The next aim was to compare profiles of cognitive impairment in both groups of patients.
The last aim was to find out a relationship between cognitive performance and severity of depressive episode during the acute state of illness.
Methods We have used neuropsychological test battery (Auditory–Verbal Learning Test, Rey-Osterrieth Complex Figure Test, Logical Memory, Digit span test, Trail making test, Verbal Fluency Test, Block Design and Benton Visual Retention Test) for the evaluation of the cognitive functions in patients with severe depressive episode with psychotic symptoms ($n = 5$) and patients with major depressive disorder ($n = 8$).
Results We found cognitive impairment in all examined domains in both groups of patients.
More profound cognitive impairment was found in patients with severe depressive episode with psychotic symptoms, particularly in visual memory, visuo-constructive abilities, speed of cognitive processing and executive functions. We found no correlation between cognitive performance and severity of depressive episodes.
Conclusions Our findings suggest a strong correlation between psychotic symptoms in depression and cognitive performance.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW0115

Maternal depressive symptom trajectories and psychosocial functioning in young adults: A 27-year longitudinal study

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Introduction Maternal depression is a well-known risk factor for child development. Longitudinal studies extending from pregnancy to adulthood, however, are rare.
Objectives The aim of the study was to investigate whether maternal high depressive symptom trajectories (chronic or intermittent depressive symptom patterns) from pregnancy to the adolescence of the children predict lower adaptive functioning or higher levels of emotional or behavioural symptoms in young adults.
Methods The sample comprised 329 first-time mothers from maternity centres in Tampere, Finland. Maternal depressive symptoms were assessed with the Edinburgh Postnatal Depression Scale (EPDS) antenatally and at two months, six months, 4–5 years, 8–9 years and 16–17 years after delivery. A model including four symptom trajectories (very low, low-stable, high-stable and intermittent) was selected to describe the symptom patterns over time. Adaptive functioning and problems of the children ($n = 144$) were assessed by the Adult Self Report forms (Achenbach & Rescorla) at the age of 27 years.
Results High maternal depressive symptom trajectories did not predict self-reported lower adaptive functioning of the children in adulthood. However, children of mothers with chronic or intermittent depressive symptom patterns reported higher levels of internalising problems as well as symptoms of depression and anxiety in young adulthood than the children of mothers with very low or low stable symptom patterns.
Conclusions High maternal depressive symptom trajectories predict higher levels of emotional symptoms of children in young adulthood. The mechanisms of intergenerational transmission are important topics for further research.
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EW0116

Quetiapine XR as add-on to antidepressants in treatment-resistant late-life major depression

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Objective To assess the efficacy and tolerability of quetiapine as add-on to antidepressant agents in treatment-resistant late-life major depression.
Methods A group of 15 patients, 8 male and 7 female, mean age 68.2, evaluated in our department for clinical symptoms that made possible a DSM 5 diagnosis of major depressive disorder, were initiated on quetiapine XR, flexible daily dose 50–300 mg QD. All patients were on treatment with an antidepressant–either a selective serotonin reuptake inhibitor (SSRI) ($n = 10$), or venlafaxine ($n = 5$)–for at least 6 weeks and presented no improvement during current treatment administered at therapeutic doses. Patients were assessed using Montgomery Asberg Depression Rating Scale (MADRS), Clinical Global Impression–Severity (CGI-S), Global Assessment of Functioning (GAF), and Columbia Suicide Severity Rating Scale (C-SSRS) every 4 weeks for 3 months.
Results After 12 weeks, patients had a mean improvement in MADRS score of $45.7 \pm 2.3\%$, with a final mean MADRS score of 13.5 ($P < 0.01$). No variations were registered depending on the specific SSRI or venlafaxine concomitant treatment. Quetiapine XR mean daily dose administered during the study was 125 mg. C-SSRS didn't registered significant variations in suicidal ideation or behavior throughout the trial. Overall GAF score increased with 22.1 points, and CGI-S decreased with a mean of 1.5 points at week 12 ($P < 0.01$). Tolerability of add-on quetiapine was very good, no serious adverse event being reported.
Conclusions Quetiapine was efficient and well tolerated in late-life resistant major depression, as add-on to SSRIs or venlafaxine, during the 12 weeks of the trial.
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EW0117

The clinical course of depression: Chronicity is the rule rather than the exception

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Introduction Major depressive disorder (MDD) is often considered an episodic disorder. However, literature might underestimate the chronicity of MDD since results depend on follow-up dura-

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tion and the extent to which psychiatric co-morbidity is taken into account.
Aim To determine, whether MDD should be considered an episodic or chronic disorder.
Objective To examine the 6 year course of MDD, incorporating data of multiple time points and taking common psychiatric co-morbidities into account.
Methods Data were from 903 patients with current MDD at baseline in the Netherlands study of depression and anxiety, with subsequent data from 2 year, 4 year and 6 year follow-up. Four course trajectories were created taking all information during follow-up into account classifying patients as (1) recovered, (2) recurrent without chronic episodes, (3) recurrent with chronic episodes or (4) consistently chronic. A chronic episode was defined as having symptoms consistently over 2 years.
Results The recovery rate of MDD was 58% at 2 year follow-up but looking at 6 year follow-up and taking into account co-morbid dysthymia, (hypo) mania and anxiety disorders reduced this recovery rate to 17%. Moreover, more than half of the patients experienced chronic episodes.
Conclusions Longitudinal data of this psychiatric cohort study showed that full recovery is the exception rather than the rule. MDD follows a chronic course and, moreover, persons are prone to switch to other psychiatric disorders.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW0118

Mirtazapine and trazodone efficacy on major depressive disorder (MDD) is moderated by patients' age and sex: A randomized, controlled trial

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Introduction NaSSA antidepressant mirtazapine and SARI trazodone has proven efficacy on MDD.
Aim To compare differences in mirtazapine and trazodone efficacy on MDD in different age and sex groups.
Methods A consecutive sample of 60 MDD outpatients were randomized to mirtazapine 30 mg/day or trazodone 150 mg/day for a 3 months stable dosing period at the department of biological psychiatry and psychogeriatrics of the university psychiatric hospital Vrapče, Croatia. Outcome was relative lowering of HAMD-17 scale result. The study was single blind: rater was blinded, while patients informed regarding prescribed medication.
Results Overall efficacy of mirtazapine and trazodone was comparable (84% lowering of HAMD–17 in both cases; difference $P = 0.754$). After adjustment for MDD baseline severity (CGI–S), education, marital and working status, interaction of age and sex significantly moderated two drugs' efficacies. In patients older than 47 years, in male patients trazodone was significantly more effective, and in female patients significantly less effective than mirtazapine. This effect was increasing by aging.
Conclusion Mirtazapine and trazodone efficacy on MDD is moderated by patients' age and sex.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW0119

Early prediction of non-response to anti-depressive treatment with an easy-to-use electrophysiological index dynamics

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Introduction The evaluation of response to pharmacological treatment in MDD requires 6–8 weeks. Therefore, the ability to predict response, and especially lack of response to treatment, as early as possible after treatment onset or change, is of major significance. Many studies demonstrated significant results regarding the ability to use EEG and ERP markers. However, these markers are derived from long EEG/ERP samples, often from multiple channels, which render them impractical for frequent sampling.
Methods We developed a new electrophysiological attention-related marker from a single channel (2 electrodes) and 1 minute samples. This work presents an initial evaluation of the ability to harness this marker, for early differentiation between responders and non-responders to anti-depressive treatment, in 26 patients with various levels of depression and heterogeneous treatment interventions and 10 healthy controls. Subjects who initiated treatment for depression were followed clinically through their Hamilton depression scores as well as their EEG activity twice a week for a period of 8 weeks. Any acceptable anti-depressive treatment been included. The improvement in Hamilton scores at the end of 8 weeks used to discriminate responders and non-responders.
Results Within two weeks, we could differentiate between non-responders and responders to anti-depressive treatment, with absolute discrimination between subjects with moderate to severe depression, and with 0.71 sensitivity and 0.96 specificity within the whole depressed subjects.
Conclusions This is a proof of concept for an easy to use, cheap and quick marker for the lack of respond to anti-depressive treatment within two weeks of anti-depressive treatment.
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EW0120

The dopaminergic polymorphisms in psychomotor retardation of depression: A pathway-based imaging genetics association study

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Introduction Several lines of evidence implicate dopamine is involved in the psychomotor retardation (PMR) in major depressive disorder (MDD). Besides, abnormal cerebral blood flow (CBF) of PMR was also found in the cortico-basal ganglia-thalamo-cortical