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Schizotypy From a Developmental Perspective

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The schizotypy construct focuses attention on the liability to develop schizophrenia-spectrum disorders, yet traditionally, the schizotypy models have put more emphasis on stress-vulnerability interactions rather than developmental dynamics of emerging risk for psychopathology. Indeed, developmental accounts of this emerging personality trait have rarely been explicitly formulated. In this position article, we wish to convey some of the basic developmental tenets of schizotypy, and how they can inform high-risk research. Firstly, we tackle the state vs trait issue to outline the possible relationship between high-risk states and trait schizotypy. Second, we review the evidence suggesting that the consolidation of schizotypy, encompassing its 3 main dimensions, could be considered as a developmental mediator between very early risk factors and transition into high-risk states. Importantly, developmental dynamics between endophenotypes, as well as transactional and epigenetics mechanisms should enter modern conceptualizations of schizotypy. Finally, we present a developmental psychopathology perspective of schizotypy sensitive to both the multifinality and equifinality of schizophrenia-spectrum disorders. We conclude that schizotypy represents a crucial construct in a fully-developmental study of schizophrenia-spectrum disorders.

Key words: high-risk/psychosis/schizophrenia/personality/developmental/psychopathology

Introduction

Researchers interested in schizotypy generally seek to examine a personality trait conferring liability to develop schizophrenia. However, 50 years after schizotypy’s first formal definition as a liability trait, which implicitly designates a developmental dimension to schizophrenia, we note that the field of schizotypy research has seldom explicitly formulated a developmental framework to conduct investigations on this putatively stable, continuous trait signaling increased probability to the unfolding of schizophrenia-spectrum disorders. Instead, developmental research has proceeded to focus on the risk of conversion to schizophrenia, by employing a high-risk state approach, focusing on transient, time-dependent shifts in functioning, or exacerbation of subclinical symptoms. Today, prodromal syndromes and basic symptoms represent the key preventive identification targets of the high-risk state approach. Epidemiological research further contributed by investigating selective schizotypal experiences during childhood and adolescence, mostly from the positive dimension, and examining their association to the unfolding of psychotic disorders during adulthood. Such psychotic-like experiences (PLEs) have been found to carry predictive value for the future development of schizophrenia spectrum disorders, although their specificity is rather low (see Debbané et al in this supplemental issue).

With regards to transition to schizophrenia, early prevention research has focused on identifying specific at-risk states that could discriminate between converters and nonconverters before the onset of the pathology. Although the early identification procedures are making significant progress, these state indicators tend to lose their predictive power when evaluated more than 36 months before putative onset of the disorder. On the other hand, the pioneering longitudinal Chapman studies have illustrated how schizotypy traits too can yield predictive power, and this over the decades of adolescence and young adulthood. These studies and others support dimensional
claims that schizotypy traits are etiologically connected to the schizophrenia-spectrum, and contain informative value to define populations at risk for these disorders. However, as it would be expected, schizotypy traits measured in nonclinical populations yield lower predictive power than clinical state indicators typically assessed in help-seeking individuals. This has sometimes been interpreted as schizotypy being a poor risk indicator, with lower sensitivity and specificity to high-risk states. Also, the developmental mechanisms linking schizotypy to clinical expression in the schizophrenia-spectrum remain to be examined longitudinally.

It thus appears that in developmental high-risk research, the different phenomena contained in the construct of schizotypy have been split and scattered over different investigation fields, yielding crucial evidence to the importance of prepsychotic states. By the same token, the different high-risk approaches are sometimes susceptible to yield piece-meal accounts that can become difficult to integrate. Furthermore, states can be predictive of future pathological development, but their articulation with schizotypal traits and their own predictive value has been understudied. The main objective of this position article will be to articulate the basic elements of a developmental model of schizotypy, and situate this model within the current research on high risk for schizophrenia. To achieve this objective, each of 3 sections seeks to provide what appear to us as critical elements in the developmental cascade of schizophrenia-spectrum disorders. The first section aims to tackle the issue of the coexistence between “clinical” or “schizotypy states and trait schizotypy, and provide evidence supporting the probable association between high-risk states and schizotypal traits during development. In the second section, we highlight how early schizotypal traits may indeed constitute a developmental mediator between endophenotypes and increased risk of conversion to adult schizophrenia-spectrum disorders. In the final section, we present a developmental psychopathology perspective of schizotypy sensitive to both the multifinality of schizotypal traits (associated to a number of different psychopathological outcomes), as well as the equifinality of schizophrenia-spectrum disorders (how different developmental interactions may lead to this class of diagnosis). Together these sections sketch what could be called a preliminary developmental framework for the schizotypy construct.

State vs Trait Schizotypy?
The first section aims to deal with the conceptual challenge arisen by the current coexistence of 2 related yet differential strands of research. On the one hand, the “prodromal” or clinical high-risk approach puts by definition a marked emphasis in the detection of at risk mental states, which are very similar to the phenomenology of overt psychosis even if they differ in dimensions such as severity, frequency, or insight about their nature. On the other hand, schizotypy research conducted in the domain of individual differences refers to it as a stable personality trait; its operationalization is not conditioned by the endeavor of detecting “true” at risk individuals for transitioning to psychosis. At the same time, both refer to “subclinical” psychosis and index risk for it, so not surprisingly some confusion seems to be present in the literature in regards to the trait or state nature of schizotypy.

As Claridge and Davis\textsuperscript{11} pointed out, the dimensional model of mental disorder takes account of the 2 “universes of discourse” [as Foulds (1965) put it] that are necessary to comprehensively describe abnormal behavior: the “trait-like personality” and the “symptom-state illness” aspects of disorder. A critical issue is that schizotypy (or any other) personality trait has a dual nature, as it defines both enduring psychological characteristics that vary along a continuum in the population, and, at the same time, a varying predisposition to psychotic disorder. Thus, there is not a clear-cut dissociation between the domains of personality and illness, between traits and states, although this is sometimes overlooked as personality tends to be conceived as a highly rigid, nondynamic construct. At the same time, it is obvious that an individual with high trait schizotypy, for instance someone who has usually felt ego-syntonic peculiar body sensations with good psychosocial functioning, is not the same as an individual who is experiencing a “schizotypal” or high-risk mental state characterized by perplexity and distress over a dismorphic concern that changes his/her usual behavior. Such state reflects a temporary fluctuation that derives from the personality trait; as Claridge and Davis\textsuperscript{11} put it, it denotes the trait “in action.” From a quasidimensional account, various latent liability models of schizophrenia have also postulated that the genetic susceptibility to schizophrenia produces a hypothetic latent liability trait that can become expressed as nonobservable endophenotypic deviances, schizotypy personality traits and/or clinical states, or schizophrenia spectrum disorders (see Meehl\textsuperscript{12}, Holzman’s latent trait model,\textsuperscript{13,14} and Lenzenweger\textsuperscript{15}). These models thus also intrinsically assume a developmental life-span perspective and articulate the relationship between trait and state manifestations. However, unlike the so-called fully-dimensional perspective referred to above, these models consider that despite there being phenomenological continuity between traits and symptoms, the latent liability (eg the genetic susceptibility) is not continuously distributed in the population, which yields a group of liable individuals (a taxon) which is qualitatively different from the rest (ie structural discontinuity). The investigation of the structure of schizotypy with taxometric analyses is a convulsive topic given its methodological complexity\textsuperscript{16} and has raised mixed findings, with some studies supporting\textsuperscript{17,18} and others not\textsuperscript{19-21} the existence of a schizotypy or schizotypal personality disorder taxon.

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There is recent evidence supporting the association between schizotypy and high-risk states in early adulthood and during development. In a recent longitudinal study spanning over a 30-year interval, Rössler et al examined the latent-state and trait structure of subclinical schizotypal signs (reduced close relationships, odd beliefs, ideas of reference and suspicious/paranoid ideation) collected over 7 semistructured interviews in subjects aged 20 years at the start of the study. They also examined the latent state and trait structure of subclinical schizophrenia nuclear symptoms (thought insertion, thought broadcasting, thought control and hearing voices), phenomena that overlap with positive schizotypal experiences. Their study analyses demonstrate the importance of schizotypal traits over 3 decades of development, during which it consistently accounted for more than half of the variance of subclinical psychotic expression. Importantly, in the high-risk age interval for developing schizophrenia, 22–30 years old, latent schizotypal-trait scores explained significantly more variance than its state counterpart. This study thus supports the hypothesis by which psychotic expression varies as a function of schizotypal traits, but that fluctuating and occasion-specific states can also contribute to its expression. The results suggest that state schizotypy explains more variance than its trait counterpart at 20/21 and 49/50 years of age. It is unclear whether developmental transitions at these ages could contribute to this finding. Furthermore, the study reveals that other personality dimensions influence both trait and state schizotypy. Low sense of mastery, depressiveness and poor self-esteem significantly associated with schizotypal signs. Employment distress, intimate relationship problems and drug-use were found to be the main time-dependent state influences to schizotypal expression. In essence, this report illustrates the multifactorial development of psychotic expression, and how schizotypal traits and states contribute to the unfolding of risk for schizophrenia-spectrum disorders.

Schizotypy: A Developmental Vehicle Towards Emerging Psychopathology

Ever since abandoning the single gene hypothesis of schizophrenia, many scientists focusing on the genetic basis of the disease have lead investigations consistent with a polygenic, endophenotype approach. Endophenotypes of schizophrenia, often identified at the neurophysiological or neurocognitive levels, consist in measurable variables that cannot be assessed at the “visible” phenotypic level, whose expression is closely tied to risk-conferring genes (risk genotype). Individual measures on these endophenotypes typically reveal abnormalities in samples of patients diagnosed with schizophrenia, but also in family members carrying the genotype underlying the impairments. It is thought that each endophenotype accounts for a small percentage of risk to develop schizophrenia, and that put together, an ensemble of endophenotypes could increase the risk for an individual to convert to schizophrenia.

Recent advances in the field of schizotypy research have shown that endophenotypes typically linked to schizophrenia are also associated with the expression of schizotypy in nonclinical youth samples, and adults from general population samples (for a recent review, see Ettinger et al). Of note, this literature has shown that, consistent with the multidimensional nature of schizotypy (and schizophrenia), there are differential associations between specific schizotypy dimensions and certain endophenotypic deviances. Importantly from a developmental standpoint, schizotypy has been found to be associated with endophenotypes and biomarkers whose dimensions can already be assessed during infancy or childhood (eg minor physical anomalies) or relate to basic functions that typically reach full maturational childhood neurocognitive development. In a study examining neurocognitive and neurodevelopmental correlates of adolescent schizotypy in a sample 270 adolescents, recruited from the regular school system and reporting elevated scores on the Chapman schizotypy scales, well recognized schizophrenia endophenotypes and biomarkers were found to correlate with executive functions and dermatoglyphic abnormalities, respectively. Similarly, in a subsequent study investigating schizotypal symptoms in adolescents at familial risk for developing schizophrenia, minor physical anomalies, fine motor dyscoordination and executive function impairments were associated with negative schizotypy scores. Visual backward masking, for which adult-like performance is generally acquired by the beginning of adolescence, represents another schizophrenia endophenotype found to be associated with schizotypal expression in youths. More specifically, deterioration in visual backward masking was found to be associated with schizotypy in a sample of late adolescence/early adulthood students, specifically with cognitive disorganization. In a similar vein, the sensorimotor gating function of prepulse inhibition, thought to reach adult-like levels around 8 years old, has not only been associated with gating deficits in schizophrenia, but has also been shown to be impaired in nonclinical youths at high genetic risk for developing schizophrenia. This wealth of reports has been collated in scientific reviews, emphasizing significant superposition in endophenotypical impairments when drawing comparison between nonclinical youth samples recruited on the basis of schizotypal expression to samples of individuals diagnosed with schizophrenia. Most importantly perhaps, the developmental evidence of such associations can be observed during childhood and adolescence. The growing number of studies thus makes it difficult to ignore the commonalities between schizophrenia endophenotypes and early expression of trait schizotypy (also in subjects without familial risk), which continue to be observed at the neurocognitive, neurophysiological, and cerebral levels all along development.
Another key area of atypical development associated with trait schizotypy concern the mentalizing functions, that is those social cognitive operations that enable oneself to employ knowledge on the mental states of self and others in order to understand social interactions. Cognitive development studies with adolescents and young adults show that mentalizing functions continue to mature during the teenage years and well into the early 20s. At the cerebral level, social cognitive specialization may indeed rely on more distal and complex internetwork collaboration, typically refined during adolescent brain maturation and specialization. To date, 2 functional neuroimaging studies suggest that schizotypal expression during adolescence is significantly associated with atypical brain activation patterns in social cognitive tasks requiring efficient self-other discrimination (reality-monitoring and self-appraisal tasks). Interestingly, adolescents with different risk etiologies (genetic vs clinical risk) may share neural markers associated with atypical social cognitive functions. In a recent fMRI study on perspective-taking, comparable patterns of atypical neural activation during a 1p-3p perspective-taking task were observed for both typical adolescents reporting transient auditory verbal hallucinations and adolescents at ultra-high risk for schizophrenia because of 22q11.2 deletion syndrome. Together, these studies would suggest that high schizotypy appears to accompany the different endophenotypical risk markers during early risk for schizophrenia spectrum disorders. In other words, and from a clinical standpoint, the significant association between valid endophenotypes and early high schizotypy may signify the establishment of early risk progress for subclinical schizotypal youths. This motivates further inquiry into the potential predictive value of an endophenotype/schizotypy trait association, at the level of the individual, for conversion to psychotic spectrum disorders. At the same time, these findings also support the validity of schizotypy as the continuous personality dimension characterized by a specific neurobiological, cognitive, and socioaffective organization that is both expressed at the level of healthy individual differences and underlies the predisposition to psychosis. Most importantly perhaps, the consolidation of schizotypy may confer a “developmental vehicle” toward psychopathology. In such a developmental account, a number of endophenotypes would consistently influence the emerging organization of personality, and each partly consolidating schizotypy. Then, towards middle childhood and through adolescence, significant metacognitive, interpersonal and social experiences may be preferred and consistently selected by the individual as a result of schizotypy (selective appraisals of perceptual aberrations, overt eccentricity in speech and behavior, decreasing social contact, etc.), thereby initiating developmental transactional influences that may augment the risk for psychopathology. As such, schizotypy may be conceived as a developmental vehicle between early endophenotypes and biomarkers, to selective transactional processes, towards adult risk for psychopathology.

To illustrate the underlying developmental interactions that position schizotypy as a developmental vehicle, we provide a figure based on the traditional account of schizotypy (Meehl) as framed by Lenzenweger, and sketch the additional developmental interactions setting the “vehicle” of schizotypy in motion during development. Although implicitly developmental in nature, the traditional model is typically illustrated in linear fashion (see left side), starting from the genetic component (the “schizogene” in Meehl’s original formulation), through the schizotaxic underlying level, toward the manifest expression of schizotypy. The model accounts for social learning opportunities as well as psychosocial stress factors and polygenic potentiators (PGP) that may influence the development of schizotypy, and admits to interactions between schizotypy and these stressors during maturation. The traditional model could be made more explicitly developmental, by including 3 possible developmental dynamics within the framework (see right side of figure 1): (1) Firstly, developmental research suggests the utility of assessing multiple endophenotypes in predicting onset of illness. Indeed cooccurring endophenotypes may signal higher risk to develop clinically relevant symptoms, in part, through interactions during development that would potentiate the genetic loading of risk for schizophrenia. These interactions are represented in red in figure 1, and may partly underlie recently evidenced interactions between schizotypy dimensions during adolescence. (2) Transactional processes during development could be captured by emphasizing possible relationships between schizotypy and both psychosocial learning and stress. Transactional models suggest that individual characteristics such as traits are not only influenced by contextual dimensions, but themselves contribute to shaping the context. These processes are captured in the blue arrows in figure 1. A relevant example for schizotypy research could be gathered from the transactional model of social withdrawal. (3) Finally, most environmental risk factors identified in relation to schizophrenia can also potentially trigger epigenetic effects. Therefore, the strictly unidirectional relationship between risk genes and psychosis could also include epigenetic mechanisms as potential mediators of gene expression, as captured by the green arrows in figure 1.

In sum, when considering endophenotype research from the perspective of developmental schizotypy, 3 critical observations come into consideration. Firstly, many of the endophenotypes associated with schizophrenia concern dimensions and functions already established during childhood. In other words, some endophenotypes of schizophrenia are fully manifest long before the first signs of the at-risk states make their way into the clinical picture. Secondly, endophenotypes associated with...
patients with schizophrenia and their family members are also associated with psychometrically defined measures of schizotypy in nonclinical youth samples. In a recent review, Ettinger et al. provide some of the overlapping behavioral and neurobiological domains of impairment between schizophrenia and schizotypy, suggesting some continuity at the endophenotypic level between schizotypy and full-blown schizophrenia. Finally, social cognitive impairments associated with schizophrenia also relate to schizotypal expression during adolescence. When this triad of observations is considered conjointly with the predictive value of schizotypy for the development of schizophrenia spectrum disorders, it positions this risk trait as a potent transactional agent, a developmental vehicle linking early endophenotypes to growing propensity of developing psychopathology. In other words, schizotypy might constitute a measurable intermediate mediator in the developmental cascade of schizophrenic disorders.

From a developmental psychopathology standpoint, developmental mediators along the trajectory of disorders usually appearing during adulthood represent key targets in longitudinal investigations. Such investigations are designed to more readily assess the intermediate points in development that appear to link early risk factors to adulthood symptom expression. The developmental psychopathology of borderline personality disorder (BPD) provides good examples of such conceptualizations. Most conceptual accounts of developing BPD will assess childhood constitutional factors (familial antecedents, motor development, temperament) early environment characteristics (trauma, attachment, life stress), as well as middle childhood/early adolescence factors (self-regulation instability, self-representation disturbance, interpersonal difficulties, etc.) as nonspecific risk factors potentially contributing to the slow unfolding of pathological manifestations at the phenotypic level (early subclinical symptoms), and opening increased probability of adult BPD psychopathology. A good example of such a longitudinal, developmental psychopathology approach to BPD is provided by Carlson et al., who prospectively assessed developmental antecedents in 162 first-born children of mothers at high parenting risk because of poverty. The authors first analyzed the correlation patterns between early risk factors and adult borderline symptoms. They found that early childhood maternal hostility and maternal life stress, as well as middle childhood/adolescent self-representations best accounted for adult borderline personality symptoms at age 28 years. In the following analysis, the authors examined which middle childhood/early adolescent self-representations potentially played a mediating role in development between early risk factors and later adult BPD symptoms. They observed that middle childhood/early adolescent self-representations significantly mediated the developmental pathway between childhood attachment disorganization and adult BPD symptoms. In this way, the authors underlined a possible developmental cascade linking early disorganized
attachment and middle childhood/early adolescence self-representations towards adult BPD symptoms. Such prospective designs also suggest that developmental mediators are construed during development through successive transactions between the individual's endogenous vulnerabilities and environmental risk factors working towards psychopathological outcomes.

To recapitulate, research on schizotypy has provided some significant clues into its developmental involvement as a distal risk marker for schizophrenia. During development, it is both related to early acquired endophenotypes and developing social cognitive functioning. As it is also correlated to later onset of psychopathology, it may be considered as a key developmental mediator along the risk trajectory. Yet, the developmental mechanisms by which this risk is maintained and or exacerbated still require further research. Critical developmental interactions between schizotypal dimensions, as reviewed in the next section, may start revealing some of these developmental mechanisms bringing distal trait schizotypy closer to proximal state schizotypy.

**Critical Interactions in the Developmental Course of Schizotypy**

A typical criticism of a developmental schizotypy approach in the field of high risk for psychosis resides in the low sensitivity and specificity provided by an assessment of schizotypy, often taking place a decade or more before significant preclinical and clinical psychotic states manifest themselves. Taking example from the Dunedin prospective sample, which followed 1007 individuals from birth to 38 years of age, a summary evaluation of positive schizotypy (clinician-rated psychotic symptoms of hallucinations and delusions) at age 11 years significantly predicts later adult development of schizophrenia-spectrum disorders. It was observed that a significant proportion of identified 11-year-olds with significant positive schizotypy (n = 13), developed schizophrenia by age 38 (3 out of 13 (23%)). However, it was also found that the total proportion of unidentified cases with weak positive schizotypy at age 11 developed schizophrenia by age 38 that developed schizophrenia was substantial (24 out of 776 (3%), which represent 24 cases out of the total 27 cases identified with the disorder (89%)). These prospective findings suggest poor sensitivity when solely examining positive schizotypal experiences in youths. Furthermore, specificity analyses of the adult sample having reported significant childhood positive schizotypy suggested that these individuals were at higher risk of meeting diagnosed criteria not only for schizophrenia, but also for posttraumatic stress disorder and attempt/complete suicide by age 38 in comparison to the rest of the sample.

From a developmental psychopathology approach of schizotypy, 3 observations may be offered with regards to the sensitivity/specificity critique. Firstly, when taking early positive schizotypy as the main variable, it is not surprising to observe multiple developmental finalities. In fact, *multifinality* represents one of the founding principles of developmental psychopathology. From this perspective, early positive symptoms act similarly as any other risk factor, such as trauma for example, associated with various developmental trajectories. Indeed, trauma has not only been associated with increased risk for the development of schizophrenia, but also to increased risk for the development of internalizing disorders and BPD. The mechanisms by which early and enduring traumata induce deleterious effects appear to be related to the developing hypothalamo-pituitary-adrenal (HPA) axis, a key axis in the regulation of developmental stressors, involved in neurobiological accounts of these psychiatric disorders. In a hypothetically similar fashion, stable positive schizotypy may reinforce atypical regulation in the mesolimbic dopaminergic system depending upon transactions with high risk environments (eg bullying), genotype and the constellation of other critical personality dimensions such as negative affect, thereby consoli- dated risk for the development of internalizing and behavior as behavioral proxies. However, most research often bypasses the significant and equally predictive value of the negative schizotypy dimension (see Debbané, Schulze-Lutter et al, this issue). In reality, the sensitivity/specificity critique cannot be confirmed nor denied until prospective cohorts evaluated on all 3 schizotypal dimensions are followed longitudinally past the critical age interval of schizophrenia onset.

This observation links directly with a third comment, which is that sparse but significant preliminary longitudinal data on developing schizotypy support the importance of assessing schizotypy in its multifactorial nature. At least 3 reports suggest that during adolescent development, crucial interactions between positive, negative and disorganized schizotypy take place to both sustain/exacerbate schizotypal expression, and augment the risk for significant psychotic outbreaks.

First, in a group of 34 nonpsychotic help-seeking adolescents assessed longitudinally on trait schizotypy, the schizotypy dimensions as measured by the Schizotypal Personality Questionnaire (SPQ) were found to be significantly associated over a 3-year period. Stability of schizotypy trait could be observed for the negative and disorganization dimension, but positive schizotypy scores
significantly declined over the study interval. In terms of developmental interactions between schizotypal dimensions, baseline negative schizotypy was found to significantly predict positive schizotypy 3 years later, through its mediating relationship with baseline disorganization. In other words, withdrawal and blunted emotion during adolescence, when associated with disorganized speech and behavior, can potentially predict the maintenance or exacerbation of cognitive-perceptual anomalous perceptions. Importantly, these results were corrected for any influences regarding internalizing and externalizing problems in these help-seeking adolescents. In a larger study of similar nature, involving 3021 youths aged 14–24 years at baseline examined for schizotypy using the Composite International Diagnostic System (CIDI), 10-year longitudinal analysis of psychotic-like positive, negative, and disorganization experiences were associated with mental health help-seeking incidence over the study interval. First, on the basis of the assessment interview, psychotic-like symptoms were clustered into a positive symptom cluster and a negative/disorganized symptom cluster. The critical outcome variable consisted in measuring help-seeking behavior in relation to symptoms. During the study interval, between 37–39% of those participants with significant positive symptom-cluster called upon professional help in relation to their symptoms. Help-seeking significantly augmented by 16.2% for those individuals with both the positive and negative/disorganized symptoms clusters. Taken together, these 2 studies suggest that the different dimensions of schizotypy significantly interact during adolescence to sustain/exacerbate and perhaps increase the need for professional help in relation to symptom expression.

A follow-up question regarding potentially pathogenic developmental interactions concerns their actual links to the emergence of categorically defined schizophrenia-spectrum disorders. To the best of our knowledge, there exists only one study testing this hypothesis. In 1 study testing this hypothesis, five-hundred and three 19-year-old college students followed over a 10-year period. A significant interaction was found between the positive and negative schizotypy dimension, specifically in determining long-term paranoid personality features. A trend like but nonsignificant interaction was also found for the development of any psychotic disorders. We note that the disorganization dimension was not evaluated here. This dimension appears to be critical in other studies examining significant longitudinal change and help-seeking behavior in relation to schizotypy. Future longitudinal studies, encompassing all 3 dimensions of schizotypy, are needed to investigate the clinical relevance of such interactions. To date however, the data available do suggest that mechanisms of natural progression, and perhaps mechanisms of change in schizotypal expression might involve maladaptive dimensional interactions between negative, disorganization and positive schizotypy. These interactions must be critically studied if promising early identification strategies are to evolve towards empirically-based and equally promising early intervention strategies.

By embracing principles of developmental psychopathology such as multifinality, the developmental schizotypy approach gains sensitivity to distal dynamic interactions that take place during development and effectively play a significant role in the transition to high-risk states. This leads to a model that is closer to the kinds of accelerated developmental dynamics observed such high-risk states. Clinically, interactions between the schizotypal dimensions, for example between cognitive disorganization and positive schizotypy, may also occur during development. Anecdotally, clinical material often supplies examples of such interactions, as illustrated in the following case example of Fred, a 16-year-old adolescent consulting in our outpatient psychiatric service, who reports the following sequence when prompted about his PLEs:

Fred: … I’ve always been quite reserved, feeling more comfortable when alone, and that people in general couldn’t really understand me...

… I think things started when sometimes, I would hear a kind of music, but I didn’t know where it was coming from. I inspected our house to see if any of the TV or the radio devices was switched on, but they weren’t… This lasted for a few months.

… so I thought it might be due to cannabis, I then decided to stop smoking joints. For more than 3 months I stopped, but the music continued, and also there started to be voices…

… at first they were kind, but then they were very mean voices. They were accusing me of all sorts of names, I think they wanted to punish me for the mean things I did as a child...

… Then there was this vision I had in my room, like an old man whose face was full of scars...

… The next day… no maybe a week before… hmmm or was it a weak after… I’m not sure… I was in store and I was sure that a couple, a man and a women then ordering a sandwich, were actually spying on me. They were suspicious; they thought I was following them. They were talking to each other with a low voice, sometimes looking at me over their shoulder. Then this other man came in the store, and he had thick scars on his face. I then thought the couple had done that to him, like they did to the old man I saw in my room, and they might do it to me. I thought I was caught and couldn’t get out....

During development, different dimensions of schizotypy are likely to interact together. In the case of Fred, an adolescent scoring high on negative schizotypy, the progression of his anomalous perception first led him (rather accurately) question whether they were linked to cannabis consumption. Then, upon their continuation, and together with disorganized and hyper associative thought processes, the quasidelirious content of beliefs...
made their way into his appraisal and explanations of what he was going through. It also appeared that confusions in temporality, as well as self-other confusions, contributed to the process. Other kinds of interactions between symptoms may occur. Another common developmental interaction can be observed between an adolescent’s odd speech, or bizarre and eccentric behavior (disorganizations schizotypy) that can sometimes be met by uneasiness, sarcasm or even hostility by his peer group. In school environments where social appraisals can make or brake adolescents, it is not rare to witness cognitively disorganized youth being bullied, or pulling away from social interactions and into social withdrawal (negative schizotypy). These interpersonal interactions make it more likely for such adolescents to increase their time spent in solitude, and away from social feedback that might balance their growing perception of the hostility of the environment.

Most clinicians would agree that such interactions between schizotypal dimensions do occur, and that emerging high-risk states in youths should be assessed within this dynamic developmental context. Other developmental trajectories, such as those observed in youth populations with “multiple complex developmental disorders” represent another potential path to psychotic disorders. These considerations are not distant from the concept of equifinality in developmental psychopathology (see figure 2). Equifinality refers to the observation that potentially different etiological interactions may lead to the same diagnostic finality. In other words, schizophrenia-spectrum disorders represent a categorical finality reached through significantly different developmental interactions. It thus appears to us that the developmental schizotypy approach may bear the ingredients to embrace multifinality and equifinality, and draw closer to the clinical complexity of emerging schizophrenia-spectrum psychopathology.

Conclusion

In this manuscript, we argue that the development of high trait schizotypy confers an increased liability to psychotic states which, contingent on endogenous and exogenous factors, as well as their dynamic transactions across especially sensitive periods, may ultimately lead to schizophrenia spectrum disorders. This integrative view of schizotypy as a liability trait implicitly confers a developmental dimension to both schizotypy and schizophrenia, and invokes a lifespan, developmental psychopathology framework, to understand the maladaptive pathways leading to spectrum outcomes.

Currently, most developmental research focuses on young adults presenting high-risk mental states very close to clinical psychosis and examines risk for transition to schizophrenia. However, little is known about the developmental pathways linking trait schizotypy, high risk mental states and the progression to clinical expression in the schizophrenia spectrum. A developmental model of schizotypy is in a good position to integrate this research. Importantly, future longitudinal research focusing on high-risk for psychosis should include schizotypy assessments as a distal risk marker. Accumulating evidence indicates that trait schizotypy (1) is related to and accounts for the liability to present subclinical psychotic states across development and (2) is already associated with many of the endophenotypes established for schizophrenia during childhood and adolescence, which suggests that trait schizotypy could be both an early distal marker to select populations of interest to conduct longitudinal studies and, importantly, a measurable developmental mediator in the pathway of risk to schizophrenia spectrum disorders. In this context, epigenetic studies focusing on schizotypy trait expression are necessary. Finally, preliminary longitudinal data points out the critical importance of taking into consideration the multidimensionality of schizotypy in such developmental framework, as mechanisms of change in schizotypal expression might involve maladaptive dimensional interactions between negative, disorganization, and positive schizotypy. Studying these interactions, the different outcomes of schizotypy traits depending on the niche of risk and protective factors (ie multifinality) and the various pathways that may conduce to a schizophrenia spectrum diagnostic entity (ie equifinality) are essential to yield empirically based early identification and intervention strategies.

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Fig. 2. An illustration of the developmental psychopathology principles of multifinality and equifinality.
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