Acute sleep disruption: does it limit performance but heighten immunity?

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Abstract

Introduction: Alterations to sleep patterns in athletes are commonly reported prior to competition and during travel to events (Juliff et al., 2015; Erlacher et al., 2011). When sleep is completely deprived decrements in performance and increased perceptions of effort are reported (Temesi et al., 2013; Oliver et al., 2009). Circulating levels of white blood cells are reported to alter during periods of sleep deprivation (Kerkhofs et al., 2007) or remain unchanged (Costa et al., 2010). Sleep disruption is perhaps more commonly experienced in athletic populations which could alter biological rhythms, immune responses and performance.

Purpose: To examine the effect of disrupted sleep on exercise-induced physiological and immunological parameters.

Methodology: Ten healthy male cyclists performed 1h of cycling at a fixed power output on an indoor cycle ergometer following either a night of DS (woken every hour of the night over an 8h period) or a night of undisrupted sleep (US) (left undisrupted for an 8h period). Heart rate (HR), perceived rate of exertion and sleep parameters were all recorded. Blood was collected before, immediately after and 1h after exercise completion. White blood cells and their subtypes were enumerated.

Results: Statistically significant differences between US and DS trials were observed for the mean heart rate during the exercise bout (t(9) = 4.80, p < 0.01) and rate of perceived exertion (RPE) (t(9) = 3.86, p < 0.01). Repeated measures ANOVA revealed a significant trial (US/DS) * time (minutes) interaction with heart rate (F(12/108) = 5.94; p < 0.01) and RPE (F(12/108) = 3.8; p < 0.05), Bonferroni corrected post hoc analysis between trials is illustrated in figure 1 & 2 below. Repeated measures ANOVA yielded a significant trial (US/ DS) x interaction with time (pre, post, 1 h post) for total lymphocytes (F(2/18) = 5.84, p < 0.05, r = 0.5) suggesting lymphocytes were significantly altered with exercise depending on whether the participants had endured a night of DS or US. Bonferroni post hoc analysis revealed this was driven by the larger increase in lymphocytes at the post time point following the DS trial (t(9) = 2.88, p < 0.01, r = 0.69). CD8+ T-cells similarly demonstrated a trial x time interaction (F(2/18) = 4.12; p < 0.03, r = 0.43) with a larger increase in cell number apparent during the DS trial, post hoc analysis revealed this was driven by a larger increase in CD8+ T-cells immediately post exercise during the DS trial (t(9) = 2.69, p < 0.05, r = 0.67). There was no time x trial interaction observed for CD4+ T-cells, NK cells or γδ T-cells between US and DS conditions.

Conclusions: Our findings indicate that short-term changes to sleep architecture lower heart rate during fixed intensity exercise while the perception of effort was increased, suggesting that acute sleep deprivation reduced cardio-respiratory function which could lead to performance decrements during endurance exercise. Sleep disruption augmented lymphocyte redeployment from the peripheral blood compartment which may ‘prime’ the system during exposure to short-term sleep disruption.

Key words: Power meter; Performance; Athlete Performance Passport

References

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Figure 1: Heart rate during 1h of intense exercise following a night of undisturbed and disrupted sleep. Values are mean ± SE. # P< 0.004.

Figure 2: RPE during 1h of intense exercise following a night of undisturbed and disrupted sleep. Values are mean ± SE. # P< 0.004.