

resistance training (RT), and 2) examine the relationship between acute changes in vascular function in response to a single bout of exercise and chronic changes following 4-weeks of exercise training. **Methods:** Using a randomised cross-over design, seven male subjects (age: 21 ± 3 yr, weight: 73 ± 9 kg, height 177 ± 4 cm) were assigned to 4-week ET and RT (randomly ordered) exercise training program (3 sessions per week), with a washout of 6 weeks. Vascular function was assessed using flow-mediated dilation (FMD), both before and immediately after the first exercise bout and after 4-weeks training. **Results:** Following an acute bout of exercise, we found a significant increase in FMD (main effect 'time': $P=0.041$), which was not significantly different between both types of exercise (interaction-effect: $P=0.11$). Brachial artery FMD following an acute bout of resistance exercise increased from $7.4 \pm 2.2\%$ to $10.6 \pm 3.6\%$, whilst endurance exercise caused an immediate increase in $6.7 \pm 1.8\%$ to $7.1 \pm 2.0\%$. We found no significant changes in FMD following the 4-week exercise training in either group (main effect 'time': $P=0.12$, $7.4 \pm 2.2\%$ to 8.36 for resistance training and $6.7 \pm 1.8\%$ to 8.7 ± 3.1 for endurance training). There was no significant correlation between acute changes in FMD after a single bout of exercise and exercise training-induced changes in FMD ($r=0.09$, $P=0.75$). **Conclusion:** Our data suggest an acute improvement in vascular function following an acute bout of 30-minute exercise, independent on the type of exercise. Despite these immediate changes in vascular function, we found that 4-weeks endurance or resistance exercise training did not alter vascular function. Moreover, the acute changes in vascular function did not relate to the change in vascular function after 4 weeks. These data suggest that adaptation to 4 week exercise training, both after endurance and resistance exercise, cannot be simply explained through examining acute changes in vascular function to a single bout of exercise.

THERMOREGULATION IN ENDURANCE TRAINED ADULTS

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THERMOREGULATION IN ENDURANCE TRAINED ADULTS Galán-Carracedo, J. 1,2, Suárez, A.2, Guerra-Balic, M. 1 1: FPCEE-Blanquerna, Ramon Llull University, Barcelona (Spain) 2: Corporació Mèdica Catalana, Cornellà (Spain) **Introduction** Thermoregulation is the ability of an organism to keep its body temperature (BT) within certain values, even when the environmental temperature is very different. The BT is related to metabolic activity and external environment conditions. The internal thermoregulation process is one aspect of homeostasis, which involves seeking homeothermic control. Many studies show how muscle activity increases our temperature due to metabolic heat production, and the importance of preventing hyperthermia. Any tendency to hypothermia ($BT < 35^\circ C$) can also cause cardiovascular disorders and exhaustion, becoming one of the main metabolic factors leading to fatigue in trained subjects. The aim of this study was to determine the evolution and the effect of BT in trained adults of endurance sports during a progressive increasing treadmill running test. Our intention was to observe if there is a characteristic pattern of temperature behavior among individuals with better functional capacity. **Methods** Ten trained adults of endurance sports (age= $38,55$, $SD= 7,08$ years), previously familiarized with the experimental procedures, performed a progressive increasing treadmill test with a constant environment temperature of $24^\circ C$. During the test, BT was obtained with axillar digital thermometer High-Speed (Microlife), while fatigue level was also obtained through Borg's RPE scale. A 6 minute warm up period at $6-8$ km/h was performed. The test began at 8 km/h with increments of 1 km every 2 minutes, until exhaustion, and $10'$ of recovery. The treadmill slope was constant at $1,5\%$. BT of each participant was obtained at basal time, at the end of the warm up, at the end of the testing and during the $5'$ and $10'$ of the recovery period. Descriptive for all variables were calculated, and non-parametric Wilcoxon matched pairs test was applied to compare the differences of the BT between phases. **Results** Mean temperatures at basal phase, end of warming up, end of testing and recovery $5'$ and recovery $10'$ were $35,9^\circ C$, $35,8^\circ C$, $35,5^\circ C$, $36^\circ C$ and $36,1^\circ C$, respectively. The mean BT present significant differences decreased at the end of the warm up and at the end of the test comparing basal data ($p < 0.01$; $p < 0,02$ and $p < 0,005$), just when exhaustion appeared. **Discussion** Results showed a decrease of BT when exhaustion appeared in trained adults of endurance sports, associated to fatigue. Endurance high intensity training could improve the metabolic response and thermoregulation of individuals. More research is needed.

THE EFFECT OF INSULIN ON HEART RATE VARIABILITY AT REST AND DURING SUBMAXIMAL EXERCISE

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Introduction The purpose of the study was to investigate the effect of insulin on heart rate variability (HRV) at rest and during submaximal exercise **Methods** Ten healthy male subjects (23 ± 1 yr) performed in different days 20 min of cycling at 35% HRR, with and without ingestion of 200 g of glucose (maltodextrin). Capillary blood glucose concentration was measured by fingerstick at rest, 20 min following glucose ingestion and every 5 min during exercise. Plasma insulin was calculated from the blood glucose measurements using the Glucosafe model (Pielmeier et al. 2012). Heart rate (HR) was measured noninvasively (Finometer) during all trials and RR intervals were processed by fast Fourier transform (FFT) for determination of low-frequency (LF, $0.045-0.15$ Hz) and high-frequency (HF, $0.15-1.0$ Hz) components. **Results** Plasma insulin was higher at 0 min ($P < 0.001$) and 5 min ($P < 0.001$) during exercise with glucose supplementation compared to baseline. Furthermore, there was higher plasma insulin between the first three measurements; 0 min, 5 min and 10 min ($P < 0.05$) during exercise with- compared to exercise without glucose supplementation. The LF and HF components expressed as absolute power (ms^2) decreased significantly at the onset of exercise ($P < 0.05$) but there was no difference between the two conditions at any time interval during exercise. The LF/HF ratio was not different with and without glucose supplementation, or during exercise and rest. **Discussion** There was no effect of plasma insulin on HRV at rest and during submaximal exercise. **References** Pielmeier et al. (2012). *J Clin Monit Comput* 26:319-28 Contact Svolian@hst.aau.dk

PERIPHERAL AND CENTRAL EFFECTS OF SMOKELESS TOBACCO ON EXERCISE ENDURANCE IN MEN

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Introduction A proliferation of nicotine use in the sport environment has been observed in recent years mainly as smokeless tobacco (Zandonai et al., 2013). Nicotine has been listed in World Anti-doping Agency's Monitoring Program from 2012 to 2015 in order to detect potential patterns of abuse. The aim of this study was to investigate the effects of Snus (SS), an oral smokeless tobacco, on the perception of fatigue during aerobic exercise to TTE. **Methods** The study was a double-blind placebo controlled (SP) crossover design. Fourteen healthy male non-smokers were recruited. Subjects were studied during three sessions on cycle-ergometer: experimental session 1 (Exp1) consisted in an incremental exercise test to determine maximal aerobic power (Wmax); Exp2 and Exp3 consisted in exercise at 65%

Wmax until exhaustion in SS or SP conditions. During the Exp2 and Exp3, muscle and cerebral oxygenation by means of NIRS (near-infrared spectroscopy) and global rating of perceived exertion (RPE) were recorded. Before and after all experiments, the Profile of Mood of State questionnaire (POMS) was administered to subjects. Subjects were then tested by means of Transcranial Magnetic Stimulation (TMS) to assess changes in cortico-motor excitability due to the prolonged exercise. Results Time to exhaustion (TTE) was not significant difference (64.4 ± 41.5 min SS; 51.6 ± 17.2 min SP) (19.2%) in paired Student's t-test. RPE in the first 30 minutes during both of the sessions showed a significant difference after 10 minutes from start exercise. POMS questionnaire values did not show any significant differences under both conditions (SS, SP). We found significant differences in the cerebral and muscular tissues oxygenation levels in the first 30 minutes of the exercise during SS and SP tests. In particular, at cerebral level, tissue oxygenation index was significantly larger in SS than in SP from the 10th to 30th min of exercise. Conclusion The study showed that the SS effect, compared to placebo condition, could not be an improvement of fatigue during an endurance exercise until exhaustion despite of an increase in tissue muscular and cerebral oxygenation. These data supported the hypothesis of a major activity induced by nicotine as a central stimulator (Mundel and Jones, 2006). References Mundel T, Jones DA. (2006). Effect of transdermal nicotine administration on exercise endurance in men. *Exp Physiol*, 91, 705-713 Zandonai T, Baraldo M, Franceschi L, Zappamiglio T, Chiamulera C. (2013). Effects of smokeless tobacco (snus) administration on exercise endurance in men. SRNT Annual Meeting Boston MA (USA) p 165

CHANGES IN SKIN-GAS ACETONE CONCENTRATIONS FOLLOWING A LOW INTENSITY CONSTANT-LOAD EXERCISE

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Introduction Ketone bodies (3-hydroxybutyrate, acetoacetate and acetone) are generated in the liver, mainly from the oxidation of fatty acids, and are exported to peripheral tissues, such as the brain, heart, kidney and skeletal muscle for use as energy fuels (Mitchell et al. 1995). Acetone is mainly generated from decarboxylation of acetoacetate. Therefore, during exercise increased acetone levels related to exercise intensity have previously reported in plasma (Balasse et al. 1989), expired air, and skin-gas (Yamai et al. 2009). On the other hand, Romijn et al. (1993) have reported that peripheral lipolysis was stimulated maximally at 25 % $\dot{V}O_{2max}$ during constant-load exercise. The purpose of the present study was to confirm changes in skin-gas acetone concentrations following a low intensity constant-load exercise. Methods Nine healthy male students performed constant-load (20% HRmax) cycle exercise for 30 min. The skin-gas samples were obtained by the covering left hand for 30 sec with a polyfluorovinyl bag (Tedlar bag; GLScience, Tokyo, Japan) in which pure nitrogen gas was introduced, and collected in a sampling bag at rest, 3, 6, 9, 12, 15, 20, 30 min after, and 5, 10, 20 min recovery of the exercise. Acetone concentration was analyzed by gas chromatography Results The skin-gas acetone concentration significantly increased 9-30 min after the exercise compared to the resting values ($p < 0.05$), then immediately returned to resting values 5 min recovery of the exercise. Thus the peak skin-gas acetone concentration in the subjects was significantly ($p < 0.05$) higher than the resting values (0.07 ± 0.02 vs 0.17 ± 0.07 ppm; mean \pm SD). Discussion Increasing skin-gas acetone concentrations after the exercise in this study indicated that low intensity constant-load cycle exercise induced the production of ketone bodies in the liver, mainly from the oxidation of fatty acids (Mitchell et al. 1995), and some of increased ketone bodies eliminated as skin-gas acetone (Yamai et al. 2009). Furthermore, the levels of skin-gas acetone concentration seemed steady state 9-30 min after the exercise. These results indicated following possibilities. First, the production of ketone bodies in the liver might be reached maximal (Romijn et al. 1993) during this constant-load study. Second, the production of ketone bodies in the liver and consumption of ketone bodies in peripheral tissues such as skeletal muscles 9-30 min after the exercise might be same. References Mitchell GA et al. (1995) *Clin Invest Med* 18: 193-216. Balasse EO and Féry F. (1989) *Diabetes Metab Rev* 5: 247-270. Yamai et al. (2009) *Redox Report* 14: 1-5. Romijn et al. (1993) *Am J Physiol* 265: E380-E391. Contact Hiroshi Itoh (itoh.hiroshi@nitech.ac.jp)

THE DEVELOPMENT OF FATIGUE MODEL IN A TISSUE-ENGINEERED MUSCLE

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Introduction Muscle fatigue is one of the typical physiological phenomena in physical exercise. Muscle fatigue can be classified as central fatigue or peripheral fatigue. Although peripheral fatigue is thought to be important contributor to muscle fatigue, the research have been limited because there are few useful model. Three-dimensional tissue-engineered muscle (3D TEM) is a powerful tool in studying muscle biology and physiology. However, it is not clear if 3D TEM can be a useful model in studying peripheral fatigue. Therefore, the aim of this study was to develop and evaluate fatigue model in 3D TEM. Methods 3D TEM having two artificial tendons were constructed from C2C12 myoblasts embedded in type-I collagen gel. 3D TEM in 3 weeks culture was used for fatigue experiment. The contraction of 3D TEM was induced by electrical stimulation. Isometric twitch force of 3D TEM was induced and measured in every 10 min by electrical stimulation. The isometric twitch contractility was evaluated after electrical stimulation. The LDH cytotoxicity assay was done to investigate muscle cell damage by electrical stimulation. Twitch force was re-measured at 24h after fatigue-induction to assess force recovery. Results The twitch tension of 3D TEM rapidly declined to nearly 10% to maximal twitch tension by 60 min with electrical stimulation. Relaxation time of 3D TEM did not significantly alter by the tension decline. There was no significant differences in LDH release between 3D TEMs stimulated and non-stimulated by electrical pulse. Twitch tension of 3D TEM could not recover from tension decline for 24h. Discussion One definition of fatigue is any decline in muscle performance associated with muscle activity (Allen et al., 2008). In this study, twitch tension in 3D TEM rapidly declined for 60 min with electrical stimulation. The decline of muscle strength with fatigue in vivo gradually recover to normal level. however, the tension decline did not recover for 24h in 3D TEM. According to the LDH cytotoxicity assay, the decline did not accompany cell damage. In case of muscle fatigue in vivo, the muscle relaxation time becomes longer but no significant alternation was observed after tension decline in 3D TEM. Taken together, 3D TEM can be highly applicable to the study of defining physiological mechanisms in peripheral muscle fatigue but the mechanisms may reflect a part of complicated pathways in peripheral muscle fatigue in vivo. References Allen, D. G., Lamb, G. D., Westerblad, H. Skeletal muscle fatigue: cellular mechanisms. (2008) *Physiol Rev*, 88, 287-332. Contact Tomohiro Nakamura tomohiro.nakamura@oit.ac.jp