Title

155. Tobacco Use and Psychosis Risk in Persons at Clinical High Risk

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Between group comparisons revealed that those with social anxiety did indeed have worse social functioning, particularly in the domains of interpersonal communication ($t(45) = 2.28$, $P = .025$) and engagement (in conversations; $t(45) = 2.89$, $P = .000$). Participants with high social anxiety also had more difficulties in recognizing neutral emotional faces, compared to individuals with schizophrenia without social anxiety ($t(31) = 2.19$, $P = .036$).
Although not specific to social anxiety, results on the facial emotional tasks were correlated with specific social functioning domains (e.g., those with poor facial recognition overall had fewer recreational ($r = -.49$) and social activities, $r = -.56$). The results were not linked to negative symptoms of schizophrenia or to psychiatric symptoms (BPRS). In the role-play, individuals with social anxiety were better than those without social anxiety at recognizing 2/4 of the emotions targeted (joy and anger, $\chi^2 = 3.58$, $P = .05$).
Conclusion: These findings suggest that specific comorbidities, such as social anxiety, might affect facial emotional recognition, particularly for neutral emotions. More studies are warranted in order to determine whether social anxiety brings an attentional bias, therefore making individuals more prone to seeing emotions (even when not present). This study did confirm that social anxiety and facial emotional recognition have an important impact on social functioning and should be targeted in treatments.

154. IMPAIRED GLUCOSE HOMEOSTASIS IN FIRST-EPISODE SCHIZOPHRENIA: A META-ANALYSIS

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Background: Schizophrenia is associated with an increased risk of type 2 diabetes mellitus. However, it is not clear whether schizophrenia confers an inherent risk for glucose dysregulation in the absence of the effects of chronic illness and long-term treatment. Our objective was to conduct a meta-analysis examining whether individuals with first-episode schizophrenia already exhibit alterations in glucose homeostasis compared with controls.
Methods: The Embase, Medline and PsycINFO databases were systematically searched for studies examining measures of glucose homeostasis in drug-naive individuals with first-episode schizophrenia compared to controls. Of 3660 citations retrieved, 16 case–control studies comprising 15 samples met inclusion criteria. The overall sample included 731 patients and 614 controls. Standardized mean differences in fasting plasma glucose, plasma glucose post-OGTT, fasting plasma insulin, insulin resistance, and HbA1c were calculated.
Results: Fasting plasma glucose (g = 0.199 (95% CI 0.022–0.376, $P = .028$)), plasma glucose post-OGTT (g = 0.605 (95% CI 0.163–1.047, $P = .007$)), fasting plasma insulin (g = 0.409 (95% CI 0.093–0.724, $P = .011$)) and insulin resistance (HOMA-IR; g = 0.334 (95% CI 0.135–0.534, $P = .001$)) were all significantly elevated in patients compared with controls. However, HbA1c levels (g = −0.08 (CI −0.340 to 0.180, $P = .547$) were not altered in patients compared with controls.
Conclusion: These findings show glucose homeostasis is altered from illness onset in schizophrenia, indicating patients are at increased risk of diabetes mellitus as a result. This has implications for the monitoring and treatment choice for patients with schizophrenia.

155. TOBACCO USE AND PSYCHOSIS RISK IN PERSONS AT CLINICAL HIGH RISK

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Background: The purpose of this study was to evaluate the role of tobacco use in the development of psychosis in individuals at clinical high risk.
Methods: The North American Prodrome Longitudinal Study is a 2-year multisite prospective case–control study of persons at clinical high risk that aims to better understand predictors and mechanisms for the development of psychosis. The cohort consisted of 764 clinical high risk and 279 healthy comparison subjects. Clinical assessments included tobacco and substance use and several risk factors associated with smoking.
Results: Clinical high risk subjects were more likely to smoke cigarettes than unaffected subjects (Light Smoking OR = 3.0, 95%CI = 1.9–5; Heavy Smoking OR = 4.8, 95%CI = 1.7–13.7). In both groups, smoking was associated with substance use, stressful life events, and perceived discrimination and in clinical high risk subjects with childhood emotional neglect and adaption to school. Clinical high risk subjects reported higher rates of several factors previously associated with smoking. After controlling for these factors, the relationship between clinical high risk state and smoking became non-significant (Light Smoking OR = 1.9, 95%CI = 0.7–5.2; Heavy Smoking OR = 0.9, 95%CI = 0.1–7.2). Moreover, baseline smoking status (HR = 1.16, 95%CI = 0.82–1.65) and categorization as ever smoked (HR = 1.3, 95%CI = 0.8–2.1) did not predict time to conversion.
Conclusion: Persons at high risk for psychosis are more likely to smoke compared to unaffected persons. Factors associated with smoking in both groups were more common in the clinical high risk cohort, and smoking status in clinical high risk subjects did not predict conversion risk. These findings did not support a causal relationship between smoking and psychosis.
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156. CONTINUED CANNABIS AND SUBSTANCE USE IN THE FIRST 2 YEARS FOLLOWING ONSET OF PSYCHOSIS: PREDICTING RISK OF MEDICATION NONADHERENCE

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Background: Medication adherence during the early period following first episode of psychosis (FEP) is a key factor that determines outcome and subsequent course of the illness. Uncertainty exists whether the use of