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Reward Function: A Promising but (Still) Under-Examined Dimension in Developmental Psychopathology

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The goal of this Special Section is to explore the ways that investigation of reward function can shed light on the development and pathophysiology of psychopathology. Reward function provides a promising starting point for clinical affective neuroscience research because, thanks to the extensive literature on the neural mechanisms of addiction, the functional neuroanatomy, cellular mechanisms, and genetic contributions to reward circuitry have been well delineated (see Russo & Nestler, 2013, for details). This knowledge has created a foundation for investigating the role of disrupted reward function in various other forms of psychopathology, beyond addiction. Among forms of psychopathology, depression has been perhaps the most frequently studied from this perspective. As illustrated by a recent meta-analytic review of 22 fMRI studies of major depressive disorder, altered neural response to reward in depression has been examined fairly widely, with remarkably consistent findings despite differences in fMRI paradigms and depression indices across studies (Zhang, Chang, Guo, Zhang, & Wang, 2013). This solid empirical literature is consistent with the longstanding conceptual view that depression involves a disruption of positive affect systems (e.g., Clark & Watson, 1991; Fowles, 1988; Gross & Muñoz, 1995; Forbes & Dahl, 2012; Phillips, Drevets, Rauch, & Lane, 2003). Developmental psychopathology perspectives on depression have also focused on reward, with strong conceptual foundations (e.g., Davey, Yücel, & Allen, 2008), although a more modest empirical literature on youth relative to the literature on adult depression.

The motivation to publish this Special Section on Reward Function and Developmental Psychopathology was driven by our perception of strong interest in the topic and a growing literature. We sought to highlight rigorous research on reward function in psychopathology, across a range of disorders, extending this examination of reward function across the field and suggesting new directions. After a promising start, with articles submitted on a variety of clinical populations, only two met the very high standards for publication in the *Journal of Abnormal Psychology*. Both articles contribute to our understanding of depression.

The articles in this Special Section report on findings indicating that altered response to reward is evident in young people at risk for depression by virtue of their mothers' history of depression. Together, these articles highlight the importance of considering altered reward responding as a potential endophenotype of depression (Hasler, Drevets, Manji, & Charney, 2004), evident before onset of the disorder or in those who are vulnerable but may never go on to develop the disorder. Both studies also build upon the extensive literature on maternal depression as a risk factor for problems in youth (see Goodman, 2007 for a review). Despite

the similar conclusions and implications, the two studies use different methods to capture different reward constructs: Kujawa, Proudfit, and Klein (in press) focus on feedback negativity, an ERP component thought to reflect reward learning (Walsh & Anderson, 2012), while Sharp et al. (in press) investigate fMRI of appetitive and consummatory reward processes. We commend both for their well characterized, difficult-to-obtain samples and their rigorous methods. The findings of these two articles are consistent with each other in that they both report blunting of reward-related physiologic responses (feedback negativity for Kujawa et al., ventral striatal response for Sharp et al.). Breaking new ground, Kujawa et al. focused on participants in middle childhood, attempted to distinguish between depression and anxiety, and examined possible distinctions between mothers' and fathers' history of psychopathology as a risk factor. Sharp et al. extended the growing literature on reward responding in adolescent offspring of depressed parents (Gotlib et al., 2010, Monk et al., 2008; Olino et al., 2013) by assessing not only a high-risk group of daughters who had never been depressed but also a high-risk group with current depression.

Both of the studies in this Special Section point to the important connection between mothers' psychopathology and altered physiology in their children. It is remarkable, for example, that both studies found that *severity* (not simply the presence or absence) of mothers' depression was associated with youths' neural responses to rewarding stimuli. To understand the ways that a risk factor translates into psychopathology, the obvious next step —and one included in too few studies of risk for depression to date—would be to examine associations between children's physiology and their own symptoms. It is puzzling, therefore, that neither of these studies found an association between physiology and either depressive severity or functioning *within* offspring themselves. Similarly, although it was not possible in these studies, it would be worthwhile to test whether altered physiologic responses mediate associations between maternal and offspring psychopathology. This null within-child association seems surprising when other studies have found that neural response to reward is associated with individual differences in symptom level in typical adolescents (Forbes et al., 2010), and that differences in striatal response between high-risk and low-risk youth are evident even after accounting for symptoms (Olino et al., 2013). Of course, differences in neural response could reflect risk rather than manifest problems, and it will be important to learn whether altered neural response to reward may be more predictive of problems at a later point in development than during childhood (for Kujawa et al.) or adolescence (for Sharp et al.). Contributing to questions on the role of fathers when mothers are depressed—or on the role of paternal psychopathology, a less-frequently examined risk factor than maternal psychopathology—Kujawa et al. also reported that blunted feedback negativity was related to mothers' history of depression but not fathers'.

These articles also remind us of the critical need to address comorbidity in this line of research. Kujawa et al.'s finding that feedback negativity was blunted in offspring of mothers with "pure" depression, but not those of mothers with both anxiety and depression, indicates that anxiety might trump depression in its influence on reward circuitry. In any case, findings with anxiety suggest that the alterations of frontostriatal response to reward may be in the opposite directions of those in depression (Guyer et al., 2012). Given that comorbidity is more the rule than the exception in psychopathology (Kessler et al., 2005),

inclusion of participants with well-characterized multiple forms of psychopathology will provide generalizable findings and therefore a strong path to knowledge about the role of disrupted reward function. And, as is evident from the focus of this Special Section, we believe that a developmental perspective is essential in general and also when investigating comorbidity. Epidemiologic findings underscore that because most disorders begin early in life, adult-onset disorders are typically comorbid and should be conceptualized as such (Kessler & Wang, 2008). From a developmental psychopathology view, investigating comorbidity invites consideration of several possible explanations (Drabick & Kendall, 2010).

With their common focus on depression risk and intriguingly overlapping findings, both articles in this Special Section raise a timely issue: whether disrupted reward function is an endophenotype of depression, with trait-like presence across risk status, episode status, and development. As these articles underscore, it appears to be present in those at risk as well as those with the disorder (see Zhang et al., 2013). Kujawa et al. point out that this alteration is evident even before the age of vulnerability for onset of the disorder. Sharp et al. report that high-risk girls have altered reward responding regardless of their own experience, as both currently depressed high-risk girls and never-depressed high-risk girls exhibited low ventral striatal response to the receipt of reward. In the literature on adult depression, disrupted reward function appears to be present in those who have remitted illness (Dichter, Kozink, McClernon, & Smoski, 2012). This pattern of findings supports a model in which reward function plays a compelling role in the pathophysiology of depression and points to the value of a dimensional approach to affective problems. Or does it? If this condition is evident in those who will never become mentally ill, or in those who are recovered and will not struggle with future episodes, what is its value? Even if it serves to distinguish those in the depression-vulnerable population from others, is it a malleable characteristic that can or should be targeted by treatment or preventive interventions? Ideally, these compelling questions will motivate future studies.

Summary and Future Directions

The focus on depression in the two articles in our Special Section closely reflects the state of the literature on reward function in psychopathology. Despite the strong conceptual framework and growing literature in depression, the affective neuroscience approach to studying reward in other disorders has been slow to emerge. Other disorders conceptualized as including affective disruption —bipolar disorder, anxiety disorders, substance use disorder, and borderline personality disorder, to name some obvious examples—would seem to be excellent candidates. For many other disorders—for example, schizophrenia, autism spectrum disorder, and attention deficit hyperactivity disorder—clinical neuroscience studies have generally focused on cognition rather than affective processes, even in the face of features that suggest affective disruption. At the same time, there is a growing literature on altered reward functioning in many of these disorders (e.g., Dichter, 2012; Dowd & Barch, 2010; Nusslock et al, 2010; Plichta & Scheres, 2014). As a result, the promise and opportunity for investigating reward function in many forms of psychopathology remains. We look forward to being able to publish the best of such work.

As we reflect on our work on this Special Section, we suggest some specific future directions. First, it will be critical to examine reward function in developmental psychopathology in terms of circuitry rather than in terms of response in specific regions. The burgeoning literature on resting state brain function in depression and other disorders has brought the value of this approach to the attention of developmental and clinical neuroscientists. Despite some differences in methods and findings (see Leibenluft & Pine, 2013 for a discussion), this area is worth pursuing. We are particularly enthusiastic about Leibenluft and Pine's suggestion to compile data across datasets, providing large samples for testing questions about resting function.

Second, it will be important to examine connections between differences in reward function and differences in behavior and experience. The two articles in this Special Section took important albeit modest steps in this direction, which can be extended more boldly to characteristics such as social functioning and affective behavior rather than exclusively self-reported experience. Similarly, examining the association of reward function with characteristics of clinical course—chronicity, age of onset, treatment history, number of episodes—will provide meaningful understanding of the mechanisms by which psychopathology develops. Namely, if those with a disorder show altered frontostriatal connectivity, for example, does this arise with experience of the disorder? Is it more pronounced in those with a greater family history density of that form of psychopathology? Is it weaker in those who have received certain pharmacologic or psychosocial treatments? And how do patterns of altered reward function interact with development, so that we might observe differences in these patterns at different points in the lifespan (aside from clinical history)?

Third, a valuable future innovation will be to examine disrupted reward function as a common feature across disorders. Anhedonia, for example, is a clinical feature of several classes of psychopathology and is developmentally intriguing because it emerges in adolescence and appears to precede several serious forms of psychopathology (see Horan, Kring, & Blanchard, 2006 for a discussion of this issue in schizophrenia). One possibility we have considered is whether disrupted reward function is a sign of more serious problems, worthy of investigation because of its prognostic role. Given the movement toward focusing on cross-disorder dimensions rather than phenomenological categories, embodied in approaches such as the NIMH Research Domain Criteria (RDoC) initiative (Cuthbert & Insel, 2013; http://www.nimh.nih.gov/research-priorities/rdoc/index.shtml), it is worth asking whether reward function might be disrupted even in psychopathology that is not typically considered "affective."

We are at an exciting and, at the same time, frustrating position in investigating the affective neuroscience of developmental psychopathology. For the study of positive affect, there are important clues to the disruption of neural circuits relevant to clinical problems. Yet, as in the research on clinical neuroscience in general, we still know little about the development, structure, and function of neural reward circuitry (see Pfeifer & Allen, 2012, for an argument against common oversimplifications and misunderstandings in this field). As discussed in a recent provocative and compelling conceptual paper by Hyman (2014), new approaches with more daring hypotheses, or even studies that are agnostic and proceed

without hypotheses, may push the field forward more quickly than the current hypothesis-driven approach. Alternatively, in the tradition of psychopathology research and its emphasis on testing hypotheses, we could look to research in other fields in an effort to find better starting points and generate innovative, unexpected hypotheses. Despite its limitations, this Special Section provides a valuable step toward our goal of understanding the development of psychopathology from an affective neuroscience perspective. The findings in these two articles offer new evidence for altered reward function as an early-emerging endophenotype of depression, suggest important associations between maternal depression and offspring physiology, and raise intriguing questions about the ways that depression develops in young people at risk for the disorder. We thank the authors for their contributions to developmental psychopathology, and we urge others to continue to submit their work on this fascinating and important topic.

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