Conclusion. Lack of consistency in the measurement of cost components and defined cohort characteristics makes comparison across the literature challenging, comparison cannot inform any meaningful economic evaluation. Despite this, the overarching theme across all studies in this review is that current service expenditure is higher for autistic people than non-autistic people. This is particularly clear when discussing accommodation, healthcare and costs due to loss of productivity. Both age and co-occurring conditions have an impact on overall cost. These findings form a strong basis for future research in this area to standardise cost calculations across specified age ranges and evaluate current government-centered financial support available to autistic people.

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The Chicken or the Egg? Understanding the Temporal Relationship Between Severe Mental Illness and Neurological Conditions in a UK Primary Care Cohort

Dr Ella Burchill*, Dr Jonathan Rogers, Professor David Osborn, Professor Joseph Hayes and Dr Naomi Launders

Division of Psychiatry, University College London, London, United Kingdom
*Presenting author.

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Aims. A significantly higher prevalence of neurological conditions has been found both before and after a diagnosis of schizophrenia, bipolar disorder and other psychotic illnesses compared with the general population.

We aimed to understand the cumulative prevalence of 16 neurological conditions in people with severe mental illness (SMI) from 5 years before to 5 years after their SMI diagnosis. We hypothesised that individual neurological conditions would have differential temporal relationships relative to SMI diagnosis.

Methods. In a longitudinal matched study, we identified a cohort of patients aged 18–100 years from Jan 1, 2000, and Dec 31, 2018, from the UK Clinical Practice Research Datalink (CPRD). Neurological conditions were classified using ICD–11 criteria into umbrella clusters of disease. Outcome of interest was a diagnosis of SMI. Each SMI patient was matched 1:4 to patients without SMI in the CPRD cohort, matching for sex, 5-year age band, primary care practice and year of practice registration. The cumulative prevalence of 16 neurological conditions was recorded cross-sectionally at 5, 3, 1 years prior to SMI diagnosis, at SMI diagnosis (index), and 1, 3 and 5 years after SMI diagnosis. Logistic regression modelling aided comparison of differential prevalence of neurological conditions, adjusting for sociodemographic variables, and with further adjustment for body mass index, smoking, alcohol and non-prescription drug use. Multiple imputation was applied in cases of missing data.

Results. We identified 68,789 patients with SMI, matched to 274,827 controls. The median age was 40.9 years, 49.05% of the overall cohort were female (33,783 SMI patients, 134,740 controls), and the majority were of White ethnicity (35,228, 51.2% SMI patients, 125,518, 45.7% controls). The most prevalent neurological conditions across seven timepoints were cerebral palsy, cerebrovascular disease, dementia, epilepsy, multiple sclerosis, paralysis and Parkinson's disease. Conditions with the highest fully adjusted odds ratios (ORs) for SMI diagnosis were dementia 3 years after SMI diagnosis (5.32, 95% CI 4.95–5.71) and Parkinson's disease 5 years after SMI diagnosis (4.26, 95% CI 3.68–4.94).

Conclusion. All 16 neurological conditions have higher prevalence in the SMI cohort compared with controls, with different prevalence patterns observed over the 10-year study period. A consistently lower OR for schizophrenia compared with other SMI warrants further exploration, as neurological conditions risk being under-recorded.

A greater understanding of the temporal relationship between SMI and neurological conditions may help promote earlier diagnosis, increased screening and better holistic management of both conditions.

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Review of Melatonin's Effectiveness and the Side Effects on Alzheimer's Disease

Dr Sui Yung Chen[1]*, Professor Farooq Khan[2], Dr Shoaib Talib[1] and Ms Suzanne Toft[3]

[1]Black Country Healthcare NHS Foundation Trust, Sandwell, United Kingdom; [2]Birmingham and Solihull Mental Health NHS Foundation Trust, Birmingham, United Kingdom and [3]Black Country Healthcare NHS Foundation Trust, Walsall, United Kingdom
*Presenting author.

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Aims. People who have Alzheimer's disease (AD) often experience sleep disturbances due to the nature of the illness. Melatonin has been prescribed for sleep disturbance in individuals with AD, although there is a lack of national guidelines for pharmacological care for this presentation. Prolonged sleep disturbances for individuals with AD tend to lead to poor quality of life for the individual, behavioural challenges, carers' exhaustion and potential placement breakdowns.

The objective of this literature review is to determine whether the available evidence supports recommending melatonin to patients with AD for sleep, along with other benefits and adverse effects.

The hypothesis for this review is that melatonin is beneficial for sleep disturbances and has neuroprotection for individuals with AD.

Methods. Literature search on the online electronic database from 2010 to November 2023, using the title of "Melatonin's effectiveness and the side effects on Alzheimer's Disease". This literature review was done by screening the 125 searched titles. The inclusion criteria included systematic review (SR), meta-analysis, randomised controlled trial (RCT), animals and cell studies. Exclusion criteria included case studies, literature and peer reviews. A total of 12 papers are included in this review.

Results. The three SRs, two meta-analyses and one RCT showed the potential effect of melatonin on ameriolating cognitive decline, improving cognition, quality of life and sleep qualities, with the conclusion that further studies are required. One combined meta-analysis and SR showed melatonin might be an effective treatment for mild AD. One Cochrane review showed melatonin has no evidence of improving sleep for moderate-to-severe AD.

One animal study and two cellular studies showed a melatonin effect in the control progression of AD. One animal study and one cellular research study concluded that melatonin has potential treatment effects.

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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.

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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

Miss Christy Cheung*, Dr Gemma Lewis, Professor Glyn Lewis and Dr Ramya Srinivasan

UCL Division of Psychiatry, London, United Kingdom
*Presenting author.

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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.

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BMAL1 Genetic Variation in Metabolic and Mental Health

Mr Hamza H Daudali[1]*, Dr Breda Cullen[1], Dr Nicholas Graham[1], Dr Joey Ward[1] and Dr Rona Strawbridge[1,2,3]

[1]Behavioural Epidemiology and Genetics, School of Health and Wellbeing, University of Glasgow, Glasgow, United Kingdom; [2]Cardiovascular Medicine Unit, Department of Medicine, Karolinska Institute, Stockholm, Sweden and [3]Health Data Research UK, Glasgow, United Kingdom
*Presenting author.

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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.

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