International Journal of Psychophysiology xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

International Journal of Psychophysiology

journal homepage: www.elsevier.com/locate/ijpsycho

Motivation, emotion regulation, and the latent structure of psychopathology: An integrative and convergent historical perspective☆

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ARTICLE INFO

Article history:
Received 3 November 2016
Received in revised form 29 December 2016
Accepted 31 December 2016
Available online xxxx

Keywords:
Motivation
Emotion
Latent structure
Emotion regulation
Executive function
History

ABSTRACT

Motivational models of psychopathology have long been advanced by psychophysiologists, and have provided key insights into neurobiological mechanisms of a wide range of psychiatric disorders. These accounts emphasize individual differences in activity and reactivity of bottom-up, subcortical neural systems of approach and avoidance in affecting behavior. Largely independent literatures emphasize the roles of top-down, cortical deficits in emotion regulation and executive function in conferring vulnerability to psychopathology. To date however, few models effectively integrate functions performed by bottom-up emotion *generation* system with those performed by top-down emotion *regulation* systems in accounting for alternative expressions of psychopathology. In this article, we present such a model, and describe how it accommodates the well replicated bifactor structure of psychopathology. We describe how excessive approach motivation maps directly into externalizing liability, how excessive passive avoidance motivation maps directly into internalizing liability, and how emotion dysregulation and executive function map onto general liability. This approach is consistent with the Research Domain Criteria initiative, which assumes that a limited number of brain systems interact to confer vulnerability to many if not most forms of psychopathology.

1. Introduction

Motivational accounts of psychopathology have a long history in the psychophysiology literature, but faded from prominence after the affective neuroscience revolution swept the field. Here, we suggest that (1) motivational constructs are as relevant as ever toward understanding psychopathology in the era of neuroscience and the Research Domain Criteria (RDoC), (2) motivation and emotion are inextricable aspects of behavioral function that were artificially separated in the history of psychology, (3) this separation yielded unnatural distinctions at both behavioral and neural levels of analysis that have impeded progress in our understanding of mental illness, and (4) re-integrating motivation and emotion represents a major advance in thinking. In demonstrating these points, we discuss the role of paradigm shifts in science, which sometimes unwittingly obscure causal mechanisms of natural phenomena. We present an historical account of motivation and emotion research in psychology, which illustrates how paradigm shifts have diminished the motivational perspective, even though it dovetails well with RDoC, neural systems accounts of mental illness, and the factor structure of psychopathology.

2. Paradigm shifts in psychopathology research

2.1. Advances and trends in science

It is a common misconception that the scientific method leads inexorably to improved understanding of natural phenomena. Kuhn (1962) noted this over 50 years ago when he described epistemological *paradigm shifts*, which comprise fundamental and often rapid changes to ways scientists measure, interpret, and/or conceptualize information. Paradigm shifts provide new ways of thinking about longstanding scientific connundra. Although some paradigm shifts advance science, as when Einstein's general theory of relativity supplanted Newton's theory of gravity, others stagnate and even impede scientific progress. This is especially likely in the social sciences, where levels of analysis are often pitted against one another (e.g., motivational vs. emotional; genetic vs. environmental; individual vs. cultural) in attempts to determine which is most relevant to understanding behavior. As a result, the history of psychopathology research is replete with paradigm shifts that have cycled between biological and environmental—sometimes following from political considerations rather than scientific discovery and innovation (see e.g., Rutter et al., 2006).

☆ Research reported in this article was supported by National Institutes of Health grants MH67192, MH63699, and UH2DE025980.

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http://dx.doi.org/10.1016/j.ijpsycho.2016.12.014
0167-8760/

Unlike theory testing in the hard sciences, where hypotheses can often be refuted with precise experiments, theories of psychopathology are difficult to falsify given the complexity of human behavior and its multiple determinants. Rather than being abandoned through refutation, theories of psychopathology therefore fade from prominence as their proponents retire or as the field loses interest and seeks new or recycled paradigms to explain mental illness. Thus, useful theories are sometimes jettisoned not because they lack explanatory power, but rather because they are no longer trendy. This can occur for a number of reasons, as described by others (e.g., Meehl, 1978). Motivational theories of psychopathology, which were prominent in the psychophysiology literature in the latter part of the 20th Century (see Beauchaine, 2001; Fowles, 1980, 1988; Gray, 1987), and are forerunners to the contemporary Research Domain Criteria (RDoC; see Beauchaine and Thayer, 2015), provide one such example. In sections to follow, we (1) briefly describe the history of such models and their fade from prominence despite clear and continued relevance to understanding diverse forms of mental illness; and (2) consider how motivational models of psychopathology dovetail with both emotion regulational and structural accounts of mental disorder. Such integration is consistent with the RDoC mission, and migrates psychopathology research away from excessive focus on description, as exemplified by the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013), toward greater emphasis on explanation—a migration that occurs as any scientific discipline matures (e.g., Popper, 1985; see also Beauchaine and Cicchetti, 2016; Beauchaine et al., 2007).

2.2. Methods-driven research and biological reductionism

Some paradigm shifts in science follow largely from methodological innovation. For example, invention and widespread use of factor analysis to reduce large numbers of observed variables to limited numbers of individual differences generated foundational advances in psychometric theory, intellectual assessment, and measurement of both personality and psychopathology. Such advances could not have been realized through continued use of rational test construction. In psychopathology research, factor analysis and related but more advanced latent variable models have provided key insights into interrelations among mental disorders. As detailed in later sections, research on the latent structure of psychopathology—as evaluated in a number of population based samples of children, adolescents, and adults—invariably yields separate but correlated internalizing and externalizing factors, which confer common vulnerability to what have traditionally been considered to be different disorders (see e.g., Achenbach and Edelbrock, 1991; Krueger and Markon, 2006). Factor analytic techniques also form the backbone of behavioral genetics (see e.g., Plomin et al., 2013), and have generated important questions about mechanisms of common vulnerabilities to psychopathology, concurrent comorbidities of psychopathology, and heterotypic continuities among psychiatric disorders across the lifespan (e.g., Beauchaine and McNulty, 2013; Krueger and Markon, 2006). Such questions, some of which are discussed in this article, have only begun to be answered by behavioral scientists.

More recently, a major paradigm shift in psychology—the so-called neuroscience revolution—was also brought about by methodological innovation. Over the past two decades, neuroimaging has been used to interrogate central nervous system substrates and correlates of virtually all psychiatric disorders. This research has produced an inordinate amount of structural and functional data, the scope and detail of which is unparalleled in the history of behavioral science. Although the significance and continued promise of neuroimaging toward understanding etiology, specifying pathophysiology, and improving treatment outcomes is clear (e.g., Akil et al., 2011; Poldrack and Yarkoni, 2016; Weingarten and Strauman, 2015), imaging techniques have been exalted to such an extent that biological reductionism now pervades the field (see e.g., Beauchaine et al., 2017; Bergner, 2016). This paradigm shift is reflected in the ascendance of RDoC, which focuses primarily on neurobiology, with no explicit representation of or accommodation for context in shaping and maintaining behavior.

From a motivational systems perspective, human behaviors and their neurobiological substrates and correlates cannot be understood when decontextualized from relevant approach-approach, approach-avoidance, and avoidance-avoidance contingencies within which they emerge (e.g., Corr and McNaughton, 2016). Almost no form of psychopathology is characterized by atypical neural or peripheral responding to all classes of stimuli. Rather, behavioral maladaptation occurs when individuals experience atypical responses to either incentives, threat cues, and/or emotion evocation in core biobehavioral systems involved in approach/active avoidance motivation, passive avoidance motivation, and/or affect regulation, respectively (see Beauchaine, 2015a; Beauchaine and Thayer, 2015; Beauchaine et al., 2017; Neuhaus et al., 2015). Accordingly, stimulus conditions must be considered carefully when interpreting temperamental response tendencies, individual differences in personality, and vulnerabilities to psychopathology, and when devising effective experiments to assess central and autonomic nervous system substrates and correlates of mental disorder.

Although psychophysiologists have historically been careful in this regard (see e.g., Beauchaine et al., 2001; Brenner and Beauchaine, 2011; Brinkmann et al., 2009; Fowles, 1980, 1988; Iaboni et al., 1997; Richter and Gendolla, 2009), it is not uncommon outside the psychophysiology literature for stimulus conditions to be ignored when interpreting behavioral and neurobiological responses exhibited by those with psychopathology (for extended discussion see Beauchaine, 2009; Zisner and Beauchaine, 2016). Moreover, although some neuroimaging research on neural substrates of psychopathology is driven by functionalist theories of motivation and emotion (see Beauchaine, 2015a), a considerable proportion of such research is atheoretical (see e.g., Sternberg, 2007). Despite certain advantages, current trends toward "bottom-up" approaches to analyzing vast amounts of imaging data threaten to further isolate neural responses from their eliciting events. As already noted, the current zeitgeist in psychopathology research favors neurobiological explanations, often ignoring and even eschewing environmental context (Beauchaine et al., 2017). One purpose of this article is to describe a contemporary functionalist account of psychopathology that emphasizes emotional response biases to motivational cues encountered in local environments. A second purpose is to outline how such an account dovetails with the well replicated factor structure of psychopathology noted above.

2.3. Misguided searches for independent causes

A third impetus for paradigm shifts in psychopathology research follows from the implicit assumption that independent causal mechanisms of large effect size will eventually be identified for individual disorders. When such effects are not found, effective theories that explain limited portions of variance in key outcomes are sometimes abandoned in efforts to articulate new theories that specify major causes. Yet with the exception of behavioral genetics studies, which account for a preponderance of variance in many psychiatric phenotypes, effect sizes are almost always modest in psychopathology research. Furthermore, behavioral genetics studies do not identify causal mechanisms, and very little of the complex phenotypes they account for is explained by molecular genetic targets (Plomin, 2013). Progress in our understanding of multifactorially inherited phenotypes will therefore require careful assessment of causal influences across many levels of analysis, including interactions among multiple genes, genes and environments, physiological response tendencies and operant reinforcement contingencies, and neural mechanisms of motivation and self/emotion-regulation, to name a few (Beauchaine & Constantino, in press; Beauchaine and McNulty, 2013; Beauchaine and Zalewski, 2016).

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2.4. Embracing etiological complexity

This multiple-levels-of-analysis perspective follows from recognition that genetic vulnerabilities, neural vulnerabilities, and environmental risk mediators almost never operate independently, and interact in complex ways to eventuate in mental illness (see e.g., Cicchetti, 2008). Many genes interact to affect temperament, personality, and psychopathology. Subcortical neural networks that facilitate approach and avoidance motivation, and cortical neural networks that facilitate self- and emotion-regulation, are interconnected and functionally interdependent (see below). Environmental risk mediators for psychopathology, such as disrupted family function, deviant peer group affiliations, and neighborhood risk factors co-occur and are often mutually reinforcing. Thus, research in which large independent effects of one or two variables on psychopathological outcomes are sought, while controlling for all other effects, is anachronistic (Beauchaine et al., 2017). Analysis of covariance and related statistical control techniques that are used to isolate "the" causal mechanism that produces a given psychopathological outcome, while controlling for all others, fails to carve the complexity of nature at its joints by creating independent variables that at best exist rarely within individuals (e.g., depression without liability to anxiety; see Beauchaine et al., 2010; Lynam et al., 2006; Miller and Chapman, 2001). Accordingly, future models of psychopathology must reflect the facts that (1) main effects of large magnitude are rarely observed for multifactorially inherited traits; (2) no single level of analysis holds primacy in explaining motivation, emotion, personality, or mental illness; and (3) interactions within and across levels of analysis must be evaluated (see Cicchetti, 2008; Panksepp, 2000).

Although examples of such research can be found, considerable effort is still spent on identifying independent causes of psychopathology. In this article, we present a model that integrates motivational, emotion regulational, and structural accounts of mental disorder. Given space constraints, we focus on neural underpinnings of motivation and emotion regulation—not molecular genetics. Such discussions can be found elsewhere (e.g., Gizer et al., 2016). Before presenting our integrated model, we first discuss the intertwined histories of research on emotion and motivation in psychology. This discussion reveals why artificial distinctions between motivation and emotion emerged, and how continued application of such distinctions distorts our understanding of the roles of bottom-up, *emotion generation* systems vs. top-down, *emotion regulation* systems in affecting behavior, including psychopathology.

3. Historical links between motivation and emotion in psychology

3.1. From Aristotle to Skinner

Associations between emotional experience and psychological adjustment were described over 2000 years ago by Aristotle, and have appeared throughout written history. In the 18th Century, Hume, 1888 devoted a large section of *A Treatise of Human Nature* to emotion, asserting that 'passions' served to motivate moral behavior—a view that was contrary to the prevailing rationalism espoused by European elites. In the late 19th Century, emotions figured prominently in writings by Charles Darwin (Darwin et al., 1872), who conducted pioneering descriptive work on interspecies expressions of affect, and William James (1884), who emphasized physiological states as precipitants of emotions rather than emotions as precipitants of physiological states. Prior to this time, outward expressions of emotion were often viewed as primitive, and sometimes as vestigial animal properties that needed to be controlled at all costs. Such views at once belied contemporary functionalist theories (e.g., Campos et al., 1994; Gendolla, 2014; Keltner and Haidt, 1999) and foreshadowed modern emotion dysregulation accounts of psychopathology (see Beauchaine, 2015a,b).

Historically, emotion remained a topic of interest to philosophers, psychologists, and sociologists until the early 1960s (see Solomon, 2000). Heidegger, 1962 formulated a philosophy in which emotions were essential to human existence, and served to adapt individuals to their local environments, consistent with modern functionalist perspectives. Similarly, constructivist theories, which were rooted in work by Elias (1982), explained emotions in terms of specific social functions they served. Emotional experience also figured prominently in major accounts of personality and psychopathology. Freud (1961), in contrast to constructivist perspectives, believed that most emotions were manifest expressions of unresolved intrapsychic conflicts. Jung (1951) adopted a similar view, asserting that emotions were byproducts of poor psychological adjustment.

Despite divergent perspectives on functions and dysfunctions of emotion, all of the accounts summarized above viewed emotions, even if implicitly, as *motivators* of behavior. In the constructivist framework, emotions motivated proper social behavior, whereas in theories of psychopathology, emotions motivated maladaptive behavior. It is worth noting that these perspectives are not necessarily exclusive, but rather focus on different intensities and expressions of emotion, and on diverse functional outcomes they engender.

By the 1950s and 60s, research on emotion fell out of favor in psychology, at the same time research on motivation expanded. This was not coincidental. Rather, it corresponded with a behavioral paradigm shift that swept psychology in the US (see Skinner, 1963). Behaviorism grew in large part from the hope that psychology might one day attain levels of prediction and control over human behavior that the physical sciences demonstrated for phenomena that fell within their purviews (see Beauchaine et al., 2008). Although the zenith of behaviorism was in the 1970s, its principles were derived from work conducted several decades earlier by authors including Hull (1943), Thorndike (1898), and Skinner (1938).

Behaviorists sought to predict actions of organisms entirely from histories of reinforcement, without appealing to endogenous mediators (Skinner, 1963). This required psychologists to specify mechanisms of learning and reinforcement that were *observable and replicable*, and to use these observations to build theories that were *falsifiable*, consistent with principles of logical positivism (Popper, 1962). Unobservable, internal mechanisms were considered to be "black box" explanations of behavior that could not be falsified with the scientific method. As a result, behavioral empiricism supplanted psychoanalysis as the dominant theoretical perspective in psychology, and research on emotions was squelched, since it could not be conducted without appealing to internal mechanisms (see Beauchaine and Zalewski, 2016). In contrast, motivation could be quantified by measures such as response latency, pursuit of incentives, frequency of operant responding, and passive avoidance of threat, among other observable behaviors.

3.2. Approach motivation, passive avoidance motivation, and changes to responding organisms

It is easy to forget that Skinner, the so-called father of radical behaviorism, acknowledged neurobiological mechanisms of learning and reinforcement. In fact, Skinner (1963) was clear in his view that operant behaviors are brought about by "changes to the responding organism" (p. 503). Thus, Skinner's behaviorism was not anti-biological. Rather, it required behavioral scientists to explicate their theories in terms that were observable and replicable, using available technologies of the day. Foundational animal research conducted in the 1960s and 1970s provided key insights into neurobiological mechanisms of learning and motivation. However, given the prevailing zeitgeist, emotion was usually not considered in such research despite (1) well established descriptive work—including observations by Darwin—demonstrating that animals experience emotion; (2) a long history of functionalist theories specifying emotions as precipitants of motivated behavior (see above); and (3) phenomenological associations between emotions and both approach and avoidance motivational states.

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Extensive and elegant pharmacological, single cell recording, and focal lesion studies conducted with animals yielded remarkable advances in our understanding of the neurobiological bases of motivation. These studies uncovered separate subcortical circuits that respond to incentive vs. threat cues in local environments. Neural substrates of incentive processing were elucidated by research conducted with rodents and nonhuman primates demonstrating that (1) electrical and pharmacological stimulation of dopaminergically-mediated mesolimbic structures including the ventral tegmental area and nucleus accumbens is reinforcing, as evidenced by trained animals engaging in protracted operant responding to obtain such incentives (see Deutsch, 1964; Milner, 1991); (2) phasic neural firing increases within mesolimbic structures during reward anticipation and reward-seeking, and following administration of dopamine (DA) agonists (e.g., Phillips et al., 1989); and (3) DA antagonists diminish and in some cases abolish incentive properties of food, water, and stimulant drugs of abuse (e.g., Rolls et al., 1974). Subsequent research demonstrated that the mesolimbic DA system responds to virtually all incentives, and is therefore the primary neural substrate of approach motivation (e.g., Holstege et al., 2003; McBride et al., 1999). Later human neuroimaging experiments demonstrated that such findings are homologous, and revealed altered structure and function of the midbrain DA system in response to reinforcement histories and other exogenous events (for extended discussion see Beauchaine et al., 2017). Thus, Skinner's supposition that reinforcement is mediated by changes to responding organisms was correct.

A temporally coinciding but largely independent stream of research identified the septo-hippocampal system—including its interconnections with the periaqueductal gray, medial hypothalamus, posterior cingulate, and amygdala—as the neural substrate of passive avoidance motivation. This perspective was summarized elegantly by Jeffrey Gray (1982) in the first edition of *The Neuropsychology of Anxiety*, and later expanded upon by Gray and McNaughton (2000). Through (1) meticulous neuroanatomical analysis, (2) extensive lesion studies, and (3) experiments demonstrating that anxiolytics—particularly benzodiazepines—alter GABA-ergic neurotransmission in the septo-hippocampal system, thereby blocking passive avoidance learning (e.g., Patel et al., 1979), Gray and McNaughton developed what is now called reinforcement sensitivity theory (RST).

RST emerged from Gray's neural model of approach-avoidance conflict resolution among organisms in their local environments (see Beauchaine, 2001; Gray, 1987). Gray specified a behavioral approach system, subserved by the mesolimbic DA pathway, and a behavioral inhibition system, subserved by the septo-hippocampal network. According to Gray, the septo-hippocampal system suppresses ongoing approach behaviors in contexts of reward cues when concurrent threat cues are detected. Under such conditions, suppressing approach allows slower neural processes to evaluate potential outcomes, so organisms can choose among less dangerous response options. More recent instantiations of RST apply to all motivational conflicts, including approach-approach, approach-avoidance, and avoidance-avoidance (Corr, 2004; see also Brenner et al., 2005).

3.3. Motivation and emotion as inextricable facets of behavioral function

Gray was not a biological reductionist. Rather, he insisted ardently that psychological reactions, both cognitive and emotional, serve to motivate behavior (Gray, 1979). During the heyday of behaviorism, when most researchers were circumspect about emotion, Gray maintained that the psychological output of septo-hippocampal activity was *anxiety*, which motivates precaution in contexts of risk. Nevertheless, the septo-hippocampal system came to be known as the "behavioral inhibition system", semantically divorcing its activation from aversive emotional states (fear, anxiety) that motivate passive avoidance. This was consistent with the dominant behavioral paradigm in which appeals to emotion and other psychological states were carefully avoided. Similarly, the midbrain DA system was referred to as either the "behavioral approach system" or the "behavioral activation system", semantically divorcing its activation from pleasurable affective states (wanting, liking) that motivate pursuit of and consummation of incentives. It was not until Robinson and Berridge (1993, 2003) developed their now famous incentive-sensitization theory of addiction that hedonic states were again linked to motivation through cue-induced patterns of activity and reactivity in mesolimbic structures.

4. Motivation, psychopathology, and psychophysiology

4.1. Peripheral markers of approach and passive avoidance motivation

Theories of motivation have long been used by psychophysiologists to describe associations between physiological responses to incentives/aversive stimuli and vulnerability to psychopathology. As reviewed elsewhere (Beauchaine, 2001; Beauchaine et al., 2001; Beauchaine and Thayer, 2015), Gray's (1982, 1987) two dimensional theory provided a framework for psychophysiologists to describe functional relations between subcortical neural systems of approach and active avoidance and individual differences in behavior, including psychopathology. Fowles (1980), in a highly influential literature review, proposed that heart rate (HR) reactivity to incentives marks behavioral activation system reactivity, whereas electrodermal responding (EDR) to extinction marks behavioral inhibition system reactivity. He then presented evidence that primary psychopaths exhibit normal incentive processing, reflected in HR reactivity to rewards, but deficient extinction processing, reflected in low electrodermal responding (EDR) to removal of rewards. According to Fowles, these data suggest that primary psychopaths do not experience punishment (i.e., frustrative nonreward) as aversive, and therefore take risks in approaching incentives when punishment outcomes are also possible.

Although more recent research suggests that cardiac pre-ejection period (PEP) reactivity to incentives is more specific than HR in marking individual differences in behavioral activation system sensitivity (Beauchaine & Gatzke-Kopp, 2012; Brenner et al., 2005; Richter and Gendolla, 2009), Fowles' perspective catalyzed a new generation of research addressing autonomic correlates of psychopathology, and continues to influence neuroimaging studies of septo-hippocampal, amygdalar, and mesolimbic substrates of internalizing and externalizing behavior (see e.g., Foti and Hajcak, 2009; Plichta and Scheres, 2014; Sauder et al., 2012, 2016).

4.2. Psychophysiology and psychopathology in multidimensional space

Fowles' (1980) asserted that primary psychopathy could be understood in terms of dysfunction in Gray's (1982, 1987) septo-hippocampally-mediated behavioral inhibition system (see also Gray and McNaughton, 2000). His 1987 Presidential Address to the Society for Psychophysiological Research foreshadowed contemporary transdiagnostic, neurobiological trait approaches to understanding psychopathology, such as RDoC, by extending his theory to several additional forms of mental illness (Fowles, 1988). Before it was trendy to do so, Fowles considered *interactive* roles of behavioral activation, behavioral inhibition, and environment on vulnerability to broad classes of psychopathology, including internalizing, externalizing, and psychotic disorders. He described how, with only approach and avoidance systems, each subdivided as weak, normal, or strong; and two environmental inputs (stimulation, regulation), each subdivided as present, absent–constantly activated, or absent–constantly inactive, a 54-cell matrix resulted, within which several forms of psychopathology were represented. Fowles acknowledged that even this multidimensional depiction was an oversimplification, since (1) motivational and environmental inputs are continuous, (2) effects of motivation and environment on behavior are interdependent, and (3) important cognitive factors were omitted.

Fowles' 1987 Presidential Address was foundational in establishing neurobiologically-informed, dimensional trait models of psychopathology.

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His integration of interactive influences spanning multiple levels of analysis (cf. Cicchetti, 2008) spawned a generation of hypothesis-driven psychophysiological research on biological mechanisms of mental illness. Among other findings (see Beauchaine and Thayer, 2015), extensions of Fowles' approach revealed that deficits in extinction learning are observed in multiple forms of externalizing conduct (e.g., Iaboni et al., 1997), and correspond with specific patterns of neural dysfunction (e.g., Gatzke-Kopp et al., 2009). Thus, Fowles' perspective reveals that RDoC thinking extends farther back than is often supposed (see Beauchaine and Thayer, 2015; Morris et al., 2015). Yet despite foreshadowing RDoC by decades, Fowles' model, and the broader psychophysiology literature, continued to treat emotion as an afterthought, since inferences about emotional processes still required appeals to endogenous mechanisms that were difficult to falsify using available technologies.

5. Resurgence of emotion research in psychophysiology and psychopathology

5.1. New technologies for interrogating central and peripheral correlates of emotion

By the mid-1990s, theoretical and technological advances made it possible to study previously unobservable correlates of psychological states among humans. These advances are exemplified in two important breakthroughs. First, the Society for Research in Child Development published a highly influential compendium of papers from a 1991 conference on development of emotion regulation (see Beauchaine, 2015a). This monograph (Fox, 1994a) demonstrated how emotional states can be inferred, verified, and quantified by measuring neurobiological systems via hormonal assays (Stansbury and Gunnar, 1994), EEG (Dawson, 1994; Fox, 1994b), and ECG (Porges et al., 1994). Collectively, papers in the Monograph challenged prevailing behavioral and cognitive paradigms that viewed emotional states as subjective, unquantifiable, and unamenable to refutation. A flood of research on emotion, emotion regulation, and emotion dysregulation ensued, and emotion became a central topic of inquiry in mainstream behavioral science. Only seven years after Fowles (1988) published his 1987 Presidential Address to the Society for Psychophysiological Research, which focused squarely on motivation with only passing reference of emotion, Porges (1995) published his 1994 Presidential Address to The Society. Porges' paper, a detailed phylogenetic account of limbic and peripheral substrates of affect regulation in the service of attention allocation and social affiliation, focused squarely on emotion. Soon thereafter, our group published two papers that integrated motivational (Gray, 1982, 1987) and emotion regulational (Porges, 1995) perspectives on vulnerability to internalizing and externalizing psychopathology (Beauchaine, 2001; Beauchaine et al., 2001).

Second, fMRI and EEG studies emerged linking behavioral approach and withdrawal tendencies to affective states and psychiatric outcomes, particularly depression. After receiving the 2000 Award for Distinguished Scientific Contributions to Psychology, Richard Davidson (2000) summarized this new, *affective neuroscience* perspective in an invited address to the American Psychological Association. Davidson outlined work on *affective style*—a term used to refer to individual differences in propensities toward approach—vs. withdrawal-related emotions (Harmon-Jones and Allen, 1997; Henriques and Davidson, 1991). Davidson's model emphasized the role of subcortical and cortical asymmetries—particularly in the amygdala and functional subdivisions of the PFC—in generating emotion (see also Davidson et al., 2000). Central to the affective style perspective is the view that emotion and motivation are inextricably intertwined: Organisms engage in motivated behavior at the behest of their emotions.

Davidson and colleagues' integration of emotion and motivation revived the historical perspectives outlined above, but now with evidence from advanced neuroscience methods. Affective neuroscience ushered in contemporary models of psychopathology that emphasize functional interactions between bottom-up *emotion generation* systems, which motivate behavior, and top-down *emotion regulation* systems, which modulate behavior (e.g., Beauchaine, 2015a; Beauchaine et al., 2017; Heatherton and Wagner, 2011; Petrovic and Castellanos, 2016; Tone et al., 2016).

5.2. Sequelae of reintegrating emotion and motivation

Reintegrating emotion and motivation in affective neuroscience served both instrumental and communicative functions. Neuroimaging enabled scientists to confirm homologies between animals and humans in associations between motivational states and neural responding in mesolimbic and septo-hippocampal brain regions (e.g., Berridge and Kringelbach, 2015; LeDoux and Pine, 2016). Even though these subcortical neural structures are preserved phylogenetically, questions always emerge when generalizing animal research to humans. Neuroimaging also provided a means of evaluating brain structure and function in regions that show little homology between animals and humans, most notably the PFC. This latter point is of utmost importance given that (1) humans are more efficient than other animals at modulating their emotional impulses and expressions—functions subserved by the PFC (see below), and (2) almost all forms of psychopathology are characterized by deficiencies in top-down prefrontal control over subcortically-generated affect (see Beauchaine et al., 2017; Caspi et al., 2014; Goodkind et al., 2015; Macdonald et al., 2016). We return to this point below.

The affective neuroscience perspective also enabled psychopathologists to speak about motivation and emotion in terms that recoupled the constructs semantically. Behavioral approach is motivated by affective states that at their extremes are manifested in *trait impulsivity* (e.g., Zisner and Beauchaine, 2016), whereas behavioral inhibition is motivated by affective states that at their extremes are manifested in *trait anxiety* (Corr and McNaughton, 2016; Gray and McNaughton, 2000). Reintegrating motivation and emotion also facilitated discussion of subcortical substrates of emotion that initiate and maintain motivated behavior. Activation of the mesolimbic DA system produces affective states such as wanting, which motivate approach (e.g., Berridge and Kringelbach, 2015), whereas activation of the septo-hippocampal system produces affective states such as fear, which motivate passive avoidance (e.g., Shin and Liberzon, 2010; Williams et al., 2015). Thus, neural systems of approach and avoidance are no longer divorced from affective states that motivate consummatory and inhibitory actions.

5.3. Subcortical emotion generation systems vs. cortical emotion regulation systems

When the emotion generation functions of subcortical approach and avoidance systems are considered, their feedforward and feedback interactions with functional subdivisions of the PFC take on greater meaning. In contrast to subcortically-generated approach- and withdrawal-related emotions, which are mediated by early-maturing brain regions, emotion regulation is subserved by later maturing cortical structures (see Brumback et al., 2016; Gogtay et al., 2004). Differing maturational time courses of subcortical and cortical networks lead to normative declines in impulsive behavior and improvements in self-regulation throughout adolescence and early adulthood (Beauchaine et al., 2017). For example, children and adolescents show stronger mesolimbic responding to incentives than adults, yet their PFC responding is weaker and more diffuse (see Macdonald et al., 2016). Moreover, adolescents with conduct disorder, who experience difficulties with impulse control, fail to exhibit normative gray matter pruning in frontal structures as they mature (De Brito et al., 2009)—a likely neural substrate of their difficulties with behavior regulation, emotion regulation, and executive function. Changing associations between amygdalar responding and ventromedial PFC function are also observed across development among those with anxiety disorders (Gold et al., 2016).

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Effortful regulation of impulsivity is effected through orbitofrontal and dorsolateral prefrontal inhibition of mesolimbic activity and reactivity, whereas effortful regulation of anxiety is effected through lateral prefrontal inhibition of amygdalar activity and reactivity (e.g., Davidson, 2002; Heatherton, 2011; Heatherton and Wagner, 2011; Tone et al., 2016). Impulsive individuals exhibit altered functional connectivity between mesolimbic and frontal structures, including the anterior cingulate (e.g., Cubillo et al., 2010; Shannon et al., 2009), whereas depressed and anxious individuals, including those with post-traumatic stress disorder, exhibit altered functional connectivity between the amygdala and ventrolateral prefrontal, ventromedial prefrontal, and anterior cingulate structures (e.g., Gold et al., 2016; Kujawa et al., 2016; Monk et al., 2008; Stevens et al., 2013; Tang et al., 2013). Furthermore, deficient top-down control of the amygdala by the medial PFC, and reduced functional connectivity between the amygdala and the orbitofrontal cortex, are implicated in emotional lability (see Churchwell et al., 2009; Hilt et al., 2011). Thus, failures in prefrontal regulation of subcortical structures involved in approach- and avoidance-related affect are observed in both externalizing and internalizing psychopathology.

Functional interactions between subcortical emotion generation systems and cortical emotion regulation systems are depicted in Fig. 1. This figure summarizes findings on neural correlates of emotion regulation, as reviewed elsewhere and outlined above (e.g., Beauchaine, 2015a; Beauchaine et al., 2017; Heatherton, 2011; Heatherton and Wagner, 2011; Shin and Liberzon, 2010). Approach-related emotions, generated by mesolimbic structures (e.g., nucleus accumbens), are regulated by frontal regions including the dorsolateral PFC, the orbitofrontal cortex, and the anterior cingulate. In contrast, avoidance-related emotions, generated by septo-hippocampal structures (e.g., amygdala), are regulated by frontal regions including the ventrolateral PFC, the ventromedial PFC, and the anterior cingulate. According to this perspective, bottom-up *emotion generation* processes occur when phylogenetically old neural structures are activated by approach- and avoidance-related motivating cues in local environments. In contrast, top-down *emotion regulation* processes occur when cortical systems modulate these neural responses. Two important corollaries of this view are that (1) disruptions in connectivity between subcortical and cortical systems confer vulnerability to different forms of psychopathology, depending on specific interconnections that are compromised; and (2) individual differences toward excessive approach- and/or avoidance-related affect/behavior may be offset by strong top-down modulating influences of functional subdivisions of the PFC (see Beauchaine et al., 2017; Heatherton and Wagner, 2011).

5.4. Emotion dysregulation and executive dysfunction: Sine quibus non of psychopathology

As this brief overview suggests, emotion dysregulation is a *transdiagnostic vulnerability* to psychopathology (see Beauchaine, 2015b; Cole et al., 2017). In fact, difficulties with emotion regulation are observed in disorders spanning the internalizing and externalizing spectra (Beauchaine, 2015a; Beauchaine and Thayer, 2015), and in various other forms of psychopathology, including autism (e.g., Neuhaus et al., 2015), borderline personality disorder (see Crowell et al., 2009), schizophrenia (see O'Driscoll et al., 2014), and self-inflicted injury (e.g., Crowell et al., 2005), among others (Cole et al., 2008). Thus, emotion dysregulation confers broad rather than specific vulnerability to psychiatric morbidity.

Consistent with thinking depicted in Fig. 1, contemporary neurobiological models of emotion dysregulation attribute its transdiagnostic nature to weak top-down, executive control over subcortical brain functions (e.g., Beauchaine, 2015a). From this perspective, both emotional and executive control are compromised among many if not most individuals with psychopathology because they represent inextricable facets of self-regulation that are mediated by overlapping neural networks (see e.g., Ochsner et al., 2012; Rohr et al., 2015). PFC dysfunction gives rise to deficiencies in both emotion regulation and executive control (see Pessoa, 2009). These deficiencies confer functional impairment—a requirement for most DSM diagnoses (APA, 2013). Importantly, neither

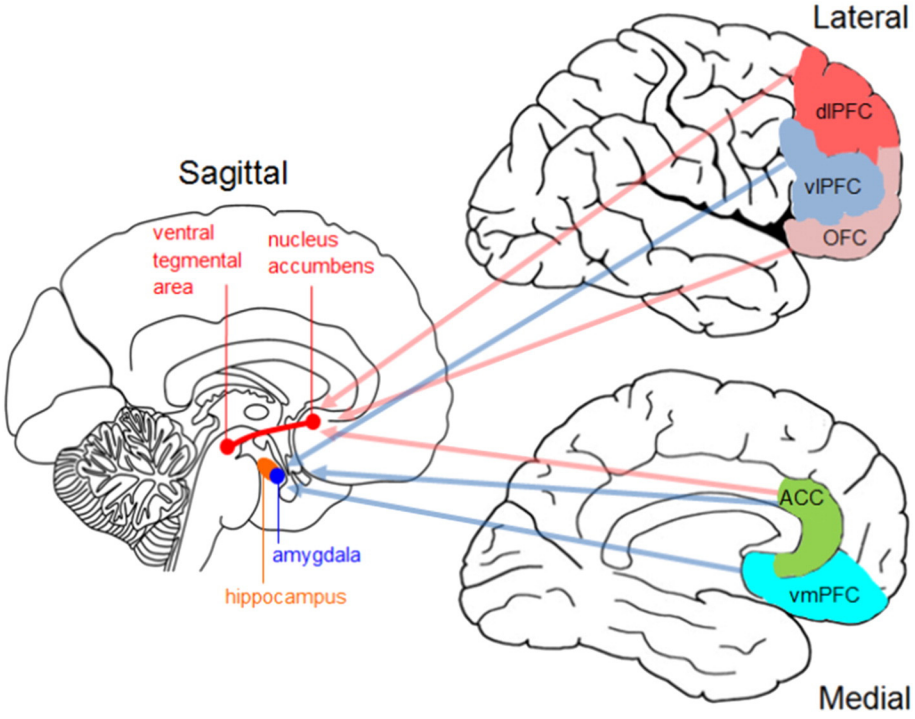

Fig. 1. Schematic representation of subcortical structures implicated in emotion *generation* (sagittal view) and cortical regions implicated in emotion *regulation* (lateral and medial views). Functional subdivisions of the PFC provide top-down modulatory influences over subcortical structures, as indicated by directional arrows. As outlined in text, these modulatory effects are compromised across diverse forms of psychopathology. Although this figure has heuristic value, it omits both feedforward connections from subcortical structures to cortical brain regions, and functional interactions within and across subcortical and cortical structures.

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affect nor cognition receive primacy in this transdiagnostic perspective, since emotional and cognitive control are effected through common neural substrates and are therefore impossible to disentangle (e.g., Pollak, 2016). For example, the ability to inhibit negative affect is mediated by connectivity in the same amygdalar-prefrontal pathway as core executive functions (Rohr et al., 2015).

This view is consistent with recent findings linking compromised structural integrity of frontal brain regions with executive dysfunction across a wide range of psychiatric disorders (e.g., Goodkind et al., 2015). Moreover, several forms of psychopathology are characterized by resting functional connectivity deficits in executive control networks, and/or compromised effective connectivity between subcortical and cortical structures during executive function tasks (e.g., Dong et al., 2015; Schlösser et al., 2003). Moreover, reduced frontostriatal connectivity is associated with executive function deficits among those who are vulnerable genetically to impulse control disorders (Gordon et al., 2015). Thus, disrupted subcortical-cortical connectivity marks both emotion dysregulation and executive dysfunction (see also Heatherton and Wagner, 2011). Encouragingly, improvements in subcortical-cortical connectivity following treatment are observed for several psychiatric disorders (e.g., Andreescu et al., 2015; Rubia et al., 2009).

6. Motivation, emotion regulation, and the latent structure of psychopathology: convergence across levels of analysis

6.1. Approach, avoidance, and the hierarchical structure of externalizing and internalizing symptoms

The historical account outlined above demonstrates increasingly sophisticated integration of motivation, motivation-associated affect, emotion dysregulation, and executive dysfunction in models of psychopathology. This *transdiagnostic* approach—perhaps the latest paradigm shift in psychopathology research—suggests that functional interactions among a limited number of neurobiological systems eventuate in diverse psychiatric outcomes. The neurobiological systems considered here can be subdivided into (1) subcortical emotion generation systems, which give rise to approach- and avoidance-related affect and behavior; and (2) cortical emotion regulation systems, which may modulate or potentiate subcortically-mediated affect and behavior, depending on their functional efficiency vs. deficiency. Although not a focus of this article, subcortical networks of approach and avoidance also exhibit functional interactions with one another (see Beauchaine et al., 2001, 2017), and cortical networks of emotion regulation interact with environmental influences across development, affecting their structural integrity and effectiveness in modulating impulsive and anxious behaviors (e.g., Hanson et al., 2010, 2012, 2013a,b, see also Beauchaine and McNulty, 2013).

In the latter part of the 20th Century, an entirely independent program of research emerged in which factor analysis and related techniques were used to specify the latent structure of psychopathology. Although structural models derive from empirical relations among symptoms, a level of analysis that is agnostic regarding etiology, recent findings demonstrate convergence across neuroscientific and structural levels of analysis. Specifying the latent structure of psychopathology began with foundational studies by Achenbach (see e.g., Achenbach and Edelbrock, 1983), who applied factor analysis to symptoms exhibited among large samples of children and adolescents. These studies, and subsequent factor-analytic evaluations of symptoms among adults (e.g., Krueger, 1999), demonstrated a remarkably consistent latent structure of psychopathology whereby two higher-order factors, internalizing and externalizing, account for a preponderance of covariation among first-order factors (behavioral syndromes). First-order internalizing factors include constructs such as anxiety, depression, withdrawal, and somatic complaints, whereas first-order externalizing factors include constructs such as impulsivity, aggression, delinquency, and (among adolescents and adults) substance dependencies. This latent structure is observed in population-based and twin studies, the latter of which indicate strong heritabilities for both the higher-order internalizing and externalizing factors (e.g., Cosgrove et al., 2011; Krueger et al., 2002; Lahey et al., 2011).

Although structural analyses are usually not informed by neurobiology, externalizing disorders, such as ADHD, conduct disorder, and alcohol/drug dependencies, share a core etiological substrate—a functional deficiency in incentive processing in the mesolimbic DA system (see Beauchaine et al., 2017; Beauchaine and Thayer, 2015). In contrast, internalizing disorders, including anxiety and depression, share a functional deficiency in threat processing in the septo-hippocampal system (Corr and McNaughton, 2012, 2016). This motivational-affective account of psychopathology converges with factor analytic findings, even though neurobiological and structural models are specified at entirely different levels of analysis. Fig. 2 depicts the latent structure of psychopathology, and adds thought problems, which emerge as a

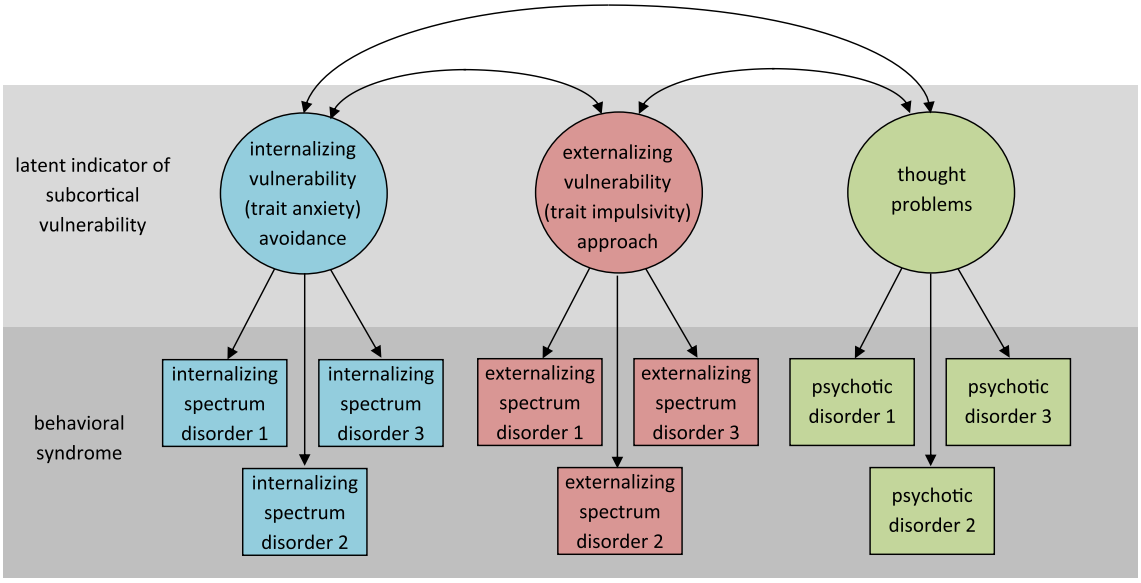

Fig. 2. The well replicated hierarchical latent structure of psychopathology. Trait anxiety emerges from aversive motivational tendencies and confers vulnerability to internalizing disorders (blue). Trait impulsivity emerges from appetitive motivational tendencies and confers vulnerability to externalizing disorders (red). Thought problems emerge as a separate factor (green). Adapted from Beauchaine and Thayer (2015).

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separate factor when psychotic symptoms are included in structural models (Wright et al., 2013).

6.2. Emotion regulation, executive function, and bifactor latent structure of psychopathology

In virtually all hierarchical models of psychopathology, broadband internalizing and externalizing factors correlate highly with one another (e.g., Krueger, 1999; Krueger et al., 1998). In more recent models, thought problems are either subsumed into the higher order internalizing factor (e.g., Keyes et al., 2013), or emerge as a separate factor that correlates highly with both internalizing and externalizing dimensions (e.g., Wright et al., 2013). Taken together, these findings suggest the possibility of a higher-order general psychopathology factor, even though no such factor was modelled in these studies.

Lahey et al. (2012) were the first to use bifactor model fitting to confirm an over-arching, general liability factor that internalizing, externalizing, and thought problems all load on. This general psychopathology bifactor accounts for significant variance in daily functioning, physical health, and future psychopathology, over-and-above independent variance accounted for by internalizing and externalizing dimensions. A nearly identical bifactor structure was reported by Caspi et al. (2014), who used confirmatory factor analysis to test multiple structural models of externalizing, internalizing, and thought disorder symptoms. Among adults who were assessed longitudinally at ages 18, 21, 26, 32, and 38 years, the bifactor model yielded good fit. Although internalizing, externalizing, and thought problems were each associated with impairment, as indicated by welfare use, violence convictions, psychiatric hospitalizations, and suicide attempts, much of this impairment was accounted for by the general vulnerability (p) factor.

Higher scores on p, which was recently replicated for a third time (Laceulle et al., 2015), were also associated with histories of child maltreatment, and with family histories of anxiety, depression, psychosis, CD, antisocial personality disorder, and substance dependence. Perhaps more importantly for purposes of this discussion, p correlated negatively with executive function, attention, memory, and IQ—all of which are subserved by the prefrontal cortex (see above). Some of these impairments were evident as early as age 3 years.

In a previous article published in this journal, we described associations between executive function and emotion regulation, and argued that these inextricably intertwined aspects of self-regulation confer general liability to psychopathology through deficient prefrontal control over behavior (Beauchaine and Thayer, 2015). This integrated model appears in Fig. 3. Direct associations between emotion regulation and p have yet to be demonstrated. However, overlapping neural substrates of emotion regulation, executive function, and self-control described above suggest that such research will be valuable.

7. Conclusions and future directions

Throughout this article, we emphasize the role of paradigm shifts in altering scientific inquiry and understanding of natural phenomena. Some paradigm shifts advance science, whereas others do not. In psychopathology research, paradigm shifts are more likely to impede scientific progress when they (1) assign priority to research conducted at single levels of analysis, (2) are driven largely by new methods rather than longstanding research questions, and/or (3) derive from misguided searches for independent causes of large effect size. When considered in these contexts, there is reason to be both optimistic and pessimistic about the current transdiagnostic focus in psychopathology research.

As noted above, a strong argument can be made that biological reductionism pervades the field. The RDoC mission, though a clear improvement over previous approaches to characterizing psychopathology, does not accommodate environmental influences, yet we know such influences affect neurobiological structure/function, and interact with

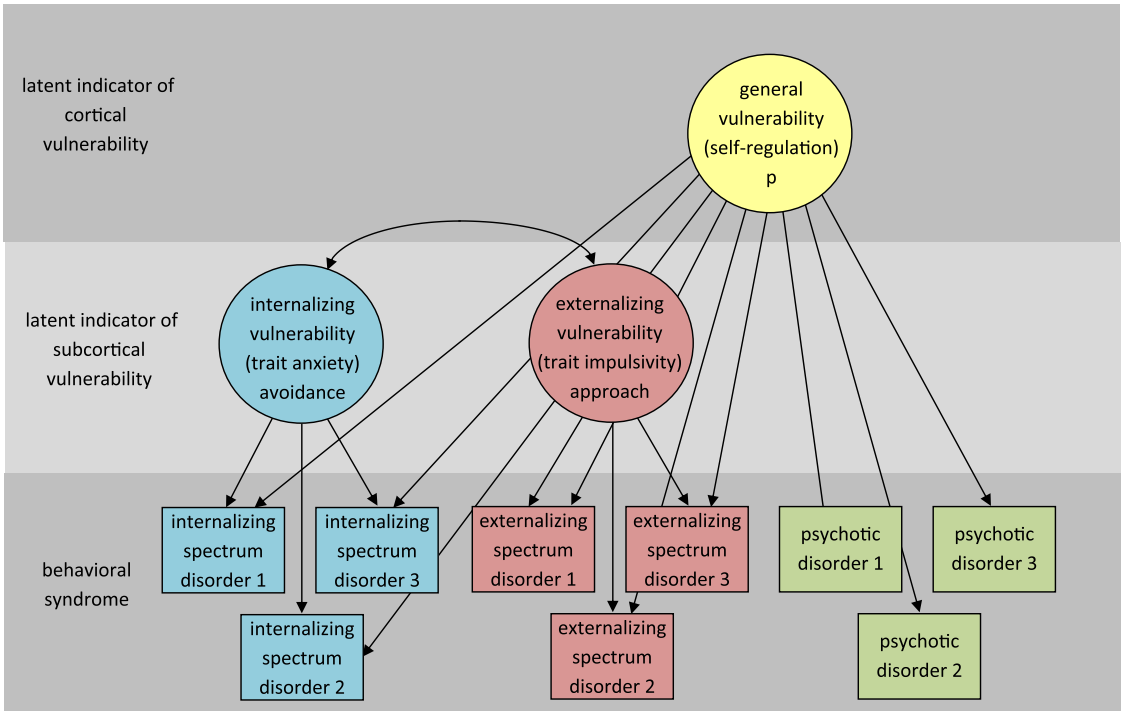

Fig. 3. The bifactor latent structure of psychopathology in which a general vulnerability (self-regulation), comprised of emotion regulation and executive function, contributes to vulnerability, over-and-above independent effects of trait anxiety, trait impulsivity, and thought problems. According to this perspective, trait anxiety and trait impulsivity arise from subcortical mechanisms, whereas general vulnerability to psychopathology arises from cortical mechanisms (see details in text). Although thought problems have primarily cortical substrates, we do not consider them here. This model integrates interactions among bottom-up, subcortical systems of approach- and avoidance-related affect with top-down, cortical systems of affect regulation.
Adapted from Beauchaine and Thayer (2015).

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neurobiological function to eventuate in psychopathology. Emotion regulation in particular is affected by familial and extrafamilial social influences (e.g., Beauchaine and Zalewski, 2016; Cole et al., 2017), and can be improved by effective interventions. Such improvements are reflected in altered neurobiological response patterns, which are sometimes normalized by effective treatment (Andreescu et al., 2015; Beauchaine et al., 2015; Rubia et al., 2009). Thus, environmental influences contribute to, maintain, and reshape neurobiological functions that subserve behavior. As stated early in this article, neural responses cannot be interpreted when decontextualized from their eliciting events, including learning histories and motivational cues in local environments.

On an optimistic note, the transdiagnostic paradigm shift represents advancing maturity of behavioral science research as we migrate from descriptive characterizations of psychopathology to explanatory models. This migration could not have been realized without dramatic advances in neuroimaging technologies, which enabled neuroscientists to confirm findings from an large body of animal research, and discover new brain-behavior relations that are unique to humans. Neuroimaging has produced astounding advances in our knowledge of psychopathology, and will continue to do so in the future. Further advances will undoubtedly be facilitated by bottom-up approaches executed on large data sets. However, such methods hold the potential to identify very small effects. Although many of these may be important, the history of psychopathology research is replete with over-interpretations and over-generalizations of small effects.

Conversely, important findings of small effect size are often eschewed in psychopathology research when they overlap with one another. This is exemplified in research on externalizing outcomes, where genetic effects, neural effects, family effects, peer group effects, and broader cultural effects are so overlapping and interactive that few if any provide strong independent prediction, even though together they account for considerable variance in delinquent outcomes (Beauchaine and McNulty, 2013; Beauchaine et al., 2017). Interest in environmental effects piqued many years ago since none are singularly or independently predictive of adverse outcomes. Yet environments interact strongly with trait impulsivity—a motivational-affective construct—to eventuate in antisocial behavior (e.g., Lynam et al., 2000). Topics discussed herein only begin to demonstrate the daunting ontogenic complexity of emerging psychopathology across the lifespan (Beauchaine and McNulty, 2013). In upcoming years, the psychopathology research community must embrace the complexity of human behavior and its myriad determinants (e.g., Cicchetti, 2008). This requires studies of large samples in which interactions within and across levels of analysis are modelled (see Beauchaine et al., 2017). The emergence of large-scale collaborative efforts is reason for continued optimism in the progression of psychopathology research.

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Please cite this article as: Beauchaine, T.P., Zisner, A., Motivation, emotion regulation, and the latent structure of psychopathology: An integrative and convergent historical perspective, Int. J. Psychophysiol. (2016), http://dx.doi.org/10.1016/j.ijpsycho.2016.12.014

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Please cite this article as: Beauchaine, T.P., Zisner, A., Motivation, emotion regulation, and the latent structure of psychopathology: An integrative and convergent historical perspective, Int. J. Psychophysiol. (2016), http://dx.doi.org/10.1016/j.ijpsycho.2016.12.014