

Early Schizotypy and Risk: The Need for Integrating Developmental Dynamics

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Important strides toward understanding the nature of risk for schizophrenia spectrum and other psychotic disorders (SSPDs) are currently being taken in the field of schizotypy research. In this issue O'Hare et al¹ elegantly contribute a further piece to the developmental puzzle by detailing how schizotypy between ages 11–12 years significantly relates to early life cumulative risk. Based on administrative records, the authors identify a group of children (“*true schizotypy*” group, 5.7% of the total sample) whom report high levels of cognitive disorganization, impulsive non-conformity, introversion, and self-other disturbance, but low levels of unusual perceptual experiences. The methodology employed through administrative records may inspire developments in early identification strategies for youths exposed to cumulative risk experiences. The low levels of unusual perceptual experiences in the “*true schizotypy*” group may appear surprising at first, yet recent research on clinical high-risk states finds that unusual perceptual experiences are only predictive of risk for conversion to SSPDs after 16 years of age.² Therefore longitudinal follow-up of the “*true schizotypy*” measure sampled by O'Hare and colleagues is needed to validate early risk indicators, and assess whether: (1) “*true schizotypy*” behaves as a stable trait measure over time; (2) a more significant proportion of individuals in the “*true schizotypy*” group develop SSPDs at later developmental stages; (3) whether youths potentially change group allocation, for example, evolve from a “*true schizotypy*” group membership to “no risk” group membership, or vice versa; and (4) whether specific types of risk experiences more significantly predict the emergence of SSPDs and/or other forms of psychopathology in this sample.

Interestingly, O'Hare and colleagues observe that cumulative risk experiences, which include potentially direct and heavily trauma-laden experiences such as “childhood crime victimization”, “childhood protection contact”, and “parental death”, as well as indirect factors such as “neighborhood crime rate”, “socioeconomic deprivation” and “financial stress”, is most prevalent for the “*true schizotypy*” group. Whilst these results are interpreted in the context of the developmental psychopathology of schizotypy, they may also be further informed by the latent vulnerability hypothesis linking childhood maltreatment to cerebral as well as social trajectories augmenting risk for psychopathology.³ The latent vulnerability hypothesis argues for a risk trajectory divided into two arms: a neurodevelopmental arm and a social thinning arm. First in terms of brain maturation, early interpersonal stress factors, which may include direct as well as indirect trauma, abuse, neglect, and environmental stress, can trigger a neurodevelopmental cascade increasing risk to psychopathology. This view is not new and resonates with previous research linking early stress and neurodevelopment to risk and resilience for SSPDs.⁴ Interestingly, the latent vulnerability theory further argues that the consequences of early trauma are not only neurodevelopmental in nature, but can in combination dynamically sculpt away the youth's social resources in “*social thinning*” fashion, that is, through an increasingly thinner and more fragile social network of interpersonal relationships and social bonds that bread security, resources for emotional, and relational needs, and belongingness. This second arm resonates with risk and resilience research, and may help us connect neurodevelopmental observations to the real impact of social deprivation, especially on a personality trait such

as schizotypy. In our view, this orients risk research on the negative and disorganization dimensions of schizotypy in childhood, because these dimensions measure early social withdrawal, blunted affect, and anhedonia, all of which may represent more potent early indicators of vulnerability in comparison to childhood unusual perceptual aberrations which yield poor predictive later conversion to SSPDs.⁵ Furthermore, the consideration of protective factors appears fundamental, yet require a shift to novel frameworks, integrating knowledge gain from latent trait models evolving to complex systems models.

Along these lines, the few longitudinal studies specifically investigating the negative dimension of schizotypy in adolescent samples appear to provide the most reliable early signals of vulnerability to SSPDs. Examining transition from clinical high-risk to SSPDs, Flückiger and colleagues⁶ found that amongst different schizotypy self-report measures, physical anhedonia significantly contributed to predict conversion to SSPDs. In terms of cerebral development, Derome and colleagues^{7,8} provided longitudinal evidence that atypical maturation trajectories of cortical thickness and key subcortical structures relate to negative schizotypy expression during adolescence, mirroring some of the alterations observed at more advanced stages of clinical continuum of SSPDs. While such reports draw from neuroscientific, clinical and epidemiological methodologies to provide increasingly earlier clues for vulnerability to SSPDs, a coherent framework targeting specific mechanisms binding the genetic, neuronal, psychological, and interpersonal levels are still needed to provide the next breakthroughs in our understanding the developmental psychopathology leading to SSPDs.

In order to articulate a coherent and integrative developmental framework in schizotypy research, one which may concretely assist in early prevention strategies, studies may need to more radically include measures tapping into dynamic psychological processes in addition to symptom-based and life event sampling methods. Unfortunately, traditional self-report measures are prone to biases, temporal drift, regression to the mean, and other issues affecting their ability to predict and explain future outcomes. Another methodological problem lies in measures that conflate, amongst others, trait expression, symptom expression, and psychological processes. In the field of risk for SSPDs, one of the best examples of such conflation can be observed when “dissociation” is under scrutiny. Dissociation is measured through a variety of instruments that prompt reports of intrusions into consciousness associated to alterations in continuity and sense of self. These tools alternatively evaluate an individual’s propensity to experience dissociation (trait dissociation), to report episodes of dissociation (such as in peritraumatic dissociation), to experience specific types of dissociative experiences (such as depersonalization and derealization), or to indicate the presence of symptoms which can entail a varying degree of dissociation (eg, perceptual aberrations, paranoid beliefs,

thought control experiences, and others).⁹ Other reports will provide experimental procedures that may induce dissociative experiences, and relate these to schizotypy measures.¹⁰ Unfortunately, the developmental psychopathology research on dissociative phenomena is itself in a state of quasi-dissociation: on one side, researchers studying risk for SSPDs have moved from adult samples to young adult, adolescent, and now samples of children enriched for psychosis risk, while researchers investigating dissociation in relation to early trauma follow infants, growing into children, adolescents, and young adults with little attention to schizotypy or psychosis-proneness indicators. While O’Hare and colleagues provide an opportunity for a much-needed interchange between these research paths, methodologies that combine both fields of interest have the opportunity to dynamically link exposure to risk experiences to their “neurodevelopment” as well as “social thinning” indicators as they prospectively evolve. Such an integrative strategy is employed in autism spectrum research, and carries the potential to better inform which early prevention strategies may incur the largest defeat in the progression of risk to clinical states of SSPDs.

To conclude, the work of O’Hare et al, directs our attention toward new challenges, entailing a shift from static to dynamic views, from symptom-based to mechanism-based models, as well as from DMS/ICD labels to contextual precision diagnosis across stages of emerging psychopathology. The field of schizotypy research is well equipped to contribute to such knowledge, provided that the tools examining the early negative and disorganization facets of schizotypy can be made sensitive to the developmental dynamics at play within a risk and resilience framework, enabled to model mental illness, and health in a prospective fashion.

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