Adverse effects were mentioned at the higher dose (10mg) with negative reaction times, sedation and confusion.
Conclusion. There is a potential favourable effect of prescribing melatonin for mild to moderate AD, but there is limited evidence for prescribing it for moderate to severe AD. Furthermore, there is emerging evidence on melatonin's neuroprotective effect and potential treatment options for mild to moderate AD; further research is required for both sleep and neuroprotection in AD.

Choices Today, Behaviours Tomorrow: Longitudinal Associations Between Childhood Risky Decision-making and Adolescent Conduct Disorder Behaviours – a Nationally Representative Prospective Cohort Study in the United Kingdom

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doi: 10.1192/bjo.2024.76

Aims. Conduct disorder carries significant individual and societal repercussions. Despite heightened risk-taking and challenges in adapting to changing probabilities of choice outcomes being linked to maladaptive behaviours such as conduct disorder, no study to date has examined the association behind childhood decision-making and adolescent conduct disorder. This study seeks to address this gap by exploring the longitudinal association between these two variables. Understanding the mechanisms underlying conduct disorder could help with developing new preventive interventions.
Methods. We used data from the Millennium Cohort Study, a nationally representative UK cohort; participants included those with complete data on exposure, outcome and confounding variables (n = 7,237). The exposure, childhood decision-making at 11 years was measured using the Cambridge Gambling Task risk-taking and risk-adjustment measures. The outcome, a binary measure of adolescent conduct disorder was created using items from the risky and antisocial behaviour interview sections at age 17. We used logistic regression to examine the association between childhood decision-making and adolescent conduct disorder and adjusted for relevant confounders.
Results. The univariable model showed that at age 11, each 20-point increase in risk-taking score increased the odds of conduct disorder behaviour at age 17 by 32% (OR = 1.32, 95% CI 1.18–1.44, p < 0.0001). In the multivariable model, there was strong evidence that a 20-point increase in risk-taking at 11 years was associated with 18% higher odds of conduct disorder behaviour at 17 years (OR = 1.18, 95% CI 1.05–1.33, p = 0.005). There was no evidence that this association differed by sex. Risk adjustment at 11 years showed no association with conduct disorder behaviours at age 17 both in the univariable model (OR = 0.96, 95% CI 0.88–1.06, p = 0.440) and the multivariable model (OR = 0.96, 95% CI 0.88–1.06, p = 0.433).
Conclusion. We found that risk-taking at 11 years was associated with conduct disorder behaviour at 17 years. If causal, our findings suggest that risk-taking might be a potential mechanism underlying adolescent conduct disorder behaviours. This may be useful in informing the design of preventive strategies, such as encouraging positive risk-taking in children and discouraging negative risk-taking behaviours.

BMAL1 Genetic Variation in Metabolic and Mental Health

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doi: 10.1192/bjo.2024.73

Aims. Epidemiological studies have previously shown a link between cardiometabolic disease and severe mental illness. The extent and mechanisms behind this link are poorly understood currently but links to impairments in the stress response and cortisol regulation have been thought to play a significant role. *BMAL1* is a circadian rhythm regulation gene found on chromosome 11 which has been associated with a variety of pro-inflammatory states as well as conditions such as depression, schizophrenia, type 2 diabetes mellitus and myocardial infarction. Our study aimed to investigate the genetic structure of the *BMAL1* gene locus and its associations with both cardiometabolic and psychiatric traits and conditions.
Methods. We used genetic data from the UK Biobank which recruited ~500,000 participants. Of these we used a population of ~430,000 self-reported white British participants and data from a variety of questionnaires and investigations looking at severe mental illness and cardiometabolic traits. We performed association analyses using Plink 1.07 with Bonferroni correction being performed for multiple testing using a number of genetic variants. Our threshold for significance was defined as a p-value $< 5.35 \times 10^{-5}$. Conditional analysis was then performed to identify if there were multiple independent signals for each phenotype.
Results. *BMAL1* variants were associated with BMI, diastolic, systolic blood pressure, waist-hip ratio and neuroticism score, and risk of anhedonia, major depressive disorder and risk-taking behaviour. Multiple significant independent signals were identified for BMI and waist-hip ratio. Linkage disequilibrium (LD) analysis showed significant coinheritance of specific traits which could suggest a role for *BMAL1* and the encoded protein as a link between cardiometabolic and mental health traits.
Conclusion. This is the first study that systematically investigated associations between the *BMAL1* locus across a variety of different mental and cardiometabolic phenotypes in a population-level cohort. Our study has shown that there is a link between the *BMAL1* locus and both cardiometabolic and mental health phenotypes. Further research is required to investigate the exact biological mechanism by which *BMAL1* connects severe mental illness and cardiometabolic disease.