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Influence of prolonged exercise and hydration status on antigen-stimulated cytokine production by whole blood culture

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ABSTRACT

Prolonged, strenuous exercise has been associated with a temporal depression of host defence, increasing susceptibility to upper respiratory tract illness (URTI). An elevated anti-inflammatory cytokine response to antigen challenge at rest has been reported as a risk factor for URTI. Chronic, strenuous exercise training appears to augment this anti-inflammatory response, with IL-10 release predicting URTI susceptibility in athletes. The purpose of this study was to determine the acute effects of a bout of prolonged exercise and hydration status on antigen-stimulated cytokine production. Twelve healthy males cycled for 120 minutes at 60% of maximal oxygen uptake on two occasions, once in a euhydrated state and once moderately hypohydrated. For the euhydrated trial, participants drank *ad libitum* during the 24 hours prior to the trial, and were provided with 250 mL water every 30 minutes during exercise. For the hypohydrated trial, fluid intake was restricted to 500 mL water during the 24 hours leading up to the trial, and no fluid was ingested during exercise. Blood samples were collected immediately before and after exercise, and following 2 hours of passive recovery. A full blood count was obtained, and plasma analysed for cortisol. *In vitro* antigen-stimulated cytokine production was determined from whole blood culture, using a multi-antigen vaccine as stimulant. Fluid restriction resulted in body mass loss of 1.3 ± 0.7 % and 3.9 ± 1.0 % before and after exercise, respectively. Exercise elicited a significant leukocytosis and elevated plasma cortisol, with no differences between trials. Post-exercise IL-10 production following stimulation was significantly higher than pre-exercise ($p<0.01$). Both IL-4 ($p<0.05$) and IL-10 ($p<0.01$) release per lymphocyte were significantly increased 2 hours post-exercise compared with pre-exercise. Antigen-stimulated IL-6 production was significantly reduced 2 hours post-exercise ($p<0.05$), an effect that remained significant when expressed per monocyte ($p<0.01$). Although not quite reaching statistical significance, antigen-stimulated IFN-γ and IL-8 release tended to decrease following exercise, as did monocyte production of TNF-α. IL-1β and IL-2 production were not significantly altered by exercise. No significant effect of hydration status was observed for any of the measured variables. Prolonged exercise appears to result in augmented anti-inflammatory cytokine release in response to antigen challenge, possibly coupled with an acute suppression of pro-inflammatory cytokine production. These findings correspond with previous studies using mitogen or endotoxin as stimulant. Neither cytokine production nor plasma cortisol was affected by moderate hypohydration induced by fluid restriction for 24 hours prior to and during exercise.