

Methods literature review and additional analyses of SFBN database.

Results BD usually begins with a depressive episode. SFBN-data reveal that an earlier AoO is associated with a less favourable prospective illness course (more depression, mood instability and rapid cycling), longer delay to first treatment, past history of suicide attempts, being abused in childhood abuse, more psychiatric and medical comorbidities. Comparison of the US sample with the European sample of SFBN showed an earlier onset in US patients.

Conclusion and early AoF of BD is associated with a poorer long-term outcome, despite adequate current treatment.

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S020

Age at the onset of a first episode of psychotic mania: Does it have an impact on outcome?

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Purpose Studies conducted in child psychiatry suggest that patients with earlier onset of psychosis have poorer outcome. Similar findings have been published regarding onset of bipolar disorder. However, few studies have been conducted in youth mental health program where these patients may actually receive treatment. Identification of subgroups with distinct need and outcome among first episode mania patients would facilitate the development of specific treatment strategies better suited to the actual needs of patients.

Methods Sixty-seven patients with a first episode of psychotic mania were followed up over 12 months after recovery from this initial episode. Syndromic and symptomatic outcome were determined with the brief psychiatric rating scale, functional outcome with the quality of life scale and premorbid adjustment scale sub items.

Results While 90% of patients achieved syndromic recovery (disappearance of manic syndrome) at 6 and 12 months, 40% had not recovered symptomatically, still presenting with depression and anxiety. Return to previous level of functioning was achieved only by 34% of patients at 6 months and 39% at 12 months. Age at the time of first manic episode with psychotic features was a significant predictor of recovery of functional level.

Conclusions While manic symptoms reduce quickly in most patients after a first episode of psychotic mania, an important number of patients still display symptoms of depression and anxiety after 12 months and 2/3 do not reach functional recovery. Younger age at first episode predicts risk of poorer functional outcome.

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Symposium: Negative symptoms: phenomenology, clinical aspects and neuroimaging

S021

Clinical psychopathology of negative symptoms: A phenomenological perspective



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Negative symptoms encompass a broad constellation of psycho-behavioral phenomena, including affective flattening, poverty of speech, alogia, avolition, social withdrawal, apathy and anhedonia. These phenomena obviously exert a substantial impact on personal autonomy, quality of life and broad functional outcomes, ultimately being an important challenge for clinical decision-making and therapeutic support. In recent years, the attention to negative symptoms in schizophrenia has revamped, boosting the development of new rating tools as well as a broader conceptualization of derivative constructs (e.g. apathy, amotivation, anhedonia). However, despite its behavioral expressivity, the in-depth phenotypic characterization of negative symptoms remains partly unaddressed. Similarly, their clinical intertwining with other non-productive clinical features (e.g. anomalous subjective experiences, cognitive-perceptual basic symptoms and schizotypal features) is generally overlooked. Therefore, the current presentation specifically offers a stratified overview of the phenomenology of negative symptoms filtered through lens of clinical psychopathology.

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S022

The Evolution of negative symptom constructs

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Introduction Negative symptoms represent a separate dimension of schizophrenia psychopathology, distinct from positive symptoms, disorganization and cognitive impairment. It is increasingly acknowledged that negative symptoms are associated with poor functional outcome and represent an unmet need in schizophrenia treatment. Improvement in definition of their phenomenology, assessment instruments and experimental models are needed in order to improve schizophrenia prognosis.

Aims The presentation will review key aspects of the evolution of negative symptom constructs. In particular, findings concerning phenomenology, clinical assessment, association with functional outcome and brain imaging correlates will be presented.

Methods We searched PubMed for English full-text publications with the keywords

Schizophrenia AND "negative symptoms"/"primary negative symptoms"/"deficit schizophrenia"/"persistent negative symptoms"/"affective flattening"/alogia/"expressive deficit"/apathy/asociality/"social withdrawal"/anhedonia/"anticipatory anhedonia"/avolition/neuroimaging.

Results The distinction between secondary negative symptoms (i.e., those due to identifiable factors, such as drug effects, psychotic symptoms or depression), and primary or persistent negative symptoms (i.e., those etiologically related to the core pathophysiology of schizophrenia) is grounded on solid research evidence and might have major implications for both treatment development and clinical care. The evidence that negative symptoms cluster in motivation- and expressive-related domains is founded on large consensus and empirical evidence and will foster pathophysiological modeling. The motivation-related domain is a stronger predictor of functional outcome than the expressive one.

Conclusions An improved definition and assessment of negative symptoms needs to translate in large-scale studies to advance knowledge. In the short-term, the improved identification of treatable causes of secondary negative symptoms can translate into better care for people with schizophrenia.

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S023

Progressive brain changes associated with persistent negative symptoms following a first episode of psychosis

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Early persistent negative symptoms (ePNS) refer to the presence of potentially idiopathic or primary negative symptoms and have been observed following a first episode of psychosis (FEP). There is evidence for cortical changes associated with ePNS and given that a FEP often occurs during a period of ongoing brain development and maturation, neuroanatomical changes may have a specific age related component. The current study examined cortical thickness (CT), hippocampal/amygdala volume and shape as a function of clinical trajectories and age using longitudinal structural imaging in FEP. T1-MRI scans were acquired for early ($n=21$), secondary ($n=30$), non- ($n=44$) PNS patients with a FEP, and controls ($n=44$). Cortical thickness and amygdalar-hippocampal volumes and surface area (SA) metrics were extracted from three time points over a two-year period. Linear mixed models were applied to test for a main effect of group, and age group interactions. Relative to the other groups, ePNS patients showed cortical thinning over time in temporal regions and a thickening with age primarily in prefrontal areas. They also exhibited reduced left amygdalar and right hippocampal volumes. Morphometry revealed decreased surface area in ePNS compared to other groups in left central amygdala. The current study demonstrates that FEP patients with ePNS show significantly different CT trajectories with age. Increased CT may be indicative of disruptions in cortical maturation processes within higher-order brain regions. Amygdalar-hippocampal changes with age are also linked to ePNS with converging results from volumetric and morphometric analyses. Taken together, these results could represent dynamic endophenotypes setting these ePNS patients apart from their non-symptomatic peers.

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Symposium: New avenues in the management of bipolar disorder

S024

Mania and depression: What's new?

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Despite the high burden of bipolar disorder and the noticeable progress in its treatment, the disorder still goes frequently mis-

diagnosed, unrecognized, or not optimally treated. To date, no medication has been specifically developed on the basis of a precise understanding of the pathophysiology of the disorder, or based on the unique characteristics of several subtypes of bipolar disorder or on the medication mechanism of action. Lithium remains one of the gold standard treatments for bipolar disorder. Its mood-stabilizing properties are thought to occur via specific cellular signaling pathways, such as inhibition of glycogen synthase kinase 3, which is considered to regulate cellular apoptosis. Divalproex, carbamazepine and several atypical antipsychotics are also approved for bipolar disease. Evidence also suggests that antipsychotics show the ability to treat and prevent mania and/or depression but are often burdened by side effects such as sedation, orthostatic hypotension and weight gain. Hence, while it is clear that there still are several unmet needs especially for what pertains tolerability, efficacy for specific subtypes, and predictability. Novel and more effective treatments are needed and researchers are currently engaging in targeted drug development for bipolar illness, aimed at improving pharmacological strategies with marked and sustained effects. A variety of newer medications are being tested. Some of these drugs target pathways that are similar to those targeted by lithium, while others focus on newer targets, such as opiate receptor and N-methyl-D-aspartate (NMDA) receptors. Newer and older treatment strategies for bipolar disorder will be presented and critically reviewed.

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S025

The role of long acting antipsychotics in bipolar disorder

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Antipsychotics are widely used for the short and long-term treatment of bipolar disorder. Depot and long-acting injectable formulations (LAIs) can be particularly useful for certain subgroups of patients. This lecture will discuss the available data from randomized controlled trials of LAIs in bipolar disorder. A recently published meta-analysis and individual studies assessing depot medications, as well as modern LAIs such as risperidone, paliperidone and aripiprazole, will be reviewed, looking carefully into the prevention of either pole of illness and tolerability. Potential indications and patient profile, based on data and clinical experience, will be discussed.

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S026

Managing cognitive dysfunction in bipolar disorder

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Cognitive dysfunction, including memory and concentration difficulty, is an emerging treatment target in bipolar disorder. However, a key challenge in the management of these cognitive deficits is the lack of treatments with robust effects on cognition. Further, it is unclear how cognitive dysfunction should be assessed and addressed in the clinical treatment of the disorder. This talk will review the evidence for cognitive impairment in bipolar disorder, including its severity, persistence and impact on patients'