p=0.930) were included as covariates in the analysis based on literature or expert criteria. The results showed that a systemic low-grade inflammation state (CRP>0.3mg/dL) (OR 13.776; p<0.001) and the CGI score (OR 2.183; p=0.012) were associated with increased gut bacterial translocation permeability state.

Conclusion: Our study found that 32.5% of adult outpatients with schizophrenia presented increased gut bacterial translocation, based on the immune activation marker LBP. This increased gut permeability was associated with a systemic low-grade inflammation state (CRP>0.3mg/dL) and with global severity of symptoms in schizophrenia.

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Is blood-brain-barrier disruption associated with cognitive deficits in first-episode psychosis? Findings from a retrospective chart analysis

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Background: In people with psychosis, cognitive deficits are present as early as disease onset and serve as a robust predictor of functional outcomes in the later course of the disease. Thereby, working memory is one of the cognitive domains most affected.

Blood-brain-barrier disruption is another well-established finding in a subgroup of people with first-episode psychosis. This alteration may be the consequence of neuro-inflammatory processes and relates to functional abnormalities and cognitive deficits. However, the implications of blood-brain-barrier disruption in first-episode psychosis have not yet been well examined.

We investigated potential associations between blood-brain-barrier disruption and working memory in the largest available first-episode psychosis cohort that also includes cerebrospinal fluid parameters. We hypothesized that different blood-brain-barrier parameters predict working memory.

Methods: We conducted a retrospective chart analysis with clinical data from 350 inpatients with first-episode psychosis admitted to our tertiary care hospital (LMU Munich, clinic for psychiatry and psychotherapy). A subsample of 147 patients (84 males, 63 females, age at cognitive testing 34.36 ± 15.57 years) underwent both a lumbar puncture and cognitive testing within the clinical routine. Regarding blood-brain-barrier disruption, age-dependent albumin ratios, IgG ratios and oligoclonal band-types were investigated. Working memory was assessed with the Wechsler intelligence test for adults (WIE) and the Attention test battery, version 2.1 (TAP 2.1).

Multiple linear regressions were performed to determine whether blood-brain-barrier is associated with working memory. We furthermore controlled for sex, age at the time of cognitive testing and type of school graduation. Prior to analysis, all non-dichotomous variables were z-standardized to establish comparability between different scales and value ranges, including age at the time of cognitive testing, type of school graduation, age-dependent albumin ratios, IgG ratios, oligoclonal band-types and working memory outcomes.

Results: In our sample 17.1% of the patients had abnormal cerebrospinal fluid findings, shown in an elevated age-dependent albumin ratio, and indicating blood-brain-barrier disruption. Oligoclonal bands in the cerebrospinal fluid were present with 10.1% of patients presenting type 3 (cerebrospinal fluid restricted oligoclonal bands and additional, identical oligoclonal bands in serum and cerebrospinal fluid) and 20.2% presenting type 4 (identical oligoclonal bands in cerebrospinal fluid and serum, “mirror pattern”).

Multiple linear regressions showed no significant associations between blood-brain-barrier and working memory when controlled for covariates (e. g. age-dependent albumin-ratios, sex, age at the time of cognitive testing and type of school graduation did not statistically significant predict working memory (TAP 2.1), $F(4, 78) = 0.46$, $p = .739$). Even when not controlling for relevant covariates, no significant associations between blood-brain-barrier and working memory could be found (e. g. age-dependent albumin-ratios did not statistically significant predict working memory (WIE), $F(1, 99) = 0.34$, $p = .560$).

Conclusions: Even though several inflammatory alterations regarding the blood-brain-barrier were found in our sample, we found no evidence for a significant relationship between blood-brain-barrier disruption and impaired working memory. The relationship studied might be present only later during the disease and should be reexamined in a multi-episode sample.

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Neuroleptic malignant syndrome and anti-NMDA encephalitis as dopamine dysregulation syndromes: a case and literature review

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Objective: Due to the rich psychiatric symptomatology, autoimmune encephalitis (AE) patients often consult a psychiatrist first [1]. Prescribing antipsychotics increase the neuroleptic malignant syndrome (NMS) risk. Neuroleptic sensitivity in AE is known, and NMS-like picture with neuroleptics is one of the red flag signs [1].

Here we report a case of NMDA-R encephalitis and a review of the literature conducted under keywords of NMS’ and ‘NMDA-R encephalitis’ in PubMed, all case reports published in English are included.

Case: A 22-year-old woman with no psychiatric history was admitted to the psychiatry clinic with anxiety, insomnia, concentration difficulty and derealization signs where she had been started on risperidone with a dissociative psychosis diagnosis. Haloperidol injection was administered at the emergency department for psychomotor agitation and the next day, altered level of consciousness, bilateral lead pipe rigidity, automatism-like movements in mouth, and autonomic instability developed. In the laboratory tests, creatine kinases (CK) was high, urine toxicology was negative, cerebrospinal fluid protein increased, leukocytes increased slightly, and a dermoid cyst was found in the right ovary on MRI. EEG was consistent with NMDAR-E, and an increased signal was found in the right insular cortex on brain MRI. Diagnosis of NMDAR-E was made upon detection of NMDA antibody positivity in serum and cerebrospinal fluid.

The patient was under antiepileptic treatment during hospitalization and given pulse steroid, IVIG, plasmapheresis, and rituximab for NMDAR-E. Amantadine, pramipexole, and bromocriptine were also given for NMS. Confusion, high CK, autonomic instability, and lead pipe rigidity continued until the 19th day in our hospital and significant improvement was seen in her clinic after the 7th dose of plasmapheresis. She was discharged in full recovery.

Discussion: Overlapping symptoms of NMDAR-E and NMS, make differential diagnosis difficult, particularly when antipsychotic medication is prescribed.

In NMDAR-E, antibodies bind to NMDA receptors which also control dopamine release by their direct effect on dopaminergic neurons in the basal ganglia, hypothalamus, and brainstem regulatory systems. The antibodies may cause disinhibition in post-synaptic neurons and dysregulation of glutamate and dopamine release by blocking presynaptic GABAergic neurons in the thalamus and prefrontal cortex [2].

NMS is a rare side effect of neuroleptics. The most accepted theory for NMS is 'Dopamine Hypoactivity' [3]. Blockade of dopaminergic signaling in motor-circuit may lead extrapyramidal signs, whereas decreased dopamine signaling to