screening instrument in the nationwide general population of South Korea.
Methods A total of 3013 adults among the 2011 Korean Epidemiologic Catchment Area survey (KECA-2011) completed face-to-face interviews using the Korean versions of the composite international diagnostic interview 2.1 and mood disorder questionnaire (K-CIDI and K-MDQ).
Results The lifetime prevalence of BPS in the South Korean adults was measured to be 4.3% (95% CI 2.6–6.9). Nearly 80% of the subjects with BPS were codiagnosed with other DSM-IV nonpsychotic mental disorders: 35.4% (95% CI 24.2–48.5) for major depression and dysthymic disorder, 35.1% (95% CI 27.7–43.3) for anxiety disorders and 51.9% (95% CI 40.5–63.1) for alcohol and nicotine use disorders. Younger age (18–34 years) was the only sociodemographic predictor of BPS positivity ($P = 0.014$) and the diagnostic overlap patterns were different between men and women.
Conclusions Positivity for BPS was estimated to be much greater than the prevalence of DSM-IV BP in South Korea. Most of the respondents with BPS were diagnosed with other major mental disorders and this might be related with mis and/or underdiagnosis of clinically relevant Sub-BP.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O018

Assessment of serum IL-4, 15d-PGJ2, PPAR gamma levels in patients with bipolar disorder

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Introduction Many hypotheses have been proposed about development of bipolar disorder including inflammatory processes due to the external and endogenous factors. There are strong evidences that immunological dysfunction is present in bipolar disorder. In the pathophysiology of bipolar disorder, there are many data that support the inflammatory hypothesis.
Objectives In this study, to clarify the etiology of bipolar disorder, based on the inflammatory process hypothesis, it is aimed to measure and evaluate serum 15d-PGJ2 and PPARγ, anti-inflammatory cytokine IL-4 levels in patients with bipolar disorder.
Methods This study was performed at Ankara Numune Training and Research Hospital. Ninety-five patients are included in the study that were in their mania or remission periods and meet the DSM-V criteria for bipolar disorder. Forty-four healthy volunteers are included in the study as well. Serum IL-4, 15d-PGJ2, PPARγ levels are measured in both groups. Young Mania Scale, Hamilton Depression Scale, demographic data form were given to patient group.
Results In our study, 15d-PGJ2, PPARγ levels were found statistically significantly lower in patients with bipolar disorder compared to healthy controls.
Conclusion There are differences in anti-inflammatory prostaglandin levels in patients with bipolar disorder who are in their mania period when compared to healthy controls and patients in their remission period. This does not show any significance according to smoking and gender. This implies that inflammation markers could be a good candidate to determine trait markers, which will provide an insight for preventing patient from mania period or prognosis after the diagnosis of bipolar disorder.
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O019

Impulsivity and brain volume in patients with bipolar disorder type I and bipolar disorder type II

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Introduction Impulsivity is a key feature of both bipolar disorder (BD) type I (BDI) and type II (BDII).
Objective Structural neuroimaging studies help clarifying brain mechanisms underpinning the regulation of impulsivity in BDI and BDII.
Aims To address the question whether grey matter (GM) alterations relate differently with impulsivity in BDI and BDII.
Methods We assessed 54 euthymic outpatients, diagnosed with BDI ($n = 28$) or BDII ($n = 26$) according to DSM-IV-TR criteria. They underwent a 3 T magnetic resonance imaging (MRI) investigation. GM brain volumes were analyzed on a voxel-by-voxel basis using Statistical Parametric Mapping 8. The Barratt Impulsiveness Scale (BIS), version 11A, was used to assess trait impulsivity.
Results BDI and BDII patients present an inverse relationship between impulsivity and GM volume in two cerebral areas: the right cerebellum (right crus I) and the interface between the left angular gyrus and the left inferior parietal cortex (Brodmann Area 39, 7, 40). More specifically, a negative relationship for BPI and a positive relationship for BPII were found in both areas.
Conclusions Results suggest that the different diagnosis between BDI and BDII could have a significant effect on GM changes according to impulsivity severity and point up the importance of considering the BP subtype distinction in neuroimaging studies on this topic.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O020

Inflammation and neurodegeneration findings in early stage bipolar disorder

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Introduction There is growing evidence about neuroinflammation in the aetiopathogenesis of bipolar disorder. Early diagnosis and intervention strategies are thought to be excessively important lately.
Objectives To check neuroinflammation levels in early stage bipolar disorder and explore the associations with clinical variables.
Aims We aimed to evaluate inflammation and neurodegeneration findings in early stage bipolar disorder.
Methods Serum interleukin 1-receptor antagonist (IL-1Ra), interleukin 6 (IL-6), tumor necrosis factor-alpha (TNF-α), high sensitive C reactive protein (hs-CRP), S100B and neuron specific enolase (NSE) levels were assessed by enzyme-linked immunosorbent assays in a total of 30 patients with bipolar disorder in the early stage and compared with 30 matched healthy controls. The clinical symptoms were rated using Montgomery Asberg Depression Scale,

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Young Mania Rating Scale, Positive and Negative Syndrome Scale and Clinical Global Impression Scale.
Results Among the patients with bipolar disorder, 14 (% 46.6) were in a manic/hypomanic state and 12 (% 40) were in a euthymic state. Serum IL-6 levels were significantly higher ($P = 0.018$), TNF-α and S100B levels were significantly lower in the early stage group ($P < 0.001$ and $P = 0.03$, respectively). After repeated analysis with only drug-naive patients, the results showed no difference. There was a positive and significant correlation between TNF-α levels and CGI, MADRS scores (all $P < 0.05$); NSE levels and MADRS scores ($P < 0.05$).
Conclusions This study supported the association of early stage bipolar disorder with inflammation and neurodegeneration. IL-6 may be a potential biomarker. Thus, early diagnosis and intervention may be crucial to prevent progressive neuroinflammation and neurodegeneration in early stages of disorder.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O021

The correlation between plasma brain-derived neurotrophic factor and cognitive function in bipolar disorder is modulated by the BDNF Val66Met polymorphism

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Objectives Brain-derived neurotrophic factor (BDNF) may be involved in the pathogenesis of bipolar disorder (BD). The functional BDNF Val66Met polymorphism (rs6265) is associated with secretion of BDNF. The current study aimed to explore the correlation between changes of plasma BDNF and cognitive function after 12 week of treatment, considering the influence of the BDNF val66Met polymorphism. The correlation of changes of plasma BDNF with quality of life (QOL) was explored.
Methods First diagnosed patients with BD were recruited. Symptom severity, plasma BDNF levels were examined during weeks 0, 1, 2, 4, 8, and 12. QOL, Wisconsin Card Sorting Test (WCST) and the Conners' Continuous Performance Test (CPT) were assessed at baseline and endpoint. The genotype of the BDNF Val66Met polymorphism was determined. The change of cognitive function and QOL measures over 12 weeks were reduced by factor analysis. Pearson's correlation was used to investigate the association between change of plasma BDNF levels with cognitive function and QOL.
Results Five hundred and forty-one BP patients were recruited. Three hundred and fifty-five (65.6%) patients completed the 12-week follow-up. A significant negative correlation was found between changes of plasma BDNF level with factor 1 (WCST) ($r = -0.25$, $P < 0.001$). After further stratification according to subtypes of BD and the BDNF genotypes, above significant correlation was found only in those with BP-I and the BDNF Val66Met Val/Met genotype ($r = -0.54$, $P < 0.008$).
Conclusion We conclude that changes in plasma BDNF significantly correlated with changes in WCST in patients with BD; such correlation is moderated by the BDNF Val66Met polymorphism and subtype of BD.
Disclosure of interest The author has not supplied his declaration of competing interest.

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O022

Cortical inhibition in symptomatic and remitted mania compared to healthy subjects: A paired-pulse TMS study

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Introduction Cortical inhibition (CI) is a neurophysiological outcome of the interaction between GABA inhibitory interneurons and other excitatory neurons. Transcranial magnetic stimulation (TMS) measures of CI deficits have been documented in both symptomatic and remitted bipolar disorder (BD) suggesting it could be a trait marker. The effects of medications and duration of illness may contribute to these findings.
Objective To study CI in BD.
Aims To compare CI across early-course medication-naive BD-mania, remitted first episode mania (FEM) and healthy subjects (HS).
Methods Symptomatic BD subjects having < 3 episodes, currently in mania and medication-naive ($n = 27$), remitted FEM ($n = 27$; YMRS < 12 and HDRS < 8) and 45 HS, matched for age and gender, were investigated. Resting motor threshold (RMT) and 1-millivolt motor threshold (MT1) were estimated from the right first dorsal interosseous muscle. Paired-pulse TMS measures of short (SICI; 3ms) and long interval intracortical inhibition (LICI; 100ms) were acquired. Group differences in measures of CI were examined using ANOVA.
Results Table 1.
Conclusions Symptomatic mania patients had the highest motor thresholds and the maximum LICI indicating a state of an excessive GABA-B neurotransmitter tone. Remitted mania patients had deficits in SICI indicating reduced GABA-A neurotransmitter tone. Putative changes in GABA-A neurotransmitter system activity with treatment may be investigated in future studies. CI has received less attention in BD as compared to schizophrenia and is a potential avenue for future research in this area.

Table 1 Measures of motor threshold and CI across the three groups.

	Symptomatic mania (n=27)	Remitted mania (n=27)	HS (n=45)	F [a]	P[b]	Posthoc LSD
RMT Mean(SD)	37.93 (8.85)	32.63 (6.19)	37.09 (7.12)	4.161	0.019	BD > FEM HS >FEM
MT1 Mean(SD)	50.97 (12.15)	41.48 (8.27)	49.00 (11.34)	5.964	0.004	BD > FEM HS >FEM
SICI (%) Mean(SD)	27.47 (33.14)	9.05 (58.65)	35.34(28.39)	3.578	0.032	FEM<HS
LICI (%) Mean(SD)	76.93 (19.52)	71.52(27.48)	56.32 (45.87)	3.215	0.045	BD > HS

[a]Degrees of freedom 2,96.
[b]Probability error for the omni-bus test.

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