We found that patients and HC could be separated very well via Neurominer (NM) was conducted. An additional VBM analysis revealed highly significant negative correlations with grey matter changes in frontotemporal cortical areas, the anterior cingulate and the insular cortex. These areas are already discussed for CT in the literature.

Discussion: CT seems to be a global risk factor for psychiatric disorders. Moreover, we could re-examine the results of our multi variate analysis successfully in a VBM procedure.

T4. IDENTIFICATION OF NEUROANATOMICAL SURROGATE MARKERS OF CHILDHOOD TRAUMA
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Background: Childhood trauma (CT) plays an important role in psychiatric disorders. It is associated with an increased risk for psychiatric disorders like major depression, anxiety disorders, dependency, post-traumatic stress disorders and even psychosis. There is a high incidence of CT in patients with psychosis, especially for physical and sexual abuse. Already in UHR-individuals increased CT could be observed. A study of Thompson and colleagues showed that 97% of their UHR sample reported a trauma in the past. 83% of the cases were physical abuse, 67% emotional abuse and 27% sexual abuse. Our aim was to investigate if there are neurobiological surrogate markers of trauma existing which can be detected by a multi pattern analysis.

Methods: PRONIA (‘Personalized Prognostic Tools for Early Psychosis Management’) is a prospective collaboration project funded by the European Union under the 7th Framework Programme (grant agreement n° 602152). Considering a broad set of variables (sMRI, rsMRI, DTI, psychopathological, life event related and sociobiographic data, neurocognition, genomics and other blood derived parameters) as well as advanced statistical methods, PRONIA aims at developing an innovative multivariate prognostic tool enabling an individualized prediction of illness trajectories and outcome. Seven clinical centers in five European countries and in Australia participate in the evaluation of three clinical groups (subjects clinically at high risk of developing a psychosis [CHR], patients with a recent onset psychosis [ROP] and patients with a recent onset depression [ROD]) as well as healthy controls; planned sample size is n=1680. CT was assessed by the Childhood Trauma Questionnaire (CTQ). To identify neuroanatomical and functional surrogate markers of CT, a multi pattern analysis via Neurominer (NM) was conducted. An additional VBM analysis was performed to evaluate the results of the NM analysis.

Results: Patients with bipolar or psychotic disorders reported higher levels of metacognitive beliefs compared to controls. Metacognitive beliefs were significantly related to depression for all patients. Higher levels of metacognitive beliefs were also related to illness-factors related to a poorer long-term outcome, specifically an earlier age at onset of affective disorder in bipolar disorders, and poorer premorbid social adjustment in psychotic disorders. Metacognitive beliefs significantly mediated the relationship between early emotional abuse and depression. The combination of metacognitive beliefs and depression significantly mediated the relationship between early emotional abuse and positive symptoms. The mediation models explained a moderate amount of the variance in symptoms (R2 = .21 and .29) compared to direct models of early emotional abuse impacting on symptomatic responses directly (R2 = .04 and .03)

Discussion: Our results show that patients with bipolar or psychotic report higher levels of metacognitive beliefs compared to controls, and that such beliefs relate to current symptoms of depression in both patient groups. Our results also suggest that metacognitive beliefs relate to factors present before or at the onset of illness, which are often linked to a poorer long-term outcome in the disorders. Further, our findings suggest that in regards to early emotional abuse, metacognitive beliefs could play a role in an affective pathway to psychosis. Metacognitive beliefs could thus be relevant treatment targets in regards to depression and positive symptoms in bipolar and psychotic disorders.

T5. LURASIDONE AND RISK FOR METABOLIC SYNDROME IN PATIENTS WITH SCHIZOPHRENIA: A COMPREHENSIVE DATABASE ANALYSIS
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Background: Patients with schizophrenia are at increased risk for developing metabolic syndrome, with an estimated prevalence of approximately 35–50% (Correll et al. Psychiatr Serv 2010;61:892–98; Vancampfort et al. World Psychiatry 2015;14:339–47). Treatment with atypical antipsychotic medications have been shown to increase rates of metabolic syndrome, with differences observed among antipsychotic agents, most notably in propensity for weight gain: higher for olanzapine, clozapine, and iloperidone; intermediate for quetiapine, risperidone, and paliperidone; and lower for amisulpride, aripiprazole, asenapine, lurasidone, and ziprasidone (Leucht et al. Lancet 2013;382:951–62). Independent of weight gain, atypical antipsychotics also appear to have direct effects on lipid metabolism and glucose regulation. The aim of this safety analysis was to assess the effects of treatment with lurasidone on metabolic syndrome risk in patients with schizophrenia.

Methods: Changes in the rate of metabolic syndrome during treatment with lurasidone (40–160 mg/d) versus active comparators (olanzapine, quetiapine, risperidone) were analyzed using pooled short-term data from 3 randomized, double-blind, placebo-controlled studies; long-term data from 2 active-controlled studies; and switch data from 2 open-label extension studies. Metabolic syndrome was defined based on the National Cholesterol Education Program criteria (NCEP ATP III; 2005 revision).

Results: In short-term studies, risk of treatment-emergent metabolic syndrome was similar for patients in the lurasidone and placebo groups (odds ratio [OR]=0.97; week 6 LOCF-endpoint); and was significantly greater for patients in the olanzapine (OR=2.68; P<0.001) and quetiapine (OR=3.70; P<0.001) groups compared to placebo. In long-term studies, risk of treatment-emergent metabolic syndrome after 12 months was significantly lower for lurasidone compared with risperidone (OR=0.97; 95% CI, 0.80–1.17; P<0.01) and non-significantly lower for lurasidone compared with quetiapine XR (OR=0.64; 95% CI, 0.44–0.93; P=0.03). In open-label switch studies, the rate of metabolic syndrome decreased in patients switched to lurasidone after 6 weeks of treatment with olanzapine or 12 months of treatment with risperidone.

Discussion: In this comprehensive analysis of the lurasidone clinical trial data base, treatment with lurasidone (40–160 mg/d) was not associated with the development of metabolic syndrome in patients with schizophrenia. Rates of metabolic syndrome increased in patients treated with olanzapine, risperidone, and quetiapine XR.