than YA. However, the meditation models did not reach statistical significance when evaluated for OA (R2 = .115, F(2, 35) = 2.275, p = .118) and YA (R2 = .010, F(2, 54) = .277, p = .759) separately.
Conclusions: Overall, these results indicated that visceral fat mediates the relationship between FAA and depressive symptomatology. In other words, the mediation model suggests that the negative effects that an increase in frontal alpha asymmetry (greater left cortical activity) has on depressive symptomatology are driven by the increment of visceral fat levels. Therefore, this evidence highlights the importance of a healthy lifestyle on neural and emotional well-being through lifespan. Additionally, further work exploring this mediation may help improving the potential use of FAA as a neural marker of depression.
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INSULIN RESISTANCE AND WORKING MEMORY EXPLORING THE ROLE OF BLOOD GLUCOSE LEVELS AND LIFESTYLE

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Abstract text
Introduction: Type 2 diabetes mellitus and dementia are among the leading causes for reduced quality of life and life expectancy worldwide and often occur comorbidly. Both diseases are linked by altered insulin signaling. Lifestyle factors and blood glucose monitoring play an essential role in the prevention and treatment of type 2 diabetes. So far, a relationship between blood glucose levels, lifestyle, and cognitive performance – a main symptom of dementia - has mainly been established in laboratory settings which reduces its ecological validity.
Objectives: This study uses ambulatory assessment and continuous glucose monitoring to explore the link between blood glucose levels, lifestyle and working memory in an ecological setting. We hypothesize that glycemic variations affect working memory performance in daily life. Second, we hypothesize that a high variance in blood glucose levels has a higher impact on working memory in insulin resistant participants. With this study, we aim to expand the knowledge on the relationship of insulin resistance and cognitive performance from the laboratory setting to everyday life.
Methods: This prospective, exploratory study will include 80 subjects with insulin resistance and 80 healthy controls. At baseline, blood indicators of insulin resistance will be measured to determine group assignment. Our ambulatory assessment includes smartphone-based sampling and sensor-based assessment. Therefore, cognitive performance will be recorded over three consecutive days using a smartphone. Four times a day, a numerical working memory task is prompted by signal-based alarms on the smartphone. Blood glucose levels are recorded in parallel by continuous glucose monitoring. In addition, lifestyle factors such as diet ad physical activity are examined. Diet is assessed by 24-h dietary protocols and movement acceleration by accelerometery.
Multilevel modelling will be used to map the relationship between blood glucose levels and working memory at the within- and between-person level. Diet and exercise are included in the analyses as additional predictors.
Results: Data collection started in March 2021 and is ongoing. Up to now, 40 insulin resistant participants and 36 healthy controls have been measured. Our preliminary results indicate a positive association between blood glucose levels and working memory performance at the within-person level (estimate = .48, 95% CI [.07, .89], p =0.022). At the between-person level the analysis revealed an inverse association between blood glucose levels and working memory performance (estimate = -.45, 95 % CI [-.86 - -.05], p = 0.029).
Conclusion: Our preliminary results are in line with studies showing that an acute rise in blood glucose levels leads to short-term improvements, while stable glucose profiles are beneficial in the long term. This might expand the understanding of the impact of insulin resistance on working memory and represent a target for early interventions. Our preliminary analysis needs to be repeated in our final dataset to confirm our results.
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A POSSIBLE PROTECTIVE EFFECT OF SLEEP CONSISTENCY AGAINST GENETIC LIABILITIES FOR MENTAL HEALTH PROBLEMS IN YOUTH

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Abstract text
Background: Adolescence is a critical period in human development, during which numerous biological and behavioral risk factors contribute to the manifestation of psychopathology. Digital phenotyping (e.g., via wearable devices) has proven to be useful in accurately indexing everyday behavioral patterns relevant to mental health. Here, we aim to characterize how genetic liabilities for psychiatric disorders and lifestyle factors individually relate to psychopathology in youth, and crucially, the interplay among these different modalities of risk factors.
Methods: We used data from a total of 11,874 participants of age 10-13 in the Adolescent Brain Cognition Development study (ABCD), of which 6,605 had reliable Fitbit-derived physical activity and sleep measures (108,973 person-days), 11,225 had guardian-reported nutrition assessment, and 4,645 unrelated European individuals were used in polygenic risk scoring analysis. We quantified the genetic risks for seven major psychiatric disorders by a set of polygenic risk scores (PRS), based on the largest and most recent genome-wide association studies of major depressive disorder (MDD, N = 480k), anxiety disorders (N = 22k), attention-deficit hyperactivity disorder (ADHD, N = 226k), autism spectrum disorders (N = 46k), schizophrenia (N = 321k), bipolar disorder (N = 51k), and cannabis use disorder (N = 358k). Investigated lifestyle variables include records of daily physical activity at different intensities, step count, sleep duration and variability at different stages, as well as nutrient intake of the youth and their biological mother around pregnancy. Psychopathology was measured by the total and subscales scores from the Child Behavior Checklist (CBCL).
Results: Increased PRS for ADHD (β=0.10, SE=0.02, t=5.89, p=4e-9) and MDD (β=0.07, SE=0.02, t=4.07, p=5e-5) were significantly associated with higher overall emotional and behavioral problems in youth, with ADHD-PRS having the largest effect on the attention problem subscale and MDD-PRS on the withdrawn/depressed subscale. Among the lifestyle factors, total CBCL score was associated the most with higher variability in daily sleep duration (β=0.16, SE=0.02, t=9.44, p=6e-21), less frequent intake of fruits, vegetables and whole grains (β=0.11, SE=0.01, t=8.81, p=2e-18), more frequent intake of saturated fat and sugar (β=0.10, SE=0.01, t=8.07, p=8e-16), and less daily steps count (β=0.09, SE=0.01, t=5.88, p=4e-9). Crucially, we found differential genetic effects for behaviorally diverse individuals. In particular, in youths with higher sleep duration variabilities, the impact of ADHD-PRS on attention problems (t = 4.09, p<.01) and ADHD symptoms (t = 3.47, p<.01), as well as the impact of MDD-PRS on somatic problems (t = 2.93, p<.01) were enhanced, whereas these effects were attenuated or even suppressed in youths with lower variabilities.
Conclusion: In a large-scale population-based cohort, we combined caregivers' reports with digital phenotyping of everyday behaviors and genotyped data, and