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Physiological Markers of Emotional and Behavioral Dysregulation in Externalizing Psychopathology

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Much of the research conducted in our lab over the past decade has focused on identifying peripheral and central nervous system markers of trait impulsivity and emotion dysregulation in preschoolers (e.g., Crowell et al., 2006), middle schoolers (e.g., Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007), and adolescents (e.g., Crowell et al., 2005). This body of work includes studies of boys with attention-deficit/hyperactivity disorder (ADHD), boys with conduct disorder (CD; e.g., Beauchaine, Katkin, Strassberg, & Snarr, 2001), and girls with borderline personality traits (e.g., Crowell, Beauchaine, et al., 2008). One key assumption of our work, which is now supported by a number of empirical findings (for a review see Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, in press), is that heritable trait impulsivity interacts with socialized deficiencies in emotion regulation to potentiate conduct problems among boys and borderline traits among girls. Although space constraints preclude us from describing the theoretical bases of this assumption in full detail (for reviews see Beauchaine, Gatzke-Kopp, & Mead, 2007; Beauchaine et al., in press; Crowell, Beauchaine, & Linehen, in press), in writing this chapter we summarize how the use of genetic data, autonomic nervous system (ANS) markers, and neuroimaging have led us to our current thinking about the development of externalizing behavior disorders. We note at the outset that our discussion of environmental risk factors is limited given the objectives of this monograph. However, we consider such risk factors to be as important in the development of psychopathology as the biological vulnerabilities discussed below.

TRAIT IMPULSIVITY AND EXTERNALIZING BEHAVIOR DISORDERS

A related assumption of our work is that heritable impulsivity, defined broadly as “behavior that is socially inappropriate or maladaptive and is emitted without forethought” (Oas, 1985, p. 142), is common to all disorders along the externalizing spectrum, including ADHD, oppositional defiant disorder (ODD), CD, antisocial personality disorder (ASPD), and many drug and alcohol dependencies (Beauchaine & Neuhaus, 2008). This assumption is supported by behavioral genetics research indicating that disorders across the externalizing spectrum share a common latent vulnerability trait (Tuvblad, Zheng, Raine, & Baker, 2009), which by adulthood is over 80% heritable (Krueger et al., 2002). One of the challenges we have faced is identifying a biological marker of trait impulsivity that can be used across a broad age range. Though functional neuroimaging can be used with adolescents, it cannot be used effectively with externalizing preschoolers, and it is difficult to use with externalizing

middle schoolers. Thus, we have developed alternative means for assessing biological vulnerability to impulsivity that can be used with younger samples. Our goal in doing so is to make valid inferences about the central nervous system substrates of impulsivity without using neuroimaging techniques. Achieving this goal requires knowledge of both structural and functional relations between the central and peripheral nervous systems.

CENTRAL DOPAMINE FUNCTIONING AND TRAIT IMPULSIVITY

It has long been known that impulsive individuals, including those with ADHD, ODD, CD, ASPD, and various addictive disorders respond differently to external rewards than controls. Across a number of monetary incentive paradigms, males with these disorders persevere in reward-responding for much longer than their peers both (a) when contingencies turn against them and they begin to lose money, and (b) when incentives are discontinued entirely (e.g., Ersche, Roiser, Robbins, & Sahakian, 2008; Giancola, Peterson, & Pihl, 2006; Matthys, van Goozen, de Vries, Cohen-Kettenis, & van Engeland, 1998; Matthys, van Goozen, Snoek, & van Engeland, 2004; Newman & Wallace, 1993). A core central nervous system substrate of aberrant reward responding appears to be underactivation in the ventral striatum, an evolutionarily old network of interconnected neural structures that have long been associated with approach motivation in rodents, nonhuman primates, and humans. This brain region is rich in dopaminergic projections, which are less responsive to reward—including monetary incentives—among impulsive individuals than among controls (see Durston et al., 2003; Vaidya et al., 1998; see also Sagvolden, Johansen, Aase, & Russell, 2005). We and others have argued that those with impulse control disorders engage in excessive reward-seeking behaviors in part to upregulate a persistently underactive mesolimbic dopamine (DA) system, which is experienced psychologically as a chronically aversive and irritable mood state (e.g., De Witte, Pinto, Ansseau, & Verbanck, 2003; Laakso et al., 2003). In addition, deficiencies in anterior cingulate cortex (ACC) activity, which are observed among impulsive individuals during extinction, also appear to contribute to perseverative responding for reward (e.g., Gatzke-Kopp et al., 2009).

CARDIAC PRE-EJECTION PERIOD: A MARKER OF CENTRAL DA RESPONDING?

Several sources of evidence now suggest that cardiac pre-ejection period (PEP)—indexed as the time interval between left ventricular depolarization and ejection of blood into the aorta—marks striatal DA responding during approach behaviors, including those elicited by monetary incentives (Brenner, Beauchaine, & Sylvers, 2005; Beauchaine et al., 2007). Pre-ejection period is controlled by the sympathetic nervous system (SNS), with shorter intervals indicating a stronger sympathetic response. The argument that PEP shortening marks central DA reactivity is based on both functional and empirical considerations. Behavioral approach requires expenditures of energy, and an important function of the SNS is to mobilize resources to meet environmental demands (e.g., Heimer, 1995). Furthermore, increases in cardiac output required for motivated behavior are mediated by SNS-induced changes in the contractile force of the left ventricle (Sherwood et al., 1990; Sherwood, Allen, Obrist, & Langer, 1986). Finally, infusions of DA agonists into striatal structures produce SNS-mediated increases in cardiac output (van den Buuse, 1998), which are similar to those

observed when normal controls participate in reward tasks. Taken together, these observations suggest that reduced SNS-linked cardiac reactivity to incentives is a likely marker attenuated DA responding. This argument is supported further by research indicating that PEP shortening among controls is *specific* to reward conditions, and is not observed during extinction or emotion evocation (Brenner et al., 2005)—two conditions in which striatal DA release is expected to be minimal.

We have now examined PEP responses to monetary incentives among male externalizing preschoolers, middle-schoolers, and adolescents ranging in age from 4–18, in several separate studies (Beauchaine et al., 2001; Beauchaine, Hong, & Marsh, 2008; Crowell et al., 2006; Mead et al., 2004). These samples included individuals with ADHD, ODD, CD, and antisocial personality traits. Without exception, the externalizing groups exhibited significantly less PEP reactivity to reward than controls. In fact, in most of our studies, no PEP responding at all is observed among externalizing groups. Importantly, these findings are not the byproduct of a generalized SNS deficiency, as electrodermal reactivity to reward among externalizers has not differed from that of controls. Assuming that our conjecture regarding PEP reactivity to incentives as a marker of central DA deficiency is correct, these findings suggest that the neural substrates of impulsivity are established in children as young as age 4. This would be expected if (a) disorders across the externalizing spectrum, including ADHD in preschoolers, share a heritable etiological substrate (see above), and (b) PEP reactivity to incentives marks the biobehavioral expression of this trait.

EMOTION REGULATION AS A MODERATOR OF EXTERNALIZING VULNERABILITY

In contrast to trait impulsivity, emotion regulation comprises the processes through which emotional experience and expression are shaped—whether volitionally or automatically—in the service of adaptive behavior (see e.g., Thompson, 1990). Following from this definition, emotion *dysregulation* might best be described as a pattern of emotional experience and/or expression that interferes with appropriate goal directed behavior. In almost all forms of psychopathology, one or more negative emotions is experienced either too intensely or for too long to be adaptive (Beauchaine et al., 2007). Thus, emotion dysregulation is a broad rather than specific risk factor for psychopathology.

Much has been learned about the central nervous system substrates of emotion regulation in the past decade. Structures that have been implicated consistently with individual differences in emotion regulation include the amygdala and the ventromedial prefrontal cortex (VMPFC; see e.g., Goldsmith, Pollak, & Davidson, 2008). The VMPFC appears to inhibit amygdala activation when individuals purposefully downregulate negative emotions. Furthermore, lesions to the VMPFC impair autonomic responses to emotionally valenced stimuli (Verbane & Owens, 1998). Much has also been written about the modulatory effects of certain brainstem nuclei—particularly the nucleus ambiguus—on the expression of emotion (see Porges, 2007). These nuclei serve as final common pathways—via the vagus nerve—from the central nervous system to the cardiovascular system and other peripheral targets.

RESPIRATORY SINUS ARRHYTHMIA AND EMOTION REGULATION

At the parasympathetic nervous system (PNS) level, the ability to regulate emotions is often marked by respiratory sinus arrhythmia (RSA), a quantification of cyclic increases and decreases in heart rate across the respiratory cycle (Beauchaine, 2001; Beauchaine et al., 2007; Porges, 2007). Since the publication of Porges' (1995) Polyvagal Theory describing links between parasympathetic responding and emotional expression, an impressively consistent body of research has emerged linking deficiencies in RSA to emotion dysregulation and psychopathology (see e.g., Beauchaine et al., 2007; Porges, 2007). As we have reviewed elsewhere, low baseline RSA and/or excessive RSA withdrawal in response to emotionally evocative stimuli have been linked with conduct problems, trait hostility, eating disorders, anxiety disorders, depression, and panic disorder—among other adverse outcomes (for a review see Beauchaine, 2001).

Although some researchers have suggested that impulsivity is a direct manifestation of emotion dysregulation, the two traits derive from distinct etiological and neural substrates, as described above. Furthermore, in contrast to impulsivity, which is almost entirely heritable, emotion dysregulation is largely socialized within families (Beauchaine et al., 2007; Linehan, 1993; Snyder, Schrepferman, & St. Peter, 1997). Indeed, behavioral genetics studies indicate that individual differences in RSA are in large part determined by environmental factors (Kupper et al., 2005; Sneider, Boomsma, van Doornen, & DeGeus, 1997).

In our own research, we typically measure RSA both at baseline and in response to emotionally evocative (e.g., sadness-inducing) stimuli. In fact, in each of the studies cited above in which PEP responding to reward was assessed, RSA data were also collected. Interestingly, attenuated baseline RSA and/or excessive RSA reactivity to emotion evocation were observed only in the in conduct-disordered middle school and adolescent samples (Beauchaine et al., 2001; Beauchaine, Hong, & Marsh, 2008; Mead et al., 2004). In contrast, neither RSA nor RSA reactivity discriminated externalizing preschoolers with ADHD and ODD from controls (Crowell et al., 2006). At first we found this perplexing because these preschoolers are at very high risk for later conduct problems and delinquency (see e.g., Beauchaine et al., in press; Campbell, Shaw, & Gilliom, 2000). However, others have demonstrated that ADHD only progresses to more serious conduct problems for children in families where emotional lability is negatively reinforced (Patterson, DeGarmo, & Knutson, 2000). Accordingly, our current thinking is that impulsivity may be 'regulated', expressed as pure ADHD, or 'dysregulated', expressed as more serious externalizing outcomes, depending on emotion regulation strategies that are socialized through recurring parent-child interactions. In the case of externalizing preschoolers, it may be too soon for negative reinforcement processes to have fully shaped emotional lability, with consequential deficiencies in RSA (see Beauchaine et al., 2007).

Following this reasoning, we have argued that trait impulsivity confers risk for the development of serious externalizing conduct only when *coupled with* familial socialization of emotion dysregulation. According to this perspective, one would expect that adolescents with pure ADHD would exhibit less emotional lability, as indexed by RSA reactivity to

emotion evocation, than adolescents with ADHD and CD. In one of our studies contrasting adolescents with pure ADHD vs. those with ADHD and CD, this is exactly what we found (Beauchaine et al., 2001).

RSA AS A MODERATOR OF EXTERNALIZING VULNERABILITY

These data suggest that strong socialized emotion regulation skills, reflected both behaviorally and in high RSA, buffer children from a core biological vulnerability for externalizing disorders—inherited impulsivity. This adds to an accumulating literature linking RSA to children's adjustment in the face of diverse familial risk factors for psychopathology, including interparental conflict, parental drinking, and parental divorce (El-Sheikh, 2005; El-Sheikh, Harger, & Whitson, 2001; Katz & Gottman, 1995, 1997). In our own research, we have demonstrated moderating effects of RSA on relations between paternal ASPD symptoms and adolescent conduct problems (Shannon et al., 2007). Children with low baseline RSA tended toward conduct problems regardless of the level of paternal ASPD symptoms, whereas children high in RSA were partially protected from the effects of their father's antisociality.

THE IMPORTANCE OF STIMULUS CONDITIONS

It is not unusual in the developmental literature for authors to mistakenly equate behavioral constructs and traits such as impulsivity with psychophysiological markers such as cardiac PEP. When this mistake is made, authors often expect the psychophysiological marker—in this case PEP—to discriminate between impulsive and non-impulsive children—regardless of stimulus conditions. However, our data show quite clearly that PEP reactivity is not observed during conditions of extinction or emotion evocation in impulsive individuals or controls. Rather, it is only during reward tasks that group differences emerge. Accordingly, our choice to use stimulus conditions of reward to assess PEP responding as a marker of impulsivity is based on strong theoretical considerations regarding the function of SNS-linked cardiac reactivity during approach behaviors, as outlined above (Beauchaine, 2001; Beauchaine et al., 2001; Beauchaine et al., 2007). A similar argument can be advanced for RSA reactivity as a marker of emotional lability. Here, one would expect better differentiation between labile individuals and controls during conditions of emotion evocation than during conditions of reward. Again, our data support this assertion. Unfortunately, I have reviewed many manuscripts and read many publications in which authors expect to find group differences in a psychophysiological marker across all of their stimulus conditions. When they do not, results are often interpreted as null findings. This illustrates how atheoretical choices of stimulus conditions can lead to considerable confusion in the literature (Beauchaine, in press). Researchers should therefore select their stimuli carefully, based on the function of the behavioral trait that they seek to mark.

FUTURE DIRECTIONS: BIOLOGY × ENVIRONMENT INTERACTIONS

As noted above in our discussion of RSA conferring protection in adverse family environments, it has become increasingly clear that certain biological vulnerabilities interact with contextual risk to potentiate psychopathology. In addition to psychophysiological markers of vulnerability, a number of Gene × Environment interactions have been reported

in the etiology of externalizing behavior disorders. Perhaps the most famous of these derives from the work of Caspi et al. (2002), who found that the combination of child maltreatment and a polymorphism in the monoamine oxidase-A gene (MAOA) predicted both juvenile and adult antisocial behavior. Those who experienced maltreatment and inherited the low MAOA activity genotype were at much higher risk for engaging in antisocial behavior than those who experienced maltreatment but did not inherit the low MAOA activity genotype.

Importantly, biological vulnerabilities and environmental risk factors are often synergistic rather than additive (Crowell, Beauchaine, & Lenzenweger, 2008; Raine, 2002). Furthermore, significant Biology $\times$ Environment interactions can be observed in the absence of main effects (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Thus, it is critical that the joint effects of vulnerabilities and risk factors be explored—even when each in isolation is only weakly associated with adverse outcomes. For example, in a recent study of biological and behavioral correlates of self-injury among adolescent females, we reported that peripheral serotonin—which is often used to mark trait impulsivity—was reduced among self-injuring teens (Crowell et al., 2005). Independently, however, peripheral serotonin was only a weak predictor of lifetime self-injurious events. Moreover, observational ratings of negativity within mother-daughter dyads failed to predict self-injury. Nevertheless, the Serotonin $\times$ Negativity interaction accounted for a remarkable 64% of the variance in self-injurious behaviors (Crowell, et al., 2008).

In addition to such moderating effects, mediational models linking genes, neural responses, and behavior are now emerging. For example, Buckholtz et al. (2008) recently reported that stronger neural coupling between the amygdala and ventromedial prefrontal cortex mediated the relation between variations in the MAOA gene and certain personality traits. This finding is particularly exciting because the mediational model spanned genes $\rightarrow$ brain $\rightarrow$ behavior. It has often been noted that genes do not affect behavior directly (see e.g., Beauchaine, Hinshaw, & Gatzke-Kopp, 2008). Mediational models specifying neural and physiological processes through which genes influence behavior are therefore an extremely important development. These models take us one step closer to understanding the complexities of human behavior and dysfunction. In our view, these studies mark a new generation in behavioral research. It is our hope that the specification of causal pathways from genes to behavior will answer important questions that behavioral scientists have been pondering for generations.

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