l)			
PLA	32.1 ± 19	25.7 ± 15	21.4 ± 18
CHOv	33.4 ± 19	31.7 ± 21	28.6 ± 33
CHOf	32.0 ± 19	37.3 ± 18	31.9 ± 19

Frayn, K.N. (1983). J. Appl. Physiol. 55, 628-634.

This study was supported by GlaxoSmithKline

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC27

Human knee-extensors architecture: diurnal rhythmicity and torque characteristics

S.J. Pearson[1], S.A. Bruce[2], P. Graham - Smith[1] and G. Onambele[3]

[1]Directorate of Sport, University of Salford, Manchester, UK, [2]School of Biomedical Sciences, Kings College, London, UK and [3]Department of Exercise & Sport Science, Manchester Metropolitan University, Manchester, UK

Time of day increments in strength have previously been reported (Deschenes et al. 1998; Martin et al. 1999), but no attempt to study this effect in conjunction with internal muscle structure has been made, even though in vivo, muscle architecture (fibre pennation angle (θ) and muscle size), are determinants of the capacity of a muscle to generate torque (Maganaris et al. 2001). The aim of the current study was therefore to examine whether muscle architecture can account for a) force changes or b) any changes in the torque-angle relationship with time of day. Sixteen healthy young men (aged 23±5.7 years) were tested at 7h45am and 5h45pm. To prevent order effects, seven subjects were tested in the order am to pm, and the rest in the order pm to am. Knee extensors test angle was randomised and the best of 3 contractions (peak isometric extension torque (PIET)) at each angle was used for analysis. The investigation was approved by the Manchester Metropolitan University Institutional Ethics Committee and all subjects gave their written informed consent to participate in the study.

PIET showed an average 7.0 ± 1.8% (p=0.023) upward shift throughout the range (30-90 deg) in the evening compared to the morning. The polynomial regressions fitted through the torque/angle relationship data showed a 10 deg shift of the angle at which PIET occurs (from ~80 deg at am to ~70 deg at pm) so

that PIET was seen at shorter muscle lengths in the evening. Also at the knee angle related to the PIET, in the contracted muscle a 14 ± 2.0% (p=0.002, paired t test) time-of-day related change in θ (to a greater θ at pm compared with am) was observed.
Muscle architecture was measured both at rest and at a standardised force level (250 N), both am and pm. In the resting muscle, increments of 8 ± 1.2% (p=0.001) in muscle thickness, and 13 ± 5.1% (p=0.046) in pennation angle (see Fig. 1), were observed at pm relative to am. In a subset (n = 5) a trend for a decrement in the pennation angle (- 5.6%) was observed from am to pm during the standardised exertions.
These results thus suggest that the time-of-day differences in muscle extensor torque are partly explained by changes in muscle architecture.

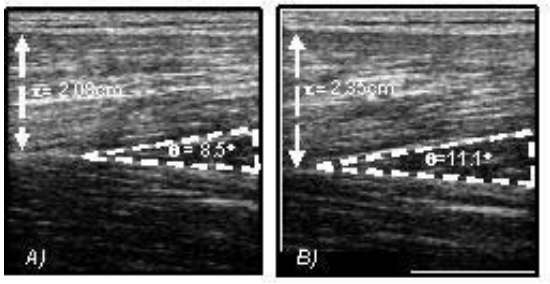

Figure 1. Typical sonographs of the sagittal plane of the vastus lateralis muscle at rest, with the knee at an angle of 85Deg (right angle is 90Deg). A) AM and B) PM, showing pennation angle (theta) and muscle thickness (t).

Deschenes MR, Kraemer WJ, Bush JA, Doughty TA, Kim D, Mullen KM and Ramsey K (1998). Med Sci Sports Exerc. 30(9): 1399-1407, 1998.

Maganaris CN, Baltzopoulos V, Ball D and Sargeant AJ (2001). J Appl Physiol. 90: 865-872, 2001.

Martin A, Carpentier A, Guissard N, Van Hoecke J and Duchateau J (1999). Muscle and Nerve. 22: 1380-1387, 1999.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC28

Lactate clearance after combining exercise and massage following a bout of maximum intensity cycling exercise

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Massage has been shown to increase blood lactate clearance during recovery from a short bout of maximum intensity cycling exercise (Jones & Cotterrell 1999; Mondero & Donne, 2000) but not as effectively as continued lower leg muscular activity, despite producing a significant improvement in perception of feeling. The present study investigated the effects of combining a short period of continued low level exercise recovery followed by leg massage on lactate clearance following a 30sec Wingate Anaerobic Test (WAnT). The recovery modes were a combination of 15min continued low level cycling exercise (CE at 60%HRtmax) followed by 30min of either continued cycling exercise (CE+CE), vibratory massage (CE+VM), manual massage (CE+MM) or supine rest (CE+SR).
There were no significant differences between the WAnT power outputs (646.5±72.4watts; mean±SD, repeated measures ANOVA) in 10 male subjects (22.3±2.3yrs, 74.7±7.6kg, height 176.0±6.9cm, and 13.3±4.2% body fat)
During the recovery period, peak finger prick blood lactate (15.9±1.5mMol·l-1) was seen at 3mins and after 15min of CE lactate had fallen to 45.6±7.3% of peak. At 45mins (following a further 30mins of CE) lactate had fallen to its pre-exercise level (89.6±1.6% of peak). Massage, either manual (MM) or vibratory (VM) following 15min CE was less effective in reducing lactate (CE+MM to 80.3±6.3% ($p<0.05$) and CE+VM to 78.4±8.1% of peak at 45min ($p<0.05$)), but was more effective than supine rest (CE+SR to 67.6±5.4% of peak at 45min ($p<0.05$). Supine rest with no CE intervention gave the slowest lactate clearance (57.5±6.7% of peak at 45min ($p<0.001$)). Therefore, attempting to increase lactate clearance by continued cycling during the period when lactate levels were highest (first 15min) was only marginally more effective than giving 45min of massage alone (MM 72.4±6.0% ($p<0.001$) and VM 73.0±3.9% of peak ($p<0.001$).
Perception of feeling at 45mins (using a 13-point scale, -6 Very Bad; 0 Neutral; +6 Very Good) was greatest with MM and CE+MM (5.8±0.6 and 5.7±0.5 respectively), and worst with either SR or CE+SR (1.6±1.7 and 2.6±2.1). There was a significant ($p<0.05$) negative correlation between perception of feeling and blood lactate ranging from r = -0.92 (SR+SR) to -0.78 (MM+MM).
The present study indicates that the positive psychological effect of manual massage and to a lesser extent vibratory massage during recovery are linked to, but cannot be attributed entirely to, increased lactate clearance.

Jones GE & Cotterrell D (1999) JPhysiol 518, 183P

Mondero J & Donne B (2000) Int J Sports Med 21, 593-597

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC29

Tendon compliance: implications for the measurement of voluntary activation levels

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The literature reports a large variation in activation capacity (AC), eg 92% (Newman et al. 2003) and 35% (Behm et al. 1995) using the twitch interpolation technique (figure 1). Loring & Hershenson (1992) suggested that the series compliance can affect the twitch force in muscle, but no report to-date has yet systematically studied this possibility. We aimed here to determine a) if tendon compliance might change acutely and b) how this may affect calculations of AC.
Twelve healthy young men (aged 27 ± 3.7 years) were tested at 7h45AM and 5h45PM. Patella tendon compliance was deter-

mined at a knee angle of 85 deg after the method by Maganaris & Paul (1999). AC of the knee extensors during maximal voluntary contractions was determined by the twitch interpolation method (see figure 1). The investigation was approved by the Manchester Metropolitan University Ethics Committee. All subjects gave written informed consent to participate in the study.

Tendon compliance increased by ~ 46.5% ($p = 0.0001$) from 4084 N.mm-1 (Youngs modulus = 2309 Pa) AM to 2184 N.mm-1 (Youngs modulus = 1235 Pa) PM. This change was in parallel with a 53 ± 20.0 % ($p = 0.039$) increase in time to relaxed twitch peak and a 37 ± 17.1 % ($p = 0.039$) increase in interpolated twitch height. The combination of the two latter effects culminated in an AC decrement (13 ± 3.8% ($p = 0.019$) to 14 ± 4.6% ($p = 0.032$), depending on whether AC calculation used the ratio of relaxed to interpolated or potentiated to interpolated twitch heights when comparing PM to AM. We propose that the measured lowered AC is an artifact of changes in tendon compliance.

In conclusion, our results indicate an increase in time to twitch peak PM, which may reflect changes in the sensitivity of the muscle to electrical stimuli but is most likely due to the decreased stiffness of the musculo-tendinous elements in series with the muscle. This mechanical change is proposed to play an important part in the calculation of activation level.

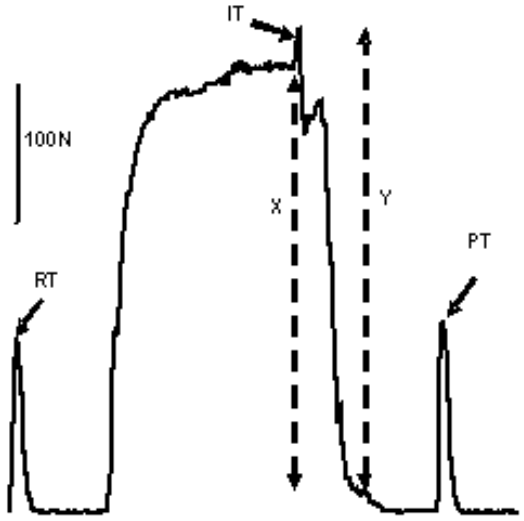

Figure 1. Sample force trace during the activation assessment protocol. Activation level calculations. A) 1-IT/RT. B) 1-IT/PT. C) 1-((Y/X)-1). Where IT is the interpolated twitch, RT is the relaxed (unpotentiated) twitch, PT is the (relaxed) potentiated twitch, X is the total isometric torque preceding an IT application, and Y is the total torque at the peak of the IT response.

Behm DG, Anderson K and Curnew RS, (2002). J Strength & Conditioning Res. 16(3): 416-422.

Loring SH and Hershenson MB, (1992). J Appl Physiol. 71: 513-521

Newman SA, Jones G and Newham DJ, (2003). Eur J Appl Physiol. 89: 49-499.

Maganaris CN, Paul JP, (1999). J Physiol. Nov 15;521 Pt 1:307-13

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC30

Exercise and Glucose Ingestion does not influence Adiponectin concentrations in Type 2 Diabetes

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Adiponectin is a protein exclusively secreted by adipocytes (Scherer *et al.* 1995). Circulating adiponectin concentrations are inversely related to obesity and Type 2 diabetes (Weyer *et al.* 2001) and a regulatory role in glucose metabolism has been suggested (Tsao *et al.* 2002). Plasma adiponectin concentrations increase ~4 fold, reaching a peak at ~60 minutes postprandially in obese individuals, yet do not change in lean healthy individuals (English *et al.* 2003) or during exercise (Ferguson *et al.* 2004). The aim of this study was to measure the effects of a glucose meal or exercise on adiponectin levels on Type 2 diabetic subjects. Six (5 male) obese dietary controlled type 2 diabetic individuals (age, 57.8±3.9yr, BMI, 31.9±1.4kg/m^2, HbA1c, 8.4±0.7%, mean±SD) and six (male) age matched lean, non-diabetic controls (age 52.1±3.6yr, BMI 24.8±0.7kg/m^2, HbA1c 4.5±0.6%) were recruited for this study. Approval was granted by the East Sussex Local Research Ethics Committee. Each subject was given a 75g Oral Glucose Tolerance Test (OGTT) at rest and following one hour of exercise (~50% VO_2max) on separate occasions. Whole blood samples were immediately analysed for glucose concentrations via the glucose oxidase reaction and plasma analysed for adiponectin and insulin using the ELISA method. A repeated measures ANOVA was used to determine statistical differences between groups and across time. Adiponectin concentrations were lower in the Type 2 diabetes group when compared with the control group ($P=0.042$) and fasting adiponectin concentrations were inversely ($r=-0.54$) related to insulin sensitivity (HOMA-IR = fasting glucose (mmol/l) x fasting insulin (μU/ml)/22.5), both group data combined. Exercise ($P=0.371$) or glucose ingestion ($P=0.898$) had no effect on fasting plasma adiponectin concentrations in either group (Table 1). Adiponectin concentrations do not seem to be affected acutely by exercise or a glucose meal in Type 2 diabetic or non-diabetic individuals.

Table 1: Adiponectin concentrations (μg/ml) at baseline, immediately post exercise and 80 min post glucose ingestion at rest and following exercise

Group	Baseline	Immediately post-exercise	Rest 80 min OGTT	Exercise 80 min OGTT
Control	7.70±0.55	7.51±0.83	7.82±0.42	8.07±0.36
Type 2 diabetes	2.63 ±0.36	2.83±0.46	2.89±0.26	2.74±0.47

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Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC31

Quantifying heat strain and its effect on intermittent sprint exercise in male games players under climatic heat stress

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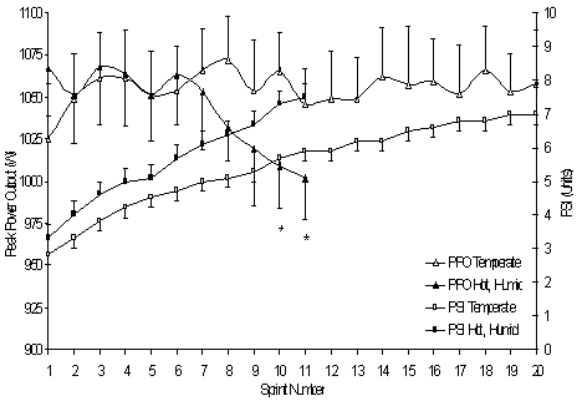
Heat *per se* (heat strain) has been suggested as a contributing factor to reduced exercise times and intermittent sprint running times while under heat stress (Morris *et al.* 1998), yet less is known about the effect of heat strain on intermittent sprint cycling. This investigation aimed to quantify the amount of heat strain (Physiological Strain Index, PSI; Moran *et al.* 1998) and its effect on peak power output (PPO) during a Cycling Intermittent Sprint Protocol in temperate (CISPtemp; 21.4±0.3°C, 62.2±2.0% relative humidity; RH;) and hot, humid conditions (CISPhot; 37.1|±1.4o°C, 74±2.4 %RH). Eight male games players (age, 23.3±0.8yr, mass 76.4±2.2kg, peak oxygen uptake 3.8±0.1L.min^{-1}, means±SEM) completed both CISPs, each of twenty, 2min periods (10s rest, 5s sprint against a resistance of 7.5% body mass and 105s active recovery at 50% of predetermined) in a randomised order. Oxygen consumption, ratings of thermal sensation (TS), perceived exertion (RPE) and fingertip blood samples (analysed for lactate concentration) were measured in all conditions. Peak PSI was greater in CISPhot (8.2±0.2) compared to CISPtemp (7.0±0.2; $P<0.01$). Subjects completed the CISPtemp without any decline in PPO. In CISPhot PPO declined by sprint 10 (fig 1; $P<0.05$) and subjects (n=5) stopped after sprint 11. Negative correlations were observed between PSI and PPO in CISPhot (r=-0.80; $P<0.01$) and PPO declined with a corresponding PSI value of 7.3 (High). PSI positively correlated with the RPE and TS in both environments ($P<0.01$). There were no differences in blood lactate or oxygen consumption between environments. Decrements in PPO while under heat stress are related to greater heat strain, rather than changes in lactate concentration or oxygen cost. Observed relationships between the quantifiable PSI and the subjective RPE/TS may indicate a centrally regulated mechanism (Noakes. T. D, 2004).



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PC31A

N-CAM and Pax-7 immunoreactive cells are expressed differently in the human vastus lateralis after a single bout of exhaustive eccentric exercise

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Neural cell adhesion molecule (N-CAM) and the paired box transcription factor, Pax-7, are both known to be markers of myogenesis. While N-CAM expression in human muscle has been well documented, few studies have investigated the expression of Pax-7. The aim of this study was to identify the immunoreactivity of these two satellite cell markers after a single bout of exhaustive eccentric exercise. Eight untrained males (22-27yrs) performed 210 maximum eccentric contractions in an isokinetic dynamometer. One leg was exercised involuntarily using electrical stimulation (ES), while the contralateral leg was exercised voluntarily (VOL). Muscle biopsies of the vastus lateralis muscle were taken from both legs on day 0 and 8 after the exercise bout. Serial sections were immunohistochemically stained for N-Cam and Pax-7. The percentage of N-CAM and Pax-7 positive cells was calculated as the number of positive cells/(positive cells + myonuclear number) x 100. Numerical data is shown as the mean ± standard deviation and a student's t-test was used to determine statistical significance. Myofibre necrosis was found in the vastus lateralis muscle of the ES leg only with a significant increase in desmin negative cells: Day 0 (0 ± 0%) and day 8 (13.7 ± 12.0%; $p<0.05$) and a corresponding inflammatory cell infiltration into the necrotic myofibres. No change above baseline was found in the VOL leg. The ES leg also showed a significant increase in N-CAM expression over time (Day 0: 2.48 ± 0.69%; Day 8: 22.53 ± 17.46%, $p<0.05$) and when compared to the VOL leg (Day 0: 2.42 ± 0.69%; Day 8: 5.26 ± 2.17%, $p<0.05$). Pax-7 immunoreactivity increased significantly over time in the ES leg (Day 0: 3.34 ± 1.56%; Day 8: 9.38 ± 6.24%*, *$p<0.05$) and when compared to the VOL leg (Day 0: 3.13 ± 1.81%; Day 8: 2.66 ± 2.19%). The pattern of expression of the N-CAM and Pax-7 was dissimilar. In particular, the Pax-7 expression 8 days after the exercise bout was markedly less than N-CAM expression. It has recently been reported that PAX-7 expression is not found in freshly regenerating myotubes or in presumed myoblasts. Additionally, Pax-7 expression remains low or undetectable at the proliferative myoblast stage but becomes prominent in an increasing proportion of mononucleate cells after the induction of differentiation (Reimann et al., 2004). We have previously hypothesised that after a single bout of exhaustive eccentric exercise there is a proliferation of satellite cells that do not undergo terminal differentiation unless an additional bout of exercise is undertaken (Crameri et al., 2004). This study suggests that the N-CAM positive cells at day 8 are satellite cells that have undergone proliferation but have not undergone terminal differentiation.

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