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SUBJECTIVE ALERTNESS, BEHAVIORAL ALERTNESS, AND PERCEPTION-ACTION COUPLING REFLECT DISTINCT ASPECTS OF NEUROBEHAVIORAL RESILIENCE DURING SIMULATED MILITARY OPERATIONAL STRESS

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Introduction: Despite exposure to operational stressors (e.g., sleep loss, caloric restriction), military personnel must maintain different aspects of neurobehavioral function (i.e., subjective alertness, behavioral alertness, perception-action coupling) to operate safely within military environments. It is unclear whether perception-action coupling, which refers to the ability to 'read and react' to ever-changing circumstances, reflects a distinct aspect of neurobehavioral resilience from subjective and behavioral alertness. Further, prior sleep may enhance resilience during subsequent exposure to operational stressors. Therefore, we examined resilience across different neurobehavioral tasks during exposure to simulated military operational stress (SMOS) and examined differences in baseline sleep between resilient and vulnerable participants.
Methods: Forty-nine military personnel (11 females, 26.6 ± 5.8 years) completed a 5-day SMOS protocol that included two days of sleep restriction and disruption (sleep opportunities: 01:00-03:00 and 05:00-07:00) accompanied by caloric restriction (50% caloric need). Participants completed tasks of subjective alertness (Profile of Mood States Vigor subscale, POMS), behavioral alertness (Psychomotor Vigilance Task) and perception-action coupling (Perception-Action Coupling Task) at baseline and at 04:00 across the two nights of sleep disruption. For each neurobehavioral outcome, a two-step decision-making process defined resilient and vulnerable participants: resilient participants demonstrated high alertness/performance during sleep disruption and minimal change from baseline during sleep disruption. Kappa coefficients were calculated to determine agreement in resilience classification across different neurobehavioral outcomes. Further, differences between resilient and vulnerable participants in baseline sleep questionnaires (Pittsburgh Sleep Quality Index; Epworth Sleepiness Scale) and polysomnography (sleep efficiency; sleep fragmentation; and slow wave activity, SWA) were examined with independent t-tests.
Results: Classification of participants as resilient or vulnerable differed across neurobehavioral outcomes, as indicated by kappa values <0.60. Resilient participants, defined by POMS, had lower baseline SWA than vulnerable participants ($t = 2.06$, $p = .04$). No other differences in sleep were observed between groups.
Conclusion: Subjective alertness, behavioral alertness, and perception-action coupling reflect distinct aspects of neurobehavioral resilience, highlighting the importance of understanding the operational relevance of different neurobehavioral measures when assessing fatigue risk. Further, more baseline SWA, indicating higher baseline sleep need, may reflect vulnerability to SMOS and subsequent sleep loss.
Support (If Any): Department of Defense Award #W81XWH-17-2-0070

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VIGILANT ATTENTION PERFORMANCE ADVANTAGE CONFERRED BY TNFA G308A POLYMORPHISM IS ASSOCIATED WITH DIMINISHED DELTA AND THETA POWER IN THE NON-REM SLEEP EEG

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Introduction: Carriers of the A allele of a single nucleotide polymorphism of the TNFα gene (G308A, rs1800629) are relatively resilient to vigilant attention performance impairment from total sleep deprivation (TSD), as compared to G/G homozygotes, and even exhibit a small performance advantage at baseline. The mechanism underlying this effect remains unclear. As TNFα is a sleep regulatory substance, we investigated whether TNFα G308A genotype is associated with systematic differences in markers of sleep homeostasis.
Methods: N=168 healthy young adults (ages 27.4±5.4y; 86 women) participated in one of seven in-laboratory TSD studies. During TSD, performance was assessed every 2–3h using a psychomotor vigilance test (PVT). The TSD period was preceded and followed by nocturnal sleep opportunities (baseline and recovery, respectively), which were recorded polysomnographically and scored according to AASM criteria. The EEG (C3-M2 derivation) of stages N2 and N3 non-REM sleep was investigated using spectral analysis.
Results: The genotype distribution of the sample was 0.6% A/A, 26.8% A/G, 72.6% G/G, in Hardy-Weinberg equilibrium (P=0.14). As documented previously, A allele carriers, compared to G/G homozygotes, had fewer PVT lapses (RTs>500ms) at baseline and during TSD, indicating greater resilience to sleep deprivation. During both baseline and recovery sleep, A allele carriers, compared to G/G homozygotes, displayed reduced power in the delta (0.8–4.0Hz; P=0.017) and theta (4.2–8.0Hz; P=0.004) bands of the non-REM sleep EEG.
Conclusion: The performance advantage of the A allele carriers brings to mind the "banking sleep" phenomenon previously observed in sleep deprivation studies with prior sleep extension, suggesting that the A allele carriers gained this advantage by essentially being able to bank sleep. If this interpretation is correct, then the diminished power in the delta and theta bands of the non-REM sleep EEG in the A allele carriers, which suggests reduced homeostatic sleep pressure during both baseline and recovery sleep, may imply that the A allele carriers operate at a lower homeostatic setpoint due to an underlying advantage in the recuperative efficiency of sleep.
Support (If Any): NIH R01HL070154, R21CA167691, R01HL105768; ONR N00014-13-C-0063, N00014-13-1-0302, FAA DTFAAC-11-A-00003, CDMRP W81XWH-16-1-0319, USAMRDC W81XWH-05-1-0099, and an SRS Elliot D. Weitzman, M.D. Research Grant.

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EXPLORING SEX AND STRAIN DIFFERENCES IN MOUSE MODELS OF SLEEP DISRUPTION

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Introduction: Sleep abnormalities are a common feature of neurodevelopmental disorders (NDDs), and can have