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Publicado por: Imprensa da Universidade de Coimbra

URL persistente: URI:<http://hdl.handle.net/10316.2/44094>

DOI: DOI:https://doi.org/10.14195/2182-7087_ex2018_31

Accessed : 26-Apr-2023 15:46:41

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VITAMIN D STATUS MODULATES INNATE IMMUNE RESPONSES AND METABOLIC PROFILES FOLLOWING ACUTE PROLONGED CYCLING

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KEYWORDS: neutrophil, elastase, inflammation, metabolomics

INTRODUCTION

The influence of vitamin D status on exercise-induced immunodepression remains unclear. The primary aim of our study was to investigate the effects of vitamin D status on innate immune responses to prolonged exercise. Secondly, we undertook a metabolomic profiling to suggest how the immune system may be modulated in relation to plasma 25(OH)D concentrations⁽¹⁾.

METHODS

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Twenty three healthy, recreationally active males (age 25 ± 7 years; body mass 76 ± 8 kg; height 179 ± 6 cm; maximal oxygen uptake ($\text{VO}_2 \text{ max}$) $56 \pm 9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) completed 2.5 h of cycling at 15% Δ (~ 55-60% $\text{VO}_2 \text{ max}$). Venous blood and unstimulated saliva samples were obtained before and after exercise. Based on available evidence, to date, on vitamin D status and innate immune responses (e.g. monocyte-derived cytokine production, plasma cathelicidin) in endurance athletes (He et al., 2013), participants were

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dichotomised using the following: plasma total 25(OH)D < 33 nmol/L (low) or > 33 nmol/L (medium-high).

RESULTS

Total lymphocyte count ($p = 0.013$) and neutrophil:lymphocyte ratio ($p = 0.033$), at rest, were lower in participants with low plasma 25(OH)D compared to those with greater plasma 25(OH)D, but the magnitude of exercise-induced changes in total and differential blood leukocytes, neutrophil-stimulated oxidative burst and salivary antimicrobial peptides were similar between groups ($p > 0.05$). Two-way mixed ANOVA revealed a significant main effect of group ($p = 0.010$) and a group X time interaction ($p = 0.003$) for bacterial-stimulated elastase release per neutrophil (neutrophil degranulation). Post hoc analysis of the low 25(OH)D group (pre-exercise: 100 ± 0 % pre-exercise, post-exercise: 83 ± 29 ; post-exercise: 58 ± 22) revealed a significant decrease from pre-exercise to 1 h post-exercise ($p = 0.007$), which at 1 h post-exercise was significantly lower compared with bacterial stimulated elastase concentration in those with higher plasma 25(OH)D (pre-exercise: 100 ± 0 % pre-exercise, post-exercise: 92 ± 20 ; 1 h post-exercise: 101 ± 31) ($p = 0.003$). Discriminant function analysis of plasma metabolomic profiles showed a clear separation of participants according to vitamin D status in post-exercise timepoints. Major sources of variation contributing to the effect of vitamin D status were markers of inflammation (linoleic acid metabolites) and metabolites belonging to the functional class of the tricarboxylic acid cycle.

CONCLUSION

These findings provide evidence of the influence of vitamin D status on exercise-induced changes in parameters of innate immune defence, and markers of inflammation and metabolic stress.

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