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DOES SLEEP PREDICT ANTIBODY RESPONSE AND MAINTENANCE TO THE COVID-19 VACCINE?

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Introduction: There is growing evidence that insufficient sleep can negatively impact the immune system, including vaccination response. Prior laboratory studies have shown that acute sleep restriction can result in impaired antibody response to the hepatitis A and influenza vaccine. Similarly, prospective studies have shown that short sleep duration, measured by self-report and wrist actigraphy, is associated with muted antibody responses. These prior findings have critical implications for the COVID-19 pandemic and the efficacy and durability of the COVID-19 vaccines currently available. Whether sleep accounts for variability in response to the COVID-19 vaccination series has not been investigated.

Methods: We recruited 530 healthy participants (mean age=52.4, SD=12.1, range: 18-88 years; 64.1% female) who were naive to the COVID-19 vaccination series. Participants completed self-report questionnaires (e.g., Pittsburgh Sleep Quality Index) and morning sleep diaries for 7-consecutive days surrounding COVID-19 vaccine administrations. Additionally, 198 participants wore a sleep tracking device (Oura ring) continuously for ~2 months beginning prior to vaccination, which provides behavioral sleep data on days prior to and following the COVID-19 vaccination series. Blood samples were collected prior to vaccination, +1 month after their final vaccine shot (peak response), and +6 months after their final vaccine shot (maintenance); neutralization assays using pseudotype virus will be carried out to quantify antibody titers.

Results: Data collection concludes December 2021, with antibody assays to be completed February 2022. Initial baseline data indicates that most participants reported poor overall global sleep quality (PSQI mean=6.3, SD=3.6; 52% PSQI>5). Linear mixed models will be conducted to test associations between habitual sleep duration (averaged over the measurement time points), sleep efficiency, and subjective sleep quality with antibody responses over time. Additionally, we will report on the relevance of sleep timing (midpoint) and vaccination timing (receiving the vaccine in the morning vs afternoon vs evening), and the role of self-reported sleep disorders (e.g., obstructive sleep apnea) and shift worker status. Covariates in these analyses will include age, gender, race, body mass index, prior COVID infection, and vaccine type (Moderna, Pfizer, Johnson and Johnson).

Conclusion: These analyses will provide new knowledge about the role of sleep in mounting and maintaining antibody response to the COVID-19 vaccination series. These findings may provide novel insights into when and for whom improvements in sleep may result in better vaccine efficacy.

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SLEEP DURATION AND TIMING IS PROSPECTIVELY LINKED WITH INCREASES IN INSULIN RESISTANCE OVER LATE ADOLESCENCE

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Introduction: During puberty, adolescents experience a period of transient insulin resistance (IR) that normalizes upon full maturation. Yet, IR continues to rise for some adolescents, increasing metabolic disease and type 2 diabetes risk in adulthood. Whether short sleep duration and/or later sleep timing are risk factors for persistently increasing IR in late adolescence has not been explored.

Methods: The study population includes 362 adolescents from Mexico City enrolled in a longitudinal birth cohort (ELEMENT study). Beginning in 2015, when participants were between the ages of 9 and 17, there were 2 clinic visits that occurred approximately 2 years apart. During the visit, a fasting blood sample and anthropometric measurements were taken. Insulin resistance was assessed with glucose and insulin via HOMA-IR. Four groups were defined using puberty-specific cutpoints for IR: normal HOMA-IR over the follow-up period (reference), transition from normal to IR, transition from IR to normal, and IR at both time points. Baseline sleep assessments (sleep duration, timing, and variability of both duration and timing) were measured with 7-day actigraphy. Multinomial logistic regression models were used to evaluate associations between sleep duration and timing with HOMA-IR categories, adjusting for age, sex, and baseline pubertal status.

Results: Seventeen percent of the sample developed insulin resistance over the follow-up period. Adolescents ≥ 1 hour below the sleep duration recommendations-for-age were over twice as likely to be in the group that developed IR compared to the normal group (95% CI 1.1, .9; P for trend=0.03). Similarly, adolescents who had a sleep midpoint later than 4:36 AM were 2.77 times as likely to be in the increasing HOMA-IR category (95% CI 1.0, 7.5; P for trend=0.05). Interestingly, there was no evidence that changes in adiposity over follow-up mediated associations between sleep and insulin resistance.

Conclusion: Insufficient sleep duration and late sleep timing were independently associated with development of IR over a 2-year period in peri-puberty. Adequate sleep during the pubertal period may promote metabolic health into young adulthood, independent of any changes in adiposity.

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PREDICTING SLEEP INERTIA IN A BIOMATHEMATICAL MODEL OF FATIGUE AND PERFORMANCE: A NOVEL APPROACH

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Introduction: Biomathematical models of fatigue typically include sleep inertia as an additive process during wakefulness. However, there is predictive information to be gained from tracking the