Methods: Eighty-five patients with schizophrenia were recruited and free-viewing, fixation stability and smooth pursuit tests were performed. First, multiple regression analysis was performed using the obtained parameters as the dependent variables, antipsychotics, illness severity, and duration of illness as independent variables. Secondly, patients were grouped into tertiles by antipsychotic dose (CPZ equivalents), then we conducted a group comparison with each parameter between the three groups. 

Results: A multiple linear regression was calculated to predict each parameter based on CPZ equivalents, illness severity and duration of illness. There was no significance in the free-viewing and fixation stability test after Bonferroni correction. In smooth pursuit test, a significant regression equation was found with the horizontal gain (F (1.81) = 15.1, p < 0.00, R2 = 0.15) and vertical gain (F (1.81) = 12.5, p = 0.02, R2 = 0.12), and both were accounted only for CPZ equivalents. In a group comparison, there were significant effects of the horizontal gain (F (2.80) = 5.32, p = 0.07) and the vertical gain (F (2.80) = 3.31, p = 0.41), but both did not survive Bonferroni correction. 

Discussion: It was found that antipsychotic treatment affects smooth pursuit eye movement. Eye movement abnormalities in schizophrenia can be a useful biomarker from previous studies, but the effects of antipsychotics must be considered. 

T9. EPIGENETIC PROFILING IN SCHIZOPHRENIA DERIVED HUMAN INDUCED PLURIPOTENT STEM CELLS (HIPCS) AND NEURONS

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Background: Schizophrenia (SCZ) is a severe psychiatric disorder affecting ~1% of the world’s population. It is largely heritable with genetic risk reflected by a combination of common variants of small effect and highly penetrant rare mutations. Chromatin modifications are known to play critical roles in the mediation of many neurodevelopmental processes, and, when disturbed, may also contribute to the precipitation of psychiatric disorders, such as SCZ. While a handful of candidate-based studies have measured changes in promoter-bound histone modifications, few mechanistic studies have been carried out to explore how these modifications may affect chromatin to precipitate behavioral phenotypes associated with the disease. 

Methods: We applied an unbiased proteomics approach to evaluate the epigenetic landscape of SCZ in human induced pluripotent stem cells (hiPSC), neural progenitor cells (NPCs) and neurons from SCZ patients vs. matched controls. We utilized proteomics-based, label free liquid chromatography mass spectrometry (LC-MS/MS) on purified histones from these cells and confirmed our results by western blotting in postmortem SCZ cortical brain tissues. Furthermore we validated our findings with the application of histone interaction assays and structural and biophysical assessments to identify and confirm novel chromatin ‘readers’. To relate our findings to a SCZ phenotype we used a SCZ rodent model of prepulse inhibition (PPI) to perform pharmacological manipulations and behavioral assessments.

Results: Using label free mass spectrometry we performed PTM screening of hiPSCs, NPCs and matured neurons derived from SCZ patients and matched controls. We identified, amongst others, altered patterns of hyperacetylation in SCZ neurons. Additionally we identified enhanced binding of particular acetylation ‘reader’ proteins. Pharmacological inhibition of such proteins in an animal model of amphetamine sensitization ameliorated PPI deficits further validating this epigenetic signature in SCZ.

Discussion: Recent evidence indicates that relevance and patterns of acetylation in epigenetics advances beyond its role in transcription and small molecule inhibitors of these aberrant interactions hold promise as useful therapeutics. This study identifies a role for modulating gene expression changes associated with a SCZ epigenetic signature and warrants further investigation in terms of how this early gene expression pattern perhaps determines susceptibility or severity of the SCZ disease trajectory.

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stable on clozapine. Between examinations there will be no planned interference with the antipsychotic treatment, but antipsychotic treatment may be changed during the period in the intention to improve symptoms. The primary longitudinal outcome is comparing changes in BBB-permeability along with symptom-fluctuations. This will be done by comparing changes in qAlb, which is the gold standard technique measuring the CSF:serum albumin ratio (QAlb).

**Results:** The study has been approved by the regional ethical committee and data collection will begin in 2020.

**Discussion:** We expect the obtained results will contribute to a better pathophysiological understanding about illness markers and their progression over time and in relation to functional outcome.

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**T11. CORONARY ARTERY CALCIFICATION IN PEOPLE WITH SCHIZOPHRENIA**

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**Background:** Coronary artery disease (CAD) is one of the major causes of premature mortality in patients with schizophrenia. Coronary artery calcification (CAC) is an independent predictor of cardiac mortality and CAD in the general population, but has not yet been investigated in patients with schizophrenia. The aim of the present study is to compare CAC quantified by cardiac computed tomography (CT) in patients with schizophrenia to the general population.

**Methods:** Baseline data from an ongoing prospective cohort study including 200 patients with schizophrenia (ICD-10 diagnoses F20 or F25) diagnosed at least 10 years prior to inclusion (chronic group) and 86 patients with schizophrenia diagnosed within two years prior to inclusion (debut group). Patients in the debut group were matched 1:1 on age, gender and smoking status with psychiatrically healthy controls (PHC). All participants underwent cardiac CT and the CAC was quantified using Agatston Score. Mean CAC in the chronic group was compared to reference CAC scores whilst mean CAC in the debut group was compared to PHC. Information on cardiovascular risk factors, illness history, social and psychiatric conditions were obtained at baseline.

**Results:** Data is currently being analyzed and results will be presented at the Congress of International Schizophrenia Research Society.

**Discussion:** If the CAC quantified by CT in patients with schizophrenia differs from the PHC population, it might act as a tool for early detection of CAD in these patients. Thus, the findings of this study might contribute to preventive strategies in order to decrease cardiovascular mortality.

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**T12. THE RELATIONSHIP OF INTESTINAL PERMEABILITY FACTORS WITH SOCIODEMOGRAPHIC AND PHYSICAL HEALTH FACTORS AND PANSS SCORES IN SCHIZOPHRENIA PATIENTS AND HEALTHY CONTROLS**

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**Background:** Recent studies have pointed to the gut-brain axis as a new venue for treatment of psychiatric disorders, with increased inflammation stemming from increased intestinal permeability to further affect brain functioning in a significant subset of patients. Yet, this line of research is still in its infancy, with multiple studies showing increased intestinal permeability in schizophrenia and bipolar disorders, demonstrated as translocation of food and bacterial antigens, as well as intestinal microbiome disturbances.

**Methods:** Therefore, we measured intestinal permeability markers soluble CD14 (sCD14) and lipopolysaccharide binding protein (LBP) in schizophrenia patients and healthy controls. Intestinal permeability markers were compared to several sociodemographic, including age, gender and BMI, and physical health variables, including CRP, glucose, cholesterol, triglycerides, HDL, LDL and non-HDL, and Positive and Negative Syndrome Scale (PANSS) scores. Of the control group (n = 43), 76.7% was male, with a mean age of 25.1 years. Of the schizophrenia group (n = 105) 75.2% was male, with a mean age of 27.4 years and an average PANSS score of 57.2.

**Results:** Levels of LBP and sCD14 were not significantly different between schizophrenia patients and controls. LBP and sCD14 levels were neither correlated in the control group, nor in the schizophrenia group. In the control group Females had elevated LBP levels compared to males (p < 0.01), but not in the schizophrenia group. Quantitative levels of LBP, but not sCD14, correlated with triglycerides in the schizophrenia group (R^2 = 0.049, p < 0.05). Furthermore, quantitative levels of sCD14, but not LBP, correlated with CRP in the schizophrenia group (R^2 = 0.078, p < 0.05). Finally, LBP levels in patients correlated with PANSS negative scores (R^2 = 0.055, p < 0.05). Neither a correlation of LBP and sCD14 with age, nor with BMI was observed in both the control and the schizophrenia group.

**Discussion:** In conclusion, these intestinal permeability markers showed few differences between the schizophrenia and the control group. We found weak, yet significant correlations with triglycerides, CRP and severity of negative symptoms, which may be caused by poor eating habits or metabolic syndrome leading to leaky gut in the more severely affected patients. These results are not in line with results of Severance et al. (2013), who performed a similar analysis and found differences in intestinal permeability markers between a control group and a schizophrenia group. Furthermore, they did observe a positive correlation between sCD14 and LBP in both the control and the schizophrenia group. The difference between that study and our current findings may stem from the different patients samples, as we assessed patients in their first five years after diagnosis, when metabolic syndrome was less abundant.

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**T13. CORNEAL CONFOCAL MICROSCOPY DETECTS NEURAL CHANGES IN SCHIZOPHRENIA**

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**Background:** A combination of neurodevelopmental and degenerative neural changes are likely to underpin positive and negative symptoms in schizophrenia. However, there are currently no validated biomarkers to accurately quantify the extent of neural changes in schizophrenia. Corneal confocal microscopy (CCM) is a non-invasive ophthalmic imaging technique that has been used to demonstrate in vivo corneal nerve fiber abnormalities in a range of peripheral neuropathies and central neurodegenerative disorders including Parkinson’s disease, multiple sclerosis and dementia. We wished to test the hypothesis that corneal nerve abnormalities occur in patients with schizophrenia, particularly those with negative symptoms and cognitive impairment.

**Methods:** Patients with DSM-V schizophrenia without other causes of peripheral neuropathy other than metabolic syndrome underwent assessment

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