

Polyvagal Theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence

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Abstract

In science, theories lend coherence to vast amounts of descriptive information. However, current diagnostic approaches in psychopathology are primarily atheoretical, emphasizing description over etiological mechanisms. We describe the importance of Polyvagal Theory toward understanding the etiology of emotion dysregulation, a hallmark of psychopathology. When combined with theories of social reinforcement and motivation, Polyvagal Theory specifies etiological mechanisms through which distinct patterns of psychopathology emerge. In this paper, we summarize three studies evaluating autonomic nervous system functioning in children with conduct problems, ages 4–18. At all age ranges, these children exhibit attenuated sympathetic nervous system responses to reward, suggesting deficiencies in approach motivation. By middle school, this reward insensitivity is met with inadequate vagal modulation of cardiac output, suggesting additional deficiencies in emotion regulation. We propose a biosocial developmental model of conduct problems in which inherited impulsivity is amplified through social reinforcement of emotional lability. Implications for early intervention are discussed.

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Scientific observation can generate an overwhelming excess of descriptive information. This is exemplified in the work of Charles Darwin, who in 5 years aboard the H.M.S. *Beagle* compiled 20 field notebooks, 8 volumes of diaries, and a 6-part catalogue of species. These works were unparalleled in both scope and detail, and uncovered several anomalies that could not be explained by prevailing theories of the day. The most famous of these is Darwin's observation of structural variation in the beaks of 13 finch species, and his subsequent recognition that each species was confined to a particular environmental niche of the Galápagos Islands.

The significance of Darwin's descriptive work can hardly be overstated. Yet description alone does not constitute science. Rather, science ultimately seeks *satisfactory explanations* for observed events or phenomena (Popper, 1985). Such explanations constitute *theories* regarding the mechanisms through which observed events or conditions occur. In Darwin's case,

the theory of evolution based on natural selection is by far his greatest contribution to science, and remains among the most important explanatory insights ever advanced. Evolutionary theory lends coherence to a perplexingly vast degree of speciation that before had been inexplicable.

Despite the importance of theories as organizing constructs in science, it is not uncommon for scientific disciplines to become mired in controversies over descriptive convention. In a public lecture in May of 2002, the late Stephen Jay Gould noted that a preoccupation with the descriptive features of dinosaurs has diverted paleontologists from pursuing evolutionary mechanisms of phenotypic variation, even though questions concerning such mechanisms are of far greater significance. In psychiatry, the proliferation of syndromes in the *Diagnostic and Statistical Manual of Mental Disorders*, from 106 in *DSM-I* (American Psychiatric Association, 1952) to 365 in *DSM-IV* (American Psychiatric Association, 1994), suggests an analogous preoccupation with description over etiological mechanisms (Beauchaine, 2003; Beauchaine and Marsh, 2006; Houts, 2002). In fact, an explicit objective of the American Psychiatric Association in constructing the *DSM-IV* (2000) was to compile criterion lists that were purposefully descriptive and

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atheoretical. This strategy was intended to move psychiatry away from psychoanalytically derived syndromes that were low in reliability, to empirically based syndromes that were replicable across raters and sites. Although successful in improving diagnostic reliability, the strictly descriptive approach places inordinate emphasis on the topography of behavior. In many cases, this results in arbitrary distinctions among seemingly different disorders that are in reality alternative manifestations of common genetic vulnerabilities (see Beauchaine and Marsh, 2006; Krueger et al., 2002). Thus, without theories regarding the etiological mechanisms of psychopathology guiding classification, we are likely to fail in our efforts to ‘carve nature at its joints’. The result is a complex list of diagnostic classes with perplexing patterns of overlap, or comorbidity (see, e.g., Klein and Riso, 1993). Resolving these perplexities will not be accomplished by engaging in ever more description. What is needed are organizing theories that specify etiological mechanisms, both common and unique, through which patterns of psychopathology emerge.

With this discussion in mind, our main objective in writing this paper is to describe the organizing role of Polyvagal Theory (Porges, 1995, 1997, 1998, 2001, 2003) for our program of research on externalizing psychopathology in children and adolescents. Before proceeding, however, it is important to juxtapose the role of theory in the behavioral sciences with the role of theory in other scientific disciplines. In physics, for example, scientists have long sought a single Grand Unified Theory to account for the strong (atomic), weak (radioactive), and electromagnetic forces in nature. The emergence of such a theory is considered by many to be a prerequisite for a comprehensive understanding of the observed universe. In contrast, grand theories will probably never emerge for phenomena studied in most other scientific disciplines. This is because causal mechanisms operate at many levels of analysis, each of which requires its own theory to explain. Consider evolution by natural selection. Selection occurs at the phenotypic level, where individual differences affect the probability of survival for organisms in their environment of adaptation. At this level of analysis, the mechanism of selection is survival based on heritable variation in phenotypic traits, the foundation of evolutionary theory. Note, however, that this tells us nothing about mechanisms of heritability, which were described by Gregor Mendel and later applied to evolution well after Darwin proposed his theory of natural selection. Thus, a full understanding of evolution requires separate but *interactive* theories of natural selection, heritability, and other implicated processes such as molecular genetics, each of which operates at a different level of analysis.

The same is true for psychopathology, which is manifested in complex interactions between individuals and their environments over time. These behavioral patterns are both affected by and effected across multiple levels of analysis, including genotypic, endophenotypic, phenotypic, behavioral, and social. Given this, no single theory can be expected to account for any particular psychiatric disorder, much less psychopathology in general. Nevertheless, Polyvagal Theory has emerged as an important explanatory construct for a wide

range of psychiatric conditions (Beauchaine, 2001). As we will demonstrate below, when used in conjunction with theories of social reinforcement and motivation, Polyvagal Theory furthers our understanding of the autonomic and central nervous system substrates of emotion regulation and emotional lability, and suggests possible intervention points for serious externalizing behavior.

1. Polyvagal Theory

We assume that most readers are familiar with Polyvagal Theory, and we therefore provide only a brief overview, focusing on aspects that are most central to our work. Readers who are unfamiliar with the theory are referred to Porges (1995, 1997, 1998, 2001, 2003, this volume) for more comprehensive accounts.

Polyvagal Theory specifies two distinct branches of the vagus, or 10th cranial nerve. These include a phylogenetically older branch originating in the dorsal motor nucleus (DMX), and a newer branch originating in the nucleus ambiguus (NA). The DMX and NA are located in the dorsal and ventral vagal complexes, respectively, adjacent neural structures in the medulla. Although both branches provide inhibitory input to the heart via the parasympathetic nervous system (PNS),¹ they do so in the service of distinct evolutionary functions. The DMX branch, sometimes referred to as the *vegetative vagus*, is rooted in the primary survival strategy of primitive vertebrates, amphibians, and reptiles, which freeze when threatened. Accordingly, the vegetative vagus functions to suppress metabolic demands under conditions of danger. In contrast, the NA branch, or *smart vagus*, is distinctly mammalian, and evolved in conjunction with the need to dynamically regulate substantially increased metabolic output. This includes modulation of fight/flight (F/F) responding in the service of social affiliative behaviors. After orienting to a conspecific, mammals must either engage in social affiliation, or initiate F/F responding. The former requires sustained attention, which is accompanied by vagally mediated heart rate deceleration (Suess et al., 1994; Weber et al., 1994). In contrast, fighting and fleeing are characterized by rage and panic, respectively, which are associated with near complete vagal withdrawal (George et al., 1989; Friedman and Thayer, 1998a,b; see also Porges, 1995, 2001). This facilitates large increases in cardiac output by the sympathetic nervous system (SNS), which is no longer opposed by inhibitory vagal influences. Thus, the smart vagus inhibits acceleratory SNS input to the heart when sustained attention and/or social engagement are adaptive, and withdraws this inhibitory influence when fighting or fleeing are adaptive.

Although the above description has clear implications for psychopathology, two additional attributes of Polyvagal Theory must be considered before proceeding. First, functional organization of the mammalian autonomic nervous system (ANS) is assumed to be phylogenetically hierarchical, with

¹ Both vagal branches also innervate other target organs that are not the focus of this paper. Interested readers are referred to Porges (1995, 1998, 2001) for further details.

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