istration did not increase overall TT power output compared to the Placebo and Control conditions (0.06-0.16 ±0.09), range of standardised differences ±90% CL). The overall times for all conditions were between 3:58.5 and 4:01.3 (min:s). The small differences between trials at specific 150m splits were not explained by quinine intake compared to control or the placebo trials. There were no substantial differences on the Feeling scale, RPE and end BLa between conditions.
CONCLUSION: Although we have previously shown that the ingestion of Quinine immediately prior to the start of a 3 km cycling TT achieves a short-lived improvement in cycling power output, in the current study, when Quinine was ingested after 2 km of effort, we failed to detect a subsequent effect on cycling power. It appears that ingesting 2mM of quinine during the last stage of a 3 km TT does not improve cycling performance and has little effect on physiological and perceptual responses.

ENHANCEMENT OF EXERCISE PERFORMANCE BY 48 HOURS, AND 15-DAY SUPPLEMENTATION WITH MANGIFERIN AND LUTEOLIN IN MEN

GELABERT-REBATO, M.1,2, WIEBE, J.C.2, MARTIN-RINCON, M.1, GALVAN-ALVAREZ, V.1, CURTELIN, D.1, PEREZ-VALERA, M.1, JUAN HABIB, J.1, PÉREZ-LÓPEZ, A.1,3, VEGA, T.2, MORALES-ALAMO, D.1, CALBET, J.A.L.1
1 UNIVERSITY OF LAS PALMAS DE GRAN CANARIA; 2 NEKTIUM PHARMA; 3 UNIVERSITY OF ALCALÁ

INTRODUCTION: The natural polyphenols mangiferin and luteolin have free radical-scavenging properties, induce the antioxidant gene program and down-regulate the expression of superoxide-producing enzymes. We have previously shown that these two polyphenols enhance sprint performance when administered every 8 h for the 48 h preceding the exercise (Gelabert-Rebato et al., 2018). However, the effects of these two polyphenols on exercise performance after prolonged supplementation remain unknown. Therefore, this study aimed at determining the acute and prolonged effects of oral supplementation with mangiferin and luteolin botanical extracts on exercise performance, muscle metabolism, and brain and muscle oxygenation in healthy young men. Given the fact that these two polyphenols may have ergogenic effects through several mechanisms, a specific exercise protocol was designed, including phases of low-intensity, high-intensity and repeated sprinting exercise combined with ischemia-reperfusion episodes.
METHODS: A combination of luteolin (peanut husk extract containing 95% luteolin, PHE) and mangiferin (mango leave extract (MLE), Zynamite®) at low (PHE: 50mg/day; and 140mg/day of MLE containing 100 mg of mangiferin; L) and high doses (PHE: 100mg/day; MLE: 420mg/day; H) was administered to twelve physically active men. Subjects performed incremental exercise to exhaustion, followed by sprint and endurance exercise, dosed every 8 h, 48 h (acute effects) and 15 days of supplementation (prolonged effects) with polyphenols or placebo, following a double-blind crossover design.
RESULTS: During sprint exercise, mangiferin+luteolin supplementation enhanced exercise performance, facilitated muscle oxygen extraction and improved brain oxygenation, without increasing the VO2. Compared to placebo, mangiferin+luteolin increased muscle O2 extraction during post-exercise ischemia, and improved sprint performance after ischemia-reperfusion likely by increasing glycolytic energy production, as reflected by higher blood lactate concentrations after the sprints. Similar responses were elicited by the two doses tested.
CONCLUSION: Supplementation with the combination of two botanical extracts of mangiferin and luteolin enhances exercise sprint performance, likely by improving brain oxygenation and allowing a higher muscle extraction of oxygen. These effects were observed following 48 h and 15 days of supplementation without significant differences between the two doses tested.
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Oral presentations

OP-PM37 Fatigue

DETERMINANTS OF TASK AND CONTRACTILE FAILURES DURING THE REPETITION OF SUSTAINED SUBMAXIMAL ISOMETRIC CONTRACTIONS

HUREAU, T.J.1,2, DUCROCQ, G.P.2, BLAIN, G.M.2
1:UNIVERSITY OF STRASBOURG, 2: UNIVERSITÉ CÔTE DAZUR

INTRODUCTION: Previous studies demonstrated that power output and muscle activation are adjusted by the central nervous system (CNS) during cycling exercise to limit the development of peripheral fatigue to an individual and task-specific critical threshold (1). However, this mechanism may not be involved in task failure of sustained isometric contractions, which might rather be determined by a deficit in muscle activation (2). Using an experimental intervention designed to potentially cause the subjects to voluntarily surpass the peripheral fatigue threshold (i.e. fatigue more), we aimed to determine whether peripheral fatigue is restrained to a critical threshold and determines time to task failure during the repetition of sustained submaximal isometric contractions involving large muscle mass.
METHODS: Seventeen healthy participants performed five sustained isometric squats until task failure interspersed with 5 min of passive recovery in between. Participants supported a barbell loaded with 50 % of individual body mass on their shoulders while maintaining their knees flexed at 90° until failure. Using supramaximal electrical femoral nerve stimulation, peripheral and central fatigue were quantified via pre- to post-exercise changes in quadriceps twitch force (Qtw) and voluntary activation (VA), respectively. To estimate quadriceps muscle activation during exercise, EMG root mean square (RMS) was normalized to the RMS recorded during pre-exercise maximal voluntary contractions (MVC) of the quadriceps (RMS%MVC).
RESULTS: Time to task failure was significantly ($P < 0.05$) reduced by 29 ± 13 % from the first (90 ± 7 s) to the second (61 ± 4 s) trial and by 11 ± 8 % from the second to the third (54± 3 s) trial, and stabilized thereafter (~50 ± 3 s, $P > 0.7$). At task failure during the first trial, quadriceps RMS%MVC only achieved 85 ± 4 % of pre-exercise values suggesting muscle activation failure from the CNS. In parallel, twitch force (– 42 ± 4 %) and VA (– 7 ± 2 %) were significantly reduced compared to baseline. During subsequent trials, gradual increases in the rate of rise in RMS%MVC and peak RMS% MVC ($P <0.05$) were found in parallel with gradual exercise-induced reductions in Qtw (– 53 ± 3 %) and VA (– 14 ± 3 %) up to the third trial, with no further change thereafter.