individuals with schizophrenia had decreased odds of having neurological autoimmune diseases (OR: 0.46; 95% CI: 0.23, 0.96). Among individuals with any psychotic disorder, females were 95% more likely to have any autoimmune disease (OR: 1.95; 95% CI: 1.72, 2.20). No racial differences were observed overall; however, compared to individuals who identified as white, individuals who identified as Black, Hispanic, and Asian had decreased odds of having gastroenterological autoimmune diseases (OR: 0.52; 95% CI: 0.35, 0.76), neurological autoimmune diseases (OR: 0.32; 95% CI: 0.10, 0.83), and systemic autoimmune diseases (OR: 0.25; 95% CI: 0.04, 0.80), respectively, while Black individuals had increased odds of having systemic autoimmune diseases (OR: 1.45; 95% CI: 1.17, 1.81).

Discussion: The prevalence of autoimmune diseases varied among people with different primary psychotic disorders, and certain associations were modified by sex and race. Clinicians may consider additional screening for autoimmune diseases among individuals with psychosis.

M88. EVIDENCE FOR INFLAMMATION AS A PUTATIVE SHARED MECHANISM FOR INSULIN RESISTANCE AND SCHIZOPHRENIA

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Background: Insulin Resistance (IR) predisposes to cardiometabolic disorders, which are common in schizophrenia and are associated with excess morbidity and mortality. The mechanisms of association remain unknown. We aimed 1) To use genetic data to examine the direction of association between IR and related cardiometabolic risk factors, and schizophrenia; 2) To examine whether inflammation could be a shared mechanism for IR and schizophrenia.

Methods: We used two-sample uni-variable Mendelian randomization (MR) to examine whether genetically-predicted IR-related cardiometabolic risk factors (Fasting insulin (FI), high-density lipoprotein (HDL), triglycerides (TG), low-density lipoprotein, fasting plasma glucose, glycated haemoglobin, leptin, body mass index, glucose tolerance and type 2 diabetes) may be causally associated with schizophrenia. We used the most recent summary statistics for genetic variants associated with schizophrenia and IR-related cardiometabolic risk factors from publicly-available large genome-wide association studies (GWAS). We used bi-directional MR to examine direction of association. To examine whether inflammation could be a shared mechanism for IR and schizophrenia, we first conducted a sensitivity analysis by performing MR using only cardiometabolic genetic variants that were also associated with inflammation, at genome-wide significance. Second, we used multi-variable MR (MVMR) to examine associations between cardiometabolic risk factors and schizophrenia after adjusting for genetically-predicted levels of C-reactive protein.

Results: In analyses using all associated genetic variants, genetically predicted levels of leptin were associated with risk of schizophrenia (OR=2.54 per SD increase in leptin; 95% CI, 1.02–6.31). In analyses using inflammation-related variants, genetically predicted levels of FI (OR=2.76 per SD increase in FI; 95% C.I., 1.31–6.17), TG (OR=2.90 per SD increase in TG; 95% C.I., 1.36–6.17), and HDL (OR=0.56 per SD increase in HDL; 95% C.I., 0.37–0.83) were associated with schizophrenia. The associations completely attenuated in MVMR analyses controlling for CRP. There was no evidence of an association between genetically-predicted schizophrenia liability and cardiometabolic factors.

Discussion: The IR phenotype of FI, TG and HDL could be associated with schizophrenia over and above common sociodemographic and lifestyle factors. This association is likely explained by a common inflammatory mechanism. Interventional studies are required to test whether inflammation could represent a putative therapeutic target for the treatment and prevention of cardiometabolic disorders in schizophrenia.

M89. PHARMACOLOGICAL INTERVENTIONS FOR SMOKING CESSATION AMONG PEOPLE WITH SCHIZOPHRENIA: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: People living with schizophrenia are 3 times more likely to smoke than the general population, and have fewer and less successful quitting attempts. In concert with psychosocial quit interventions, there is a need for evidence based pharmacological interventions to assist people living with schizophrenia achieve smoking abstinence.

Methods: We systematically searched PubMed, PsycInfo, EMBASE and Cochrane for randomised controlled trials of pharmacological interventions for reducing smoking among people living with schizophrenia. We conducted pairwise and network meta-analyses of effectiveness of interventions for achieving abstinence and reduction in smoking. We also examined psychiatric and physical adverse events of interventions.

Results: Nineteen studies were included in the systematic review. Data was available for buproprion, varenicline and nicotine replacement therapy (NRT). Buproprion (RR 3.4, 95%CI 1.6–7.3, p=0.002), varenicline (RR 3.8, 95%CI 2.0–7.2, p<0.001) and NRT (RR 4.3, 95%CI 1.7–10.7, p=0.002) were all associated with increased rates of abstinence in pairwise meta-analyses. In a network meta-analysis varenicline was superior to buproprion (RR 2.0, 95%CI 1.0–3.9), however there was no statistically significant difference between varenicline and NRT or buproprion and NRT. Varenicline was associated with higher rates of nausea than placebo.

Discussion: Buproprion, varenicline and NRT were all superior to placebo for achieving abstinence. Varenicline appears to be superior to buproprion for achieving abstinence, however varenicline is associated with higher rates of nausea.

M90. CANNABIS USE, CIGARETTE SMOKING, AND PSYCHOTIC EXPERIENCES IN ADOLESCENCE AND DIAGNOSIS OF PSYCHOSIS IN EARLY ADULTHOOD. A BIRTH-COHORT STUDY

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Background: Recent studies indicate that adolescent cannabis use (1) and cigarette smoking (2) increase the risk for psychosis. However, less is known about symptom profile associated with cannabis use and cigarette smoking prior to the psychotic episodes. Our aim was to study the associations between daily smoking, life-time cannabis use, and psychotic experiences in adolescence, and their relationship with psychotic disorders in early adulthood.

Methods: The Northern Finland Birth Cohort 1986 study includes 99% of all births (n=9432) in the region. At age 15–16, data on self-reported daily cigarette smoking and cannabis use was gathered using questionnaires. Psychotic experiences during past 6 months were evaluated using PRODscreen (3). Psychiatric diagnoses were collected from four Finnish nationwide health-care registers until year 2016, when participants were 30–31 years old. Individuals with information on daily smoking, cannabis use and psychotic experiences (n=6037, 47.7% male, 64.0% of the total