Reward-related cognitive vulnerability to bipolar spectrum disorders

Bipolar spectrum disorders (BSDs) are characterized by extreme swings of mood (euphoria or irritability versus sadness), cognition (grandiosity and racing thoughts versus worthlessness and concentration difficulties), and behavior (supercharged energy and excessive goal-striving versus anhedonia and lethargy) occurring within the same individual. They are prevalent, associated with significant disability, and occur on a continuum of severity, from milder cyclothymia to bipolar II to full-blown bipolar I disorder.

Individuals' cognitive styles (i.e., the general filters they use to process information and construe events in their lives) may provide vulnerability to BSD symptoms and episodes. Indeed, individuals with BSDs exhibit cognitive styles with certain unique reward-relevant features[1,2] and these cognitive styles have been shown to predict the onset and course of BSDs[3]. According to the reward hypersensitivity theory[3,4], individuals with or vulnerable to BSDs possess a hypersensitive reward system, linked to a dopaminergic fronto-striatal neural circuit subserving approach motivation and goal-directed behavior, that overreacts to goals or reward-relevant cues. This hypersensitivity leads to excessive approach-related affect and incentive motivation in response to life events involving goal-striving and attainment, which in turn leads to hypomanic/manic symptoms. It also can lead to excessive downregulation or decrease in approach-related affect and motivation in response to non-attainment of goals or rewards (e.g., irreconcilable losses or failures), which in turn leads to bipolar depressive symptoms.

Thus, a propensity toward excessive reward system activation and deactivation is the hypothesized vulnerability to BSDs in this model. The model also proposes that vulnerable individuals' reward hypersensitivity leads to behaviors that increase their exposure (via "stress generation" processes) to the very goal- and reward-relevant events that, in turn, precipitate excessive responses from their reward systems. To date, extensive self-report, behavioral, cognitive, life event, neurophysiological and neural evidence supports this reward hypersensitivity model of BSDs[3,4].

High reward sensitivity may be a temperament trait that contributes to the development of reward-relevant cognitive styles[1]. In line with this hypothesis, euthymic bipolar individuals have been found to exhibit a distinctive profile of cognitive styles characterized by perfectionism, self-criticism and autonomy rather than the dependency and approval-seeking styles observed among unipolar depressed individuals[1]. Additionally, controlling for current mood symptoms, individuals with BSDs exhibit higher achievement motivation, goal-attainment dysfunctional attitudes (e.g., "A person should do well at everything") and ambitious goal-striving styles than controls[1,2].

The strongest evidence confirming that reward-relevant cognitive styles provide vulnerability to BSDs comes from a prospective study[5], which found that, controlling for initial mood symptoms and family history of bipolar disorder, adolescents with no prior history of BSD who exhibited an ambitious goal-striving cognitive style at baseline had a greater likelihood and shorter time to first lifetime onset of BSD than those without that cognitive style. Additionally, a cognitive style characterized by ambitious goal-striving mediated the predictive association between high self-reported reward sensitivity and shorter time to first onset of BSD in this adolescent sample[5], further suggesting that ambitious goal-striving is a vulnerability trait to BSDs that may account for some of the risk associated with reward sensitivity.

Ambitious goal-striving cognitive styles, perfectionism, and a tendency to overgeneralize from success (rewards) have also been observed in individuals with no prior history of BSD but at behavioral risk for developing a bipolar disorder[6,7]. Further, controlling for baseline hypomanic symptoms, a cognitive style to overgeneralize from success interacted with self-reported reward hypersensitivity to predict increases in hypomanic symptoms among adolescents with no prior history of BSD[8].

Reward-relevant cognitive styles also affect the course of BSDs. In individuals with bipolar I disorder, ambitious goal-striving for financial success and popular fame predicted increases in manic symptoms over a three-month follow-up[2]. In addition, controlling for past history of mood episodes and baseline symptoms, late adolescents with bipolar II disorder or cyclothymia who possessed self-critical or autonomous reward-relevant cognitive styles at baseline had a greater likelihood of hypomanic and manic episodes over a three-year follow-up than adolescents who did not exhibit these styles[1]. Moreover, an autonomous cognitive style mediated the predictive association between self-reported reward hypersensitivity and prospective occurrence of hypomanic and manic episodes in this sample[1].

Finally, in the same sample, reward-relevant life events interacted with reward-related cognitive styles to predict bipolar mood symptoms[9]. Specifically, controlling for initial mood symptoms and total number of life events experienced, baseline perfectionistic and self-critical cognitive styles interacted with reward system-activating positive events to predict increases in hypomanic/manic symptoms, and with reward system-deactivating negative events (e.g., certain failures) to predict increases in depressive symptoms over follow-up[9].

Reward-relevant cognitive styles may not always be maladaptive. Indeed, the high achievement motivation and ambitious goal-striving may contribute to high levels of creativity and achievement also exhibited by many individuals with BSDs or at behavioral risk for developing a bipolar disorder[6].

The role of reward-relevant cognitive styles in the onset and course of BSDs has implications for psychosocial interventions for these disorders, particularly for cognitive-behavioral therapy (CBT), which has been shown to have efficacious prophylactic effects for BSDs[10]. There may be added value to CBT interventions that specifically target achievement, ambitious goal-striving, and reward-oriented cognitive schemas in man-

aging BSDs[10]. For example, the therapist might develop a plan in which surges of ambitious goal-setting and overconfidence are identified and challenged during prodromal periods to lessen the likelihood of a manic episode onset[10].

In summary, ambitious goal-striving cognitive styles appear to be involved in the vulnerability to onset and recurrences of mood episodes in individuals with BSDs. Thus, these styles may be an excellent target for preventive and therapeutic interventions for individuals with bipolar disorders.

Lauren B. Alloy[1], Robin Nusslock[2]
[1]Temple University, Philadelphia, PA, USA; [2]Northwestern University, Evanston, IL, USA

1. Alloy LB, Abramson LY, Walshaw PD et al. J Abnorm Psychol 2009;118:459-71.
2. Johnson SL, Carver CS, Gotlib IH. J Abnorm Psychol 2012;121:602-9.
3. Alloy LB, Nusslock R, Boland EM. Annu Rev Clin Psychol 2015;11:213-50.
4. Nusslock R, Alloy LB. J Affect Disord 2017;216:3-16.
5. Alloy LB, Bender RE, Whitehouse WG et al. J Abnorm Psychol 2012;121:399-51.
6. Murray G, Johnson SL. Clin Psychol Rev 2010;30:721-32.
7. Stange JP, Shapero BG, Jager-Hyman SG et al. Cogn Ther Res 2013;37:139-49.
8. Stange JP, Molz AR, Black CL et al. Behav Res Ther 2012;50:231-9.
9. Francis-Raniere EL, Alloy LB, Abramson LY. Bipolar Disord 2006;8:382-99.
10. Nusslock R, Abramson LY, Harmon-Jones E et al. Clin Psychol Sci Pract 2009;16:449-69.

DOI:10.1002/wps.20494

Prevention of child maltreatment: strategic targeting of a curvilinear relationship between adversity and psychiatric impairment

Child maltreatment – which includes physical, emotional and sexual abuse as well as neglect – is the single most influential known cause of lifetime mental health impairment that is preventable (the other high-impact causes being primarily genetic), with conservative estimates of prevalence of about 15% in high-income countries[1,2].

Its deleterious impact arguably accounts for 25% or more of the population-attributable risk for child psychopathology[1,3], and in severe cases can extend to the lack of the minimum requirements for normative human development (food, hygiene, human interaction), physical injury, sexual exploitation and mutilation, permanent brain injury and death[4], or be associated with perpetration of child abuse by victims when they reach adulthood[5].

Maltreatment most commonly first occurs in infancy, particularly when adult caregivers are too stressed or functionally incapacitated to attend to the needs of the children under their care. The long-term cost for each yearly cohort of children abused in the US alone has been conservatively estimated to exceed $124 billions[6].

Our ability to predict child maltreatment on the basis of risk indicators that can be feasibly ascertained on the first day of an infant's life (including indices of parental mental health or substance use impairment, concentrated poverty, and a range of socio-economic stress indicators) has considerably advanced[7], and specific risk profiles can be delineated identifying a subgroup of children who have an up to 70% likelihood of ultimately being detected in official governmental records for child abuse/neglect. In spite of this, hospitals and health agencies rarely systematically screen for child maltreatment risk.

Child maltreatment is preventable. Its prevention requires the coordinated application of interventions that address key lapses in "species-typical" mechanisms of protection of the young: caregiving knowledge and competence, resource acquisition, surrogacy (i.e., the family or adult "village" surrounding a child to assist when a parent needs help), and close surveillance of the child[3].

A prototypic, yet remarkably common risk scenario is that of a single parent with multiple young children, isolated by poverty, under-educated in the modeling of appropriate caregiving (or whose own experience in being parented was traumatic or deficient) and with either an untreated mental health impairment or substance use disorder.

An effective, evidence-informed approach to reduce the risk of child maltreatment imposed by this set of circumstances would include nurse (or paraprofessional) home visitation, parenting education, parental mental health care, a support resource for times of crisis, and reproductive health planning. This is analogous to the level of comprehensive intervention that is afforded to patients with complex medical disorders in most health systems, encompassing cost-efficient, evidence-based interventions that could be prioritized for families at risk and coordinated by efficient, targeted case management.

Yet, rarely does any family at risk receive a full complement of these necessary supports[3,8]. In the US, fragmentation across health agencies, state departments, and local bureaucracies, together with a lack of ownership of systematic risk surveillance by health systems, all but ensure that almost no family at risk ever receives this level of support. The end result is that child maltreatment is perpetrated at epidemic proportions: a conservative estimate of prevalence based on official records is that, in the US, one out of every six children is a victim[2].

Not all children succumb to the deleterious impact of maltreatment. Rather, the effects of trauma on brain and behavioral development are moderated by factors such as timing of occurrence over the course of childhood; severity, type and chronicity of maltreatment; and genotypic variation of victims. These factors render children more or less prone to becoming overwhelmingly biologically stressed by the adverse experience. It is the phenomenon of being stressed beyond capac-