Contemporary classification is striving for simplicity and reliability, with much research being performed by for-the-purpose-trained lay interviewers. The disappointment with the slow progress of pathogenetic research encourages critical voices advocating abandonment of phenotypic categories altogether. However, the story of self-disorder research may inspire us to reconsider the phenotypic classification with a more refined psychopathological approach.

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1. Parnas J, Henriksen MG. Harv Rev Psychiatry 2014;22:251-65.
2. Bleuler E. Dementia praecox oder Gruppe der Schizophrenien. Leipzig: Deuticke, 1911.
3. Parnas J. World Psychiatry 2012;11:67-9.
4. Stephensen H, Parnas J. J Phenom Cogn Sci (in press).
5. Jaspers K. Allgemeine Psychopathologie, 1st ed. Berlin: Springer, 1913.
6. Nordgaard J, Nilsson LS, Sæbye D et al. Eur Arch Psychiatry Clin Neurosci (in press).
7. Parnas J, Henriksen MG. Conscious Cogn 2016;43:75-88.
8. Henriksen MG, Parnas J. Schizophr Bull 2013;40:542-7.
9. Zandersen M, Henriksen MG, Parnas J. J Pers Disord (in press).
10. Zandersen M, Parnas J. Schizophr Bull (in press).

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The schizophrenia spectrum anhedonia paradox

Anhedonia, defined as a diminished capacity to experience pleasure, has been considered a core symptom of schizophrenia since the earliest descriptions of the disorder. It is longitudinally stable and associated with a range of poor clinical outcomes[1]. Unfortunately, interventions targeting this symptom have produced minimal benefits, and no drug has received US Food and Drug Administration's approval for this indication.

Limited progress in effectively treating anhedonia results in part from a lack of conceptual clarity regarding the nature of the symptom. Evidence for anhedonia in schizophrenia has primarily come from data obtained via clinical interviews, which indicate that the majority of those diagnosed with that disorder are anhedonic. Clinicians have long assumed that such self-reports indicate that individuals with schizophrenia have a diminished capacity to experience positive emotion. However, laboratory-based studies provide evidence that contradicts this notion, indicating that schizophrenia patients self-report as much positive emotion as healthy controls in response to pleasant stimuli[2] and show intact neurophysiological responses in key reward circuits during receipt of rewarding outcomes[3].

It has been argued that this apparent discrepancy can be resolved if one examines the anchors and probes used in negative symptom interviews[4]. Upon careful inspection, it is clear that what interviewers are rating is the frequency of reward-seeking behavior, rather than the extent to which patients enjoy pleasurable activities when engaged in them. Based on this evidence, as well as on results from ecological momentary assessment studies, the field has gradually shifted away from the view that schizophrenia patients have a reduced hedonic capacity. Rather, schizophrenia appears to be associated with a behavioral deficit characterized by a reduction in the frequency of pleasurable activity[4].

The disconnect between behavior and hedonic capacity has been termed the "liking-wanting anhedonia paradox", and spurred research attempting to determine why apparently normal hedonic responses do not translate into motivated behaviors aimed at obtaining rewards in schizophrenia. Several conceptual models attempted to answer this question, proposing that impairments in various aspects of reward processing (e.g., reinforcement learning, value representation, effort-cost computation, reward anticipation), that rely on cortico-striatal circuitry, prevent fully intact hedonic responses from influencing decision-making processes needed to guide action selection and initiate motivated behavior[4]. These models have received significant empirical support and are beginning to influence the development of treatments targeting these underlying mechanisms.

However, there is a second "anhedonia paradox" that has emerged over recent years. We refer to this as the "schizophrenia spectrum anhedonia paradox". Specifically, there is growing evidence that, although patients with schizophrenia have intact hedonic capacity[4], individuals with schizotypy and youth in the prodromal phase of illness do not. People with schizotypy self-report less positive emotion in response to pleasant stimuli than healthy controls and show reduced neurophysiological response during the receipt of reward outcomes[5]. Youth at clinical high risk for psychosis also have diminished neurophysiological and self-reported responses to pleasant stimuli[6]. Since schizophrenia is a more severe form of psychopathology in nearly every conceivable way, this apparent discrepancy is paradoxical: why would the less severe forms of pathology show deficits in hedonic capacity, whereas the more severe form does not? Below we discuss some plausible explanations, hoping to promote future studies aimed at resolving this paradox.

A first possibility is that mood and anxiety symptoms produce diminished hedonic response in schizotypy and clinical high risk youth more than in schizophrenia. Consistent with this notion is evidence indicating that youth at clinical high risk for psychosis and those with schizotypy have higher rates of comorbid depression and anxiety than people with schizophrenia, and that greater severity of depression and anxiety is associated with reduced hedonic response in those individuals[6].

A second possibility is that antipsychotics have a normalizing effect on reward processing. Studies examining the neural response to rewarding stimuli in schizophrenia suggest that second generation antipsychotics are associated with intact response to reward outcomes in the ventral striatum[7]. Since individuals with schizotypy and youth at clinical high risk for psychosis are much less likely to be prescribed antipsychotics, the apparent paradox may reflect medication effects that become evident with more severe pathology.

Third, schizophrenia is associated with more severe cognitive impairment and poorer insight into clinical symptomatology than schizotypy or clinical high risk states. It is possible that impaired cognition and insight are paradoxically protective, causing schizophrenia patients to have less awareness of hedonic deficits that may actually exist. Those with schizotypy and clinical high risk youth may be better able to accurately report their hedonic state because of higher cognitive function and insight.

Fourth, environmental and stress effects may have a greater impact on youth at clinical high risk for psychosis and those with schizotypy. Schizophrenia is associated with impoverished quality of life, which for many patients reflects an environment and daily routine with restricted social, cognitive and affective demands. For individuals with schizotypy and clinical high risk youth, environments and daily routines are generally more complex and stressful. It is possible that this stress attenuates reward system responsivity. Supporting this, individuals with schizotypy seem to enjoy solitary activities, yet report activities with others as being taxing, stressful and unenjoyable[8]. Animal models and studies on humans support the notion of a "stress-induced anhedonia"[9]; however, this phenomenon has yet to be directly investigated in the schizophrenia spectrum.

The schizophrenia spectrum anhedonia paradox harkens back to the seminal writings of P. Meehl[10], who proposed that anhedonia is one of several polygenic potentiators that comprise the endophenotype for schizotaxia. Meehl distinguished between primary and secondary anhedonia. Primary anhedonia refers to one's hedonic capacity. This capacity is polygenically determined and dependent on neurotransmitter function and neural circuitry responsible for reward responsivity. Capacity varies on a continuous dimension, but may be taxonic at the extreme end. It seems that only a small proportion of schizophrenia patients would fall at the extreme end of the continuum, whereas substantially more individuals with schizotypy and clinical high risk youth would display primary anhedonia. Primary anhedonia may be a risk factor for the development of many forms of psychopathology, with schizophrenia being a less common outcome than others.

Meehl also proposed the existence of "secondary anhedonia", which is measured via verbal report (through clinical interview or questionnaire) of one's hedonic response. He proposed that such reports are influenced by "aversive drift", which reflects heightened trait negative affect that becomes increasingly more prominent as the course of illness progresses. The aversive drift construct has yet to receive significant empirical attention; however, there is evidence from laboratory and ecological momentary assessment studies that those on the schizophrenia spectrum display elevated negative emotion that is contextually invariant[2]. Thus, even in instances where the neural machinery for hedonic response is intact, co-activation of negative emotion may bleed over into potentially rewarding situations, lowering the overall net hedonic value of stimuli that would otherwise be rewarding.

Whether secondary anhedonia worsens with illness progression has yet to be determined. However, a greater frequency of secondary anhedonia in the chronic phase of schizophrenia than in earlier phases of illness could be another viable explanation for the schizophrenia spectrum anhedonia paradox.

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1. Herbener ES, Harrow M. J Abnorm Psychol 2002;111:237-48.
2. Cohen AS, Minor KS. Schizophr Bull 2010;36:143-50.
3. Radua J, Schmidt A, Borgwardt S et al. JAMA Psychiatry 2015;72:1243-51.
4. Kring AM, Barch DM. Eur Neuropsychopharmacol 2014;24:725-36.
5. Cohen AS, Callaway DA, Najolia GM et al. J Abnorm Psychol 2012;121:407-15.
6. Gruber JM, Strauss GP, Dombrecht L et al. Schizophr Res (in press).
7. Juckel G, Schlagenhauf F, Koslowski M et al. Psychopharmacology 2006;187:222-8.
8. Kwapil TR, Silvia PJ, Myin-Germeys I et al. J Res Pers 2009;43:103-6.
9. Pizzagalli DA. Annu Rev Clin Psychol 2014;10:393-423.
10. Meehl PE. J Abnorm Psychol 2001;110:188-93.

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Peer delivered services in mental health care in 2018: infancy or adolescence?

Peer support is now considered to be a central component of the behavioral health care system in countries such as the US, Canada, Australia and the UK. Professionals looking to improve their ability to promote recovery have strategies and training programs that include collaborating with peers in their services (e.g., Boston University's Recovery Promoting Competencies Toolkit).

In 2012, Davidson et al[1] characterized in this journal peer delivered services as being still in their infancy. They pointed out that, while there was a proliferation of peer support work-