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Invited commentary

# Navigating the evolving landscape of catatonia research

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## 1. Introduction

The past decade has witnessed a meteoric rise in the pace of catatonia research, and recent findings are dramatically reshaping our understanding and approach towards this unique psychiatric disorder. The multifactorial nature of catatonia encompasses epidemiology, history, phenomenology, genetics, immunology, and neurobiological components, presenting a formidable challenge but also an opportunity for developing innovative diagnostic markers and therapeutic interventions. The latest findings guided our understanding towards a holistic and more nuanced understanding of catatonia and even led to catatonia being listed (again) as a separate diagnosis in the ICD-11. This is an important renewal, both clinically and scientifically, and we have taken it (next to the 150th anniversary of the first clinical description of catatonia in 2024) as an opportunity to invite numerous renowned researchers in the field of catatonia to present their latest results. We hold deep appreciation for numerous prominent investigators in the realm of catatonia research who have made substantial contributions to this Special Issue. Among them are distinguished psychiatrists whose perspectives and methodologies originate from their extensive clinical experience including Gregory Fricchione, Stanley Caroff, Gabor Ungvari, Victor Peralta, Manuel Cuesta, Jonathan Rogers, Jo Ellen Wilson, Sebastian Walther, Scott Beach and Jack Foucher. All are (or were) involved in routine clinical care and have adapted their groundbreaking studies to meet catatonia patients' needs.

As Guest Editors of this Special Issue, our aim is to foster a comprehensive understanding of catatonia, bridging the gap between historical, conceptual, animal, neuroimaging and epidemiological findings and translating them to clinical applications. Each article underscores a great commitment to advancing knowledge, promoting scientific dialogue, and catalyzing further research in this rapidly evolving field. A total of 31 articles were accepted for publication as part of this Special Issue. These articles can be divided into the following thematic categories (for overview see Fig. 1):

## 2. Historical and conceptual origins

Tracing the history of catatonia understanding and treatment offers insight into how far the field has advanced, both in terms of neurobiological underpinnings, diagnostic precision and therapeutic approaches. This historical context reminds clinicians and researchers of the struggles and achievements in the research on catatonia over the past 150 years. Therefore, it was important for us to include studies on historical origins and conceptual developments of catatonia.

In a unique systematic review of the historical literature, Hirjak et al. showed that catatonic symptoms were described and studied decades before Karl Ludwig Kahlbaum's catatonia concept of 1874. Furthermore, Hirjak et al. (2023a) also examined closely the Kahlbaum's 11 autopsy reports. This study found that, based on postmortem examination, Kahlbaum and other historical researchers postulated several pathogenic components of catatonia including increase in cerebral volume or atrophy, anemia, inflammation, suppuration, serous effusion, or dropsy as well as alterations of brain blood vessels such as rupture, distension or ossification in the pathogenesis of catatonia. These two papers were complemented by a narrative review on the history of benzodiazepines and lorazepam in the treatment of catatonia (Hirjak et al., 2023d). Lorazepam has been used successfully in the treatment of catatonia since 1983. Taking into account an important contemporary witness - Gregory Fricchione - the initial indication and application of lorazepam was described and important conclusions were drawn.

In another series of articles by the research group led by Jack Foucher and Fabrice Berna, various forms of hypertonia were examined in certain psychoses and catatonic disorders, such as paratonia, Gegenhalten, and psychomotor hypertonia. For instance, Foucher et al. (2022b) discussed the fact that after the introduction of antipsychotic medication, these motor disorders fell out of favor in psychiatry, and drug-induced parkinsonism became the prevailing explanation for all resistance to passive motion. Recently, there has been a rediscovery of hypertonia in antipsychotic-naive patients, referred to as *"spontaneous*

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*parkinsonism"*, implying that intrinsic and drug-induced forms might be the same. This century-old knowledge is not only of historical interest but also has relevance in understanding motor symptoms in psychiatric disorders. Another article by Foucher et al. (2022a) discussed abnormal movements intrinsic to certain endogenous psychoses, specifically focusing on a phenomenon known as parakinesia. Parakinesia was documented by neuropsychiatrists decades before antipsychotics were introduced but has been largely neglected in the mainstream psychiatry literature. Interestingly, this study conducted a Delphi consensus exercise with clinicians familiar with parakinesia and proposed two distinct forms: hyperkinetic parakinesia (HPk), characterized by dyskinetic-like expressive movements predominantly in the upper part of the face and body, and parakinetic psychomotricity (PPM), involving awkward, stiff, and bizarre gestures and mimics. According to authors, these parakinesias are highly specific to endogenous psychoses, have prognostic value, and are distinct from self-dystonic or self-alien movements. The study by Schorr et al. (2022) aimed to compare the clinical and neuropsychological characteristics of SSD patients with different forms of catatonia, namely progressive periodic catatonia (PPC), chronic system catatonias (CSC), and non-catatonic SSD patients (NC-SSD). The study found that PPC patients had a more frequent schizo-affective presentation with higher levels of depression but fewer positive psychotic symptoms compared to both CSC and NC-SSD patients. CSC patients had an earlier onset of illness, poorer cognitive functioning, and higher antipsychotic doses than PPC and NC-SSD patients. This study suggested that there is substantial clinical heterogeneity within the concept of 'catatonia' in SSD, and distinguishing between at least two chronic catatonic phenotypes (PPC and CSC) may help better understand this diversity, offering a more parsimonious approach than considering numerous distinct catatonic presentations based on ICD-11 criteria. In line with the previous studies, Foucher et al. (2023) aimed to establish a common framework for understanding clinical phenomena like drug-induced parkinsonism (DIP), locomotor paratonia (LMP), and psychomotor paratonia (PMP) in SSD. The authors also highlighted points of disagreement, such as the definition of DIP and the sensitivity of these phenomena to anticholinergic drugs, and proposed using measures like torque, EMG, and joint angles to investigate these issues in treated SSD patients, potentially leading to important neurobiological, clinical and therapeutic implications.

In addition to work on the historical origins of catatonia, three articles on contemporary concepts and current diagnostic criteria of catatonia were also contributed: First, Marc Oldham (2022) discussed the challenges in diagnosing catatonia due to variations in rating scales and diagnostic criteria. While the diagnostic criteria in DSM-5-TR and ICD-11 are somewhat aligned, the absence of item thresholds poses a fundamental limitation. His review highlighted the need for clear and consistent definitions for catatonia features to improve reliable detection. He also suggests that certain scales like the Bush-Francis (BFCRS)

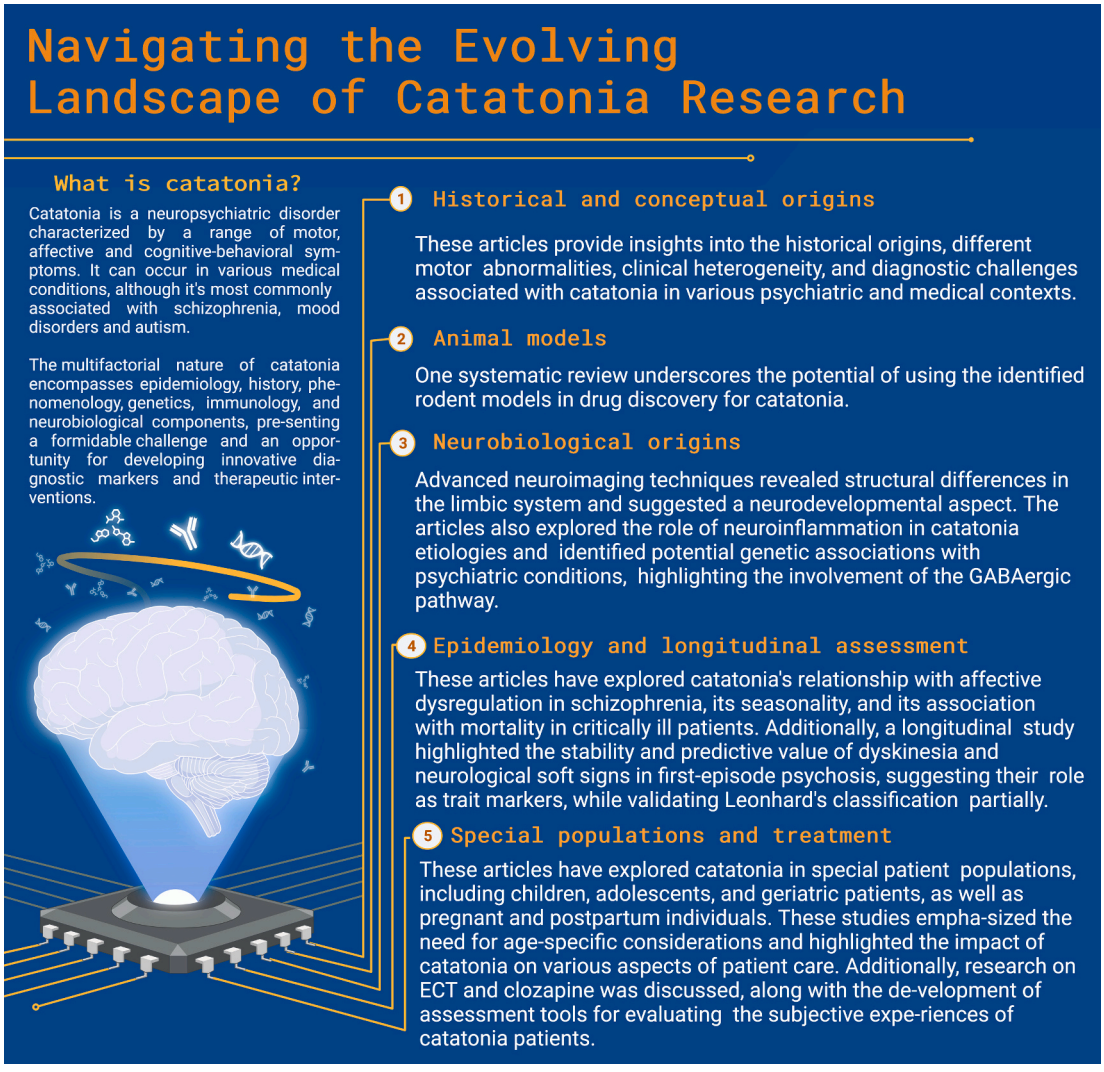


**Fig. 1.** Overview of thematic categories within the Special Issue Catatonia.

and Northoff (NCRS) Catatonia Rating Scales could be adapted for diagnostic criteria with some modifications. Second, Hirjak et al. (2023b) conducted a systematic review of scientific articles related to catatonia assessment and found that DSM and ICD criteria were the most commonly used clinical criteria, while the BFCRS and NCRS were the most frequently utilized rating scales. However, nearly half of the identified articles did not systematically assess catatonia. Hirjak et al. (2023b) recommended DSM and ICD criteria, along with BFCRS and NCRS, as valid instruments for evaluating catatonia symptoms. Third, John Waddington (2022) discussed the challenges in psychiatry, particularly in defining conditions like catatonia within evolving dimensional-continuum approaches to mental health disorders. Waddington suggested that conditions like catatonia may not neatly fit into categorical diagnoses but rather represent points of intersection between different dimensions of psychopathology and intrinsic movement disorder within a continuous landscape of mental health and dysfunction.

## 3. Animal models

Animal models of catatonia are scarce. Therefore, it is all the more gratifying that Mallien et al. (2023) has contributed a systematic review paper on rodent models of catatonia. The authors examined existing literature on rodent models of catatonia, highlighting their potential in uncovering the disorder's pathophysiology. The identified evidence suggested that these animal models exhibit similar neuronal abnormalities as seen in human patients, particularly within cortical-striatal-thalamocortical networks and associated dopaminergic, GABAergic, and glutamatergic neurotransmitter systems. This congruence underscores the potential of using these models in drug discovery for catatonia.

## 4. Neurobiological origins

Leveraging advanced neuroimaging technologies, a number of researchers endeavored to decode the neurobiological substrates underpinning catatonia: In a structural Magnetic Resonance Imaging (sMRI) study by Fritze et al. (2022) catatonia patients had significantly smaller anterior inferior hypothalamus, cortical nucleus of amygdala, and hippocampal fimbria volumes when compared to SSD patients without catatonia. In another sMRI study by Walther et al. (2022), catatonia patients showed increased local gyrification in premotor, motor, and parietal cortices compared to healthy controls and hypergyrification of the motor cortex and higher order cortical areas when compared to patients without catatonia. The authors suggested an early neurodevelopmental insult in the pathogenesis of catatonia. In line with this, Gregory Fricchione (2023) suggested that the concept that catatonia's core meaning is deeply rooted in the evolution of the vertebrate brain, originally proposed in 2004. Fricchione explored advances in catatonia theory and research, drawing from various thought leaders in neuropsychiatry and incorporating contemporary insights from neurophysiology, molecular biology, neuroimaging, and neurotherapeutics. This article helped to gain a deeper understanding of catatonia's origins and its impact on human life, with the aim of improving diagnosis and treatment while also shedding light on the fundamental nature of human fear and the challenges it presents. Keeping the limitations of previous neuroimaging studies in mind, Northoff and Hirjak (2022) proposed an integrated brain-mind approach to catatonia. This said, catatonia is based on aberrant neural topography and dynamic, e.g., inner time and space, that are shared by the mind's mental topography and dynamic, e.g., time-space experience, as their “common currency”. To better understand white matter (WM) tracts changes over time, Hirjak et al. (2023c) introduced the rationale and preliminary clinical findings of a new longitudinal study on catatonia (whiteCAT). If this study will be successful, it might be the largest longitudinal MRI study to date that has investigated WM tracts in catatonia patients.

Furthermore, in a review article, Beach et al. (2023) suggested that catatonia can arise from various medical causes, including both psychiatric and neurologic factors. The authors found an emerging evidence suggesting a potential link between catatonia etiologies and neuroinflammation. In particular, this connection is seen in conditions involving infections, inflammation, delirium, depression, and autism-spectrum disorders, all of which exhibit symptoms overlapping with catatonia. Syndromes like neuroleptic malignant syndrome and akinetic mutism, which share features with catatonia, may also have neuroinflammatory origins. Additionally, the activation of immune responses and its impact on specific brain regions, particularly the anterior and mid-cingulate cortex, medial prefrontal cortex, paralimbic cortico-striato-thalamo-cortical circuit, could play a crucial role in generating the motor and behavioral symptoms of catatonia. To the best of our knowledge, Wilson et al. (2023) conducted the first genome-wide association study of catatonia. This study found that psychiatric conditions like anxiety, bipolar affective disorder, and SSD and cross disorder polygenic risk scores were significantly associated with catatonia status in the European Ancestry. These associations suggested a potential shared genetic risk among these disorders, particularly in catatonia patients, even after controlling for relevant covariates. However, this shared genetic risk was not observed in the African Ancestry set, indicating potential ethnic differences in the genetic associations with catatonia. In a unique case study, Legrand et al. (2023) detected a pathogenic de novo rare variant in GABRB2, a gene involved in GABAergic transmission. This case report shed light on the genetic bases of catatonia. Moreover, by highlighting the involvement of the GABAergic pathway in this condition, the authors stressed the interest in appropriate treatments targeting GABA, like lorazepam. Finally, a systematic review by Cattarinussi et al. (2022) identified various structural, functional, metabolic and perfusion brain abnormalities associated with catatonia, including atrophy, signal hyperintensities, alterations in fronto-parietal and limbic regions, and abnormalities in the orbitofrontal, medial prefrontal, motor cortices, cerebellum, and brainstem. Further, this review also highlighted the need for caution due to considerable heterogeneity in the populations and neuroimaging techniques used in these studies.

## 5. Epidemiology and longitudinal assessment

Epidemiological studies help us understand the prevalence, risk factors, and distribution of catatonia in populations, enabling better resource allocation, prevention strategies, and advancements in mental healthcare. Special analyses were conducted as part of this Special Issue:

Kline et al. (2022) aimed to investigate the relationship between affective dysregulation and catatonia by analyzing electronic medical records of 36,839 patients with schizophrenia, using anxiety and depression diagnoses as proxies for affective dysregulation. The results showed that catatonia was present in 4.7 % of schizophrenia patients. The analysis revealed that catatonia was significantly associated with co-existing anxiety and depression; individuals with schizophrenia and catatonia were 1.71 times more likely to have anxiety and 1.80 times more likely to have depression compared to those without catatonia. Furthermore, the use of benzodiazepines was notably more common among schizophrenia patients with a catatonia diagnosis, suggesting a potential connection to GABAergic dysfunction underlying these conditions and affective dysregulation. In another study using electronic medical records, Mastellari et al. (2023) examined the seasonality of catatonia patients and its potential relationship with season of birth. The authors analyzed clinical records of catatonia patients and a control group of psychiatric inpatients from 2007 to 2016 in South London. The findings revealed that catatonia episodes increased during winter, peaking in February, and also showed a rise during summer, with a second peak in August. However, there was no evidence to suggest a link between the month of birth and the risk of developing catatonia. The authors concluded that recent triggers may play a role in catatonia development rather than distal events like birth season. Sexton et al.

(2023) investigated the associations between catatonia, delirium, coma, and mortality in 378 critically ill adults. The findings indicated that catatonia and delirium were not associated with increased mortality, but the occurrence of coma was significantly linked to both in-hospital and one-year mortality. These results emphasized the prognostic significance of suppressed arousal in critically ill patients and suggest that further research is needed to better understand the clinical impact of catatonia in this context.

A very unique longitudinal study by Peralta et al. (2022) followed 243 individuals with first-episode psychosis (FEP) for 21 years and assessed the stability of neuromotor domains such as dyskinesia, parkinsonism, neurological soft signs (NSS), and catatonia, as well as their predictive value for long-term outcomes. The study found that dyskinesia and NSS ratings demonstrated good stability over time, while parkinsonism and catatonia were less stable. Dyskinesia and NSS at baseline were robust predictors of various long-term outcomes, including symptoms, functioning, personal recovery, and clinical staging, suggesting that they serve as trait markers of the disease process in FEP. The same study also aimed to empirically validate Leonhard's four classes of psychoses: systematic schizophrenia (SSch), unsystematic schizophrenia (USch), cycloid psychosis (Cyclo), and manic-depressive illness (MDI). The findings presented by Cuesta et al. (2023) partially supported Leonhard's classification, with SSch displaying more significant impairments across these variables compared to USch, Cyclo, and MDI. However, the study did not find strong empirical evidence to distinguish between Cyclo and MDI, suggesting some limitations in Leonhard's classification in this context.

## 6. Special populations and treatment

This Special Issue featured very unique studies and reviews of catatonia in various patient populations across the lifespan. For instance, Karl et al. (2023) addressed the benefits of ECT in treating catatonia in children, adolescents and geriatric patients. Based on recent evidence, the authors suggested that it's vital to consider age-specific needs when administering ECT. Further, Delvi et al. (2023) examined electronic healthcare records from a large mental health trust in South-East London focusing on catatonia in the peripartum period. This study identified 21 individuals who experienced postpartum catatonia. All cases presented with typical catatonic features, and many had obstetric complications. Most of the patients received antipsychotic treatment and benzodiazepines, with a significant number being diagnosed with depressive disorders after the catatonic episode. These findings suggested that the postpartum period may carry a heightened risk of catatonia, especially in cases with obstetric complications. Focusing on the same topic, Csihi et al. (2022) conducted a comprehensive review combining the terms “catatonia” with various terms related to pregnancy and postpartum periods. The authors did not identify any prospective or controlled studies. Only a retrospective chart review, a small case series, and twenty individual case reports were identified. Based on this evidence, the authors concluded that catatonia during pregnancy presents similarly as in other contexts and could severely impact a mother's ability to care for and bond with her baby. Further, Caroff et al. (2022) suggested that the real significance of catatonia in schizophrenia lies in its ability to predict the disease's progression and treatment outcomes. While most modern antipsychotic drug trials broadly addressed schizophrenia without considering symptom-specific responses, early studies indicated catatonia might poorly respond to first-generation antipsychotics, with potential risks. This review highlighted the effectiveness of second-generation antipsychotics, especially clozapine, and electroconvulsive therapy (ECT) for schizophrenia patients with catatonic symptoms. In line with these findings, Saini et al. (2022) conducted a systematic review on clozapine treatment of catatonia and included 182 patients from cohort studies, case reports, or case series where at least one patient with catatonia was treated with clozapine. The findings suggested that over 80 % of reported catatonia patients experienced at least partial remission following treatment with clozapine. However, the study acknowledged the major limitations due to the reliance on case reports and small cohort studies, calling for future research using large healthcare databases to better understand outcomes in individuals treated with clozapine for catatonia. Although benzodiazepines, antipsychotics and ECT are the cornerstones of catatonia therapy, better understanding of subjective experience in catatonia patients could stimulate the development of disorder-specific psychotherapeutic modules for post-acute catatonia. Brandt et al. (2023) laid the initial groundwork by modifying, expanding, and validating the Northoff Scale for Subjective Experience in Catatonia (NSSC) in 33 catatonia patients according to ICD-11. The new version of NSSC showed good psychometric properties and hence, it is a useful tool for everyday clinical work to assess the subjective experience of catatonia patients.

## 7. Conclusion

After almost 150 years after the first clinical description of catatonia, the horizon of catatonia research is brighter than ever, illuminated by the latest findings from various international research groups. As we stand at this juncture, it is imperative to integrate these revelations into clinical practice while continuing to foster an environment of scientific curiosity. The quest to fully comprehend catatonia is far from over, but with each study published, we move one step closer to offering better care and hope to thousands worldwide. It was our great pleasure and honor to assemble this Special Issue with the close collaboration of eminent researchers and colleagues.

## CRediT authorship contribution statement

DH and GN: original idea, discussion of the articles, writing and manuscript revision.

## Declaration of competing interest

The authors have declared that there are no conflicts of interest in relation to the subject of this Guest Editorial.

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