

Schizotypy as An Organizing Framework for Social and Affective Sciences

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Schizotypy, defined in terms of commonly occurring personality traits related to the schizophrenia spectrum, has been an important construct for understanding the neurodevelopment and stress-diathesis of schizophrenia. However, as schizotypy nears its sixth decade of application, it is important to acknowledge its impressively rich literature accumulating outside of schizophrenia research. In this article, we make the case that schizotypy has considerable potential as a conceptual framework for understanding individual differences in affective and social functions beyond those directly involved in schizophrenia spectrum pathology. This case is predicated on (a) a burgeoning literature noting anomalies in a wide range of social functioning, affiliative, positive and negative emotional, expressive, and social cognitive systems, (b) practical and methodological features associated with schizotypy research that help facilitate empirical investigation, and (c) close ties to theoretical constructs of central importance to affective and social science (eg, stress diathesis, neural compensation). We highlight recent schizotypy research, ie providing insight into the nature of affective and social systems more generally. This includes current efforts to clarify the neurodevelopmental, neurobiological, and psychological underpinnings of affiliative drives, hedonic capacity, social cognition, and stress responsivity systems. Additionally, we discuss neural compensatory and resilience factors that may mitigate the expression of stress-diathesis and functional outcome, and highlight schizotypy's potential role for understanding cultural determinants of social and affective functions.

Key words: schizophrenia/schizotypy/emotion/affect/social/motivation/drive/negative

Introduction

Schizotypy, defined as a set of both genetically and environmentally influenced personality traits related to

schizophrenia spectrum pathology, has been an important construct for understanding the neurodevelopment and stress-diathesis of schizophrenia.¹⁻⁵ Indeed, Lenzenweger¹ noted its importance as an “organizing framework” for understanding schizophrenia spectrum pathology. Schizotypy also has a rich empirical literature outside of psychiatry research, and has been of interest to a diverse set of disciplines, including neuroscience, genetics, evolution, personality, experimental psychology, parapsychology, religion, industrial and organizational psychology, anthropology, art, music, education, and philosophy. This multidisciplinary interest highlights the expanding utility of the schizotypy construct well beyond the borders of schizophrenia spectrum pathology. A particularly important focus of recent work has involved social and emotional anomalies, as both are core components of schizotypy and integral to human functioning and civilization. The primary purpose of this article is to evaluate schizotypy as an “organizing framework” for social and affective sciences beyond its utility for understanding schizophrenia. To this end, we will (a) briefly highlight the literature on social and affective functions in schizotypy, (b) discuss how understanding social and affective anomalies in schizotypy can uniquely provide insights about these functions more generally, and (c) highlight some recent exemplars of how schizotypy research informs our understanding of the neurobiological, neurodevelopmental, psychological, and cultural systems underlying social and affective functions.

Social and Affective Functions in Schizotypy

Social and affective abnormalities in relation to schizotypal traits appear across a widely distributed and interconnected set of systems and adversely impact quality of life. Both positive and negative schizotypal traits have been associated with fewer self-reported social activities

as well as difficulties in occupational, recreational, relational, and academic domains assessed using structured interviews.⁶⁻⁸ Social abnormalities, notably in the frequency and intensity of social interactions, have also been associated with elevated positive and negative schizotypy in daily life as documented by ecologically valid mobile assessment methods.⁹ These social dysfunctions appear to reflect reduced social competence; not merely as a consequence of avoidant or introverted tendencies, in that they have been demonstrated during structured laboratory-based interactions with confederates, at least in individuals with social anhedonia.^{10,11} Importantly, schizotypy is not associated with general impairments across all domains of functioning, as shown by relatively intact academic performance,¹² cognitive abilities,¹³ mating success¹², and abnormally enhanced creativity¹⁴ in at least some studies.

Closely tied to the social dysfunctions in schizotypy are affective anomalies, in particular, “social anhedonia”—defined in terms of a trait-like disturbance in the experience of affiliative states. Cross-sectional and laboratory studies provide evidence that social anhedonia is important to the schizotypy construct, and is associated with relatively poor social functioning and schizophrenia-like abnormalities—though typically attenuated in severity.^{6,7} Mobile assessment⁹ and longitudinal^{15,16} studies have also provided evidence that social anhedonia is tied to the emergence of schizophrenia spectrum pathology. Social anhedonia, and negative schizotypy more generally, reflect reduced interest and drive to participate in social activities, ie separable from negative affect, such as social anxiety and depression.^{9,17} Moreover, social anhedonia is distinguishable from low positive affect and extraversion.¹⁸ Thus, social anhedonia appears to reflect a relatively specific anomaly in social and affective reward processes, ie related to schizophrenia pathology.

Affective anomalies in relation to schizotypy manifest well beyond affiliative states. First, general abnormalities in subjective experience of emotion are particularly striking in schizotypy, with low levels of positive affect (eg, “physical” anhedonia, reduced extraversion) and high levels of negative affect being reported across multiple domains of measurements and in response to a wide range of stimuli. For example, negative schizotypal traits and reduced positive affect have shown significant associations using trait questionnaires⁸ and on state questionnaires following laboratory-based emotion manipulations.¹⁷ With respect to the latter, it is noteworthy that this literature includes over a dozen published studies collectively employing stimuli with social and nonsocial features and presented across a range of sensory domains.¹⁷ Mobile assessment studies have provided evidence that schizotypy is associated with abnormally low levels of positive and high levels of negative affect in daily life.⁹ Affective anomalies are by no means trivial as they appear to partly determine quality of life^{7,19} and

the magnitude of subjective experience of abnormalities exceeds that observed in chronic outpatients with schizophrenia.¹⁷ Dovetailing these findings is evidence that psychophysiological and neurobiological responses to emotional stimuli are sometimes exaggerated in people with schizotypy.²⁰ In short, anomalous subjective emotional experiences are closely tied to schizotypy.

Related to the social and affective abnormalities in schizotypy are reductions in communication and expressive behaviors. Constricted affect, characterized by reduced facial, vocal, and gestural expressions, is central to schizotypy, and is a symptom of schizotypal personality disorder per Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM 5).²¹ Self-report measures of expressivity indicate that individuals with high social anhedonia express themselves less intensely and frequently than their peers.²² Observers’ behavioral ratings of social anhedonics engaged in social interactions also suggests that their expressive gestures are abnormally low.^{10,22} Moreover, measures of constricted affect have been associated with reductions in social functioning and quality of life.^{7,19} Studies employing objective methods, such as computerized facial or vocal analysis, have not uniformly supported the notion that individuals with elevated schizotypal traits show constricted affect,^{23,24} though there is evidence that high negative schizotypal traits may be associated with more sparse and flatter speech under cognitively challenging conditions²³ and that highly schizotypal individuals who are reporting symptoms of psychosis tend to show reduced facial expressions.²⁴

Finally, disruptions in social cognitive processes have been evaluated in relation to schizotypy. With respect to emotion perception, laboratory studies of faces, bodies, and prosody in relation to schizotypy have yielded mixed and relatively nuanced findings. Elevated schizotypy is not necessarily associated with deficits in identifying facial emotional expressions^{25,26} although specific facial emotion perception impairments have been associated with increased positive,²⁷ negative^{25,28}, and disorganized²⁹ schizotypal traits in individual studies. Interestingly, under perceptually challenging conditions (ie, an emotional chimeric faces task), subtle impairments can be observed with respect to positive schizotypy; individuals with elevated positive schizotypy classified emotional faces such that they misidentified angry faces as happy, and happy faces as angry or fearful.³⁰ Such misidentification of facial emotions could lead to inappropriate social interactions and contribute to social impairments. Positive schizotypy has also been associated with impaired identification of emotion through vocal prosody.²⁵ Since prosody plays a central role in effective communication, abnormal prosody perception is likely to contribute to social misunderstanding.

Theory of mind (ToM)—the ability to attribute mental states to oneself and others (eg, understanding others’

actions, feelings, and intentions), and its link with schizotypal traits have also yielded mixed results. Depending on task and schizotypal traits examined, high schizotypy has been associated with intact, impaired, and even enhanced performances. For example, in subjects with high schizotypy scores (reflecting positive, negative, and disorganization traits), intact performance has been reported on a task assessing sarcasm;²⁶ poorer performance on a task assessing social sensitivity,²⁸ and enhanced performance on a ToM task that involved detection of irony.⁶ These observations are in contrast to earlier findings of deficits in irony and deception detection^{31,32}—which also used similarly broad definitions of schizotypy. It seems that behaviorally, individuals with elevated schizotypy cannot yet be reliably distinguished from those low on schizotypy on measures of ToM. As discussed later, it is possible that compensatory mechanisms may be mitigating social cognitive performance in those with elevated schizotypy who may be using alternative strategies to solve daily social cognitive challenges.

Schizotypy as An Organizing Framework for Affect and Social Sciences

The last decade has seen increasing emphasis on understanding social and affective functions from multidimensional (eg, covering multiple levels of complexity) and neurodevelopmental perspectives. This emphasis is seen in the recent formation of professional organizations devoted to these topics (eg, Society for Social Neuroscience, Society for Affective Sciences) and is the thrust of the new Research Domain Criteria (RDoC) initiative from the National Institute of Mental Health (NIMH)³³ in the United States. The latter program reflects an attempt to redefine psychopathology in terms of distinct mechanisms across levels of complexity from the genetic and molecular to the neuroanatomical and subjective/behavioral. It is noteworthy that 3 of the 5 currently defined RDoC domains, namely negative valence systems, positive valence systems, and systems for social processes, directly pertain to abnormalities discussed in this article. Note also that a fourth domain, cognitive systems, is also directly relevant to schizotypy.^{34,35} Thus, the abnormalities associated with schizotypy reflect those of most interest to NIMH as well as a critical frontier of science more generally.

Schizotypy is a particularly useful construct for expanding the scope and impact of affective and social sciences. From a practical perspective, schizotypal traits are pronounced in a large segment of the population—approximately 10% using categorical conceptualizations¹ and a much larger segment using dimensional conceptualizations.⁴ Thus, subjects with schizotypal traits are readily accessible for empirical study. Moreover, there is nothing inherently debilitating about schizotypy that might interfere with a standard research protocol. Thus,

motivationally demanding, high burden and difficult experimental protocols that may pose challenges to those with debilitating psychiatric disorders can be undertaken, and the results of such studies could shed light on more severe conditions without the confounds of illness, institutionalization, and medications. From a statistical and methodological perspective, there is considerable variability associated with schizotypy in terms of social and affective functioning, development of psychopathology, and outcome. Some of the variability arises from the multidimensional nature of schizotypal personality.⁵ Thus, a dimensional approach can add considerable variability in outcome measures beyond what is seen when averaging performance of the general population.

The social and affective anomalies associated with schizotypy have close conceptual and empirical ties to clinical pathology, notably with psychosis and schizophrenia spectrum disorders, but also depression, anxiety, substance use, personality disorders, attention deficit hyperactivity disorder, and other disorders.^{15,16,21} Thus, examining the link between schizotypy and clinical pathology can provide a mechanistic understanding of clinical symptoms more generally. This can be particularly informative for understanding how specific neurodevelopmental factors mitigate symptom expression. Insofar as schizotypy is theorized to follow a fairly predictable temporal course, particularly in terms of a relatively well-defined window of risk for expression of psychosis, one can potentially glean critical information about the interplay between diathesis-stress factors, as well as risk and resilience variables. Relatedly, links between mental illness risk and well-being, psychological outcome, and adaptive traits (eg, creativity, genius) can be understood within the context of schizotypy.³⁶ The construct of schizotypy can also contribute to our understanding of how social and affective processes are influenced by culture. That is, exploring how affective and social processes manifest as a function of schizotypy across cultures can provide a unique perspective for understanding how culture shapes individuals. In the following sections, we provide exemplars of how the study of schizotypy is providing important insights for social and affective sciences.

Schizotypy and the Nature of Social Cognitive Processes

The study of schizotypy offers insights into the mechanisms underlying social cognition. Of particular importance is empathy, which refers to interpreting and reacting to the experiences of others, and is central to social functioning. Empathy is a multifaceted construct that involves both cognitive and emotional components and requires intact representations of self, others, and appropriate distinctions between them. Disturbances in the self-other boundary were central to early conceptualizations of schizophrenia and schizotypy³⁷ and both positive and

negative schizotypy have been associated with anomalous self-other boundaries.^{38,39} Positive schizotypy has been associated with high self-reported affective empathy⁴⁰ and enhanced performance on a visuospatial imagery task assessing perspective taking abilities.⁴¹ Conversely, individuals with elevated negative schizotypy have reported experiencing relatively low levels of self-reported empathy and poor performance on empathy tasks.^{40,41} The neural correlates of empathy implicate the right temporoparietal junction (TPJ) a multisensory brain region implicated in self-processing. Interestingly, highly schizotypal individuals appear to allocate greater resources to this region when processing self-related stimuli than those with lower schizotypy scores,⁴² suggesting a compensatory mechanism at work. Additionally, negative, but not positive, schizotypal traits have been associated with greater right TPJ thickness.⁴³ Although the meaning of cortical thickness in relation to personality traits is unclear, it is possible that altered structural asymmetry of the parietal region could reflect neural compensation arising from prolonged functional hyperactivity over the course of development while engaged in self-other processing. Regardless, schizotypy offers an important window into understanding the neural mechanisms underlying empathy, and how these mechanisms may respond in the face of neural liabilities or challenges.

Schizotypy and the Nature of Hedonic Capacity

The study of schizotypy also provides insight into the neurobiological mechanisms underlying hedonic capacity. There has been considerable progress from affective science in defining a coherent network of brain structures related to the experience of pleasure, though the relationships between these structures and their individual functions are far from clear. Investigations into the neural signature of hedonic experience in schizotypy indicate a complex interplay between limbic and cortical structures. For example, there is evidence of reduced ventral lateral prefrontal cortex (PFC) activation in individuals with high trait social anhedonia while actively evaluating videos of pleasant social interactions.³⁹ Similarly, trait anhedonia in a nonpsychiatric sample has been associated with reduced activity in left medial PFC and rostral anterior cingulate cortex (ACC) while actively processing a range of positively valenced images.⁴⁴ In contrast however, an earlier study by Harvey et al⁴⁵ found that trait anhedonia was associated with *increased* ventromedial PFC activity when participants were engaged in passive social information processing (eg, subjects provided dichotomous ratings regarding the presence or absence of human stimuli in an image still). Importantly, none of these studies found abnormal limbic activation. In explaining this apparent inconsistency, some have noted that the control conditions (ie, processing neutral stimuli) for the active tasks required greater frontal resources than those for the

passive task.^{44,46} Hence, the differences in frontal activity in relation to social anhedonia could reflect baseline differences. Indeed, default activity in the PFC structures has been reduced in individuals with high negative schizotypy^{46,47} and there is evidence that the PFC and ACC can upregulate activity in limbic systems.⁴⁴ Thus, it is possible that intact engagement of limbic structures (or of attentional systems more generally) during passive social processing requires greater effort to recruit PFC and ACC in anhedonic individuals. This line of research could shed light on how hedonic experience varies as a function of contextual factors and task demands, and how components of the hedonic system can potentially upregulate activity to compensate for other neural anomalies.

Schizotypy and the Diathesis-Stress Model

Schizotypy is a useful construct for understanding how genetic, neurobiological, familial, psychological, and other factors influence functional outcome as well as the emergence of psychopathological symptoms. In evaluating the literature on factors influencing “outcome” in schizotypy from a diathesis-stress perspective, social, and affective variables are emerging as particularly important. For example, stressful experiences have been linked to more severe schizotypal traits in healthy adults,⁴⁸ and some longitudinal studies have reported that social anhedonia is a particularly important predictor of development of schizophrenia spectrum disorders.^{15,16} Similarly, impaired social functioning predicts the emergence of psychosis among high schizotypal individuals—notably in recent large scale clinical high-risk studies.⁴⁹ A more nuanced understanding of the effects of social and affective anomalies on psychopathology is provided by recent mobile assessment studies. Following even minor daily hassles and stressors, individuals high in schizotypy tend to show increases in psychotic-like and paranoid symptoms.⁹ Of particular interest, in contrast with the general population, individuals with high social anhedonia report feeling better when alone and consider socializing to be stressful.⁵⁰ These findings highlight an important potential contribution of studying the stress-symptom link in schizotypy—that “stress” is idiosyncratic. Of note, the effects of social support, a long-heralded buffer of stress, may not be ubiquitous across people.

Relatedly, schizotypy can offer insights into how stress-response systems operate—as these systems are important for mitigating hypothalamic pituitary adrenal (HPA) activity and the deleterious effects of corticosteroids and important for explaining psychosis proneness more generally.⁵¹ Temporal structures play an important role in HPA activity, and there is evidence for the role of striatal dopaminergic abnormalities under states of psychosocial stress in schizotypy. In a laboratory study employing a social-evaluative mental arithmetic stressor in individuals with high trait anhedonia, Soliman et al⁵² reported significant associations between

anhedonia and increases in striatal dopamine release, suggesting individuals with high anhedonia show exaggerated stress responses. Interestingly, in a follow-up study, Soliman et al⁵³ found that trait anhedonia was associated with greater *deactivation* in medial temporal and striatal structures. In healthy adults, deactivation in these regions during the same social stressor task has been associated with increases in cortisol,⁵⁴ providing some assurance that the findings are not spurious. While the meaning of these findings is unclear, the possibility that dopamine may inhibit striatal activity during social stress in some individuals is important, particularly in light of evidence that individuals high in (positive) schizotypy may show a compensatory mechanism for an excess of available dopamine (pharmacological levodopa challenge) that might balance out otherwise anomalous cognitive functioning.⁵⁵ Clinically speaking, abnormal striatal dopaminergic release with concomitant deactivation may also serve as an index of stress sensitivity.

Schizotypy and Neurodevelopment

Further insight into adaptive abilities and compensatory neurodevelopmental factors in relation to schizotypy could elucidate the complex interplay between diathesis and stress. Insofar as the vast majority of individuals with high schizotypy are protected from severe psychopathology, investigation of the mitigating factors that buffer symptom onset may provide insight into diathesis-stress more generally. At a behavioral level, the use of adaptive coping strategies, reduced negative affect, and increased positive affect have been associated with better functioning and less pathology in cross-sectional studies of schizotypal individuals.⁵⁶ Relatedly, schizotypal individuals with healthy and more secure attachment styles, reflecting the ability, and tendency to form intimate emotional bonds with others, tend to have better outcomes.⁵⁷ Investigations into the neurobiology of schizotypy have suggested that certain neural structures, and/or the ability to recruit them, may be bolstered in individuals with high schizotypy. A recent meta-analysis of the neurobiology of schizotypal personality disorder found that medial temporal lobe structures were compromised and that prefrontal structures were abnormally large,⁵⁸ which may reflect a compensatory role of the prefrontal regions for neural inefficiencies^{34,58,59} that may underlie social and affective processing difficulties. Indeed, recent studies examining neural correlates of ToM functioning in schizotypy observed a lack of behavioral differences but increased activation in the inferior frontal gyrus (IFG) in individuals with high positive schizotypy⁶⁰ and negative schizotypy.⁶¹ Given that the IFG is associated with mirror neuron mechanism,⁶¹ as well as inhibitory functions,⁶⁰ particularly with respect to self-referential information, increased activation in this region is speculated to reflect increased resources needed for balancing self vs other

information. In sum, schizotypy offers a unique opportunity to understand how psychological and neural systems compensate for neural challenges, and potentially serve as a mitigating factor for expression of genetic and environmental vulnerability.

Schizotypy can also contribute towards a more nuanced understanding of the neurodevelopment of social and affective systems. Some theorists have noted the importance of the prenatal environment in the development of schizophrenia spectrum disorders, and this has led to inquiry of how developmental instability during critical gestational epochs may contribute to schizotypy. In particular, the second half of fetal development is critical for development and programming of the HPA axis,⁶² and abnormalities in HPA activity are likely part of an expansive set of physical abnormalities that can be detected and studied. Support for a link between developmental instabilities and schizotypy comes from associative studies linking schizotypal traits to minor physical anomalies (MPAs). MPAs are defined in a range of physical abnormalities, eg, wide-spaced eyes, low-set ears and anomalies of the fingers and toes, and are thought to reflect second trimester development. Of note, social anhedonia has been associated with a host of developmental instabilities.⁶³ There is also evidence that, within schizotypal samples, MPAs are linked to HPA anomalies. Mittal et al⁶⁴ found significant associations between MPAs and salivary cortisol levels within an adolescent schizotypy group—and these relationships were specific to schizotypy in that they were not significant for control groups. These data suggest that prenatal factors play an important role in the expression of individual differences in affiliative emotions, and might be part of a network of abnormalities affecting stress-response systems and physical development.

Schizotypy and Culture

Social and affective variables vary considerably across cultures, and recent evidence from affective sciences is raising questions about the long-held notion that emotional expression and experience is structurally similar across cultures.⁶⁵ In this regard, the study of schizotypy as it manifests across cultures can provide insight into the environmental and cultural determinants of social and affective functions. While it is the case that the factor structure of schizotypy scales is generally invariant across cultures, there is considerable evidence that individual scales, particularly those related to negative schizotypy, vary as a function of culture. For example, in samples from the United States, Asian-Americans have reported experiencing higher levels of negative schizotypy (ie, constricted affect, no close friends) compared to Caucasians; the opposite pattern was seen with African-Americans.^{7,66} Relatedly, British-Caucasians have shown lower levels of negative schizotypy than Afro-Caribbeans,⁶⁷ and

individuals from Spain report higher levels of negative schizotypy than individuals from Switzerland.⁶⁸ Cross-cultural differences in affective experiences are well illustrated in a recent psychometric investigation of a measure of anticipatory and consummatory anhedonia. While available evidence suggests a 2-factor solution for individuals from the United States, a more complicated factor structure was found in a healthy Chinese sample.⁶⁹ The results suggest that contextual and abstract factors are important for understanding hedonic experience in Chinese cultures in ways less relevant for US cultures. For example, in the US sample, items assessing “contextual” hedonic experiences (eg, “when I think of something tasty, like a chocolate chip cookie, I have to have one”) loaded commonly with items assessing more “abstract” hedonic experiences (eg, “looking forward to a pleasurable experience is in itself pleasurable”), but did not in the Chinese sample. The authors propose that Chinese culture emphasizes and fosters harmony and low arousal states and adjustment to immediate external environment. From this perspective, Chinese individuals may structure and possibly even define hedonic experience based on contextual information very differently from Western cultures. Clinical utility of these factor structures has been demonstrated in a group of patients with and without prominent negative symptoms in the Chinese setting.⁷⁰ As yet, there has been limited investigation of how culture influences social and affective functions within the context of schizotypy. However, this is a potentially fruitful line for future research to further understand protective and risk factors for psychosis, particularly given the similar rates of schizophrenia across different cultures.

Towards a More Concerted and Systematic Schizotypy Research

In this article, we highlight ways that the study of schizotypy in the general population can provide a useful framework for understanding emotional and social systems outside the context of illness or pathology. It is worth briefly considering how this endeavor can be advanced further. On one hand, improving the visibility of schizotypy research outside mental illness and schizophrenia research venues seems important. Increasing the multidisciplinary focus of schizotypy research, to focus even further on genetic, neurobiological, behavioral, cognitive, phenomenological, and cultural systems should help broaden its appeal, particularly as clinical science becomes increasingly translational in nature.³³ At the same time, we see 3 core issues regarding schizotypy research that, at the present time, could serve as obstacles for advancing its progress into novel research frontiers. Addressing these issues could help facilitate a more concerted and systematic effort for applying schizotypy to understanding human nature more generally. We consider in more detail the following 3 core issues.

A first issue concerns the heterogeneity of schizotypy. Much like schizophrenia, schizotypy shows considerable phenotypic variability. Statistical examination of commonly used schizotypy measures suggests that it is comprised of positive, negative, and disorganized traits,³ and there is considerable evidence that these traits show discrepant outcomes and cognitive, behavioral, and neurobiological correlates, eg⁵ with respect to social and emotional processes, eg, negative, but not positive, schizotypal traits have been associated with greater right TPJ thickness⁴³ and negative and positive schizotypal traits are associated with different types of anomalies in empathy.^{40,41} Differences aside, there is little understanding of how positive, negative and disorganization traits are related to each other in terms of neurodevelopmental trajectory and in terms of underlying mechanisms. Moreover, it is unclear whether the traits underlying the “superordinate” positive, negative, and disorganization trait factors should also be considered separately. For example, should constricted affect and social anhedonia be considered separately or should they be combined as part of a negative schizotypy factor? Insofar as most of our understanding of schizotypy’s structure is based on cross-sectional trait questionnaire data, studies investigating the mechanisms underlying various schizotypal traits can provide critical information about their structural overlap. Exploring how social and affective processes converge and diverge as a function of schizotypal traits seems a particularly fruitful approach for clarifying the structure of schizotypy.⁷¹

A second issue concerns variability across studies in how schizotypy is measured. At present, there exists a large number of questionnaires validated for schizotypy research,⁷² and these measures vary considerably in their scope, length, and content.⁷³ Subscales from different schizotypy measures that cover similar constructs typically correlate very highly with each other, so it seems reasonable to assume that these measures are generally measuring the same construct. For example, schizotypy subscale scores from the Chapman scales highly correlate with those with matching content from the Schizotypal Personality Questionnaire (r 's > 0.75).⁷⁴ As observed in this article, anomalies in social and affective functions have been observed across studies using a variety of measures, and we are aware of no reason to champion one measure over another at the present time. It is also the case that schizotypy research is an increasingly international endeavor, so it will be important to ensure that measures are validated across different cultures. Relatedly, attention to how culture contributes to expression of schizotypal traits will be important, as the content of schizotypal scales may have to be adjusted based on cultural mores, technology, and values. Future research would be helpful for addressing these issues; with a potential goal of

promoting consensus on specific items and measures of schizotypy for concerted international use.

A third issue involves the unresolved debate over whether schizotypy reflects a categorical or dimensional construct.⁷⁵ The preponderance of evidence from studies employing taxometric and mixture modeling analysis suggests that schizotypy is a categorical construct with a population incidence on the order of 10%.⁶¹ On the other hand, concerns have been raised about the test-retest reliability of taxometric procedures within individuals more generally,⁷⁶ and it is clear that very few individuals in the schizotypy taxon develop schizophrenia.⁷⁴ Moreover, there is evidence that both schizophrenia and schizotypy are determined by incremental contributions from a wide array of demographic, obstetric, genetic, psychosocial, environmental, and other factors; and hence, may be continuous in nature.⁷⁷ With respect to emotional and social anomalies, it is clear that they manifest regardless of categorical^{10,11,13,15,16} or dimensional^{6,7,9,12,14} conceptualizations, as a sizeable literature exists employing both approaches. Regardless, resolving this issue is an important step in applying schizotypy research to understanding emotion and social functions beyond those associated with mental illness.

Conclusions

The schizotypy construct is nearing its sixth decade of existence, and it has contributed to an impressively voluminous and diverse literature. While schizotypy continues to be primarily examined within the framework of schizophrenia spectrum pathology, its application to understanding human nature more generally is beginning to take form. On some scientific frontiers, schizotypy is providing important information about how individual differences in social and affective functions develop, how they are expressed at a genetic, neurobiological, and psychological level, and how they uniquely contribute to both adaptive and maladaptive features of humanity. In this article, we attempt to highlight some of these contributions with the hope that a more concerted and systematic effort can be mustered.

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References

1. Lenzenweger MF. Schizotypy: an organizing framework for schizophrenia research. *Curr Dir Psychol Sci*. 2006;15:162–166.
2. Meehl PE. Schizotaxia, schizotypy, schizophrenia. *Am Psychol*. 1962;17:827–838.
3. Raine A. Schizotypal personality: neurodevelopmental and psychosocial trajectories. *Annu Rev Clin Psychol*. 2006;2:291–326.
4. Claridge G. Schizotypy and schizophrenia. In: Bebbington P, McGuffin P, eds. *Schizophrenia: The Major Issues*. Oxford, UK: Heinemann Medical Books/Heinemann Professional Publishing; 1988:187–200.
5. Tarbox SI, Pogue-Geile MF. A multivariate perspective on schizotypy and familial association with schizophrenia: a review. *Clin Psychol Rev*. 2011;31:1169–1182.
6. McCleery A, Divilbiss M, St-Hilaire A et al. Predicting social functioning in schizotypy: An investigation of the relative contributions of theory of mind and mood. *J Nerv Ment Dis*. 2012;200:147–152.
7. Cohen AS, Davis TE 3rd. Quality of life across the schizotypy spectrum: findings from a large nonclinical adult sample. *Compr Psychiatry*. 2009;50:408–414.
8. Horan WPB, Jack J, Clark LA, Green MF. Affective traits in schizophrenia and schizotypy. *Schizophr Bull*. 2008;34:856–874.
9. Kwapil TR, Brown LH, Silvia PJ, Myin-Germeys I, Barrantes-Vidal N. The expression of positive and negative schizotypy in daily life: an experience sampling study. *Psychol Med*. 2012;42:2555–2566.
10. Llerena K, Park SG, Couture SM, Blanchard JJ. Social anhedonia and affiliation: examining behavior and subjective reactions within a social interaction. *Psychiatry Res*. 2012;200:679–686.
11. Gibson CM, Penn DL, Prinstein MJ, Perkins DO, Belger A. Social skill and social cognition in adolescents at genetic risk for psychosis. *Schizophr Res*. 2010;122:179–184.
12. Nettle D. Schizotypy and mental health amongst poets, visual artists, and mathematicians. *J Res Pers*. 2006;40:876–890.
13. Chun CA, Minor KS, Cohen AS. Neurocognition in psychometrically defined college Schizotypy samples: we are not measuring the “right stuff.” *J Int Neuropsychol Soc*. 2013;19:324–337.
14. Acar S, Sen S. A multilevel meta-analysis of the relationship between creativity and schizotypy. *Psychol Aesthet Creat Arts*. 2013;7:214–228.
15. Kwapil TR, Gross GM, Silvia PJ, Barrantes-Vidal N. Prediction of psychopathology and functional impairment by positive and negative schizotypy in the Chapmans’ ten-year longitudinal study. *J Abnorm Psychol*. 2013;122:807–815.
16. Gooding DC, Tallent KA, Matts CW. Clinical status of at-risk individuals 5 years later: further validation of the psychometric high-risk strategy. *J Abnorm Psychol*. 2005;114:170–175.
17. Cohen AS, Callaway DA, Najolia GM, Larsen JT, Strauss GP. On “risk” and reward: investigating state anhedonia in psychometrically defined schizotypy and schizophrenia. *J Abnorm Psychol*. 2012;121:407–415.
18. Watson D, Naragon-Gainey K. On the specificity of positive emotional dysfunction in psychopathology: evidence from the mood and anxiety disorders and schizophrenia/schizotypy. *Clin Psychol Rev*. 2010;30:839–848.

19. Wang Y, Yeh Y-h, Tsang S-m, et al. Social functioning in Chinese college students with and without schizotypal personality traits: An exploratory study of the Chinese version of the First Episode Social Functioning Scale. *PLoS One* 2013;8.
20. Premkumar P, Ettinger U, Inchley-Mort S, et al. Neural processing of social rejection: the role of schizotypal personality traits. *Hum Brain Mapp.* 2012;33:695–706.
21. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Publishing. 2013.
22. Leung WW, Couture SM, Blanchard JJ, Lin S, Llerena K. Is social anhedonia related to emotional responsivity and expressivity? A laboratory study in women. *Schizophr Res.* 2010;124:66–73.
23. Cohen AS, Hong SL. Understanding constricted affect in schizotypy through computerized prosodic analysis. *J Pers Disord.* 2011;25:478–491.
24. Cohen AS, Morrison SC, Callaway DA. Computerized facial analysis for understanding constricted/blunted affect: initial feasibility, reliability, and validity data. *Schizophr Res.* 2013;148:111–116.
25. Shean G, Bell E, Cameron CD. Recognition of nonverbal affect and schizotypy. *J Psychol.* 2007;141:281–291.
26. Jahshan CS, Sergi MJ. Theory of mind, neurocognition, and functional status in schizotypy. *Schizophr Res.* 2007;89:278–286.
27. Miller AB, Lenzenweger MF. Schizotypy, social cognition, and interpersonal sensitivity. *Personal Disord.* 2012;3:379–392.
28. Morrison SC, Brown LA, Cohen AS. A multidimensional assessment of social cognition in psychometrically defined schizotypy. *Psychiatry Res.* 2013;210:1014–1019.
29. Brown LA, Cohen AS. Facial emotion recognition in schizotypy: the role of accuracy and social cognitive bias. *J Int Neuropsychol Soc.* 2010;16:474–483.
30. van't Wout M, Aleman A, Kessels RPC, Laroi F, Kahn RS. Emotional processing in a non-clinical psychosis-prone sample. *Schizophr Res.* 2004;68:271–281.
31. Langdon R, Coltheart M. Recognition of metaphor and irony in young adults: the impact of schizotypal personality traits. *Psychiatry Res.* 2004;125:9–20.
32. Malcolm S, Keenan JP. My right I: Deception detection and hemispheric differences in self-awareness. *Soc Behav Pers.* 2003;31:767–772.
33. Insel T, Cuthbert B, Garvey M, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *Am J Psychiatry.* 2010;167:748–751.
34. Ettinger U, Meyhöfer I, Steffens M, Wagner M, Koutsouleris N. Genetics, cognition, and neurobiology of schizotypal personality: a review of the overlap with schizophrenia. *Front Psychiatry.* 2014;5:18.
35. Ettinger U, Mohr C, Gooding DC, et al. Cognition and brain function in schizotypy: a selective review. *Schizophr Bull.* 2015;41(suppl 2):S417–S426.
36. Mohr C, Claridge G. Schizotypy—do not worry, it is not all worrisome. *Schizophr Bull.* 2015;41(suppl 2):S436–S443.
37. Sullivan HS. *Schizophrenia as a Human Process*. New York, NY: W W Norton & Co.; 1962.
38. Debbané M, Vrtička P, Lazouret M, Badoud D, Sander D, Eliez S. Self-reflection and positive schizotypy in the adolescent brain. *Schizophr Res.* 2014;152:65–72.
39. Hooker CI, Benson TL, Gyurak A, Yin H, Tully LM, Lincoln SH. Neural activity to positive expressions predicts daily experience of schizophrenia-spectrum symptoms in adults with high social anhedonia. *J Abnorm Psychol.* 2014;123:190–204.
40. Henry JD, Bailey PE, Rendell PG. Empathy, social functioning and schizotypy. *Psychiatry Res.* 2008;160:15–22.
41. Thakkar KN, Park S. Empathy, schizotypy, and visuospatial transformations. *Cogn Neuropsychiatry.* 2010;15:477–500.
42. Arzy S, Mohr C, Michel CM, Blanke O. Duration and not strength of activation in temporo-parietal cortex positively correlates with schizotypy. *Neuroimage.* 2007;35:326–333.
43. Kühn S, Schubert F, Gallinat J. Higher prefrontal cortical thickness in high schizotypal personality trait. *J Psychiatr Res.* 2012;46:960–965.
44. Harvey PO, Armony J, Malla A, Lepage M. Functional neural substrates of self-reported physical anhedonia in non-clinical individuals and in patients with schizophrenia. *J Psychiatr Res.* 2010;44:707–716.
45. Harvey PO, Pruessner J, Czechowska Y, Lepage M. Individual differences in trait anhedonia: a structural and functional magnetic resonance imaging study in non-clinical subjects. *Mol Psychiatry.* 2007;12:767–775.
46. Wacker J, Dillon DG, Pizzagalli DA. The role of the nucleus accumbens and rostral anterior cingulate cortex in anhedonia: integration of resting EEG, fMRI, and volumetric techniques. *Neuroimage.* 2009;46:327–337.
47. Lagioia A, Van De Ville D, Debbané M, Lazeyras F, Eliez S. Adolescent resting state networks and their associations with schizotypal trait expression. *Front Syst Neurosci.* 2010;4.
48. Kocsis-Bogár K, Miklósi M, Forintos DP. Impact of adverse life events on individuals with low and high schizotypy in a nonpatient sample. *J Nerv Ment Dis.* 2013;201:208–215.
49. Tarbox SI, Addington J, Cadenhead KS, et al. Premorbid functional development and conversion to psychosis in clinical high-risk youths. *Dev Psychopathol.* 2013;25:1171–1186.
50. Silvia PJ, Kwapil TR. Aberrant asociality: How individual differences in social anhedonia illuminate the need to belong. *J Pers.* 2011;79:1013–1030.
51. Walker E, Kestler L, Bollini A, Hochman KM. Schizophrenia: etiology and course. *Annu Rev Psychol.* 2004;55:401–430.
52. Soliman A, O'Driscoll GA, Pruessner J, et al. Stress-induced dopamine release in humans at risk of psychosis: A [11C]raclopride PET study. *Neuropsychopharmacology.* 2008;33:2033–2041.
53. Soliman A, O'Driscoll GA, Pruessner J, et al. Limbic response to psychosocial stress in schizotypy: a functional magnetic resonance imaging study. *Schizophr Res.* 2011;131:184–191.
54. Pruessner JC, Dedovic K, Khalili-Mahani N, et al. Deactivation of the limbic system during acute psychosocial stress: evidence from positron emission tomography and functional magnetic resonance imaging studies. *Biol Psychiatry.* 2008;63:234–240.
55. Mohr C, Krummenacher P, Landis T, Sandor PS, Fathi M, Brugger P. Psychometric schizotypy modulates levodopa effects on lateralized lexical decision performance. *J Psychiatr Res.* 2005;39:241–250.
56. Horan WP, Blanchard JJ, Clark LA, Green MF. Affective traits in schizophrenia and schizotypy. *Schizophr Bull.* 2008;34:856–874.
57. Sheinbaum T, Bedoya E, Ros-Morente A, Kwapil TR, Barrantes-Vidal N. Association between attachment

- prototypes and schizotypy dimensions in two independent non-clinical samples of Spanish and American young adults. *Psychiatry Res.* 2013;210:408–413.
58. Fervaha G, Remington G. Neuroimaging findings in schizotypal personality disorder: a systematic review. *Prog Neuropsychopharmacol Biol Psychiatry.* 2013;43:96–107.
 59. Siever LJ, Koenigsberg HW, Harvey P, et al. Cognitive and brain function in schizotypal personality disorder. *Schiz Res.* 2002;54:157–167.
 60. van der Meer L, Groenewold NA, Pijnenborg M, Aleman A. Psychosis-proneness and neural correlates of self-inhibition in theory of mind. *PLoS One.* 2013;8:e67774.
 61. Lenzenweger MF, McLachlan G, Rubin DB. Resolving the latent structure of schizophrenia endophenotypes using expectation-maximization-based finite mixture modeling. *J Abnorm Psychol.* 2007;116:16–29.
 62. Matthews SG. Early programming of the hypothalamo-pituitary-adrenal axis. *Trends Endocrinol Metab.* 2002;13:373–380.
 63. Blanchard JJ, Aghevli M, Wilson A, Sargeant M. Developmental instability in social anhedonia: an examination of minor physical anomalies and clinical characteristics. *Schizophr Res.* 2010;118:162–167.
 64. Mittal VA, Dhruv S, Tessner KD, Walder DJ, Walker EF. The relations among putative biorisk markers in schizotypal adolescents: minor physical anomalies, movement abnormalities, and salivary cortisol. *Biol Psychiatry.* 2007;61:1179–1186.
 65. Gendron M, Roberson D, van der Vyver JM, Barrett LF. Perceptions of emotion from facial expressions are not culturally universal: evidence from a remote culture. *Emotion.* 2014;14:251–262.
 66. Kwapil TR, Crump RA, Pickup DR. Assessment of psychosis proneness in African-American college students. *J Clin Psychol.* 2002;58:1601–1614.
 67. Mason OJ, Medford S, Peters ER. Ethnicity, violent offending, and vulnerability to schizophrenia: a pilot study. *Psychol Psychother.* 2012;85:143–149.
 68. Ortuño-Sierra J, Badoud D, Knecht F, et al. Testing measurement invariance of the Schizotypal Personality Questionnaire-Brief scores across Spanish and Swiss adolescents. *PLoS One.* 2013;8:e82041.
 69. Chan RC, Shi YF, Lai MK, Wang YN, Wang Y, Kring AM. The Temporal Experience of Pleasure Scale (TEPS): exploration and confirmation of factor structure in a healthy Chinese sample. *PLoS One.* 2012;7:e35352.
 70. Chan RC, Wang Y, Huang J, et al. Anticipatory and consummatory components of the experience of pleasure in schizophrenia: cross-cultural validation and extension. *Psychiatry Res.* 2010;175:181–183.
 71. Kwapil TR, Barrantes-Vidal N. Schizotypy: looking back and moving forward. *Schizophr Bull.* 2015;41(suppl 2):S366–S373.
 72. Fonseca-Pedrero E, Paino M, Lemos-Giráldez S, et al. Schizotypy assessment: State of the art and future prospects. *Int J Clin Health Psychol.* 2008;8:577–593.
 73. Mason OJ. The assessment of schizotypy and its clinical relevance. *Schizophr Bull.* 2015;41(suppl 2):S374–S385.
 74. Wuthrich VM, Bates TC. Confirmatory factor analysis of the three-factor structure of the schizotypal personality questionnaire and Chapman schizotypy scales. *J Pers Assess.* 2006;87:292–304.
 75. Lenzenweger MF. Thinking clearly about schizotypy: hewing to the schizophrenia liability core, considering interesting tangents, and avoiding conceptual quicksand. *Schizophr Bull.* 2015;41(suppl 2):S483–S491.
 76. Ruscio J, Walters GD, Marcus DK, Kaczetow W. Comparing the relative fit of categorical and dimensional latent variable models using consistency tests. *Psychol Assess.* 2010;22:5–21.
 77. Myin-Germeys I, Krabbendam L, van Os J. Continuity of psychotic symptoms in the community. *Curr Opin Psychiatry.* 2003;16:443–449.