psychosis symptoms did not predict GRF (R-squared = .31, F[5, 92] = 8.09, p < .001). Age significantly correlated with GRF (r = -.31, p = .002). Poverty remained a significant predictor of GRF after including age (R-squared = .37, F[6, 89] = 8.84, p < .001). Additional cognitive correlates, collateral report of childhood ADHD symptoms, prior diagnoses, date of psychosis illness onset, and medication history will also be examined.
Discussion: Social functioning was predicted by childhood self-rated inattention and current negative and disorganized symptoms; however, the relation with childhood inattention did not remain after controlling for race/ethnicity. Additional analyses will be conducted to assess if race is presenting as a proxy for other social determinants, including insurance designation, in this sample. Individuals with ADHD experience more difficulty in social settings compared to typically developing peers, possibly due to increased need to use environmental cues; for individuals who go on to develop psychosis, these childhood events are possibly perceived as more stressful, adding to risk for psychosis. However, it is unclear if self-report childhood inattention – captured here as a putative symptom of ADHD – may be better accounted for by premorbid cognitive impairment associated with risk for psychosis. Additional research is required to establish this connection.

T77. ASSOCIATION BETWEEN INTRACELLULAR INFECTIOUS AGENTS AND SCHIZOPHRENIA

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Background: A number of studies have reported association between Toxoplasma gondii (T. gondii) and Chlamydia infection and the risk of schizophrenia. The aim of the present study was to compare the prevalence of T. gondii and Chlamydia infection between the schizophrenia and normal control subjects and to compare the clinical features between seropositive and seronegative schizophrenia patients.
Methods: The rate of serum reactivity to T. gondii, Chlamydia trachomatis (C. trachomatis), Chlamydia pneumonia in 96 schizophrenia and 50 control subjects was investigated using enzyme-linked immunosorbent assay and indirect fluorescent antibody technique. The clinical symptoms of the schizophrenia patients were scored with Positive and Negative Syndrome Scale and a comparative analysis was carried out.
Results: A significant positive association between immunoglobulin G (IgG) antibodies to T. gondii and C. trachomatis in schizophrenia was found, and the odds ratio of schizophrenia associated with IgG antibody was found to be 3.22 and 2.86, respectively. The Toxoplasma-seropositive schizophrenia patient had higher score on the negative subscale N1 and N7 and general psychopathology subscale G13, while C. trachomatis-seropositive schizophrenia patient had higher score on the general psychopathology subscale G10.
Discussion: The results from the present study suggest significant association between T. gondii, C. trachomatis infection and schizophrenia. In future, further studies are needed to elucidate the correlation between the two types of infection and schizophrenia.

T78. MORTALITY IN PATIENTS WITH SCHIZOPHRENIA ADMITTED FOR INCIDENT ISCHEMIC STROKE: A POPULATION-BASED COHORT STUDY

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Background: Evidence shows that schizophrenia is associated with increased incidence of cardiovascular diseases (CVD), including stroke. The relationship between schizophrenia and post-stroke mortality was understudied, and mixed findings were observed. Of note, none of these studies specifically explored the association of schizophrenia with short-term mortality after incident ischemic stroke. One of them specifically examined short-term mortality following ischemic stroke in schizophrenia patients, but it did not address potential confounding by patients who had past history of stroke. The only study which included solely incident stroke patients indicated that patients with psychotic disorders experienced higher short-term mortality ensuing incident stroke.
Methods: We conducted a retrospective cohort study to investigate short-term mortality of schizophrenia patients after incident ischemic stroke. All individuals admitted for incident ischemic stroke between 2006 and 2016 in Hong Kong were identified using a territory-wide electronic health record database. 817 patients with an ICD-10 diagnosis of schizophrenia (F20) or schizoaffective disorder (F25) (termed schizophrenia henceforth) prior to index admission constituted the study group. The comparison group comprised 8170 patients (10:1 matched to schizophrenia patients on age, sex, treatment sites and calendar-period for index admission) without any non-affective psychoses, mania or bipolar disorder (F20, F22-25, F28-31).
Results: Multivariate logistic regression revealed that schizophrenia patients had higher 1-year (OR [95% CI] = 1.51 [1.22 – 1.85]) and marginally higher 30-day (OR [95% CI] = 1.34 [1.00 – 1.79]) mortality following incident ischemic stroke, after adjusting for medical comorbidities, including hypertension, diabetes, hyperlipidemia, alcohol and substance use disorders and other comorbidities quantified by Charlson-Deyo comorbidity index. Additional age- (<65 years and ≥65 years) and gender-stratified analyses revealed similar results. Elevated 1-year mortality was exhibited by all schizophrenia subgroups, being more pronounced in younger patients (OR [95% CI] = 2.02 [1.38 – 2.96]). Increase in 30-day mortality was only seen in younger (OR [95% CI] = 1.75 [1.04 – 2.95]) and male (OR [95% CI] = 1.63 [1.06 – 2.50]) schizophrenia patients.
Discussion: Our results of heightened short-term post-stroke mortality in schizophrenia were in line with the only previous study which compared short-term mortality ensuing incident stroke in patients with and without psychotic disorders. This intuitive result may be explained by some studies which demonstrated that schizophrenic stroke patients were less likely to receive reperfusion treatments and prophylactic medications. The absence of data on lifestyle factors, antipsychotic treatment and post-stroke management is a major limitation in our study. In conclusion, our results indicated that schizophrenia is associated with increased short-term mortality after incident ischemic stroke. Further research is warranted to clarify the contribution of possible risk factors to post-stroke mortality in schizophrenia patients.

T79. BALANCING EFFECTS WITH SIDE-EFFECTS: EXAMINING COMPARATIVE METABOLIC CONSEQUENCES OF 18 ANTIPSYCHOTICS IN TREATMENT OF SCHIZOPHRENIA USING NETWORK META-ANALYSIS

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Background: Antipsychotic treatment is associated with metabolic disturbance. However, the relative degree to which metabolic alterations occur

in treatment with different antipsychotics remains unclear. Furthermore, predictors of metabolic dysregulation are poorly understood, and association between metabolic-change and change in psychopathology is uncertain.
Methods: We searched Medline, EMBASE and PsychINFO from inception until June 30, 2019. We included blinded randomised controlled trials (RCTs) comparing 18 antipsychotics and placebo in acute-treatment of schizophrenia. We performed frequentist random-effects network meta-analyses (NMAs) to investigate treatment-induced changes in body weight, BMI, total/LDL/HDL-cholesterol, triglycerides, and glucose. We performed meta-regressions to examine relationships between metabolic change and age/gender/ethnicity/baseline-weight/baseline-metabolic parameter level. We examined the association between metabolic change and psychopathology change by estimating the correlation between symptom severity change and metabolic parameter change.
Results: Of 6532 citations, 100 RCTs met inclusion criteria, including 25,952 patients. Median treatment-duration was 6-weeks. According to our NMAs, mean differences for weight-gain compared to placebo ranged from -0.23 (95% CI: -0.83, 0.36) for best (haloperidol) to +3.01kg (1.78, 4.24) for worst (clozapine); for BMI from -0.25 (-0.68, 0.17) for best (haloperidol) to +1.07kg/m2 (0.90, 1.25) for worst (olanzapine); for total-cholesterol from -0.09 (-0.24, 0.07) for best (cariprazine) to +0.56mmol/L (0.26, 0.86) for worst (clozapine); for LDL-cholesterol from -0.13 (-0.21, -0.05) for best (cariprazine) to +0.20mmol/L (0.14, 0.26) for worst (olanzapine); for HDL-cholesterol from +0.05 (0.00, 0.10) for best (brexpiprazole) to -0.10mmol/L (-0.33, 0.14) for worst (amisulpride); for triglycerides from -0.01 (-0.10, 0.08) for best (brexpiprazole) to +0.98mmol/L (0.48, 1.49) for worst (clozapine); for glucose from -0.29 (-0.55, -0.03) for best (lurasidone) to 1.05mmol/L (0.41, 1.70) for worst (clozapine). Greater increases in glucose were predicted by higher baseline-weight (p=0.001) and male-gender (p=0.008). Non-Caucasian ethnicity was associated with greater increases in total-cholesterol (p=0.04). Improvements in symptom severity were associated with increases in weight (rho=0.36, p=0.002), BMI (rho=0.84, p<0.0001), total-cholesterol (rho=0.31, p<0.05), and LDL-cholesterol (rho=0.42, p=0.01), and decreases in HDL-cholesterol (rho= -0.35, p=0.04).
Discussion: There are marked differences between antipsychotics in terms of metabolic side-effects, with olanzapine and clozapine exhibiting the worst profiles. By contrast, compared with placebo, lurasidone and cariprazine respectively reduce fasting glucose and LDL-cholesterol, while aripiprazole and brexpiprazole increase HDL-cholesterol. Baseline weight, male gender, and non-Caucasian ethnicity predict vulnerability to antipsychotic-induced metabolic change. Considering the increased prevalence of metabolic syndrome, cardiovascular disease, and cardiovascular mortality in schizophrenia, these data may be used to inform antipsychotic-prescribing, especially in those at-risk groups we have identified. However, clinical decisions to preferentially use an antipsychotic with fewer metabolic side effects should consider that clinical improvement appears to be associated with development of these side effects.

T80. CARDIOMETABOLIC RISK PREDICTION ALGORITHMS AND THEIR APPLICABILITY FOR YOUNG PEOPLE WITH PSYCHOSIS: A SYSTEMATIC REVIEW AND ILLUSTRATIVE EXAMPLE USING ORIGINAL DATA FROM A POPULATION-BASED BIRTH COHORT

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Background: Cardiometabolic risk prediction algorithms are used in clinical practice. Young people with psychosis are a high-risk group for developing cardiometabolic disorders, but it is unclear whether existing algorithms are suitable for this group.
Methods: We conducted a systematic review employing PRISMA criteria to identify studies reporting the development and/or validation of cardiometabolic risk prediction algorithms for general or psychiatric populations. A narrative synthesis was conducted to compare algorithms and consider their suitability for young people with psychosis. In addition, we used data from 3,470 young adults aged 18 years from the ALSPAC birth cohort to illustrate the impact of age on model performance of QDiabetes, an established algorithm.
Results: Having screened 6,609 studies, we included 57 risk algorithms designed for type 2 diabetes, cardiovascular disease or stroke, all of which were developed/validated in relatively older participants. Three algorithms featured psychiatric predictors and could be used for young people with psychosis. However, in all of three, age was weighted to a much greater extent than other risk factors. Furthermore, using ALSPAC data, we report that QDiabetes significantly under-predicted cardiometabolic risk in young people. Increasing the sample age to 50, leaving all other predictors unchanged, improved algorithm calibration markedly.
Discussion: Existing cardiometabolic risk prediction algorithms are heavily weighted on age and so under-predict risk in young people. A new or recalibrated algorithm is required for young people with psychosis that appropriately balances the weighting of relevant risk factors.

T81. MULTIPLE DRUG USE IN SCHIZOPHRENIA - THE ROLE OF EARLY ENVIRONMENTAL RISK ACCUMULATION AND GENETIC PREDISPOSITION

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Background: Drug (ab)use and substance use disorders are frequently observed in patients with psychiatric illness, but the underlying causes remain widely unknown. A number of environmental risk factors have been proposed to affect the use of one or multiple drugs in the general population and adolescents. Whereas most previous studies focused on the influence of single risk factors on the use of one or a few selected drugs, the effect of accumulated environmental risk in early life on multiple drug use remains to be studied. Similarly, evidence on genetic susceptibility to the (ab)use of a single drug, e.g. nicotine, alcohol, cocaine, is abundant, while the role of genetic predisposition for multiple drug use - in particular during early life - is yet to be explored. Thus, the current work aims to study the role of environmental as well as genetic risk factors for multiple drug abuse ('polytoxicomania') in a large sample of schizophrenic/schizoaffective patients.
Methods: Information from ~2000 schizophrenia/schizoaffective patients on (preadult) multiple drug use (> 2 drugs) and environmental risk factors was extracted from the Göttingen Research Association for Schizophrenia (GRAS) data collection – currently the largest data base of deeply phenotyped patients with schizophrenia/schizoaffective disorder or other neuropsychiatric diseases. In addition, genetic data from these patients and 2111 healthy blood donors were used in a novel genetic approach that employs multiple genome-wide association studies (GWAS) to identify genetic associations with preadult multiple drug use. Genotyping was performed on a semi-custom Axiom MyDesign Genotyping Array (Affymetrix, Santa Clara, CA, USA), based on a CEU (Caucasian residents of European ancestry from UT, USA) marker backbone.
Results: The accumulation of environmental risk factors, i.e. sexual abuse, physical abuse, migration, urbanicity, together with alcohol and cannabis consumption as secondary risk factors, in early life (< 18 years) were strongly associated with lifetime multiple drug use (p = 3.48 x 10^-44, extreme group comparison odds ratio (OR) = 31.8). When the sample was split into preadult and adult multiple drug users, there was a remarkable association of the number of preadult environmental risk