EUROPEAN COLLEGE OF SPORT SCIENCE

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Letter of Attendance

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GALLEGO SELLES, ANGEL

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attended the

27th Congress of the European College of Sport Science in Sevilla - Spain

between 30 August - 2 September 2022.

Prof. Jørn Wulff Helge
ECSS Congress President

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Confirmation of Presentation

This is to certify that the following title has been presented at the 27th Annual Congress of the European College of Sport Science between 30 August - 2 September 2022 in Sevilla - Spain.

GALLEGO SELLES ANGEL

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Abstr.-ID: 1852, Presentation format: Oral , Session name: OP-PN08 - The importance of Oxygen
Title: Nrf2 and NF-kB signalling, and antioxidant enzyme adaptations to sprint interval training are potentiated by brief ischaemia application during the recovery periods
Authors: GALLEGO-SELLES, A., GALVAN-ALVAREZ, V.1, MARTINEZ-CANTON, M.1, GARCIA-GONZALEZ, E.1, GELABERT-REBATO, M.1, GARCIA-PEREZ, G.1, SANTANA, A.2, MORALES-ALAMO, D.1
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Presentation date: 31.08.2022, 15:30, Lecture room: AUDITORIUM 3, No: 2

European College of Sport Science

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27th Annual Congress of the

EUROPEAN COLLEGE OF SPORT SCIENCE

30 Aug - 2 Sep 2022, Sevilla, España

Hosted by: Faculty of Sport Science - Universidad Pablo de Olavide

BOOK OF ABSTRACTS

Edited by:

Dela, F., Piacentini, M.F., Helge, J.W., Calvo Lluch, Á.,
Sáez, E., Pareja Blanco, F., Tsolakidis, E.

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at baseline (PRE) and following training (POST). Paired samples t-tests using changes in serum renin and copeptin concentrations (pre to post BFR/free-flow walking) were used to determine whether greater increases were observed following BFR walking. A repeated measures ANOVA was performed to compare changes in aerobic capacity and blood volume.
RESULTS: BFR walking resulted in significantly greater changes in serum copeptin (BFR: 5.5±2.9 to 57.8±99.4 pmol/L; free-flow: 6.3±3.8 to 6.7±4.0 pmol/L; p=0.02) and renin (BFR: 19.1±8.2 to 44.5±27.1 ng/L; free-flow: 19.41±7.2 to 28.5±15.1; p=0.001). Significant changes in maximal aerobic capacity were observed following training (p=0.03). Increases in VO2max were observed from PRE to POST for both BFRH (46.7±8.8 to 50.7±9.5 ml.kg-1.min-1, p=0.004) and BFRL (48.1±7.4 to 52.5±10.2 ml.kg-1.min-1, p=0.02) groups but not CON (48.8±9.4 to 49.2±8.6 ml.kg-1.min-1, p=0.6). No changes in blood volume were evident (BFRH: 83.4±12.3 to 82.0±10.5 ml.kg; BFRL: 76.6±12.8 to 78.2±13.9 ml.kg; CON: 77.3±9 to 78.4±8.7 ml.kg; p=0.7).
CONCLUSION: The current results support findings of improved maximal aerobic capacity following BFR exercise. Of note, mean improvements in VO2max were of a similar magnitude in both the high- (+9.6%) and low-frequency (+9.0%) BFR training groups. Despite large increases in circulating fluid retention hormones following an isolated bout of BFR exercise, blood volume over the training period did not increase in either group. The notable adaptations promoting greater endurance capacity with low intensity BFR exercise require further investigation to elucidate the mechanisms responsible.

NRF2 AND NF-KB SIGNALLING, AND ANTIOXIDANT ENZYME ADAPTATIONS TO SPRINT INTERVAL TRAINING ARE POTENTIATED BY BRIEF ISCHAEMIA APPLICATION DURING THE RECOVERY PERIODS

GALLEGO-SELLES, A., GALVAN-ALVAREZ, V.1, MARTINEZ-CANTON, M.1, GARCIA-GONZALEZ, E.1, GELABERT-REBATO, M.1, GARCIA-PEREZ, G.1, SANTANA, A.2, MORALES-ALAMO, D.1

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INTRODUCTION: *more authors: FERNANDEZ-GARCÍA, B.3, BOUSHEL, R.4,HALLEN, J.5, CALBET, J.A.L.1,4,5, MARTIN-RINCON, M.1
Reactive oxygen and nitrogen species (RONS) stimulate signalling pathways essential for the adaptative response to exercise. Nrf2 and NFkB transcription factors regulate over 150 genes involved in redox homeostasis, inflammation, and the antioxidant response. High-intensity exercise increases RONS and activates Nrf2, NFkB, and CaMKII signalling in human skeletal muscle (HSM), an effect potentiated by immediate ischaemia application (PMID: 32863217). It remains unknown how sprint interval training (SIT) modulates antioxidant enzyme expression and regulatory transcription factors. It is uncertain whether additional metabolic and RONS-mediated stress could further stimulate the adaptive response to SIT. We hypothesized that SIT would upregulate the basal and exercise-induced Nrf2 and NFkB signalling, with this effect being exacerbated by post-exercise ischaemia.
METHODS: Ten active subjects were tested before and after SIT (4-6 30s sprints, 4min recovery, 6 sessions in 2 weeks). Immediately after each sprint, the circulation of one leg was instantaneously occluded (300mmHg) for 30-50s. The main PRE and POST-training tests consisted of an incremental exercise to exhaustion (IE) followed by 90min rest and 6 bouts of supramaximal exercise to exhaustion at 120%VO2max (SPE) interspaced with 20s recovery, during which circulation of both legs was fully occluded. In addition, VL biopsies were taken at rest, 90 min after IE, and immediately after SPE unilaterally at PRE and bilaterally at POST from the leg training with free circulation (FCL) and the leg training with ischaemia (IS) for Western Blotting. Statistics: repeated-measures ANOVA
RESULTS: After SIT, basal protein levels of pSer536p65, IkBalpha, IkBbeta, pSer40Nrf2, Nrf2, Nrf2/Keap1 ratio, Catalase, SOD1, GR, pThr287CaMKII, and CaMKII were largely increased (~2.3-fold) solely in IS ($p<0.05$). Besides, SIT increased basal protein levels significantly more in IS for pSer176/180IKK, p50, p65 (~1.9-fold vs. FCL) ($p<0.05$). The acute upregulation following SPE observed at PRE and after SIT in FCL was blunted in IS ($p<0.05$) for pThr287CaMKII, IkBbeta, TrxR1, Nrf2, pSer40Nrf2, p65, pSer536p65, pSer176/180 IKK and pTyr705STAT3.
CONCLUSION: These findings show that Nrf2 and NFkB signalling and their downstream antioxidant enzymes are largely upregulated by additional time under oxidative stress during 2 weeks of SIT in HSM. Furthermore, we have shown that a marked elevation of the basal antioxidant response with training suppresses the acute exercise-induced activation of Nrf2 and NFkB signalling otherwise required in the non-trained state. This was achieved by employing a novel experimental approach where immediate ischaemia is applied after each bout in only one leg, exacerbating RONS and metabolite build-up, which may enhance the antioxidant capacity of HSM. Whether these adaptations are beneficial for performance or long-term adaptation to training remains to be evaluated.

PREMATURITY AND MAXIMAL EXERCISE CAPACITY AT HIGH-ALTITUDE: EVIDENCE OF HYPOXIC PRECONDITIONING?

MANFERDELLI, G.1, NARANG, B.J.2,3, BOURDILLON, N.1, DEBEVEC, T.2,3, MILLET, G.P.1

1: UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND; 2: JOŽEF STEFAN INSTITUTE, LJUBLJANA, SLOVENIA; 3: UNIVERSITY OF LJUBLJANA, LJUBLJANA, SLOVENIA

INTRODUCTION: Premature birth induces several long-term sequelae on the pulmonary and cardiovascular systems that contribute to reduced exercise capacity. However, preliminary data suggest that prematurity might, at least in-part, protect against some altitude-induced physiological alterations. Accordingly, we aimed to investigate the cardiorespiratory and muscle oxygenation responses to incremental exercise to exhaustion performed at sea level and at altitude in preterm and full-term born adults, to clarify the mechanism(s) underpinning prematurity-related impairments in exercise capacity.
METHODS: Healthy preterm (n=17; age, 21±1 years; gestational age, 29±1 weeks) and age-matched full-term (n=17; gestational age, 40±0 weeks) adult men underwent a hypoxic sensitivity test (i.e., random 1 to 8 consecutive breaths with 100% N2) to assess the hypoxic ventilatory response (HVR) and two incremental exercises to exhaustion on a cycle ergometer – one at sea level and the other at altitude (3375 m). Gas exchange, hemodynamics, and muscle oxygenation were assessed continuously by metabolic cart, transthoracic impedance, and near-infrared spectroscopy, respectively. Intramuscular matching between O2 delivery and utilization was assessed by the presence of a deoxygenation (Δ[deoxy(Hb+Mb)]) overshoot in the transition from rest to exercise.
RESULTS: The HVR was comparable between preterm and full-term adults (0.383±0.061 vs. 0.270±0.029 L/min/%, p=0.11). In normoxia, preterm demonstrated lower peak power output (276±10 W vs. 312±12 W, p=0.04) compared to full-term adults, but not in hypoxia (248±10 vs. 279±10 W, p=0.07 and 2.62±0.11 vs. 2.84±0.09 L/min, p=0.47), despite similar peak O2 uptake in both normoxia (48.5±2.6 mL/kg/min vs. 51.9±1.9 mL/kg/min, p=0.36) and hypoxia (36.4±1.6 mL/kg/min vs. 37.9±1.1 mL/kg/min, p=0.87). At peak, stroke volume (116±6 vs. 138±5 mL, p=0.02) and cardiac output (21.7±1.2 vs. 26.0±1.0 L/min, p=0.01) were lower in preterm compared to full-term adults in normoxia, but not in hypoxia (113±7 vs. 122±5 mL, p=0.53 and 21.1±1.2 vs. 23.2±0.9 L/min, p=0.35). Peak Δ[deoxy(Hb+Mb)] was increased from normoxia to hypoxia in full-term (11.0±1.5 vs. 16.2±2.1 µM, p=0.04) but not in preterm (13.5±2.5 vs. 13.7±2.0 µM, p=0.84)

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