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EDITED BY
Joni L Mihura,
University of Toledo, United States

REVIEWED BY
James Kleiger,
Independent Researcher, Bethesda, MD, United States
Smita Krishnaswamy,
Yale University, United States

*CORRESPONDENCE
Michele Poletti
michele.poletti@ausl.re.it

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Developmental perspectives on HiTOP psychosis superspectrum: Unveiling pitfalls and theoretical fallacies

Michele Poletti[1]*, Antonio Preti[2] and Andrea Raballo[3,4]

[1]Department of Mental Health and Pathological Addiction, Child and Adolescent Neuropsychiatry Service, Azienda USL-IRCCS di Reggio Emilia, Reggio Emilia, Italy, [2]Department of Neuroscience, University of Turin, Turin, Italy, [3]Chair of Psychiatry, Faculty of Biomedical Sciences, University of Southern Switzerland, Lugano, Switzerland, [4]Cantonal Sociopsychiatric Organisation, Mendrisio, Switzerland

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Introduction

The Hierarchical Taxonomy of Psychopathology (HiTOP) consortium recently defined the psychosis superspectrum (1) and focused on its nosology, etiology, and lifespan development (2). In the HiTOP bottom-up conceptual model, the psychosis superspectrum comprises two spectra - psychoticism and detachment - each consisting of traits and symptoms.

Psychoticism includes four traits (fantasy proneness, unusual experiences, unusual beliefs, and peculiarity), two core symptom components (reality distortion and disorganization), and two additional symptom components (mania and dissociation), which are provisionally included pending further investigation (3).

Detachment is composed of four traits (emotional detachment, anhedonia, social withdrawal, and romantic disinterest) and two symptom components (inexpressivity and avolition).

The HiTOP model of psychosis is not without critical points, particularly regarding the two-spectrum structure of the superspectrum (4); for example, a meta-analysis found that detachment was consistent with general psychopathology rather than with the negative dimension of psychosis (4, 5) and in a recent developmentally-informed HiTOP model on a symptom-based, large-scale study with youth (6), psychotic symptoms were included in the externalizing spectrum rather than each forming separate factors.

Acknowledging these limitations (4–6) - which warrant further empirical investigation and model refinement (7) - this contribution focuses on the dimensional nuances of the psychosis superspectrum as articulated by the HiTOP consortium (1), outlining a possible longitudinal development of psychosis across developmental and clinical stages (2). This developmental perspective presents potential shortcomings and misconceptions that require further examination to guide future empirical research, inspired by the HiTOP model, on psychosis.

Developmental features of psychoticism

A first oversimplification may lie in the overall conceptualization of the developmental trajectory leading to the emergence of psychoticism, i.e. its ontogenesis. HiTOP authors (2) stated that individual differences in psychoticism are apparent by middle school, citing a study on childhood psychosis (8). However, it is well known that even childhood-onset schizophrenia does not begin with the early (or very early) appearance of diagnostic psychotic symptoms (9). Therefore, the low prevalent condition of childhood psychosis may not be the optimal model for tracing the earliest emergence of individual differences in psychoticism. Similarly, the moderate rank-order stability stated for traits of psychoticism from ages 7 to 12 (2) is based on a longitudinal study of the offspring of individuals with schizophrenia or bipolar disorder (10). Familial high-risk vulnerability is a questionable model for generalizing the hypothesis of developmental stability in psychoticism to the broader general population, which, by definition, is presumably not at familial-genetic risk (11).

These examples illustrate a tendency toward oversimplification in describing the development of the psychotic superspectrum, i.e. its ontogenetic unfolding along developmental stages. This is particularly evident in the conflation of more stable features - putative traits of psychoticism - with more transient features - symptoms of psychoticism, such as psychotic-like experiences. In contrast, a more realistic and clinically faithful developmental perspective suggests that vulnerability traits for psychoticism may already emerge in childhood as a consequence of neurobiological constraints related to genetic and/or environmental risk factors (11, 12). These traits may phenotypically manifest as, for example, childhood oddity or intersubjective difficulties with peers, which could later evolve and structure into avoidant or cluster A-like (paranoid, schizotypal, schizoid) personality traits, laying the groundwork for the eventual emergence of reality-distortion symptoms (13).

In this regard, also the statement that the Clinical High-Risk for Psychosis (CHR-P) stage serves as a bridging link - or intermediate stage - between childhood subclinical manifestations of psychoticism and full-blown reality distortion (hallucinations and delusions) in early adulthood (2) represents another oversimplification, despite its apparent conceptual linearity. As highlighted in the clinical staging model, the prodromal stage indexed by CHR-P is characterized by the first appearance of psychotic manifestations in the form of attenuated symptoms (14). In contrast, childhood premorbid stages may only involve endophenotypic features that are relatively nonspecific as prognostic precursors of psychosis (12).

Contemporary conceptualizations of psychosis, shaped by its prevailing reduction to positive symptoms, could tend to overemphasize delusional-hallucinatory features in risk assessment and transition prediction. This often comes at the expense of attention to negative symptoms, assessed but poorly considered in specific instruments as CAARMS and SIPS while determining the putative psychometric transition to psychosis in CHR-P individuals (15). Only about one-third of CHR-P individuals undergo a psychometric transition to psychosis, suggesting that prognostic accuracy based solely on the symptomatic level of attenuated positive psychotic symptoms in adolescence is significantly lower than what might be achieved by also considering levels of negative symptoms (15). Therefore, the CHR-P construct could only partially fit to study prodromal or intermediates stages of psychoticism according to the HiTOP model.

Moreover, in examining the psychoticism spectrum, it is essential to distinguish traits from symptoms, as they likely differ in ontogenesis, timing of phenotypic onset, longitudinal course, neurobiological determinants, and prognostic significance (16, 17). Finally, further refinements of the HiTOP model should be more selective in the supporting evidence used and more rigorous in articulating a clinically coherent interpretation. Childhood-onset schizophrenia, familial high-risk, CHR-P, and psychotic-like experiences are not interchangeable constructs, nor are they equally central proxies for the ontogenesis of psychoticism across developmental and clinical stages.

Developmental features of detachment

Keeping in mind the critiques regarding the loading of detachment onto the negative dimension of the psychosis superspectrum (5), a similar reasoning can be applied to the development of detachment itself, whose accurate diagnosis presents greater clinical challenges than that of psychoticism - particularly in younger individuals.

Recognizing the early roots of detachment is more complex than for psychoticism. Detachment may originate from early disruptions in intersubjective attunement with peers, beginning in childhood. These disruptions can later manifest as social anhedonia when peer relationships become central to identity formation during adolescence (18). Therefore, detachment typically precedes the symptomatic features of psychoticism, such as reality distortion (19). Detecting detachment before adolescence - and distinguishing it from the secondary effects of affective symptoms, which belong to the emotional dysfunction superspectrum (20) - is especially difficult and often relies on observable behaviors such as social withdrawal. In this perspective EPA guidelines on assessment of negative symptoms (21) clearly encouraged that clinical observation of social withdrawal should also focus on inexpressivity and should be accompanied by the assessment of subjective experience of social amotivation, not limited to self-report as in the HiTOP model.

Developmental features of cognition

Although the shortcoming of not including cognitive impairment in the HiTOP model of psychosis, it has been suggested that cognitive deficits within the psychosis superspectrum emerge more than a decade before the onset of clinically significant psychotic symptoms (2). From a neurodevelopmental perspective on the ontogenesis of psychosis,

traits of psychoticism and detachment may be present from childhood but typically become more pronounced and structured during adolescence, forming the foundation for related symptoms. According to this developmental framework, cognitive impairment should be understood as a developmental lag due to neurodevelopmental constraints rather than a decline from a previously normative developmental trajectory, particularly when compared to healthy age-matched controls (22).

Thus, in individuals with a presumed transgenerational and neurobiological vulnerability (e.g., schizotaxia), nonspecific cognitive symptoms may represent the earliest phenotypic premorbid expressions (12), particularly in the neurocognitive domain of motor coordination (23–25).

Conclusions

The HiTOP model's depiction of the psychosis superspectrum through the constructs of psychoticism and detachment offers a compelling framework (1). If the HiTOP perspective is robust, though not yet conclusive (4–6), in capturing the structure of psychosis once it has manifested, its dynamic characterization across development - considering developmental phases (childhood, adolescence, adulthood) and clinical stages (premorbid phase, prodromal phase, clinical phase) - remains preliminary (2) requiring further refinement.

To address the ontogenesis of psychosis while adhering to the HiTOP model's framework, particular attention must be paid to the selected populations and measurement tools used when comparing them to healthy controls. As previously noted, childhood-onset schizophrenia, familial high-risk, clinical high-risk, and psychotic-like experiences are not interchangeable constructs for assessing psychosis risk across development and clinical stages.

Finally, it is important to emphasize that while psychosis appears to be a transdiagnostic construct, the ontogenesis of the process culminating in its phenotypic manifestation is not necessarily transdiagnostic - that is, it may not be analogous across different mental disorders. For example, Self-disorders affecting the Minimal or Basic Self, in terms of anomalous subjective experiences, are specific to the schizophrenia spectrum and underlie the emergence of psychotic manifestations (26).

Concluding, to advance and deepen our understanding of developmental unfolding of psychoticism and detachment, future longitudinal studies must employ tailored instruments and include a range of pre-test risk populations.

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References

1. Kotov R, Jonas KG, Carpenter WT, Dretsch MN, Eaton NR, Forbes MK, et al. Validity and utility of Hierarchical Taxonomy of Psychopathology (HiTOP): I. Psychosis superspectrum. *World Psychiatry*. (2020) 19:151–72. doi: 10.1002/wps.20730

2. Jonas KG, Cannon TD, Docherty AR, Dwyer D, Gur RC, Gur RE, et al. Psychosis superspectrum I: Nosology, etiology, and lifespan development. *Mol Psychiatry*. (2024) 29:1005–19. doi: 10.1038/s41380-023-02388-2

3. Kotov R, Krueger RF, Watson D, Achenbach TM, Althoff RR, Bagby RM, et al. The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *J Abnorm Psychol*. (2017) 126:454–77. doi: 10.1037/abn0000258

4. Mihura JL, Boyette LL, Görner KJ, Kleiger JH, Jowers CE, Ales F. Improving dependability in science: A critique on the psychometric qualities of the HiTOP psychosis superspectrum. *Schizophr Res*. (2024) 270:433–40. doi: 10.1016/j.schres.2024.06.051

5. Ringwald WR, Forbes MK, Wright AGC. Meta-analysis of structural evidence for the Hierarchical Taxonomy of Psychopathology (HiTOP) model. *Psychol Med*. (2023) 53:533–46. doi: 10.1017/S0033291721001902

6. Forbes MK, Watts AL, Twose M, Barrett A, Hudson JL, Lyneham HJ, et al. A hierarchical model of the symptom-level structure of psychopathology in youth. *Clin Psychol Sci*. (2024) 13:278–300. doi: 10.1177/21677026241257852

7. Forbes MK, Ringwald WR, Allen T, Cicero DC, Clark LA, DeYoung CG, et al. Principles and procedures for revising the hierarchical taxonomy of psychopathology. *J Psychopathol Clin Sci*. (2024) 133:4–19. doi: 10.1037/abn0000886

8. Bettes BA, Walker E. Positive and negative symptoms in psychotic and other psychiatrically disturbed children. *J Child Psychol Psychiatry*. (1987) 28:555–68. doi: 10.1111/j.1469-7610.1987.tb00223.x

9. Driver DI, Thomas S, Gogtay N, Rapoport JL. Childhood-onset schizophrenia and early-onset schizophrenia spectrum disorders: an update. *Child Adolesc Psychiatr Clin N Am*. (2020) 29:71–90. doi: 10.1016/j.chc.2019.08.017

10. Gregersen M, Møllegaard Jepsen JR, Rohd SB, Søndergaard A, Brandt JM, Ellersgaard D, et al. Developmental pathways and clinical outcomes of early childhood psychotic experiences in preadolescent children at familial high risk of schizophrenia or bipolar disorder: A prospective, longitudinal cohort study - the Danish high risk and resilience study, VIA 11. *Am J Psychiatry*. (2022) 179:628–39. doi: 10.1176/appi.ajp.21101076

11. Raballo A, Poletti M, Preti A. Applying transgenerational scientific evidence to the next wave of early identification strategies for psychopathological risk-transdiagnostic, developmental, and personalized. *JAMA Psychiatry*. (2021) 78:1067–8. doi: 10.1001/jamapsychiatry.2021.1901

12. Poletti M, Raballo A. Developmental psychotic risk: toward a neurodevelopmentally informed staging of vulnerability to psychosis. *Harv Rev Psychiatry*. (2020) 28:271–8. doi: 10.1097/HRP.0000000000000266

13. Verbeke L, De Clercq B. Integrating oddity traits in a dimensional model for personality pathology precursors. *J Abnorm Psychol*. (2014) 123:598–612. doi: 10.1037/a0037166

14. Tsuang MT, Van Os J, Tandon R, Barch DM, Bustillo J, Gaebel W, et al. Attenuated psychosis syndrome in DSM-5. *Schizophr Res*. (2013) 150:31–5. doi: 10.1016/j.schres.2013.05.004

15. Raballo A, Poletti M, Preti A. Attenuated psychosis syndrome or pharmacologically attenuated first-episode psychosis?: an undesirably widespread confounder. *JAMA Psychiatry*. (2020) 77:1213–4. doi: 10.1001/jamapsychiatry.2020.1634

16. Cicero DC, Jonas KG, Li K, Perlman G, Kotov R. Common taxonomy of traits and symptoms: linking schizophrenia symptoms, schizotypy, and normal personality. *Schizophr Bull*. (2019) 45:1336–48. doi: 10.1093/schbul/sbz005

17. Moriyama TS, van Os J, Gadelha A, Pan PM, Salum GA, Manfro GG, et al. Differences between self-reported psychotic experiences, clinically relevant psychotic experiences, and attenuated psychotic symptoms in the general population. *Front Psychiatry*. (2019) 10:782. doi: 10.3389/fpsyt.2019.00782

18. Poletti M, Gebhardt E, Iannuzzi V, Tortorella A, Raballo A. Social dysfunction in preclinical, at risk stages of psychosis: A developmental view. *Schizophr Res*. (2019) 206:456–7. doi: 10.1016/j.schres.2018.11.020

19. Jones HJ, Stergiakouli E, Tansey KE, Hubbard L, Heron J, Cannon M, et al. Phenotypic manifestation of genetic risk for schizophrenia during adolescence in the general population. *JAMA Psychiatry*. (2016) 73:221–8. doi: 10.1001/jamapsychiatry.2015.3058

20. Watson D, Levin-Aspenson HF, Waszczuk MA, Conway CC, Dalgleish T, Dretsch MN, et al. Validity and utility of Hierarchical Taxonomy of Psychopathology (HiTOP): III. Emotional dysfunction superspectrum. *World Psychiatry*. (2022) 21:26–54. doi: 10.1002/wps.20943

21. Galderisi S, Mucci A, Dollfus S, Nordentoft M, Falkai P, Kaiser S, et al. EPA guidance on assessment of negative symptoms in schizophrenia. *Eur Psychiatry*. (2021) 64:e23. doi: 10.1192/j.eurpsy.2021.11

22. Poletti M, Raballo A. Clinical implications of slower cognitive growth in the psychosis spectrum. *JAMA Psychiatry*. (2018) 75:755–6. doi: 10.1001/jamapsychiatry.2018.1218

23. Burton BK, Hjorthøj C, Jepsen JR, Thorup A, Nordentoft M, Plessen KJ. Research Review: Do motor deficits during development represent an endophenotype for schizophrenia? A meta-analysis. *J Child Psychol Psychiatry*. (2016) 57:446–56. doi: 10.1111/jcpp.12479

24. Poletti M, Gebhardt E, Kvande MN, Ford J, Raballo A. Motor impairment and developmental psychotic risk: connecting the dots and narrowing the pathophysiological gap. *Schizophr Bull*. (2019) 45:503–8. doi: 10.1093/schbul/sby100

25. Burton BK, Krantz MF, Skovgaard LT, Brandt JM, Gregersen M, Søndergaard A, et al. Impaired motor development in children with familial high risk of schizophrenia or bipolar disorder and the association with psychotic experiences: a 4-year Danish observational follow-up study. *Lancet Psychiatry*. (2023) 10:108–18. doi: 10.1016/S2215-0366(22)00402-3

26. Raballo A, Poletti M, Preti A, Parnas J. The self in the spectrum: A meta-analysis of the evidence linking basic self-disorders and schizophrenia. *Schizophr Bull*. (2021) 47:1007–17. doi: 10.1093/schbul/sbaa201