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Modelling the effects of sleep deprivation – from physiological to biochemical analyses

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Introduction

Sleep deprivation is unavoidable in certain professions but negatively affects well-being, health and performance. We are interested in the relation between sleep deprivation and cognitive performance. While sleep deprivation decreases cognitive performance in general, it does so at very different degrees between individuals. As of yet, we cannot predict decreased performance due to sleep deprivation, and we do not understand what causes this decrease. If we better understand the reasons, we may predict (momentary) resilience of individuals to sleep deprivation and develop (personalized) counter measures against the negative effects. Our working hypothesis is that sleep deprivation-induced inflammatory processes underlie cognitive decline due to sleep deprivation. These processes are reflected by biochemical analytes such as cytokines, lipids and cortisol in blood and saliva. Skin conductance and heart rate may be of interest as well since they reflect the level of arousal which may also have explanatory value. The advantage of the physiological measures is that they are non-invasive and continuous.

In the current study, we compare 1 night sleep deprived and control individuals on a number of physiological, biochemical and performance markers. To evaluate the biochemical and physiological effect of sleep deprivation, we use machine learning models to select features that best describe the difference between sleep deprived and control participants.

Methods

This study was approved by the METC Brabant (approval no. NL74961.028.20). In total, 102 participants were recruited through the institute's participant pool and through direct recruitment advertisements. Ages ranged from 19 to 55 years old (M = 28.5, SD = 10.3). They were randomly assigned to spend the night awake at the research institute (n=58) or at home sleeping (n=44). All participants underwent the same measurement procedures including cognitive tests (SYNWIN multi-tasking, Psychomotor Vigilance task, Go-no go inhibition task, Sternberg working memory task, TAP-M flexibility of task switching), exposure to social stressors (Sing-a-Song Stress Test and Trier Social Stress Test) and had standardized breakfasts the morning before and after the night. Heart rate and skin conductance were recorded throughout using a Tickr chest-strap (Wahoo Fitness, Atlanta, GA, USA) and EdaMove 4 (Movisens GmbH, Karlsruhe, Germany), respectively. Sampling of saliva and blood occurred both before and after the social stressors.

Heart rate and phasic skin conductance values were determined for several intervals before, during, and after the social stressors. The concentration of cortisol and cytokines were determined in saliva and blood. Via metabolomics analyses, using the Lipidyzer lipidomics platform, a large panel of lipid species were analyzed in blood (only samples before the social stressors).

For the modelling, we used logistic regression with an elastic net penalty. We trained the model with data of 90% of the participants using 10-fold cross validation and tested the model with the remaining participants that were completely kept separately from the training procedure. This procedure was followed once with the physiological data and once with the biochemical data from blood and saliva. In total, 40 input features were used for the physiological model and 753 input features were used for the biochemical model (729 of which concern lipid species from blood, 2 cortisol metabolites from saliva, and 22 cytokines from blood and saliva). All features concern values from the second morning, baselined for each individual participant by the values on the first morning.

Results

For all cognitive tests, we found (strongly) significant effects of sleep deprivation, showing a decrease in performance for the sleep deprived group of participants compared to the controls.

The biochemical model distinguished sleep deprived versus control participants with an accuracy of 100% for the training data; for the test data the accuracy was 90%. In total, 114 out of the 753 features contributed significantly to the model. One of these was a cortisol feature, 7 cytokine features and the remainder were lipids from various classes. By contrast, the physiological model could not distinguish sleep deprived versus control participants (accuracy of 70% for the training data, but for the test data the accuracy was 44%).

Discussion

Our study resulted in a rich database that can help elucidate associations between cognitive performance, sleep deprivation and a range of biochemical, physiological and psychological factors. Our first analyses as described here suggest that sleep deprivation does not so much affect physiological parameters (indicative of physiological arousal), but rather biochemical markers, many of which are associated to inflammatory processes. In fact, biomarkers from blood and saliva allow for a very accurate estimate of whether or not an individual is sleep deprived. Further interpretation and analyses linking effects on biochemical markers to decreases in performance are required.