CBT significantly decreased GP appointments at 6 months follow-up compared with PPT with a large effect size ($\eta 2 = 0.5$, $p < 0.05$). A similar trend was seen for total cost ($\eta 2 = 0.5$, $p < 0.06$) with each PPT patient costing £790 more on average than their CBT counterparts during the 6 months after therapy.

Conclusion. Whilst CBT appears to be efficacious in the short-term, PPT caused significantly increased healthcare utilisation compared with CBT in the 6 months after therapy. This aligns with similar studies that demonstrate a 'sleeper effect' in which patients who receive PPT, but not CBT, deteriorate before improving over long-term follow-up.

Additional research is needed to correlate this data with symptoms and capture the long-term benefits of these psychotherapies for MUS.

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The Effects of Developmental Stress on Dopaminergic Function in Adulthood: A Systematic Review and Meta-Analysis

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doi: 10.1192/bjo.2024.219

Aims. Exposure to traumatic experiences during childhood and adolescence is a significant risk factor for the development of psychiatric disorders in adulthood. An estimated 50% of the worldwide incidence of depression and anxiety can be attributed to childhood maltreatment (Li et al., 2016). In addition, approximately one-third of psychotic experiences are attributable to a history of developmental trauma (McGrath et al., 2017). It is thought that long-lasting, trauma-induced adaptive changes in neurobiological function may lead to a predisposition towards pathophysiology (McCrory and Viding, 2015). However, the precise mechanisms through which developmental trauma exposure alters brain function on cellular and circuit levels remain poorly elucidated.

Methods. A systematic literature search and meta-analysis was performed to establish how dopaminergic functioning in adulthood is affected by developmental stress in rodents. Three databases, Medline®, Embase®, and PsycINFO®, were systematically searched initially on 2nd December 2023. Terms for three superordinate concepts ('childhood' terms, 'trauma' terms, and 'dopamine' terms) were combined. Cohen's d statistic was used for effect sizes. This protocol is pre-registered on PROSPERO® (ID: CRD42018106382).

Results. A total of 104 studies met our inclusion criteria. Meta-analysis indicated that developmental stress exposure leads to complex and long-lasting effects in basal and post-amphetamine extracellular dopamine concentrations in the medial prefrontal cortex, amygdala, and nucleus accumbens. In addition, there is a significant downregulation of D1 receptors and upregulation of D2 receptors in prefrontal and striatal regions involved in threat and reward processing. Effect sizes ranged from 0.36 to 1.55.

Conclusion. These findings strongly suggest that dopaminergic dysfunction is a mechanistic link between developmental trauma and vulnerability towards mental illness in adulthood.

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White Matter Microstructure Abnormalities in Individuals at High Risk for Psychosis: A Meta-Analysis of Fractional Anisotropic Changes Associated With Transition to Psychosis

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doi: 10.1192/bjo.2024.220

Aims. Recent studies have focussed on detecting white matter abnormalities in subjects who transition to psychosis (UHR-T). Research suggests that fractional anisotropy (FA), may be decreased in UHR-T. However, global and regional findings have been inconsistent. By objectively combining data in a meta-analysis, we have investigated white matter alterations associated with transition, by comparing FA in UHR-T with subjects that do not transition (UHR-NT) and healthy volunteers.

Methods. The meta-analysis was registered on PROSPERO (ID: CRD42021265348) and followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses PRISMA guidance. A systematic database search of PUBMED and EMBASE identified reports, which were screened by 2 independent researchers (CN and DD) for inclusion, from inception to 20 July 2021. Discrepancies were decided on consensus with a third researcher (KM). Reference lists of eligible studies were also screened. Authors of screened reports were contacted to provide parametric maps. Coordinate-based meta-analysis was conducted using Seed-based *d*-Mapping software to combine parametric map and coordinate data from reports, using a random-effects model. Quality and risk of bias analysis were conducted using the Newcastle-Ottowa Scale. Heterogeneity and sensitivity analyses were also conducted.

Results. The search strategy identified 889 potential studies, from which 6 met eligibility criteria. A total of 71 UHR-T, 142 UHR-NT and 148 healthy volunteers were included. Weighted-mean decreases in FA were observed in UHR-T compared with: UHR-NT ($d = -0.99$; $p < 0.0001$; 95% CI −1.43 to −0.55); and healthy volunteers ($d = -0.91$; $p = 0.04$; 95% CI −1.78 to −0.05). The level of heterogeneity for the former was not significant. For UHR-T, regional FA decreases were observed in areas including the left genu of the corpus callosum (Z-score = −1.76, 204 voxels, $p < 0.0001$) compared with UHR-NT, while FA increases were most observed in the white matter region adjacent to the left postcentral gyrus (Z-score = 1.64, voxels = 16, $p < 0.0001$). These findings persisted despite sensitivity analyses.

Conclusion. The findings suggest that white matter alterations, specifically in left frontotemporal tracts, are associated with an increased risk of transition to psychosis. The neurobiological implications of these findings, and their contribution to UHR-T prediction efforts, are explored, as are avenues for further research.

Additional Author: Mr Gunnar Gronlid.[1]

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https://doi.org/10.1192/bjo.2024.221 Published online by Cambridge University Press

Adolescent Psychopathology and Cognitive/Academic Functioning: Impact of Comorbidity Using a Genetically Sensitive Design

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doi: 10.1192/bjo.2024.221

Aims. To determine: i. the nature of the associations between three domains of psychopathology (depressive, hyperactivity and conduct symptoms) and cognitive/academic performance among adolescents i.e., whether these reflect causal processes and/or common genetic effects; ii. The extent to which these associations vary by comorbidity.
Methods. The sample comprised participants in the UK Twins Early Development Study (TEDS; n≈12,000 individuals) assessed for depressive, hyperactivity and conduct symptoms using standardised questionnaires. Cognitive and academic performance were assessed using Standard Progressive Matrices and GCSE scores respectively. Comorbidity was derived as a count of borderline/high psychopathology scores present per individual. Twin modelling was used to investigate preliminary correlations and moderation effects. Genetic models were further used to determine the most likely direction of causal effects with/without genetic correlations.
Results. There were small to moderate negative correlations between adolescent psychopathology domains and cognitive performance ($-0.01 \leq r \leq -0.15$) and academic performance ($-0.06 \leq r \leq -0.23$). Correlations were smallest for depressive symptoms and larger for hyperactivity/conduct symptoms. The correlation between hyperactivity symptoms and cognitive performance was significantly more negative as comorbidities increased (moderation coefficient – $\beta_{mod} = 0.07$, 95% CI: 0.02, 0.12). Similarly, the association between depressive symptoms and academic performance also became more negative as comorbidities increased ($\beta_{mod} = -0.08$, 95% CI: −0.11, −0.05). Twin modelling indicated that hyperactivity symptoms were causally associated with poorer cognitive and academic performance. In contrast, poorer cognitive performance was causally associated with conduct symptoms.
Conclusion. These preliminary findings indicate the impact of comorbidity on the functioning of adolescents with hyperactivity and depressive symptoms. They further suggest the need to specifically recognise these comorbidities during assessment and treatment planning to promote optimal functioning. Our findings also suggest differential mechanisms for the links between different psychopathology domains and impaired functioning. Further analyses will investigate moderation of the causal links and/or genetic correlations and whether these associations vary by indicators of marginalisation (sex and ethnicity).

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Modelling Co-Occurring Mental Health Conditions Among Autistic Individuals Using Polygenic Scores

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doi: 10.1192/bjo.2024.222

Aims. This study investigated the relationship between common genetic variation and co-occurring mental health conditions among autistic individuals.
Methods. The study was conducted with the Simons Foundation Powering Autism Research (SPARK) dataset, V9 release, and included probands [n = 17,582] with confirmed diagnosis of autism, who were also in the SPARK iWES1 array genotyping dataset. Six co-occurring mental health conditions (attention deficit hyperactivity disorder or ADHD, bipolar disorder, depression, schizophrenia, anxiety disorder and disruptive behaviour disorders) were analysed. Polygenic scores (PRS) were generated with PRScs software, using summary statistics from the most recent genome wide association studies (GWAS) for autism, ADHD, schizophrenia, bipolar disorder, depression, anxiety, neuroticism, p-factor, intelligence, educational attainment and hair colour (negative control). General linear models (GLM) and Cox proportional hazards models were computed, with age at registration, sex, cognitive impairment and genetic principal components included in both sets of models. Multiple testing correction was done using the Benjamini-Yekutieli method. Results were calculated using odds ratios (OR), 95% Confidence Intervals (CI) and corrected p values (p).
Results. There were similar patterns of association and interaction for both GLMs and Cox models. Polygenic scores for educational attainment were significantly lower for those with co-occurring ADHD (GLM: OR=8.85E-01, 95% CI=8.48e-01–9.23e-01, p = 2.91E-07; Cox: OR=8.94E-01, 95% CI=8.66e-01–9.22e-01, p = 4.76E-11), bipolar disorder (GLM: OR=7.45E-01, 95% CI=6.54e-01–8.49e-01, p = 2.40E-04; Cox: OR=7.25E-01, 95% CI=6.39e-01–8.23e-01, p = 3.96E-05), depression (GLM: OR=8.63E-01, 95% CI=8.04e-01–9.26e-01, p = 5.13E-04; Cox: OR=8.56E-01, 95% CI=8.03e-01–9.12e-01, p = 2.80E-05), schizophrenia (GLM: OR=6.94E-01, 95% CI=5.71e-01–8.42e-01, p = 3.99E-03; Cox: OR=6.67E-01, 95% CI=5.52e-01–8.05e-01, p = 1.41E-03), anxiety disorder (GLM: OR=8.77E-01, 95% CI=8.37e-01–9.20e-01, p = 9.88E-07; Cox: OR=8.81E-01, 95% CI=8.49e-01–9.15e-01, p = 1.46E-09) and disruptive behaviour disorders (GLM: OR=7.10E-01, 95% CI=6.63e-01–7.60e-01, p = 3.22E-21; Cox: OR=7.10E-01, 95% CI=6.67e-01–7.57e-01, p = 1.35E-24).
Conclusion. Polygenic scores for educational attainment were associated with the co-occurrence of several mental health conditions among autistic individuals.

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How Are Inpatient Psychiatric Ward Rounds Understood in Research Literature? A Scoping Review

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https://doi.org/10.1192/bjo.2024.221 Published online by Cambridge University Press