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Schizotypy, Schizotypic Psychopathology, and Schizophrenia: Hearing Echoes, Leveraging Prior Advances, and Probing New Angles

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The nature and definition of schizotypy, as the latent liability for schizophrenia capable of generating various phenotypic and endophenotypic outcomes, is reviewed. The proceedings of the 2017 meeting of the *International Consortium on Schizotypy Research* are included in this *Special Section* and they are presented as illustrations of current research work on schizotypy. The potential leverage of the schizotypy framework for schizophrenia research continues to be realized and these articles present current research efforts that explore new angles of inquiry while building upon past advances. Methodological and substantive areas of concern are highlighted and suggestions for improvement of future schizotypy research are made.

Key words: schizotypy/schizophrenia/liability/latent structure/schizotype

The value of schizotypy as an organizing framework for schizophrenia research[1,2] continues to reveal itself through laboratory studies. The proposal that schizotypic psychopathology represents an alternative manifestation of schizophrenia liability[3] resonates clearly and is gaining substantial traction (a view echoed in the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* [*DSM-5*] through placement of schizotypal personality disorder [SPD] with schizophrenia).

With this *Special Issue* on schizotypy, we are presented with a rich collection of articles that emerged from the 2017 meeting in Beijing (China) of the relatively newly formed *International Consortium on Schizotypy Research*. This sampling reveals that schizotypy research continues to thrive in the laboratories of a new generation of scientists that are leveraging prior advances and probing new angles while attending to theoretical and empirical echoes from the past 100 years. These reports further illuminate the heuristic potential of schizotypy as a psychopathology research vector in schizophrenia studies. The *Bulletin* reader that typically focuses only on the expressed illness of schizophrenia as a unit of analysis is invited and encouraged to sample these articles most of which share the goals of extracting meaning and direction from empirical data for understanding the full schizophrenia spectrum.

Background and Context

In my 2010 monograph on schizotypy,[4] I had the opportunity to explore the long history of the schizotypic psychopathology/schizophrenia connection, reviewing insights from the phenomenological masters that I had been inspired by nearly 30 years earlier. Both Kraepelin[5] and Bleuler[6] spoke to this connection when describing the relatives of schizophrenia patients, to wit:

> ... in the families attacked there comes under observation with relative frequency besides dementia praecox a series of other anomalies, especially manic-depressive insanity and *eccentric personalities* [italics added]. ... the latter are probably for the most part to be regarded as "latent schizophrenias" and therefore essentially the same as the principal malady. (Kraepelin[5])
>
> There is also a latent schizophrenia, and I am convinced that this is the most frequent form, although admittedly these people hardly ever come for treatment. ... In this form we see *in nuce* all the symptoms and all combinations of symptoms which are present in the manifest types of the disease. (Bleuler[6])

These guiding conjectures, proffered over 100 years ago, subsequently influenced many of those tangling with the schizotypic psychopathology/schizophrenia connection.[7] Deriving from detailed phenomenological observation, based on direct experience with patients with clinical schizophrenia, and bold efforts at rich theoretical integrations, schizotypy research was effectively launched in earnest by Rado[8] and Meehl.[9] Schizotypy, largely attributed

to Meehl,[9,10] refers to a latent (underlying) psychological organization that putatively harbors the liability for schizophrenia. Thus, there is a clear-cut and intimate connection between schizotypy as a construct and the liability for a clinical illness, namely schizophrenia. Schizotypy, therefore, is *not* akin to a normative personality dimension such as extraversion, conscientiousness, or agreeableness, rather it derives its meaning from an illness. Schizotypy, which is determined by any number of as-yet-unknown schizophrenia-related genetic influences acting against a background of polygenic assets and liabilities as well as impacts from the environment (eg, stressors, epigenetic inputs), can manifest itself phenotypically variously, ranging from clinically diagnosable schizophrenia through pathological personality manifestations (eg, schizotypal, paranoid, avoidant, and schizoid personality disorders [PDs]) to subtle, subclinical psychotic-like phenomenology (eg, perceptual aberrations, magical ideation, referential thinking, and interpersonal aversiveness). Schizotypy may also manifest itself in an imperceptible manner, undetectable by the unaided naked eye, through deviance on endophenotypes that have established valid relations with schizophrenia. Moreover, schizotypy as a latent construct is centrally embedded in a diathesis-stressor theoretical model that has considerable utility as an organizing framework for the study of schizophrenia, schizophrenia-related psychopathology (eg, delusional disorder, psychosis-not otherwise specified (NOS) schizotypal, paranoid, and other related personality disorders) as well as putative schizophrenia endophenotypes, a view I have advocated for several decades[2–4,11–13] (see Supplementary Figure 1). Note, the term schizotypy is *not* restricted to describe only those clinical manifestations that are associated with SPD, eg, rather it is a broader construct (see references [4], [12], and [13] for detail). Nor is the term reserved to indicate a *methodological* preference such as self-report psychometric assessments. Schizotypy manifestations can be assessed using a variety of approaches such as interviews, psychometric inventories, familial risk, and/or laboratory measures. Schizotypic persons may indeed display some of the phenomenology associated with SPD, but they may also show other indications as well.[13–15] (In this context, I also note that although embraced by many theoreticians and researchers, the endophenotype concept is *not* universally accepted and the field awaits definitive data on the validity of this construct.)

On the Value of a Schizotypy Model Grounded in Schizophrenia: Empirical Rungs on the Schizotypy–Schizophrenia Ladder

Unraveling the connections among schizotypy as a latent construct and its phenotypic manifestations (see the Supplementary Figure) necessarily engages a substantive model that serves a foundational function for an emerging web of predicted empirical relations anchoring schizotypic indicators to schizophrenia. The synergistic interplay between theory and data in attempts to understand schizophrenia liability (ie, schizotypy) has long guided this effort. For example, this interplay was illustrated, completely by happenstance, in the October 1989 issue of the *Archives of General Psychiatry* (now *JAMA Psychiatry*) through something of a schizotypy hat trick. Meehl[16] provided a synopsis of his current views on schizotypy in relation to schizophrenia. Gottesman and Bertelsen[17] presented their seminal findings confirming the existence of unexpressed genotypes for schizophrenia, a view inspired in part by the schizotypy model of Meehl. Lenzenweger and Loranger[18] demonstrated that elevated levels of schizotypy deviance in previously nonpsychotic individuals predicted increased morbid risk for treated schizophrenia in their first-degree biological relatives, as one would predict from the schizotypy model. The theory and data dance across those three articles reflected the synergism, and that theory/data synergism is seen in this *Special Issue* too.

In the ensuing 30 years post-1989, working from a model that argues schizotypic deviance should reveal itself in laboratory studies in a manner consistent with what is known about schizophrenia, we now know that indicators of schizotypy have been associated with a wide range of schizophrenia-related findings (with schizotypy defined variously using clinical, laboratory, and/or familial risk) including increased risk for schizophrenia, sustained attention deficits, working memory deficits, smooth pursuit eye movement dysfunction, Minnesota Multiphasic Personality Inventory (MMPI) schizophrenia-related psychometric deviance, executive functioning deficits, dysfunctional antisaccade performance, subtle formal thought disorder, clinical schizotypal and paranoid personality features, schizophrenia-related social cognition deficits, exteroceptive and proprioceptive somatosensory deficits, psychomotor abnormalities, and candidate polymorphisms (eg, ZNF804A, Val158Met-COMT, neuregulin-1, with more genes surely to come).

An area of continued speculation concerns the underlying structure of schizotypy and the precise nature of the variation expressed in that latent construct. In my view, considerable statistical evidence (over 3 dozen studies), derived from numerous empirical investigations using a variety of latent structure methods (taxometric analysis, finite mixture modeling, latent class analysis), points to the existence of possible underlying discontinuities *or* severe threshold effects in the latent structure of schizotypy. Such an abundance of quantitative evidence stimulates the methodologic–substantive caveat: The use of continuous measures to assess *phenotypic* manifestations of schizotypy should not be taken to confirm that the *latent* (underlying) schizotypy construct (or unfolding schizotypic disease process) is fully quantitative or uniformly graded by degree. Whether or not schizotypy is fully quantitative at the latent level is an empirical question and can only be answered with proper statistical

methods with probative value (*caveat venditor*: Factor analysis cannot answer this question); it is not one of methodological preference or *zeitgeist*.

The long-term course and clinical outcome for those designated as harboring schizotypy remains an area of active inquiry. It is entirely conceivable that many individuals possessing schizotypy may traverse the life-course, escaping psychotic illness as well as other diagnosable schizotypic manifestations. The expectation that some people, though validly at risk for schizophrenia, may never manifest the illness is well established and buttressed by the reality of schizophrenia outcomes in the offspring of discordant monozygotic twins (ie, the classic Gottesman and Bertelsen[17] study noted earlier), in which one twin is affected by schizophrenia and the co-twin is not psychotic (perhaps not even diagnosable as having a nonpsychotic, but detectable, clinical schizotypic condition such as schizotypal or paranoid PD). In long-term follow-up (greater than 15 years), individuals that achieve elevated scores on psychometric measures of schizotypy have been shown to be at increased risk for schizophrenia-related psychoses later in life as well as a variety of other related outcomes (M.F.L., unpublished data, 2018). Such individuals also display poorer psychosocial functioning, lower rates of marriage, increased use of psychiatric medications, and increased utilization of psychiatric services (M.F.L., unpublished data, 2018). It is entirely conceivable that many individuals designated as "prodromal" for schizophrenia, but who do not convert to schizophrenia (which is 60%–70% of such subjects), are in fact harboring schizotypy and will, even if not psychotic, show impairments across the life span, perhaps adopting an eccentric or odd manner of interpersonal functioning.

The schizotypy model has also helped to adjust the phenotypic boundaries of schizophrenia phenotype in the *DSM-5* as noted earlier (eg, schizotypal pathology is now included with schizophrenia). Furthermore, illuminating the nature of schizotypy more deeply may aid in unraveling the current puzzle of the very low conversion to schizophrenia rates seen in "prodromal" schizophrenia research.[19] Finally, I have argued that the schizotypy framework may be useful in understanding *configurations* (rather than simple additive summation) of genes relevant to schizophrenia variants[4(pp234–235)], an idea that is beginning to gain traction. There is no doubt that incorporation of schizotypy indicators into genomic studies of schizophrenia increases their statistical power, principally by properly defining the phenotypic spectrum that corresponds with fidelity to the breadth of the underlying liability. The advantages of a cleaner unit of analysis (the schizotype) free from the effects of medication, institutionalization, and neurocognitive decline are axiomatic. However, the understanding (and misunderstanding) of the schizotypy model as well as alternative approaches to the construct require vigilance in order to ensure the approach continues to yield the fruit that it can.

Reflections on the Special Issue Articles

There is a range and diversity of articles in this *Special Issue*. My intent is not to review these articles, rather I raise points of interest, reflection, and/or suggestions for future study. Docherty et al announce the creation of their data-sharing initiative and it helps to set the tone for the collaborative nature of the articles in this issue as well as bodes well for cross-laboratory work on schizotypy in the future. The Fonseca-Pedrero et al article illustrates the value of such a data-sharing approach. In their article, a team of 33 authors share their data and explore the cross-sectional correlation relationships in a large dataset of Schizotypal Personality Questionnaire (SPQ) scales using the emerging statistical modeling approach known as network analysis. The Fonseca-Pedrero et al results highlight the interactive nature of underlying schizotypic dimensions within the SPQ indicator space, results that accord well with clinical realities seen in schizotypes, the factorial structure of the SPQ, and what is known about the factorial structure of schizophrenia symptoms. Future research will be required to adequately probe whether any factors/items in this indicator space indeed have a genuinely causal relationship to any other, mindful of the limitations imposed by cross-sectional correlational data. Irrespective of network modeling parlance (eg, "causal correlations"), readers will necessarily remind themselves that correlation does not imply causation.

Armando et al have sought to pull in the important psychosocial vector of stressful life events (SLE) as well as coping strategies grounded in their unit of analysis the 22q11.2 Deletion Syndrome, which they postulate as a genetically influenced model for psychosis pathogenesis. Future advances in this realm will hinge critically on the *specificity* of the relations between SLE and negative coping strategies to schizotypy vs many other forms of psychopathology. Derome et al explore the impact of depersonalization (an alteration of conscious experience of the self), artificially induced through a mirror gazing task, on resting-state networks using resting-state neuroimaging (resting-state functional magnetic resonance imaging) assessments. This is an interesting line of inquiry given the close connection between depersonalization and anxiety as well as the profound and disorganizing levels of anxiety often reported by schizotypes, indeed anxiety so profound as to lead to what Meehl termed interpersonal aversiveness. Their data raise several questions: (1) How would task-induced depersonalization differ from genuine clinical depersonalization that is naturally occurring (the validity of the analog issue)? and (2) Would other measures of near-neighbor constructs be correlated similarly with resting-state findings as well (eg, anxiety, depression, dissociation, derealization, post-traumatic stress phenomena)? In another connectivity-oriented study, Wang et al report on findings linking negative schizotypy in a facial emotion processing task with a focus on associated reduced functional activity in the amygdala and

reduced connectivity of the amygdala with the medial prefrontal cortex (mPFC). The connectivity/activation findings from this imaging study are interesting; however, we must remind ourselves that they were found within a context in which there was *no* difference between study groups on the focal behavioral task (a facial emotional valence discrimination task). This pattern (absence of behavioral task differences) is not unusual in functional neuroimaging psychopathology research today and sets the stage necessarily for another follow-up experiment to help nail down their explanation of the data. To be clear, the interpretation offered by Wang et al is heuristic and in need of further study. A future study might also employ more ecologically valid measures for the study of social cognition deficits and interpersonal functioning dysfunction using more lifelike stimuli (eg, video).

Steffens et al take a review focus on cognitive control (parsed as updating, shifting, and inhibition components) and the subdimensions of schizotypy measures in a meta-analysis. The observed variation in correlations across the positive, negative, and disorganized schizotypy components in relation to updating, shifting, and inhibition emphasizes the importance of examining relationships between differing aspects of cognition and these different facets of schizotypy. Madsen et al join the two powerful tools of functional neuroimaging with machine-learning methods in a novel classification effort. Although their study schizotypy sample is relatively small (N = 14), defined only in terms of social anhedonia, and activated by a social stimulus (a comic strip), the findings are indeed stimulating and suggestive for further exploration. The use of rigorous computational methods to try to discern order in data that may have classification value is indeed worthy of the effort and echoes earlier work using discriminant function and other statistical techniques using quantitative data to achieve a comparable classification goal.

Lui et al have approached the classification issue from a more traditional perspective and have used the familial/biological approach[3] in assembling their general study sample, namely first-degree biological relatives of individuals diagnosed with schizophrenia. This research is clearly motivated by the heterogeneity challenge in the study of schizotypy. What Lui et al demonstrate effectively is that considerable heterogeneity exists within schizotypy, just as it exists within schizophrenia. Their choice of dependent variables for study echoes the well-established neurocognitive measures including, but not limited to, many we studied in relation to schizotypy some time ago (Wisconsin Card Sorting Test (WCST),[20] sustained attention,[21] and working memory[22]). An issue of potential concern with this effort is that any cluster analytic study of relatives must always take into account the relatedness of the individuals sampled (typically more than one relative comes from a single family) and the use of classification methods that can or cannot tolerate such nonindependence (K-means clustering assumes independence of observations and relatives from the same families are *not* independent of one another). Resolving heterogeneity, whether related to features or laboratory performance measures, remains a worthy goal in ongoing schizotypy research. In our laboratory, we have sought to develop statistical methods to handle such dependencies in data through the development of a hierarchical finite-mixture model that accommodates nonindependence as well as other data features often found in the study of first-degree relatives.[23] With regard to the utility of resolving subgroups of relatives within a larger sample, it will be important for future research to illustrate the gains achieved by using *across* subgroups, rather than dimensional scores *within* persons. Schizophrenia research largely abandoned the "positive vs. negative vs. mixed" *type* approach in favor of a dimensional of pathology model in which reality distortion and disorganization (positive symptoms) and negative symptoms were assessed within every person. The latter dimensional approach follows the seminal contributions of Strauss et al[24] as well as Crow.[25]

Finally, the Grant et al article provides a cursory overview of different conceptualizations of schizotypy. Does it represent a normal personality trait or does it represent an indicator of schizophrenia liability? What is the nature of the underlying structure of schizotypy (quantitative vs qualitative latent structure)? Is it an asset or liability? The diminished relevance of Eysenck's model for understanding schizotypy and schizophrenia is correctly suggested as his psychoticism construct bears no persuasive substantive or empirical connection to schizophrenia liability. Grant et al also succinctly provided their understanding of Claridge's view on schizotypy, one that postulates schizotypy as part of normal personality, continuous in underlying nature, and yielding conceptualizations such as so-called "happy schizotypes," "healthy psychosis," "psychotic traits [that] constitute an essentially healthy dimension of personality," and "benign schizotypy" (BS). Regular readers of *Schizophrenia Bulletin* might juxtapose the many "*First Person Accounts*" found in the *Bulletin* with the notion of "healthy psychosis" to evaluate the plausibility of this conjecture. The Claridge BS model, as is well known, argues for latent continuity in the schizotypy construct, largely inferred from continuous measures of *phenotypic* features of schizotypy and related factor analytic studies. This model, in my view, unfortunately fails to take into account a large corpus of countervailing empirical *latent* structure studies.

Grant et al also provide a brief general overview of Meehl's model of schizotypy, though not without shortcomings. Meehl's model has clearly been the substantive engine driving most schizotypy research over the last 50 years, particularly in experimental psychopathology. However, as I noted in 2006,[26] despite the impact of his propositions and the attention his model garnered, Dr. Meehl frequently remarked to me (and others, I am told reliably) that he felt that many in the fields of clinical

psychology and psychiatry did not take the time to truly understand his model of schizotypy, noting that many people frequently "get it wrong!" Those interested in a full and rich explication of his model are directed to his opus on the topic.[10] To clarify vis-à-vis the Grant et al article, Meehl himself rejected the notion that his model was "quasi-dimensional." He saw schizotypy as yielding different phenotypic outcomes, all of which were valid representations of the underlying taxonically structured liability. He did *not* view these phenotypic outcomes as arrayed in some dimensional manner, quasi or otherwise. An illustration from another perspective might be helpful here. Consider Von Recklinghausen's neurofibromatosis, which is a disease resulting from a single major locus gene that shows various expressions phenotypically. The disease can reveal itself in a full syndrome (elephant man disease), albeit less common, or it can be more commonly revealed as tan spots on the skin, areas of depigmentation, and skeletal abnormalities. The appearance of such mild symptoms in neurofibromatosis does *not* make it a *quasi-dimensional* model, rather all of these phenotypic outcomes reflect an underlying taxonic entity (single major locus gene). By way of necessary correction, Grant et al appear to have misunderstood an assertion by the present author regarding the compatibility of Meehl's model with a polygenic formulation for genetic influences. Lenzenweger argued that Meehl's "cascade of processes and outcomes in the model is entirely compatible with *multiple* genes contributing to the underlying schizotaxic brain/neural pathology … and could incorporate a *threshold effect* [italics added][12(pS489)]." The concept of a threshold (cut-off) effect in an underlying continuum of liability is crucial to properly understanding this plausible alternative view of liability structure that would make it broadly compatible with Meehl's developmental model. Indeed, the existence of a threshold effect, advocated strenuously by Gottesman over the decades, in the multifactorial polygenic threshold model of the genetics schizophrenia is widely accepted, whereas a simple polygenic model without a threshold is generally not. As the years went by, Meehl himself agreed that the evidence for multiple gene involvement in schizophrenia liability was strong and, importantly, was aware of the utility of the threshold (cut-off) assumption (P. E. Meehl, personal communications).

Parting Reflections: Seeking Clarity Moving Forward

This collection of articles is rich with diversity, insight, and creativity. I offer a few parting reflections. These comments, to my mind, do not merely reflect "inside baseball," but rather each of the following issues touches on a critical theoretical or methodological issue for fruitful future development of schizotypy research.

1. The term "schizotypy" is used loosely at times in the psychopathology research literature. We must bear in mind that schizotypy gives rise to schizotypic psychopathology, and schizotypal personality is but one possible manifestation of schizotypy. Moreover, the terms schizotypy, schizotypic, and schizotypal are not fungible as they refer to different levels of scientific analysis.
2. Future schizotypy studies should venture to incorporate a third subject group in all study designs in order to evaluate the specificity of obtained group differences on dependent variables of interest (ie, the well-known psychiatric control group). Of course, if one is a committed "dimensionalist," then one should *not* create subject groups at all in studies and all data should be analyzed within a correlational/regression/individual differences framework.
3. Going forward, it makes good sense to study all three subdimensions of schizotypic features that correspond broadly to reality distortion, disorganization, and negative features in schizophrenia. I would recommend the inclusion of some measure of social relations as well in addition to the 3 well-known symptom dimensions.[27]
4. A major hole in the schizotypy research corpus concerns long-term longitudinal outcome data. Future long-term follow-ups of 15 years or more would be most welcome. We hope to publish our 17-year follow-up data on schizotypy in the near term (M.F.L, unpublished data, 2018).
5. Future studies that do employ case–control designs and rely upon the use of comparison of means on dependent measures of interest should abandon the old-fashioned, unfocused-ANOVA approach and adopt the method of contrast analysis, which confers both greater statistical power and theoretical precision to such statistical analyses.
6. Conceptual clarity is needed when formulating schizotypy research. Future workers should endeavor to make clear their substantive position on the nature of schizotypy. Schizotypy, as traditionally defined in psychopathology and appreciated in normal personality science, is *not* a normal personality trait *per se*. Schizotypy, rather, represents the construct capturing the underlying liability for a disease state, namely schizophrenia. Reports that simultaneously describe schizotypy as (1) a personality trait, (2) a disease liability, (3) a risk enhancer, (4) a symptom or sign, and/or (5) a personality disorder run the risk of sowing confusion as well as appearing confused.

Supplementary Material

Supplementary data are available at *Schizophrenia Bulletin* online.

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