

Understanding the effects of sleep deprivation and acute social stress on cognitive performance using a comprehensive approach

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Objective

Different professionals (e.g. in the military) have to perform cognitive challenging tasks in multi-stressor environments. However, our understanding how combined stressors interact and affect cognitive performance is limited (Van Dongen & Belenky, 2009). This study examined how sleep deprivation (SD) and acute social stress affect cognitive performance in isolation and in combination, and used a comprehensive approach to find evidence for a (shared) mechanism. Recent research suggests that SD leads to higher amounts of proinflammatory markers (i.e. cytokines) in the blood, which assumedly contribute to a decline in cognitive performance (Irwin, 2019; Shields et al., 2017). In addition, acute social stressors have also been shown to elicit an immune response, as reflected by circulating cytokines in blood (Marsland et al., 2017; Prather et al., 2014). These findings suggest that different stressors may affect cognitive performance through an effect on the immune system. We therefore hypothesize that individuals showing a high proinflammatory response to a combination of two stressors (SD and acute social stress) are more vulnerable to cognitive decline compared to individuals showing a lower proinflammatory response. To test this hypothesis, we measured not only cognitive performance, but also the physiological response and biochemical determinants of metabolism and inflammation at baseline and after SD, but also in response to an acute social stressor (Tkacheenko & Dinges, 2018).

Method

97 Participants took part in a two-day study. An approval for this study was granted by an accredited medical research ethics committee (MREC Brabant, reference number: P2045). All participants gave written informed consent. The SD group (52) underwent a night of controlled SD and the control group (45) had a normal night of sleep at home. In the morning before and after this night, but also before and after a social stressor, both groups performed a cognitive test battery to assess cognitive performance. Vigilant attention was measured using the Psychomotor Vigilance Task (PVT) and executive functioning using the SYNWIN, Go/No-Go task (GN), Task Switching task (TS) and Sternberg Working Memory task (SB). The widely used Trier Social Stress Test (TSST) was used as social stressor. Inflammatory, as well as autonomic nervous system (ANS), endocrine and subjective responses to the social stressor were also recorded prior to and after the night.

Results

First analyses show clear negative effects of SD on the cognitive performance of all tasks in the task battery, except for task switching. The next step is to analyze and present the physiological and metabolic-inflammatory parameters that are associated with cognitive decline after SD and acute social stress.

Discussion

To our knowledge, this is the first study that took a comprehensive approach to characterize the metabolic-inflammatory, psychological and autonomic state at baseline and under acute social stress conditions. This research

can contribute to predictions of stressor effects and development of mechanism-based strategies to intervene and cope with negative effects of stressors in conditions where stressors are unavoidable.

References

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