Development and Psychopathology **28** (2016), 1071–1088

doi:10.1017/S0954579416000705

Development of self-inflicted injury: Comorbidities and continuities with borderline and antisocial personality traits

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Abstract

Self-inflicted injury (SII) is a continuum of intentionally self-destructive behaviors, including nonsuicidal self-injuries, suicide attempts, and death by suicide. These behaviors are among the most pressing yet perplexing clinical problems, affecting males and females of every race, ethnicity, culture, socioeconomic status, and nearly every age. The complexity of these behaviors has spurred an immense literature documenting risk and vulnerability factors ranging from individual to societal levels of analysis. However, there have been relatively few attempts to articulate a life span developmental model that integrates ontogenenic processes across these diverse systems. The objective of this review is to outline such a model with a focus on how observed patterns of comorbidity and continuity can inform developmental theories, early prevention efforts, and intervention across traditional diagnostic boundaries. Specifically, when SII is viewed through the developmental psychopathology lens, it becomes apparent that early temperamental risk factors are associated with risk for SII and a range of highly comorbid conditions, such as borderline and antisocial personality disorders. Prevention efforts focused on early-emerging biological and temperamental contributors to psychopathology have great potential to reduce risk for many presumably distinct clinical problems. Such work requires identification of early biological vulnerabilities, behaviorally conditioned social mechanisms, as well as societal inequities that contribute to self-injury and underlie intergenerational transmission of risk.

Self-inflicted injury (SII), which includes both suicidal and nonsuicidal self-harm behaviors, is a significant public health problem. These behaviors are associated with high rates of primary care, outpatient, inpatient, and emergency department utilization (Shepard, Gurewich, Lwin, Reed, & Silverman, 2016). Nonsuicidal self-injury (NSSI) is especially prevalent among adolescents and young adults and is linked to poor academic performance, disrupted peer relationships, and increasing risk for psychopathology across development (Crowell, Beauchaine, & Linehan, 2009; Klonsky, 2011). Suicidal behaviors are associated with similar problems and often require costly and restrictive interventions. Moreover, the devastation of losing a loved one to suicide is unquantifiable (Centers for Disease Control, 2015). The full range of SII behaviors are also associated with some of the most impairing psychiatric diagnoses, such as major depressive disorder, borderline personality disorder (BPD), eating disorders, posttraumatic stress disorder, conduct disorder (CD), and antisocial personality disorder (ASPD; American Psychiatric Association [APA], 2013). SII appears to be a transdiagnostic manifestation of underlying vulnerability for psychopathology. Nearly every psychiatric diagnosis is associated with elevated suicide risk (Hoertel et al., 2015).

At the same time, certain diagnoses appear to be associated with higher rates of SII. For example, suicidal thoughts/behaviors can be a symptom of major depressive disorder, and repetitive self-mutilation is a diagnostic criterion for BPD (APA, 2013). More recently, however, researchers have sought to understand associations between SII and specific psychiatric diagnoses by examining common developmental processes, vulnerabilities, and risk factors, rather than simple symptom-level co-occurrence. Consistent with this approach, we take an ontogenic process perspective to outlining the genetic, epigenetic, and developmental mechanisms of shared vulnerability for SII, BPD, and other related conditions such as ASPD. A central tenet of the ontogenic perspective is that many phenotypically distinct clinical problems emerge from a smaller number of underlying and often interdependent biological vulnerability factors (Beauchaine & McNulty, 2013). These biologically based individual differences are shaped by complex transactions with the environment and manifest differently across development and context (e.g., Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). For example, early impulsivity may progress into a multitude of presumably distinct psychiatric diagnoses depending upon interactions with other biologically based traits, socioeconomic factors, neighborhood risks, sex-specific socialization experiences, parenting practices, peer influences, and many other correlated contextual risk factors (DeYoung, 2010; Neuhaus & Beauchaine, 2013).

Most models of SII and related diagnoses outline a developmental trajectory that begins with childhood or early adolescence, when proximal risk factors for psychopathology emerge and become readily identifiable (e.g., child psychopa-

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thology or family conflict; Crowell et al., 2009). There have been fewer attempts to move earlier in development to predisposing genetic, epigenetic, and temperamental factors that contribute to vulnerability. However, identifying prenatal and childhood risks is enormously important for elucidating the origins of psychopathology. Relative to many other medical conditions, psychiatric problems often have clear roots in early development, a more chronic course, and are fairly unique in the requirement for ongoing administration of medication in order to achieve optimal clinical benefits (Tsankova, Renthal, Kumar, & Nestler, 2007). Many chronic physical health problems (e.g., Type II diabetes and cardiovascular disease) can also have origins in childhood psychological problems and environmental adversity (see e.g., Crowell, Puzia, & Yaptangco, 2015). It is now well established that psychopathology is a product of cumulative environmental forces interacting with biological systems beginning prior to conception. Accordingly, a major challenge in the field is to identify epigenetic, neurochemical, morphological, and physiological mechanisms underlying stable traits and behaviors while simultaneously elucidating modifiable contextual risk factors impinging upon these biological systems.

Understanding the emergence and developmental course of SII is an urgent priority and foundational for future prevention efforts. In this review, we synthesize a mounting literature to suggest that the developmental trajectory leading to SII is increasingly well understood. Specifically, SII appears to be associated with identifiable temperamental vulnerabilities early in development and with personality traits and disorders later in life. Developmental models linking SII to temperament and personality can reveal fruitful targets for intervention at different points in the life span and across levels of analysis (e.g., biological, contextual, and societal). As our review will highlight, developmental psychopathology (DP) theories are essential to understanding SII because they help explain diagnostic comorbidity and continuity. In lieu of traditional models that examine co-occurrence of psychiatric symptoms statically, DP scholars seek to understand comorbidity and continuity as emerging through dynamic longitudinal processes. This perspective is especially relevant for understanding transdiagnostic clinical problems such as SII that typically co-occur with different psychiatric conditions across distinct stages of development, such as anxiety in childhood, oppositional defiant disorder in adolescence, and severe depression and/or personality disorders in adulthood.

Foundational Concepts

Self-inflicted injury

Historically, all self-injuries were viewed as functionally similar. NSSIs were often incorrectly treated as suicide attempts, and all such behaviors were believed to emerge from an underlying "death wish" or misplaced homicidal urges (see Simpson, 1950; Zilboorg, 1936). Over time, researchers and clinicians have begun to take a more nuanced view of SII, differentiating such acts based upon their function(s), lethality of means, suicidal intent, as well as physical and interpersonal consequences (e.g., Linehan, 1997). For example, therapeutic strategies may differ depending upon whether the primary function of SII is to regulate negative emotions, communicate with others, self-punish, or cause death. Similarly, access and/or desire to use highly lethal means may result in more or less time intensive or restrictive interventions (e.g., access to guns is considered in hospitalization decisions). Most important, researchers and clinicians distinguish between behaviors with *zero* suicidal intent (i.e., NSSI) and behaviors with any *nonzero* level of intent to die (i.e., suicidal behaviors). We now know that even though lethality and suicidal intent often align such that high-lethality means are chosen for high-intent behaviors and low-lethality means are chosen for low-intent behaviors, these two factors can also covary in unexpected patterns. Some individuals report high intent to die but use low-lethality means and vice versa. It is essential to assess the function, lethality, intent, and consequences of SII across many different self-injurious behaviors in order to characterize research/clinical samples and tailor interventions accordingly.

Unfortunately, the utility of distinguishing between suicidal and nonsuicidal behaviors led many scholars and practitioners to believe that this distinction is also an appropriate way of categorizing people. This assumption is often inaccurate and has affected clinical research and practice. Recent studies of SII frequently restrict samples to a narrow phenotype of participants (e.g., nonsuicidal self-injurers or suicide attempters only) and thus fail to capture the full range of self-injurious behaviors. Many self-report measures also focus exclusively on one category or the other, for example, assessing NSSI while neglecting suicidal self-injury. Similarly, the current *Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition* (DSM-5; APA, 2013) now has two distinct diagnoses outlined in the section on "Conditions for Further Study": nonsuicidal self-injury disorder and suicidal behavior disorder. If adopted, these diagnoses will further reify the notion that people can be slotted reliably into one category or the other, in spite of data to suggest that many individuals who self-injure will eventually engage in both suicidal and nonsuicidal self-harm (although associations between SII behaviors are undeniably complex; Andover, Morris, Wren, & Bruzzese, 2012). The DSM approach of creating more diagnostic categories with increasingly narrow phenotypes is inconsistent with the DP perspective and contributes to excessive comorbidity.

Comorbidity

Early versions of the DSM included diagnostic hierarchies that precluded clinicians from assigning multiple disorders to patients (Beauchaine, Klein, Erickson, & Norris, 2013; First, 2005). *Comorbidity*, the simultaneous co-occurrence of multiple clinically significant problems or disorders within the same individual, has been a significant concern within the

https://doi.org/10.1017/S0954579416000705

field ever since the hierarchical system was abolished with the introduction of DSM-III-R (APA, 1987; Klein & Riso, 1993). Since that time, epidemiological research indicates that approximately 55% of adults with psychopathology have a single diagnosis, 22% have two, and 23% have three or more disorders (Kessler, Chiu, Demler, & Walters, 2005). In clinical samples, comorbidity rates are far higher (e.g., Fusar-Poli, Nelson, Valmaggia, Yung, & McGuire, 2014). Although some comorbidity is understandable and valid given that underlying biological systems are associated with a wide range of clinical problems (e.g., dopaminergic hypoactivity underlies both impulsivity and irritability; Beauchaine, Klein, Crowell, Derbridge, & Gatzke-Kopp, 2009), our current diagnostic system vastly overestimates both the number of existing diagnoses and the frequency of their co-occurrence (e.g., Caspi et al., 2014).

Furthermore, the criterion sets that make up the DSM are largely atheoretical and primarily descriptive, placing a disproportionate emphasis on the topography of behavior rather than latent vulnerability traits (Beauchaine, Gatzke-Kopp, & Mead, 2007; Beauchaine & Marsh, 2006). This creates arbitrary distinctions and obscures biological and trait-based vulnerabilities for psychopathology, which are distributed continuously in the population (see Beauchaine & Marsh, 2006; Krueger et al., 2002). Many argue that this artificial splitting of latent vulnerabilities into multiple diagnoses represents a failure to "carve nature at its joints" (e.g., Beauchaine & McNulty, 2013). For example, most externalizing spectrum disorders share a common heritable vulnerability, as do many internalizing disorders (see Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007; Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002; see also Krueger & Markon, 2006), which may contribute to comorbidity within the externalizing or internalizing spectra. Developmental psychopathologists label this overlap *homotypic comorbidity*, or the co-occurrence of multiple within-spectrum disorders within an individual (e.g., depression and anxiety; Beauchaine, Neuhaus, et al., 2008). In contrast, *heterotypic comorbidity* is the co-occurrence of disorders across the internalizing/externalizing spectra (e.g., depression and CD).

Relative to homotypic patterns, heterotypic comorbidity is less simply explained. There is less overlap in diagnostic criteria, biological vulnerabilities, and contextual risks. Thus, heterotypic comorbidity may reflect distinct disease processes transmitted separately via complex biological and environmental mechanisms (Kopp & Beauchaine, 2007). Alternatively, such comorbidity may represent moderating influences on biological vulnerabilities (Krueger & Markon, 2006). Biologically based sex differences or sex- and gender-based socialization processes are one especially interesting moderating influence. For example, vulnerability to negative affectivity may lead to depressive affect or to aggressive behavior depending on sex-specific genetic and/or socialization mechanisms (see e.g., Beauchaine, Hong, & Marsh, 2008). Etiology-based diagnosis and treatment is more likely to be fruitful for disentangling the source of comorbidity and the developmental course of complex clinical problems such as SII (see Beauchaine & Marsh, 2006; Crowell et al., 2009; Preskorn & Baker, 2002). In order to develop such an alternative approach, researchers must attend to longitudinal transactions between biological and contextual mechanisms of risk. This is a primary goal of DP perspective.

DP

As a field, DP emerged initially from the union of developmental and child clinical psychology perspectives (Sroufe & Rutter, 1984). Researchers in the new field adapted the methods and measures of each founding discipline, creating a rich and novel approach to studying the emergence of psychopathology over the life span. Central tenets of the DP perspective are that (a) adaptive and maladaptive developmental processes are mutually informative, (b) biological and contextual processes are constantly transacting to produce observed behavior, and (c) traits and behaviors are best conceptualized as continuous, both from adaptive to maladaptive presentations across people and throughout development within a single person. These tenets have encouraged DP scholars to identify unique vulnerability, risk, and protective factors, allowed for more flexibility when studying problematic behaviors and outcomes, and have improved the ability to describe intraindividual change across development (Hinshaw, 2015; Schmeck, Schluter-Muller, Foelsch, & Doering, 2013; Sroufe & Rutter, 1984; Widiger & Trull, 2007).

Adherents to the DP perspective take a multiple levels of analysis approach to understanding the etiology of behavior through examining mechanisms of continuity and discontinuity across the life span (Rutter & Sroufe, 2000). Several key constructs have arisen in the DP literature in order to describe these complex phenomena and developmental processes (e.g., heterotypic and homotypic comorbidity). Many DP researchers are interested in the emergence of different disorders within the same person at distinct points in development. In some cases, a new diagnosis supersedes prior diagnoses yielding a pattern of sequential comorbidity; in other cases, multiple diagnoses are accrued additively across development. Regardless, the term *homotypic continuity* is used to describe an enduring pattern of symptoms or behaviors that are consistent in their behavioral manifestation across development. For example, an adolescent diagnosed with anxiety may continue to be anxious as an adult. However, the concept of homotypic continuity not only is used to describe a person with a stable diagnosis over time. This term also is used more broadly to describe enduring dimensional traits, characteristics, or behavior patterns within a person. In contrast, *heterotypic continuity* denotes a developmental trajectory in which symptoms, behaviors, or diagnoses change across time within an individual, such as when a child with separation anxiety later develops oppositional defiant disorder.

A related set of concepts is multifinality and equifinality. *Multifinality* is used to describe trajectories by which a single

https://doi.org/10.1017/S0954579416000705

biomarker, contextual factor, or diagnosis is associated with multiple distinct outcomes measured later in development (e.g., adolescents who engage in SII may later develop BPD, ASPD, depression, or have no significant psychopathology; Crowell et al., 2009). *Equifinality* is a term used when many diverse developmental pathways lead to a single outcome (e.g., many different trajectories lead to suicide; see Crowell, Derbidge, & Beauchaine, 2014). These four developmental concepts (homo/heterotypic continuity, multifinality, and equifinality) are highly important for understanding the emergence of SII. Because SII does not emerge before late childhood or early adolescence (except in rare cases), we must examine complex trajectories characterized by both continuities and discontinuities across development. One fruitful avenue for prevention research is in understanding how early temperament links to personality and later psychopathology.

Temperament, personality, and psychopathology

Temperament, or early-emerging endogenous traits, influences a range of individual differences from later personality functioning to pathological behavior (see Bates, Schermerhorn, & Petersen, 2014; Davidson, Jackson, & Kalin, 2000; McCrae et al., 2000). Temperament researchers propose that heritable biologically based differences, stemming from both structural and functional neural processes, contribute to the emotional and behavioral response tendencies that make up a child's disposition. These response tendencies, in the context of myriad environmental exposures, may ultimately develop into symptoms of psychopathology (Kagan, 2013). There is increasing evidence to suggest that a range of outcomes can be predicted from a relatively small number of underlying temperamental characteristics. For example, trait impulsivity has been implicated in disordered behaviors across the externalizing spectrum (e.g., Beauchaine & McNulty, 2013), whereas trait anxiety (rooted in early behavioral inhibition) has been linked to internalizing problems (e.g., Beauchaine, 2015; Williams et al., 2009). Thus, temperament and subsequent personality traits appear to underlie trajectories to internalizing versus externalizing psychopathology.

Temperamental traits map on well to dimensional conceptualizations of personality, spanning models of typical personality functioning to personality disorders (Cloninger, Svrkic, & Przybeck, 1993; Costa & Widiger, 1994; Kendler, Myers, & Reichborn-Kjennerud, 2011; Kochanska, 1997; Rothbart, 2007; Rothbart, Ahadi, & Evans, 2000; Tackett, Slobodskaya, et al., 2012; Trull & Widiger, 2015). From a dimensional perspective, personality disorders reflect extreme and maladaptive variants of basic personality traits (see, e.g., Gore & Widiger, 2013). This assertion is supported by empirical work demonstrating that elevations on five-factor model trait profiles are as accurate as DSM criterion sets for diagnosing personality disorders (Glover, Crego, & Widiger, 2012). Many scholars favor continuous approaches to research and clinical diagnosis, because such approaches are better able to capture both stability and change in personality over development. Robust empirical evidence also demonstrates moderate to high stability of temperamental and subsequent personality traits, even when diagnostic continuity is low across time (see, e.g., Durbin & Klein, 2006; Jylhä et al., 2013). Furthermore, prospective longitudinal and epidemiological research shows temperamental characteristics and personality dimensions can effectively predict a host of important outcomes, such as behavior problems, interpersonal functioning, employment, psychiatric disorders, and criminal behavior (Caspi, 2000; De Fruyt et al., 2006; Hampson, 2008; Shiner & Caspi, 2003; Tackett, Kushner, De Fruyt, & Mervielde, 2013).

There is a growing theoretical and empirical literature linking trait impulsivity to SII, BPD, ASPD, and other diagnoses along the externalizing spectrum such as attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder, and CD (see, e.g., Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Crowell et al., 2009; Hinshaw et al., 2012; Swanson, Owens, & Hinshaw, 2014). Those who are high in trait impulsivity often act on urges with less deliberation, engaging in reactive, rapid, undercontrolled approach behavior to rewarding stimuli without appropriate consideration of potential negative consequences (Eisenberg, Spinrad, & Eggum, 2010; Martel, 2013, Nigg, 2006). In addition to reward-related approach, those high in trait impulsivity may also be more likely than those who score low on this trait to engage in active rather than passive avoidance strategies when distressed (e.g., SII rather than withdrawal). Research with clinical samples indicates impulsive and/or high-risk behaviors are associated with (a) the desire to upregulate a chronically aversive negative mood state (e.g., boredom) through approach behaviors or (b) avoid emotional pain through active avoidance (e.g., Beauchaine, Hinshaw, & Pang, 2010; Linehan, 1993). Thus, it is important to clarify that behavioral impulsivity can emerge due to both reward seeking and pain avoidance motivations. These processes may have different biological correlates (e.g., Carver & Miller, 2006).

Although trait impulsivity is linked to SII, BPD, and ASPD, multifinal pathways invariably lead to these conditions. Thus, trait impulsivity is unlikely to be the only developmental antecedent to these complex conditions. A less thoroughly explored temperamental feature within the SII, BPD, and ASPD literature is trait anxiety and its temperamental precursor behavioral inhibition. Those high in behavioral inhibition display an overarching tendency toward negative emotionality and reactivity to novelty in infancy (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). By childhood, these individuals tend to avoid stimulating situations and are wary in novel contexts (Kagan, 2013). Longitudinal studies find that 3-year-old children characterized as undercontrolled (i.e., impulsive) *or* inhibited are more likely to report suicidal behavior by age 21 relative to those who were well adjusted at the age of 3 (Caspi, Moffitt, Newman, & Silva, 1996). Thus, it appears that early trait anxiety, which is linked to later symptoms of depression and anxiety, may represent an inter-

https://doi.org/10.1017/S0954579416000705

nalizing trajectory to SII and BPD (Beauchaine, 2015; Neuhaus & Beauchaine, 2013).

On the surface it may seem that trait impulsivity and anxiety represent extreme ends along a single dimension of behavioral control. However, these traits appear to be mediated by unique genetic and neural substrates (see further discussion below) and are also correlated. Nonetheless, males appear to be more genetically predisposed to trait impulsivity and externalizing problems, whereas females are typically more susceptible to trait anxiety and internalizing psychopathology (Caspi et al., 2014; Eme, 2015). Accordingly, in our ontogenic model, we highlight sex-specific mechanisms contributing to different rates of internalizing and externalizing disorders in females and males, respectively. We also examine contextual factors that shape biological risk for psychopathology including in utero stress exposure, parent–child dynamics, and societal inequities that perpetuate the intergenerational transmission distress among disadvantaged and chronically stressed populations.

The ontogeny of self-injury and related conditions

As articulated elsewhere, ontogenesis is defined as the scientific description of an organism or of a behavioral/anatomical feature across development (Crowell et al., 2015). A key tenet underlying the study of ontogenic processes is that the same disease or condition may manifest differently over time (Beauchaine & McNulty, 2013). In other words, the phenotypic expression of a disorder is likely to look quite different during infancy versus older adulthood even though core biological dysfunctions are similar across both time points. Just as a person's facial features show continuity *and* discontinuity across the life span, the connections between early temperament and later psychopathology are often both readily apparent and nuanced.

The central thesis of this review is that there are identifiable developmental precursors to SII that can be shaped into distinct presentations and comorbid disorders. Even though there are multifinal pathways to SII, there are a few common trajectories leading probabilistically to this outcome. Such pathways frequently begin in early development with trait impulsivity, trait anxiety, or more important, through the co-occurrence of these early vulnerabilities (Beauchaine, 2015). In turn, SII is an early-emerging feature of later psychopathology. Thus, an ontogenic perspective elucidates key points for prevention and early intervention, not only for SII, but for other forms of psychopathology as well. We hypothesize that the following developmental processes increase risk for SII and several related clinical conditions:

- Early vulnerability for psychopathology is transmitted at conception via heritable genetic and epigenetic transmission of parental vulnerabilities.
- Epigenetic processes in utero, including maternal stress, teratogen exposure, and sex-linked hormonal processes, affect newborn neurobehavior and temperament.
- Two temperamental tendencies (trait impulsivity and anxiety) are identifiable in infancy and display some sex-specific segregation. These temperaments are associated with biological differences in monoamine neurotransmitter systems, such as serotonin, dopamine, and norepinephrine.
- Parenting strategies and gender-specific socialization processes shape internalizing and externalizing developmental trajectories among vulnerable infants. Risk may be especially high for youth with both internalizing and externalizing vulnerabilities and/or for girls presenting with traditionally masculine traits/behaviors and boys presenting with traits/behaviors typically associated with females.
- Across development, coercive parenting practices, emotional invalidation, and conflict escalation lead to more severe emotion dysregulation among vulnerable youth. These interpersonal processes are replicated in peer relationships, which further increases risk for psychopathology.
- By adolescence or young adulthood a constellation of emotional and behavioral characteristics begins to stabilize, which includes shared features of SII and antisocial/borderline personality disorders (e.g., interpersonal problems, aggression, identity dysfunction, and emotional lability).

In this review we provide evidence of these developmental processes (see Figure 1). Of note, some aspects of this theory have been articulated in prior work (see, e.g., Beauchaine et al., 2009; Crowel et al., 2009). Accordingly, we emphasize new developments and recent findings on the emergence of SII, ASPD, and BPD, with a particular focus on early development.

Intergenerational Transmission of Psychopathology

Vulnerability for psychopathology is transmitted within families, yet the precise mechanisms of this transmission are exceptionally difficult to disentangle. For example, it is well established that genes contribute to intergenerational transmission of disease vulnerability, including risk for complex psychiatric disorders (Thompson, Hammen, Starr, & Najman, 2014). At the same time, the environment also exerts a powerful influence on gene expression from conception through death (Natsuaki et al., 2013). Furthermore, contextual forces not only have a powerful effect on genetic methylation patterns (i.e., the epigenome) but also moderate the extent to which specific genes are linked to risk versus resilience (e.g., Boyce & Ellis, 2005; Bridgett, Burt, Edwards, & Deater-Deckard, 2015). These two areas of research (epigenetics and gene–environment interactions) have received significant attention in the psychopathology literature. However, intergenerational transmission of vulnerability is affected by numerous additional processes, including active, passive, and evocative gene–environment correlations, selective breeding, and intergenerational transmission of nongenetic or complex environmental stressors/teratogens, many of which are not entirely separable from potential genetic influences

https://doi.org/10.1017/S0954579416000705

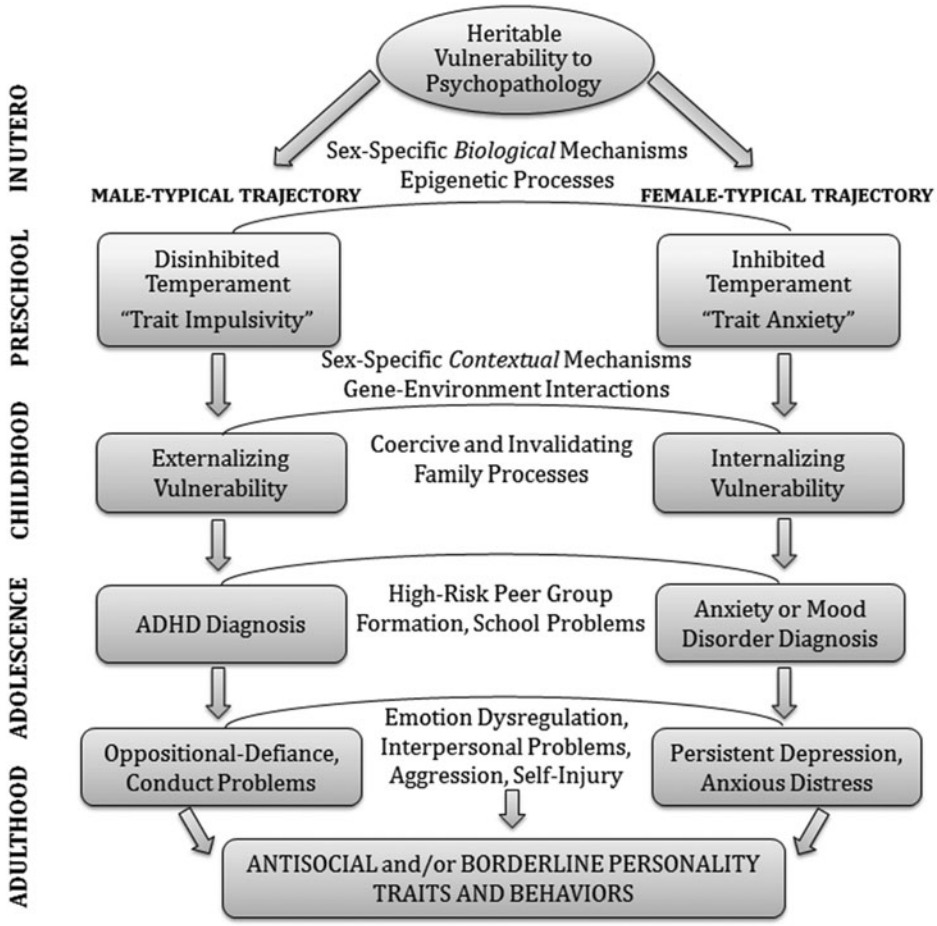

Figure 1. Ontogenic model of self-inflicted injury and developmental continuities with borderline and antisocial personality traits.

(e.g., poverty, sexual abuse, partner violence, and exposure to pollutants; see Beaver & Belsky, 2012; Miller & Barnes, 2013; Scarr & McCartney, 1983; Stith et al., 2000). Because it is difficult to disentangle these complex influences through observational and quasi-experimental methods, it is necessary to consult animal models and acknowledge the limitations of human studies.

Methodological caveats aside, there is clear evidence that psychopathology is familial but that diagnoses do not "breed true" (i.e., depressed parents have offspring with disorders other than depression; Starr, Conway, Hammen, & Brennan, 2014). As stated above, it is essential to examine broad vulnerability and risk factors for intergenerational transmission rather than one-to-one correspondence between parent and child diagnoses (Bridgett et al., 2015). This approach is consistent with factor analytic studies of psychopathology, which have found that DSM diagnoses cluster reliably into broader categories of internalizing, externalizing, and thought disorders (e.g., Krueger, 1999). As reviewed elsewhere (Beauchaine & Thayer, 2015), internalizing disorders are characterized by excessive behavioral inhibition, which has been linked to septohippocampal dysfunction and deficits of the serotonin (5HT) and norepinephrine systems. In contrast, externalizing disorders are characterized by mesolimbic dysfunction and dopamine deficits. Although relevant to broad understanding of psychopathology, we do not focus on thought disorders in this review.

In essence, it appears that several genetically linked neurobiological vulnerabilities to psychopathology may account for broad transmission of risk from parent to child. The specific neural mechanisms underlying this transmission remain unknown. However, there is strong evidence of prefrontal cortex (PFC) dysfunction across internalizing, externalizing, and thought disorders (e.g., Menon, 2011). There is also evidence of an association between PFC dysfunction and lower respiratory sinus arrhythmia (RSA), a biomarker of poor parasympathetic regulation over heart rate (Beauchaine, 2001; Thayer & Lane, 2009). Both PFC dysfunction and low RSA have been linked to deficits in self-regulation, especially inhibition of prepotent responses and effortful regulation of emotions (Beauchaine & Thayer, 2015). Two early-emerging aspects of poor self-regulation include temperamental traits of impulsivity and trait anxiety. It is important to note that these traits are defined variably in the literature reviewed here. However, it is widely accepted that impulsivity is characterized by undercontrolled behaviors that emerge early in

https://doi.org/10.1017/S0954579416000705

development and that, by later development, are expressed without appropriate forethought or consideration of potential consequences (Bridgett et al., 2015; Neuhaus & Beauchaine, 2013). In contrast, trait anxiety is marked by negative emotionality and overcontrolled or fearful behaviors, which are especially activated in novel contexts (Kagan, 2013).

Researchers examining parent to child transmission of psychopathology have examined a number of potential risk factors, several of which predate conception. For example, maternal (and possibly paternal) diet and adiposity, maternal (and possibly paternal) toxicant exposure, and maternal stress have each been linked to newborn neurobehavioral outcomes (see Nigg, 2016). We hypothesize that such factors increase risk for all forms of psychopathology, including trait impulsive and anxious profiles as well as their correlation. Kagan (2013) identified a group of infants that he labeled as "high-reactive" who later developed both internalizing and externalizing problems. High-reactive babies had observed behavior patterns characterized by back arching, excessive limb activity, and crying. Thus, it is possible that high-reactive infants represent a subgroup of youth with high vulnerability to psychopathology that is shaped into internalizing, externalizing, or correlated trajectories across development. As noted above, biological sex is an important moderating factor when examining developmental trajectories. In one longitudinal study, the externalizing pathway was associated with male sex and personality traits such as extraversion, low conscientiousness, and low agreeableness (Caspi et al., 2014). Internalizing, however, was associated with female sex and traits of introversion and neuroticism. Thus, vulnerability to internalizing and externalizing psychopathology appears to be driven, in part, by sex-linked personality styles with roots in early temperament.

Epigenetic processes in utero

Over the past two decades, there has been strong interest in identifying epigenetic mechanisms of disease (Dawson & Kouzarides, 2012). Epigenetics is the scientific study of meiotic and mitotic changes in gene *expression* rather than DNA sequence. These changes occur through environmental processes shaping DNA methylation, histone modification, and RNA-linked silencing (Egger, Liang, Aparicio, & Jones, 2004). More recently, scholars have begun to explore epigenetic consequences of prolonged stress exposure, which is a potential source of individual variation in lifelong vulnerability to psychiatric diagnoses, such as ADHD and depression (Crowell et al., 2015; Nestler, 2012; Rice et al., 2010; Talge, Neal, Glover, & Translational Research Prevention Science Network, 2007). Evidence is accumulating that risk for most psychiatric problems begins early in life, and individuals are most vulnerable to disease during periods of rapid developmental change (Network Pathway Analysis Subgroup of the Psychiatric Genomics Consortium, 2015). The fetal period is unmatched in terms of neuronal growth and development, and the fetus is particularly vulnerable to prenatal stress (Nestler, 2012). However, the processes involved in epigenetic transmission of risk for psychopathology are understudied and poorly understood. Early identification of high-risk infants may improve prevention efforts targeted at reducing stress exposure prenatally (Mulder et al., 2002), inform parent education on sensitive care (Pluess & Belsky, 2010) for exposed newborns, and/or promote novel therapeutic targets (Abel & Poplawski, 2014) to buffer the fetus against maternal stress.

Extensive animal research supports the theory that maternal stress has a lasting influence on offspring phenotype and that changes in gene expression mediate this association (Champagne & Curley, 2009). Most animal studies, which inform human epigenetics research, examine the effects of severe and repeated stressors, including subjecting the mother to dominant males, restraining her under a bright light, or exposing the mother to random loud noises (Champagne & Curley, 2009; Darnaudéry & Maccari, 2008). However, translating such findings from bench to clinic has proven difficult, making it unclear whether environmental stressors have lifelong transgenerational epigenetic effects in humans (Bollati & Baccarelli, 2010). A common translational approach is to select mothers with clinical diagnoses, such as depression, as one proxy of maternal stress exposure (Conradt, Lester, Appleton, Armstrong, & Marsit, 2013). However, depression alone may not capture the full range of maternal distress (Lester, Conradt, & Marsit, 2014). There is an emerging consensus that several environmental factors, including nutrition, stress, behavior, toxins, and stochasticity (i.e., unknown, random effects), contribute to phenotypic alterations among animals and humans (Faulk & Dolinoy, 2011). In other words, extending animal findings to humans will necessitate identifying mothers whose health and well-being are compromised across multiple domains.

There are several challenges associated with linking epigenetic findings to complex outcomes such as SII or personality disorders. Foremost among these is the lack of longitudinal studies linking prenatal influences (e.g., methylation patterns in placental tissue) with adult outcomes. A related challenge is the complexity involved in interpreting adult findings when distal causal factors are unknown or poorly specified. Finally, epigenetic results are most interpretable when tissue is assayed at the site of influence (i.e., directly in the brain), which makes findings from deceased samples more readily understandable epigenetically but which precludes further assessment with the subject. Nonetheless, there is a growing body of research examining potential epigenetic effects in SII and personality pathology. For example, McGowan et al. (2009) found differences in mRNA and mRNA transcription in a glucocorticoid receptor (NR3C1) promoter in the postmortem hippocampus of suicide victims with a history of childhood abuse relative to suicide victims with no abuse and controls. This finding replicated rat studies examining quality of parental care and lasting effects on offspring development. Yet another interesting line of research examines epigenetic changes among adults with depressive symptoms or

https://doi.org/10.1017/S0954579416000705

BPD in response to treatment. Findings indicate that both medication and behavioral interventions can produce changes in methylation patterns (see Perroud et al., 2013). Taken together, there is a growing literature to suggest that gene expression patterns are maleable across the life span and that early life experiences can have a powerful effect on later development. Across several prospective studies, maternal depression, anxiety, or stress during preganacy is shown to potentiate infant emotional and behavioral problems, such as ADHD. Although genetics and the postnatal environment can also affect developmental trajectories, one recent review estimated that prenatal stress exposure may contribute 10%–15% of the variance in emotional/behavioral outcomes (Glover, 2015). Thus, future research must examine the specific biolgoical changes linking maternal stress to early temperament and later psychopathology.

Temperament and early development

During infancy, temperamental vulnerabilities begin to express phenotypically (see, e.g., Beekman et al., 2015; Schwartz et al., 2012; Sheese, Voelker, Posner, & Rothbart, 2009). A number of unique models propose higher order temperamental dimensions and lower order dispositional traits that interact to produce observable affective, behavioral, and physiological responses (e.g., Buss & Plomin, 1975, 1984; Chess & Thomas, 1966; Rothbart, 1981; Rothbart & Ahadi, 1994; Zentner & Bates, 2008). Although no single model is accepted universally, there is general consensus that (a) individual differences in temperament have a basis in neurophysiological substrates and (b) extreme expressions of these traits confer vulnerability to psychopathology (see Martel, 2013, for a review). A comprehensive review of the temperament literature is beyond the scope of this paper. However, three constructs (approach/withdrawal, affective sensitivity, and effortful control) appear regularly in the temperament literature and may be relevant to the later development of SII, BPD, and ASPD. Each of these domains and its biological substrates show some sex-specific segregation that, in combination with socialization practices, may contribute to "male typical" and "female typical" trajectories over time (Beauchaine et al., 2009). It is important to note that the literatures on approach/withdrawal, affective sensitivity, and effortful control emerged from independent schools of thought, even though they describe overlapping phenomena. Thus, we highlight areas of convergence, especially at the biological level of analysis, but do not attempt to integrate these different frameworks.

Approach/withdrawal. Researchers have long described endogenous motivational systems that modulate appetitive and aversive behaviors in terms of *approach* and *withdrawal* (e.g., Cloninger et al., 1993; Gray & McNaughton, 2000; McNaughton & Corr, 2004). Approach tendencies reflect readiness to seek potential incentives and rewarding contexts, whereas withdrawal tendencies reflect an individual's propensity to retreat from potentially unrewarding or ambiguous contexts (Nigg, 2006). These motivational tendencies have been termed the behavioral approach system (BAS; or behavioral activation) and behavioral inhibition system (BIS; Fowles, 1980). The BAS facilitates appetitive behaviors in response to reward and plays a central role in reinforcement-based learning and goal-directed activity (Barrós-Loscertales et al., 2010; Fowles, 1980). When faced with divergent motivational objectives, the BIS suppresses prepotent behaviors (whether approach or avoidance related), and is experienced as an anxiety response. Activating this system is adaptive under appropriate circumstances. Anxiety may facilitate behaviors consistent with effective risk assessment, resolve competing motivational goals, and generate defensive action (Brenner, Beauchaine, & Sylvers, 2005; Gray, 1987).

Researchers using the BIS-BAS framework theorize that under- or overactivity of the neurotransmitter systems that underlie BIS and BAS may each confer risk for psychopathology (see Brenner et al., 2005). Moreover, combinations of atypically low and high functioning of these systems may predict different disorders. Although some researchers have presented approach/withdrawal as a single latent dimension (see, e.g., Martel, 2013), evidence indicates that BAS and BIS are mediated by distinct neurobiological mechanisms beginning in infancy. The primary central nervous system (CNS) substrates underlying BAS functioning are *dopaminergic* (DA) pathways originating in the ventral tegmental area and projecting to the nucleus accumbens and the ventral striatum (Swartz, 1999). This mesolimbic DA network matures exceptionally early in development, and it is a key CNS mechanism underlying disinhibition over the life course (see Beauchaine, Katkin, Strassberg, & Snarr, 2001; Castellanos, 1999; Gatzke-Kopp & Beauchaine, 2007; Kalivas & Nakamura, 1999; Sagvolden, Johansen, Aase, & Russell, 2005). Specifically, *hypo*dopaminergic functioning is associated with low positive affectivity, trait irritability, and trait impulsivity (see Beauchaine et al., 2009; Crowell et al., 2009; Gatzke-Kopp & Beauchaine, 2007; Laakso et al., 2003; Sagvolden, Russell, et al., 2005). Thus, abnormally low BAS functioning has been linked to deficient motivation and depression (e.g., Takahashi, Ozaki, Roberts, & Ando, 2012) and also to externalizing diagnoses such as ADHD, CD, ASPD, and substance use disorders (Neuhaus & Beauchaine, 2013). In addition, hypodopaminergic functioning has also been linked to heterotypic comorbidity across the internalizing and externalizing spectra (e.g., comorbid conduct problems and depression; Beauchaine, 2012).

In contrast, the BIS is supported by neural structures including the amygdala and septohippocampal system and is innervated primarily by *serotonergic* (5-HT) pathways. This system is thought to inhibit approach behaviors under conditions of threat and mediate trait anxiety (Gray & McNaughton, 2000; McNaughton & Corr, 2004; Neuhaus & Beauchaine, 2013). When underactive, the septohippocampal system can result in an impulsive phenotype that is similar behaviorally to that produced via the DA pathway (i.e., equifi-

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nality; see Beauchaine et al., 2001). Those who are low in trait anxiety often fail to attend to and respond effectively to punishment cues or are unable to desist from problematic ongoing behaviors (Beauchaine & Neuhaus, 2008). Early deficiencies in the 5-HT system predict aggressive and antisocial behavior later in the developmental trajectory (Flory, Newcorn, Miller, Harty, & Halperin, 2007; Kruesi et al., 1992). There is also robust evidence that 5-HT dysfunction is associated with self-injury, suicide, borderline pathology, and impulsive aggression (e.g., Crowell et al., 2008; Gollan, Lee, & Coccaro, 2005; Joiner, Brown, & Wingate, 2005; Kamali, Oquendo, & Mann, 2001; Lee & Coccaro, 2007; Lis, Greenfield, Henry, Guile, & Dougherty, 2007; van Goozen, Fairchild, Snoek, & Harold, 2007).

The appeal of the BIS-BAS framework lies in is its clear connections to CNS neurotransmitter systems, which can be measured across development. These biological systems underlie early-emerging trait anxiety and impulsivity, which are both highly heritable traits and which confer vulnerability to the developmental risk trajectories depicted in Figure 1 (see also Beauchaine, 2015). However, these biological systems and their behavioral concomitants are also shaped over time by environmental inputs. Contextual factors play a key role in the early emergence of emotion regulation and effortful control and the consolidation of these self-regulation skills into adolescence and adulthood.

Affective sensitivity and emotion dysregulation. A propensity toward negative affectivity in infancy is another vulnerability factor for maladaptive outcomes. This trait is related to later emotion dysregulation when met with environmental risks (Beauchaine, 2001). As described above, vulnerability to negative affectivity is mediated by central DA functioning, with low levels contributing to irritability and high levels (e.g., infusions of DA into mesolimbic structures) producing pleasurable affective states (Ashby, Isen, & Turken, 1999; Berridge, 2003; Berridge & Robindon, 2003; Forbes & Dahl, 2005). By the time diagnosable psychopathology emerges, vulnerable children struggle to regulate negative affect. In most forms of psychopathology, one or more emotions is either too prolonged or experienced too intensely (e.g., panic, rage, euphoria, or depression) to be adaptive (Beauchaine et al., 2007).

At the autonomic level, RSA is a widely used measure of early emotional sensitivity and later emotional lability (Beauchaine, 2001, 2015). RSA is a measure of the ebb and flow of heart rate across the respiratory cycle and an established index of parasympathetic influences on heart rate via the vagus nerve (Berntson et al., 1994; Cacioppo et al., 1994). When the stimulus conditions are carefully controlled, resting RSA can serve as a biomarker of emotion regulation capacity, whereas RSA reactivity is associated with emotional lability (Beauchaine, 2015). Theoretical and empirical evidence suggests high RSA is protective, whereas low RSA renders individuals vulnerable to emotion dysregulation (Crowell et al., 2009; Porges, 1995, 2007; Thayer & Lane, 2009). Self-injuring adolescents show attenuated RSA and a negative slope on RSA reactivity during sad emotion induction and interpersonal conflict (Crowell, Baucom, et al., 2014; Crowell et al., 2005). Although low baseline RSA and excessive RSA reactivity to emotion are associated with psychopathology (Beauchaine, 2001), the development of specific externalizing and internalizing disorders is influenced by other neurobiological systems, temperamental traits like effortful control and approach/withdrawal tendencies, and environmental factors (see Beauchaine et al., 2001, 2007; Beauchaine & Gatzke-Kopp, 2012; Chambers & Allen, 2007; El-Sheikh et al., 2009; Mead, Beauchaine, & Shannon, 2010; Porges, 2007).

Effortful control. The construct of effortful control (EC; Nigg, 2000) is another widely examined temperamental trait with clear ties to SII and later related psychopathology. Similar to emotion regulation, EC refers to broad self-regulatory elements of temperament, especially under conditions in which exercising self-restraint is difficult (Diamond, 2013; Rothbart, 2012; Rothbart & Bates, 2006). Current theory suggests functioning in this domain is facilitated by executive attention systems, because EC includes the capacity for appropriately shifting and focusing attention, enacting effortful behaviors when adaptive (even when not preferred), and inhibiting desired behaviors when maladaptive (Eisenberg et al., 2010, 2013; Petersen & Posner, 2012). The propensity to exert EC is theorized to depend on the prefrontal cortex (especially the dorsolateral, orbitofrontal, and anterior cingulate cortex; Derryberry & Tucker, 2006; Rothbart & Posner, 2006; Whittle, Allen, Lubman, & Yucel, 2006) and anterior neural systems such as frontal–striatal neural loops (Nigg & Casey, 2005). EC dysfunction emerges early in development, is relatively stable, and is linked to externalizing problems and behavioral impulsivity. Low EC is also associated with internalizing problems and anxiety when combined with excessive negative affectivity (see Nigg, 2006, for a review). Research indicates that functioning in the EC domain moderates symptom severity among those with BPD such that higher EC functioning is associated with better outcomes (Hoermann, Clarkin, Hull, & Levy, 2005).

Sex differences. The dispositional traits described above and many lower order temperamental dimensions demonstrate well-validated sex differences (Costa, Terracciano, & McCrae, 2001; Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006; Feingold, 1994; Gartstein & Rothbart, 2003; Hyde, 2005; Schmitt, Realo, Voracek, & Allik, 2008). Such differences appear to have biological origins (see Eme, 2015, for a review), and are often canalized into male-typical and female-typical trajectories to SII, BPD, and ASPD via socialization practices (described below). During infancy, males typically exhibit higher approach tendencies (i.e., more activity and high-intensity pleasure), and lower negative affectivity and EC compared to females (Gartstein & Rothbart, 2003; see Martel, 2013, for a review). Given this constellation, males

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tend to be at greater risk for externalizing pathways to psychopathology, and eventual ASPD. Females are more likely to demonstrate high negative emotionality, low approach, and high EC (Eme, 2015; Martel, 2013) and are more often at risk for internalizing pathway to SII and BPD. Although we often see these sex-typical trajectories, evidence suggests that externalizing problems during childhood and early adolescence are prospectively associated with BPD symptoms in *both* adolescent girls and boys (Burke & Stepp, 2012; Stepp, Burke, Hipwell, & Loeber, 2012; Swanson et al., 2014). Similarly, some boys may arrive at BPD and SII via a more traditional internalizing pathway. The combination of internalizing and externalizing vulnerabilities may place youth at exceptionally high risk (Crowell et al., 2009).

Early socialization and sex-typical trajectories

Gene–environment interaction models have highlighted the significance of an individual's unique social context for moderating genetic effects on developmental outcomes (Cacioppo, Berntson, Sheridan, & McClintock, 2000). According to the DP perspective, functional impairment arises when youth's biologically based temperamental traits are ill suited to the environment (Cicchetti & Posner, 2005; Linehan, 1993; Rutter & Sroufe, 2000). Thus, in order to better understand prospective risk for SII, BPD, and ASPD, and their continuity/discontinuity, we must examine salient developmental contexts and socialization practices by which the child's dispositional characteristics manifest and are shaped over time (e.g., Hallquist, Hipwell, & Stepp, 2015; Hopwood Schade, & Pincus, 2014; Schaffer, Barak, & Rassovsky, 2015).

The family system and especially the parent–child relationship serve as important environmental contexts for temperamentally vulnerable infants (Hughes, Crowell, Uyeji, & Coan, 2012; Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012). Parent–child transactions can have a lasting influence on youth affective and self-regulatory behavior via social and biological mechanisms (Diamond, Fagundes, & Butterworth, 2012; Laurent, 2014; Lyons-Ruth, 2008). For example, conflictual parent–child relationships can worsen internalizing and externalizing symptoms among youth who are high on emotional instability (Feinberg, Kan, & Hetherington, 2007; Huh, Tristan, Wade, & Stice, 2006). Furthermore, caregiver behavior influences the neural regions such as the prefrontal cortex, limbic structures, and the hypothalamus, which affect youth social affiliation, executive functioning, and emotion regulation (see Hughes et al., 2012, for a review).

However, youth are not only receiving and responding to social cues but also actively evoking others' behavior. Thus, children help to create their own environments from infancy onward (Cicchetti & Cohen, 2006; Hallquist et al., 2015). Temperamentally vulnerable youth often make substantial demands on caregivers, which has an effect on socialization and skill acquisition (Crowell, Yaptangco, & Turner, 2016; Stepp, Whalen, Scott, et al., 2014). For example, youth with impulsive personality traits have an evocative effect on parent–child interactions (Burt, McGue, Krueger, & Iacono, 2005), and infant emotional reactivity and negative affectivity interact with maternal caregiving strategies to predict child emotion regulation strategies (Mirabile, Scaramella, Sohr-Preston, & Robison, 2009; Spinrad, & Stifter, 2002). Thus, temperamental vulnerabilities shape parent response, which in turn may further exacerbate the child's maladaptive behaviors (Stepp, Whalen, & Pedersen, 2014).

Although there appear to be biologically based temperamental differences between males and females (see above), sex-differentiated socialization at parental and cultural levels of analysis further shape early traits into internalizing and externalizing psychopathology. Previous research demonstrates that parenting practices predict externalizing behavior for boys and internalizing behavior for girls (Rothbaum & Weisz, 1994). However, the mechanisms promoting these sex-typical trajectories are complex and fairly nuanced. Some findings indicate that parents interact with their children in ways that reinforce stereotypic gender role-consistent emotional displays (Brody, 1999; Chaplin, Cole, & Zahn-Waxler, 2005; Lytton & Romney, 1991). For example, parents use a greater variety of emotion-related words and employ more specific emotional terms when communicating with daughters compared to sons (see Fivush, 2007), which in turn predicts individual differences in children's use of emotion labels (Cervantes & Callanan, 1998). Parents also (a) use more anger-related words with boys and refer more to sadness and happiness with girls (for a review, see Chaplin et al., 2005), and (b) attend and respond more frequently to daughters' emotions of sadness and anxiety and sons' expressions of anger. Furthermore, parental attention to these emotions predicts the expression of sadness and anxiety 2 years later (Chaplin et al., 2005).

Peer groups and differentiated patterns of play also elicit and reinforce sex-segregated emotional styles (Rose & Rudolph, 2006). Males tend to play in larger groups, engage in physical aggression and rough-and tumble games, and engage in less extended dyadic interactions compared with girls (Brody & Hall, 2010). Females tend to disclose more feelings to peers, engage in more prosocial behaviors, and report greater friendship stress compared with male youth. All of these factors may further reinforce girls' facility in expressing and decoding emotions, especially in expressing vulnerable feelings (Brody, 1999; Rose & Rudolph, 2006).

Parents are also less accepting of atypical gendered misbehavior from their children or symptoms that violate social expectations and norms for children's conduct based on their sex. Kim, Arnold, Fisher, and Zeljo (2005) found that internalizing symptoms in girls and externalizing symptoms in boys predicted lax parenting approaches, whereas females' externalizing behaviors and boys' internalizing symptoms were each associated with overreactive disciplinary practices and parental hostility. Thus, children who display gender-inconsistent symptoms may be at particularly high risk for conflictual environments and associated negative outcomes such as SII.

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Contextual risk for emotion dysregulation and psychopathology

As outlined in Figure 1, many youth traverse along an externalizing or internalizing trajectory leading to increasingly severe psychopathology. Early temperamental vulnerabilities are shaped over time into diagnosable disorders of childhood, such as ADHD or separation anxiety disorder. During adolescence, these youth are at increased risk for oppositional behavior and/or major depressive disorders (Caspi et al., 2014). There is extensive research examining the social and contextual mechanisms that increase risk for continuing along these heterotypically continuous developmental pathways. Much of this research began by delineating processes that lead some youth with ADHD to develop oppositional defiant disorder or CD, whereas others desist from this course. Researchers identified specific familial processes related to problematic developmental outcomes, which they labeled coercion (see Crowell et al., 2016).

Coercive family processes are problematic dyadic interaction patterns that promote and maintain emotion dysregulation, interpersonal conflict, and psychopathology (Patterson, 1976, 1982). Specifically, coercion theory posits that maladaptive behaviors emerge in part due to early parent–child relationships characterized by harsh, inconsistent parenting practices and negative reinforcement patterns (Dodge, Greenberg, & Malone, 2008; Patterson, DeBaryshe, & Ramsey, 1989). Coercive processes have been studied extensively among externalizing youth, and researchers have found that these parent–child interaction patterns contribute to risk for antisocial behavior in adulthood via social learning processes and biological adaptations to adversity. There have been fewer attempts to characterize family patterns leading to SII. However, there is emerging evidence that similar social interactional dynamics may operate in the relationships of those with BPD or self-injuring behaviors (Crowell et al., 2013). Specifically, these relationships also appear to be characterized by emotional invalidation in which the emotional needs of the child are ignored, minimized, or rejected. Individuals in these relationships also engage in reciprocal reinforcement of negative affect (i.e., increasingly aversive behaviors are necessary in order for each person to get their needs met; see Crowell, Beauchaine, & Linehan, 2009; Linehan, 1993).

Coercive interactions can become a recurrent pattern within families due to intermittent negative reinforcement of aversive behaviors. Negative reinforcement is characterized by temporary relief when an undesired stimulus is stopped or removed (Baldwin & Skinner, 1989; Patterson, Dishion, & Bank, 1984). For example, a tantrum may lead a parent to withdraw his or her demand (e.g., to go to bed), effectively ending the argument and temporarily sparing the dyad further relationship problems. However, coercion is reciprocal in nature, and parents are also reinforced when yelling or other aversive behaviors are effective at ending child misconduct (El-Sheikh & Erath, 2011). Recent empirical work is consistent with this theory. In a study of mother–daughter dyads, self-injuring adolescents and their mothers expressed higher levels of aversive verbal behavior during conflict (Crowell et al., 2013). Furthermore, relative to controls, both members of the dyad were more likely to match one another's aversive behavior, only deescalating the conflict when their dyad partner became highly dysregulated. In contrast, control dyads were more likely to deescalate conflict in all instances, especially the mothers. This suggests that aversive and dysregulated behavior may be an effective means of ending conflict temporarily. Over time, however, such patterns likely contribute to interpersonal problems, poor emotion regulation, psychophysiological dysregulation, and more severe psychopathology.

There is evidence that coercive family processes can shape biological development across multiple systems. For example, chronic environmental stress can affect neurodevelopment of prefrontal cortical regions involved in top-down regulation of emotions and behaviors, potentially increasing risk for externalizing behavior problems (see Beauchaine & Zalewski, 2016). In our research examining mother–daughter conflict, self-injuring and depressed adolescents showed a high-risk psychophysiological pattern in response to maternal aversiveness relative to typical controls. Specifically, self-injuring and depressed adolescents showed RSA decreases during minutes when their mother became more aversive. In contrast, controls showed RSA increases in response to maternal aversive behavior (Crowell, Baucom, et al., 2014). This finding is consistent with theories that coercion, invalidation, and aversive escalation may contribute to emotion dysregulation among vulnerable youth via negative reinforcement processes (Beauchaine et al., 2009; Crowell et al., 2009).

It is possible that negative and positive reinforcement cycles also serve to shape and maintain suicidal thoughts and behaviors. For distressed adolescents, the thought of death can provide powerful relief from the ongoing pain of daily life. Similarly, some research suggests that SII can reduce negative emotions and serve a calming function (Klonsky, 2007). Suicidal communications and SII can be positively reinforced when these behaviors are met with additional warmth or support by parents, therapists, or systems (e.g., residential treatment facilities). In essence, family dynamics may inadvertently potentiate emotion dysregulation, and once SII is initiated, the reinforcing properties of self-injurious behaviors may maintain it. Although there is relatively little research examining the peer relationships of self-injuring adolescents, it is possible that problematic interpersonal interaction styles are replicated in the peer relationships of self-injuring adolescents, contributing to lasting interpersonal problems (Hughes et al., 2012; Prinstein et al., 2010).

The emergence of SII, BPD, and ASPD

Adolescence is a critical developmental stage characterized by reorganization and consolidation of both biological and social structures. Research on parent–child relationships during this stage reveals that it is a period of proximity seeking

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toward and also individuation from caregivers (Ruhl, Dolan, & Buhrmester, 2015). Peer and romantic relationships become central and form a foundation for adult attachment patterns (Hughes et al., 2012). Several key skills are canalized during the adolescent years, which can affect the probability of adaptive or maladaptive developmental outcomes. Specifically, adolescence is marked by cognitive development, the emergence of more sophisticated emotion regulation skills, challenging interpersonal situations that require navigation, identity formation tasks, onset of risky health behaviors, and frequent emotional distress due to endogenous and exogenous stressors. Although few researchers have examined all of these challenges in conjunction, strengths and deficits across these core skills are correlated (Linehan, 1993). This suggests that common developmental processes may contribute to dysregulation of emotions, behaviors, interpersonal relationships, cognitions, and identity.

According to our ontogenic model and consistent with the DP perspective, a stressed caregiving environment leads to dysregulation across multiple domains, especially in a biologically vulnerable child. Thus, youth who struggle to navigate the developmental tasks of childhood and/or who are not scaffolded through this stage by caregivers are at elevated risk for psychopathology during the teenage years. There are a number of factors that may account for important changes during adolescence that either promote or protect against psychopathology. The predominant biological model of healthy adolescent development is one of increasing cortical control over subcortical brain structures (i.e., "frontal cortical immaturity" or "linear advances in PFC development" theory; Crone & Dahl, 2012, p. 639). This model can partially account for self-regulation improvements across normative adolescent development. However, this oversimplifies complex biological and social processes among high-risk adolescents (Crone & Dahl, 2012, Hughes et al., 2012). More recent work has emphasized interdependence of neural systems related to reward and incentive processing, motivation, affective and social processes, and cognitive control (Crone & Dahl, 2012). Thus, future research must integrate findings at the nexus of DP, social and affective neuroscience, and personality theory in order to understand the emergence of high-risk health problems and personality disorders during this stage (see Crowell & Kaufman, in press).

Complex interactions between hormone levels and neural functions contribute to systemic changes in the brain during pubertal development. These changes begin around age 9 and continue well into adolescence. Pubertal changes in gonadal hormone levels appear to produce reorganization of the adolescent brain in a sex-specific fashion. For example, gray matter thickening occurs about 1 year earlier in girls than in boys, which correlates with earlier pubertal onset in girls (for a review, see Sisk & Zehr, 2005). Similarly, during adolescence the volume of the hippocampus increases for girls, whereas amygdala volume increases for boys, which may contribute to sex-specific trajectories in internalizing and externalizing symptoms for girls and boys, respectively. In rat studies, males and females also differ in brain region-specific reorganization of D1 and D2 DA receptors during adolescence, although this appears to occur independent of gonadal hormone levels (Sisk & Zehr, 2005). Specifically, dopamine receptors are initially overexpressed in the striatum and prefrontal cortex and then are pruned in later adolescence. Dopamine receptors in the nucleus accumbens, however, increase around the onset of puberty but are not pruned. The overexpression and later pruning of dopamine receptors is more pronounced in males relative to females (Sisk & Zehr, 2005), which may partially contribute to risk taking and externalizing symptoms among boys (Steinberg, 2008).

Researchers examining brain development have recently begun to focus on the connectome, or the study of neural connections in the brain. Adolescence to early adulthood is an interesting stage for connectome research given the extent of brain development during this stage (second only to fetal and early childhood development; DiMartino et al., 2014). Doll and colleagues (2013) recently examined intrinsic functional connectivity within and between three brain networks (the salience network, default mode network, and central executive network) in a small sample of patients with BPD. Relative to controls, those with BPD showed aberrant patterns of resting connectivity such that networks involved in emotion were activated relative to those involved in cognitive control. Similar findings were reported for adults with ASPD, who showed uncoupling between the default mode network and the attention network relative to healthy controls (Tang, Jiang, Liao, Wang, & Luo, 2013). The authors hypothesize that this may account for poor self-regulation among those with ASPD. Although this research is preliminary, it is clear that a network of brain structures underlies complex clinical problems such as SII, BPD, and ASPD (e.g., Peled, 2013).

Personality disorders emerge in late adolescence and show relatively high stability into young adulthood (see Stepp, 2012). In addition, core traits such as aggression, interpersonal problems, identity dysfunction, emotional lability, and self-injury also appear during this stage (Crowell et al., 2009). Changes in brain maturation likely contribute to much of the "stable instability" observed during adolescence. However, social pressures and ongoing problems in parent–child relationships must not be overlooked. Adolescence can be viewed as a sensitive period characterized by increased neural plasticity. Thus, this may be a particularly fruitful stage for treatment of SII and prevention of personality pathology.

Conclusions and Future Directions

In this review we have outlined an ontogenic process model of intentional self-injury, including comorbidities and continuities with borderline and antisocial personality traits. Consistent with other papers examining the ontogeny of psychological problems, we hypothesize that early markers of psychopathology (e.g., trait impulsivity) are different phenotypically from later psychiatric diagnoses (e.g., ASPD). Thus, individuals at risk for SII and related conditions follow a heterotypically continuous trajectory beginning with genetic

https://doi.org/10.1017/S0954579416000705

vulnerabilities and epigenetic processes that shape early temperament. Over time, these early biomarkers shape infant temperament, which interacts with child sex and potentiating environmental experiences to predict psychopathology and other health problems in adolescence and adulthood. Without intervention, adolescents and adults with antisocial and/or borderline traits are at heightened risk for suicide and transmission of risk to the next generation.

Developmental psychopathologists view early intervention as an effective form of prevention. There are two key points of intervention that are often neglected in the DP literature and in suicide prevention research: (a) treating adults who may later become parents and (b) improving prenatal and infant health. There are important advantages to furthering this work. Adults in their childbearing years make important contributions to the workforce and society. Thus, there are economic and social benefits associated with providing accessible treatment options for adults, including suicide prevention, lower rates of incarceration, reduced recidivism, lower reliance on other forms of government assistance, and improved physical health. It is also critical that we provide better support to parents during the prenatal and infant stages of child development, particularly among those who are vulnerable and/or under significant environmental stress. There is increasing evidence that maternal stress produces biological adaptations in her unborn child, which may be one mechanism contributing to difficult infant temperaments and intergenerational transmission of psychopathology. Decades of research have established the challenges of raising a difficult child. Such youth appear to have an evocative effect on parenting behaviors and are also uniquely sensitive to their caregiving environment (Boyce & Ellis, 2005; Burt et al., 2005).

Youth with a highly reactive temperament are at increased risk for childhood forms of psychopathology, such as ADHD and/or separation anxiety disorder. This in turn affects relationships with peers and educators. During this stage, few youth express suicidal urges or self-injurious behaviors. When such behaviors occur, they typically include lower lethality behaviors, such as head banging or hitting oneself without suicidal intent. Nonetheless, this is a critical stage for early treatment and ultimately suicide prevention due to probabilistic links with later SII. Childhood interventions almost always involve some form of parent coaching and support, which increases the likelihood that initial benefits will be maintained in the environment. Relative to intensive and costly interventions provided in adulthood (especially imprisonment or hospitalization), providing evidence-based therapy to children is a cost-effective and highly beneficial option. However, many third-party payers do not cover the expense of behavioral intervention for ADHD, leaving parents to rely upon medication-based treatments for symptom reduction. As a result, many families are unable to access expert psychological care during an important stage of child development.

Over the past several decades, research delineating developmental trajectories to SII, BPD, and ASPD has increased exponentially. As noted above, in some of the earliest work, researchers identified clear diagnostic antecedents to antisocial behavior problems, a heterotypically continuous trajectory leading from ADHD to oppositional defiant disorder then CD. Scholars also elucidated the contextual risks that predicted who would continue along this high-risk pathway versus who would desist, revealing coercive family processes as a predominant familial risk factor. More recent research has outlined a similar trajectory leading to BPD (Crowell et al., 2009). Temperamentally reactive, impulsive, and emotionally sensitive youth are at high risk for later BPD, especially when raised in coercive and invalidating environments. There is extensive evidence to suggest that these two developmental trajectories are fundamentally related (Beauchaine et al., 2009). ASPD and BPD are both diagnoses that lie at the intersection between impulsivity and emotion dysregulation. Thus, there can be little doubt that adults with these diagnoses followed a developmental trajectory characterized by internalizing and externalizing features. The extent to which impulsive versus inhibited traits predominate is likely shaped by many factors, especially sex- or gender-specific biological and social processes. It is clear that BPD and ASPD are phenotypically distinguishable and must not be confounded. Nonetheless, antisocial populations would likely benefit from dialectical behavior therapy, and treatment for BPD could be enhanced by attention to externalizing problems, such as substance use and anger. Many individuals within both populations would benefit from targeted intervention for self-injury and suicide prevention.

There is now a wealth of research validating components of the developmental trajectory from ADHD to self-injury and/or conduct problems and then to borderline and/or antisocial traits (Hinshaw et al., 2012; Kaufman, Crowell, & Stepp, 2014; Swanson et al., 2014). Similarly, there is evidence linking early temperament to later behavior problems, including psychopathology in adulthood. However, there is no work linking prenatal epigenetic adaptations to later self-injury, BPD, or ASPD. Evidence reviewed here suggests that adults with these diagnoses have different methylation patterns, which is consistent with early epigenetic effects (e.g., McGowan et al., 2009). Researchers should ultimately seek to link early biological development to adult outcomes through longitudinal follow-up of samples identified prenatally. This review offers theory-guided targets for this type of work, which is necessary in order to identify limitations and inaccuracies of our developmental model and to test it fully. Even without such longitudinal research, it is clear that many families lack access to the early interventions that could prevent SII, BPD, ASPD, and countless other forms of adult psychopathology. This could be one factor contributing to ongoing societal inequities in health outcomes (Saxena, Thornicroft, Knapp, & Whiteford, 2007). Similarly, suicide rates have remained relatively unchanged over the past century in spite of extensive research into proximal risk factors (Centers for Disease Control, 2015). We argue that earlier identification of risk could improve these outcomes by promoting change during sensitive biological periods and enhancing health and well-being across development.

https://doi.org/10.1017/S0954579416000705

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https://doi.org/10.1017/S0954579416000705