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

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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 concentrations⁴.

METHODS

Twenty three healthy, recreationally active males (age 25 ± 7 years; body mass 76 ± 8 kg; height 179 ± 6 cm; maximal oxygen uptake (VO₂ max) 56 ± 9 mL·kg⁻¹·min⁻¹) completed 2.5 h of cycling at 15% Δ (~ 55-60% VO₂ 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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dichotimised 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.

REFERENCES