Results: Overall, AT8 levels were reduced in the SD groups compared to the NS ones ($p<0.05$), while Tau-1 levels were not significantly affected. A clear trend towards higher p[Thr205]-Tau levels was also observed after SD. The decrease in PPTau induced by SD was accompanied by an increase in both p[Ser9]-GSK3β and p[Ser473]-Akt2 levels, although statistical significance was reached only for the latter ($p<0.05$). Also, PP2A levels were lower in R3SD *vs*. R3NS ($p< 0.05$). Finally, melatonin levels were higher in R3SD vs. R3 ($p <0.05$).
Conclusions: The present results indicate that SD soon after ST enhances PPTau dephosphorylation, coherently with the observed concomitant increase of p[Ser9]-GSK3β and p[Ser473]-Akt2. This molecular pattern is known as being neuroprotective, and may be mediated by melatonin that can activate Akt2 and, consequently, inhibit GSK3β by acting on the PI3K/Akt2/mTOR antiapoptotic pathway (Risso et al., 2015). These findings open interestingly translational perspectives in the use of sleep deprivation in patients suffering from hypothermia-induced brain PPTau formation due to general anesthesia (Whittington et al., 2013).
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ELITE ATHLETES PILOT STUDY OLYMPIC GAMES RIO 2016

L. Bonamassa [1]. [1] *UNIVE - Università Cà Foscari Venezia, Venice, Italy*

Introduction: The data suggest that shifting the time with late-night race simulation tends to desynchronize circadian rhythms, compromising the physiological and psychological state of athletes. The results indicated that there are effects on HRV (cardiac variability), SQ (quality) and TMD (mood disorders).
Materials and Methods: Three male swimmers (age 21± 1), qualified in the sprint races for the 2016 Olympic Games in Rio. Monitored over the course of a week of night training sessions with the same time schedule as the Olympic semi-finals and finals. Athletes were monitored during sleep for quality assessments (SQ,), where the ratio of total time in bed was calculated using actigraphy (SenseWear, BodyMedia USA). The athletes were monitored in the two days before the start of the protocol (T0) and in the following days, respectively after 1, 3, 5 days (T1,T2,T3). Resting Heart Rate (HRR) and Cardiac Variability (HRV) were evaluated upon awakening (Minicardium, Hosand - Italy). All athletes responded to an "Evening/Morning" questionnaire in relation to the chronotype profile. The assessment of mood disorder using the "Total Mood Disturbance" (TMD) was conducted before and after the week of night training. Nutrition has been specially adjusted to contribute to the maintenance of sleep quality. For each variable (mean and DS) a time-factor univariate ANOVA (T0-T1-T2-Y3) was performed to compare the significant effects between the individual sessions. At the same time, a t-test was performed to verify any differences in the TMD (at the beginning and at the end of the protocol). The significance level was set at $p<0.05$ using SPSS 15.0 Software.
Results: The data suggest that shifting the time with late-night race simulation tends to desynchronize circadian rhythms, compromising the physiological and psychological state of athletes. The main results indicated that there are effects on HRV (cardiac variability), SQ (quality) and TMD (mood disorders). The sleep detected is below the required physiological limit and higher than the alert levels of partial deprivation, starting from the second day of the protocol. Similar results were reported with a study that predicted partial sleep deprivation for four days for 2.5h, with a mood alteration, increased depression, tension, confusion, fatigue, rabies, decrease in strength.
Conclusions: Increased sleep disorders, increased fatigue and tiredness, as well as alterations of the nervous system are present in 82% of athletes before competitions and seem to be assimilated to a state of overreaching in the short term even in swimmers. The time shift was shown to have a significant effect on the observed psychological and physiological variables, indicating potential effects on athletes' behaviour as early as day two. Therefore, to limit the effects of an induced change in circadian rhythms, proper education of the athlete seems to be particularly effective.
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ARC GENOTYPE MODULATES SLOW WAVE SLEEP AND EEG SPECTRAL POWER FOLLOWING TOTAL SLEEP DEPRIVATION

B. Satterfield [1,2], M. Finlay [1,2], L. Skeiky [1,2], D. Lawrence-Sidebottom [1,3], M. Schmidt [1,2], J. Wisor [1,2], H. Van Dongen [1,2]. [1] *Washington State University, Sleep and Performance Research Center, Spokane, United States;* [2] *Washington State University, Elson S. Floyd College of Medicine, Spokane, United States;* [3] *Naval Post Graduate School, Operations Research, Monterey, United States*

Introduction: The activity-regulated cytoskeleton associated protein (*ARC*) gene is an immediate early gene that is involved in synaptic plasticity. Evidence from a rodent model suggests that *Arc* may also be involved in sleep homeostasis. In humans, sleep homeostasis is manifested by a marked increase in slow wave sleep (SWS) following acute total sleep deprivation. There are large, trait individual differences in the magnitude of this SWS rebound effect, with concomitant changes in EEG spectral power. However, little is known about the molecular mechanisms regulating the sleep homeostat and the expression of individual differences therein. We sought to determine whether a single nucleotide polymorphism (SNP) of the *ARC* gene is associated with individual differences in SWS rebound and EEG spectral power.
Materials and Methods: 50 healthy normal sleepers (27.3±4.9 years; 28 females) participated in one of two in-laboratory studies. Each participant had a 10-hour baseline sleep opportunity (22:00–08:00), 38 hours of consecutive wakefulness, then a 10-hour recovery sleep opportunity (22:00–08:00). Sleep periods were recorded polysomnographically and scored visually according to standardized criteria. Genomic DNA was assayed for the *ARC* c.*742+58C>T non-coding SNP, rs35900184. The genotype effect on time spent in SWS was assessed using mixed-effects ANOVA with fixed effects for *ARC* genotype, night, and their interactions. Log-transformed spectral power over 0.2 Hz frequency bins in each of four frequency bands – delta (0.8–4.0 Hz), theta (4.2–8.0 Hz), alpha (8.2–12.0 Hz), and beta (12.2–16.0 Hz) – was analyzed by band using a mixed-effects ANOVA with fixed effects for *ARC* genotype, night, frequency bin, and their interactions. All analyses included study and age as covariates and a random effect over subjects on the intercept.
Results: The genotype distribution in this sample was 33 C/C homozygotes, 11 C/T heterozygotes, and 6 T/T homozygotes. There was a significant *ARC* x night interaction on SWS rebound ($F_{2,47}=4.84$, $p=0.012$). C/C homozygotes exhibited 60.4±3.0 minutes more SWS, whereas C/T heterozygotes exhibited only 46.0±5.2 minutes and T/T homozygotes only 42.4±7.1 minutes more SWS during recovery sleep compared to baseline. There was also a significant *ARC* x night interaction on theta ($F_{2,1833}=5.94$, $p=0.003$) and alpha ($F_{2,1833}=8.58$, $p<0.001$) power. C/C homozygotes had 18.9% more theta power and 8.7% more alpha power, whereas C/T heterozygotes had 17.9% more theta and 7.6% more alpha power and T/T homozygotes had 20.0% more theta power and 15.1% more alpha power during recovery sleep compared to baseline sleep.
Conclusions: Our results show that *ARC* genotype is associated with individual differences in SWS and in NREM EEG spectrum responses to sleep deprivation. *ARC* appears to mediate these physiological responses to sleep loss through two seemingly distinct dynamics, with homozygosity for the T allele being associated with a blunted SWS response and an amplified increase in spectral power in the theta and alpha bands. The functional implications of these *ARC* effects remain to be determined.
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