

# The ontogeny of chronic distress: emotion dysregulation across the life span and its implications for psychological and physical health

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Development is characterized by continuity and change across the lifespan. This is especially true of emotions and emotion regulation strategies, which become increasingly complex and variegated over development. Recently, researchers have begun to characterize severe emotion dysregulation (ED) across the life span. In particular, there is increasing data delineating mechanisms by which emotional distress leads to poor health, early mortality, and intergenerational transmission of psychopathology. In this review, we present converging evidence that many physical and psychological problems have identifiable and treatable origins in childhood ED. When the literature is examined from an ontogenic process perspective it becomes clear that many phenotypically distinct forms of mental and physical distress emerge from the same underlying emotional processes expressed differently across development.

## Addresses

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Current Opinion in Psychology 2015, 3:91–99

This review comes from a themed issue on **Emotion regulation**

Edited by **Amelia Aldao** and **Matthew T Tull**

For a complete overview see the [Issue](#) and the [Editorial](#)

Available online 6th May 2015

<http://dx.doi.org/10.1016/j.copsyc.2015.03.023>

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## Introduction

Ontogenesis is the characterization of an organism or a behavioral/anatomical feature from early development through maturity [1]. In medicine the study of ontogenic processes is common. For example, researchers studying cardiovascular disease have found that higher sympathetically mediated tachycardia during adolescence can mark a nascent stage of a disease that emerges decades later [2]. Only recently have researchers begun to take a similar approach to understanding psychological problems, such as emotion dysregulation (ED), across the life span. The ontogenic model outlined by Beauchaine and McNulty [3\*\*] encourages the examination of psychopathology

from a multiple-levels-of-analysis perspective across development. This reveals processes by which early, biologically-based trait vulnerabilities interact with complex contextual factors, heightening risk for multiple conditions. From this perspective, many diagnoses that are perceived as distinct can be demonstrated to have common origins and, therefore, to co-occur at higher-than-expected rates at single time-points and over the life span.

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5 [4]) is the predominant tool for cataloging and diagnosing mental disorders. Diagnoses are listed as discrete entities, in spite of a wealth of research questioning the categorical system [5]. This obscures etiological commonalities across conditions, with implications for research and treatment. In particular, this approach artificially increases homotypic comorbidity (i.e., co-occurrence of more than one internalizing or externalizing disorder within a person [6]) and heterotypic continuity (i.e., emergence of presumably different disorders across development [7]). However, comorbidity is better understood by examining a small number of trait vulnerabilities ontogenically rather than studying each phenotypically distinct disorder separately.

Researchers in psychology and medicine have each examined shared etiologies across comorbid conditions within their respective disciplines. However, there have been few attempts to articulate an integrated life span model of co-occurring psychological *and* physical distress. For example, many scholars disregard early emotional origins of non-genetic obesity, addiction, cardiovascular disease, and personality disorder, in spite of evidence that these conditions have many common features. Indeed, these diagnoses appear quite different from one another and from temperamental difficulties in infancy or behavior problems in childhood. However, there is increasing evidence that ED is a core feature of these seemingly distinct forms of distress.

Subjective distress is the most common criterion in the DSM followed closely by impulsivity [8]. *Distress* is extreme unhappiness, pain, or fatigue and the word has physical and emotional implications. Among those with severe psychopathology, distress is an enduring psychological state punctuated by painful moments of crisis [9,10]. This raises questions regarding how chronic physical and emotional distress emerge, from the earliest stage of embryonic development through death. To what

extent should hundreds of psychiatric diagnoses be conceptualized and treated as distinct versus developmentally-driven manifestations of the same underlying vulnerability? Many scholars believe that ED is the predominant thread connecting early life experiences to later morbidity, mortality, and intergenerational transmission of psychopathology [11–13]. The specific manifestations of this thread are shaped into distinct diagnoses via complex interdependent contextual risks, producing a tapestry of emotional and physical conditions. We assert that ED underlies many forms of chronic distress (e.g., from early behavioral dysregulation to later cardiovascular disease). Thus, when chronic distress is studied ontogenically it is possible to observe relations across psychiatric and medical diagnoses that have previously been studied by different scholars across distinct scientific disciplines.

### ED, psychopathology, and health

Clinically significant ED is typically viewed as a problem that emerges in childhood, potentiates psychopathology by adolescence, and worsens in adulthood [14–16]. It can be defined as the absence of: first, understanding and awareness of emotions; second, acceptance of emotions; third, the ability to control impulsive behavior when upset; and fourth, the ability to apply regulation strategies to meet goals and demands [17]. The links between ED and psychopathology are robust. It is a core feature of internalizing disorders, including depression and anxiety [13,18], panic disorder symptoms [19], suicidal ideation [20], suicidal and non-suicidal self-injury [21,22], and borderline personality disorder [16]. In addition, ED predicts prospective increases in internalizing disorders, rather than vice versa [23]. Research on borderline pathology and self-injury has focused extensively on ED, which is a key etiological factor in the emergence and maintenance of self-harming behaviors [24,25]. Indeed, there is evidence that a predominant function of self-injury is to provide relief for emotional distress [26].

ED, especially emotional impulsivity, has also been linked to externalizing disorders such as ADHD [27,28]. Longitudinal research suggests stressful life events and peer victimization are associated with later aggression, mediated by higher ED [29]. Dysregulation is also linked to alcohol-related problems and substance use disorders [30,31]. Early identification of ED among externalizing youth could also help predict risk for later antisocial behavior [32]. There is strong theoretical and empirical evidence linking early ADHD to later conduct problems, self-injury, borderline and antisocial personality disorders, substance use, and completed suicide [7,33,34]. Contextual processes shaping increased emotional lability appear to underlie this heterotypic developmental trajectory leading from ADHD to more severe psychopathology. There are several contextual factors that are associated with increased emotional lability over time, such as emotional invalidation, abuse, and coercive

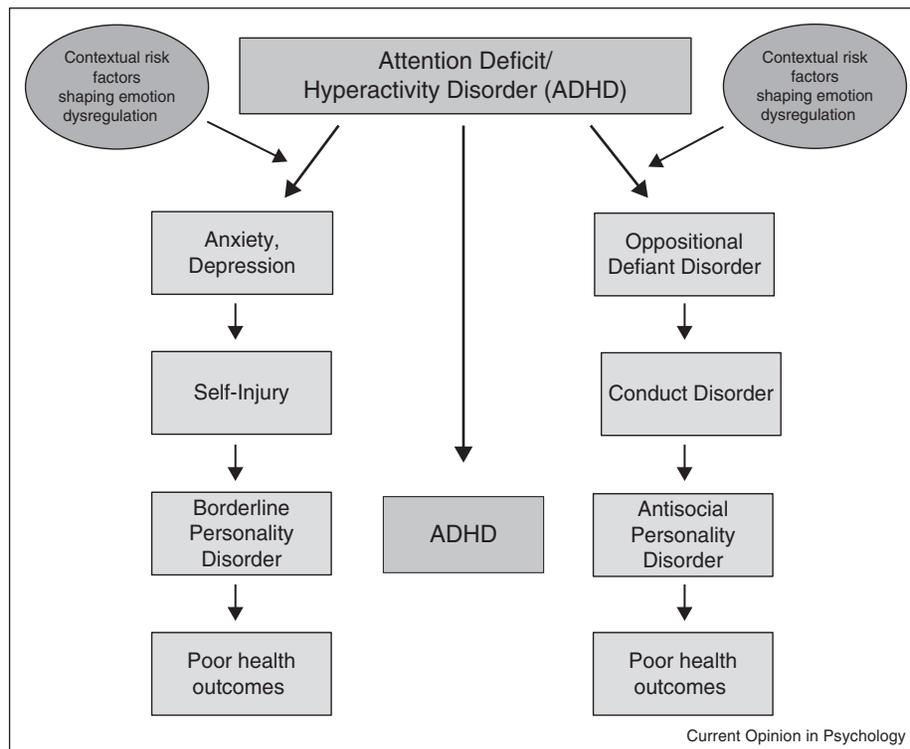
family processes characterized by negative reinforcement of conflict escalation [3\*\*]. Broadly, these contextual factors increase ED via two interrelated processes: first, communication that emotions are invalid or inappropriate and second, intermittent reinforcement of extreme negative affect. Together, these processes increase ED by failing to teach emotional awareness and by reinforcing behavior dysregulation, especially during conflict. There is now considerable evidence that contextual factors shaping ED play a significant role in the development of borderline and antisocial personality disorder among impulsive (i.e., vulnerable) children (see Figure 1).

More recently, it has become clear that ED is associated with negative health outcomes such as cardiovascular disease, arthritis, type II diabetes, osteoporosis, periodontal disease, certain cancers, Alzheimer's disease, frailty, and functional decline among older adults [35,36]. Dysregulation also contributes to sleep difficulties among those with generalized anxiety disorder and borderline personality disorder [37,38]. The mechanisms linking ED to negative health outcomes are complex [39]. Although there is some evidence that emotions can have a direct effect on health via epigenetic [40,41] and biological changes (e.g., inflammatory processes [36]), indirect mechanisms also drive these associations. This could include unhealthy stress management (e.g., smoking), poor situation selection (e.g., into stress generating environments), or risk-taking behaviors (e.g., drunk driving; self-injury). Furthermore, ED has interpersonal origins and consequences [42]. Dysregulated emotions and behaviors may increasingly alienate sources of emotional support, reducing access to healthy friends and romantic partners.

### Life-span development

Although a fully articulated life span model is beyond the scope of this review, key elements of our perspective are depicted in Figure 2 (see also [3\*\*,16]). We acknowledge that all infants lack regulation strategies. However, some infants are also biologically and/or temperamentally vulnerable (e.g., trait impulsive) or difficult to sooth. These infant vulnerabilities interact with contextual stressors to shape ED. Because regulatory skills and strategies are socialized, problems within proximal attachment relationships are uniquely devastating. Over time, temperament–environment interactions alter underlying biology (e.g., fight-flight systems, parasympathetic regulation, functional connectivity of brain circuits, DNA methylation) via allostatic mechanisms. Poor self-regulation further affects parent–child, peer, and romantic relationships, which can lead to risky health behaviors, severe psychopathology, and physical illness by adulthood. Finally, stress and poor health alter biological systems among unborn offspring of vulnerable parents, increasing risk for intergenerational transmission of ED.

Figure 1



Heterotypic continuity from ADHD to internalizing and externalizing disorders.

### Infancy and early childhood

Timely acquisition of emotional control in infancy is associated with later psychosocial functioning [43,44]. Infants possess relatively little ability to independently regulate emotions, depending upon external sources of co-regulation, especially parents [45]. Indeed, negative parenting behaviors and low sensitivity in infancy are associated with poor psychosocial outcomes throughout childhood [46]. Furthermore, babies who exhibit more frequent and intense negative emotions are more difficult to soothe [47,48], receive less sensitive parenting [49,50], and are more likely to develop behavior problems (e.g., tantrums) with unresponsive parenting [51,52]. In the context of highly responsive parenting, however, emotionally reactive infants can develop better self-regulation [53].

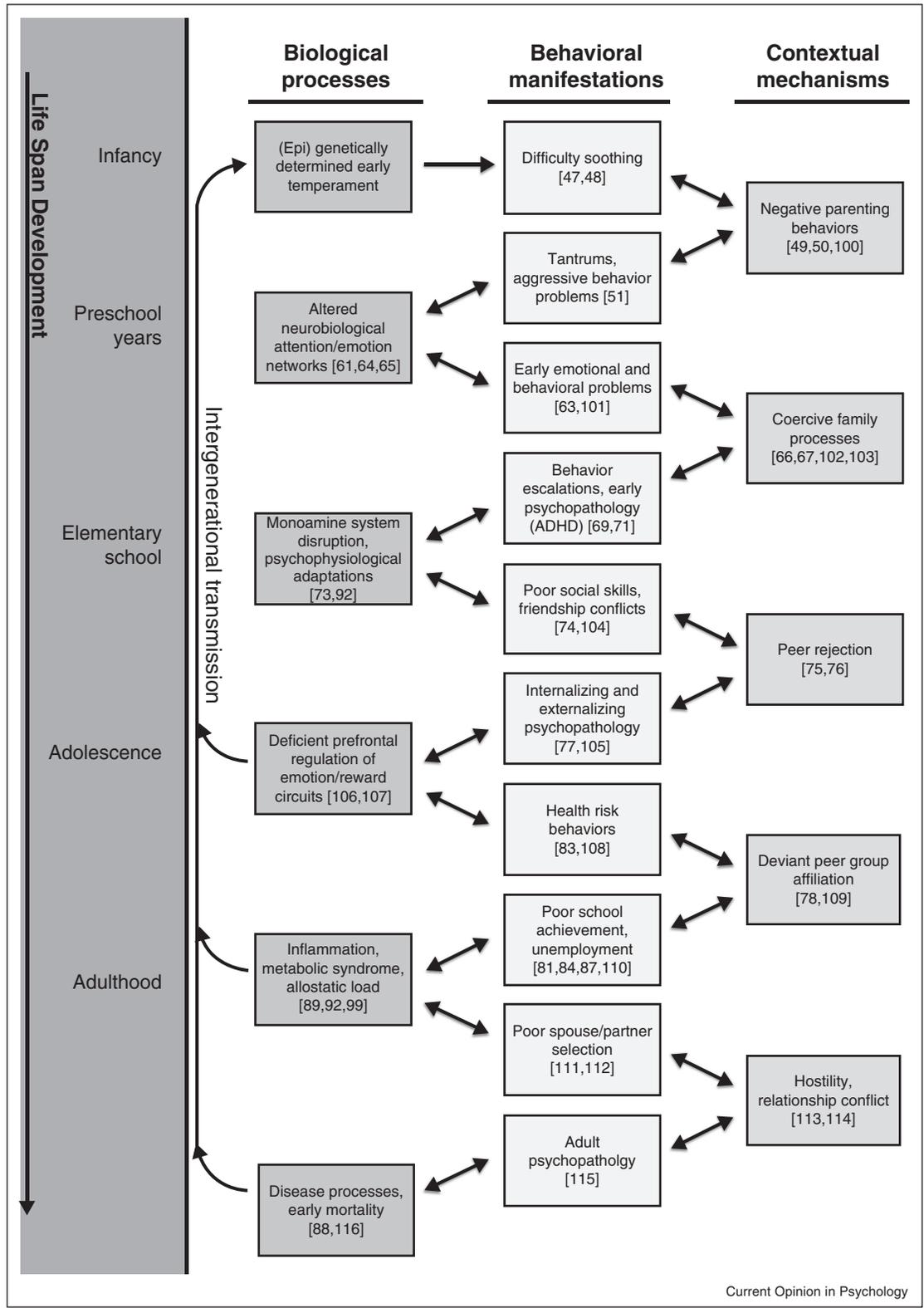
Early caregiving experiences affect developing biological systems [54–56] and these differences persist into young adulthood [57–59]. In particular, unsupportive early environments affect neurobiological systems related to attention and self-regulation [60]. For example, parental responsiveness during infancy is associated with higher respiratory sinus arrhythmia (RSA) in childhood [61], a biological index of attention and emotion regulation capacity [11]. Low parental sensitivity in infancy is associated with lower RSA in childhood and less effective

emotion-regulation [62]. Similarly, delays in executive control over attention are associated with regulatory problems later in childhood [63]. This is because executive attention abilities scaffold later behavioral, emotional, and cognitive regulation [64,65]. In other words, early experiences initiate a cascade of biological and social processes that shape ED.

### Later childhood

Research in later childhood reveals that biology  $\times$  environment interactions potentiate severe ED across development [54]. Moreover, dysregulated children can elicit caregiver reactions that are similarly problematic [66]. This results in coercive family processes, which have been linked to emergence and maintenance of ED [67]. Coercion is a process whereby parent and child use increasingly extreme displays of negative affect to get needs met [7,68]. Extreme behaviors are intermittently reinforced when one party relents, which increases the frequency of dysregulated conflict-resolution tactics. Complex transactions between early externalizing problems and parent–child conflict can lead to externalizing behaviors and the development of internalizing symptoms [69,70]. Other problematic parent–child conflict interactions (e.g., limited emotional variability [71,72]) are also associated with adolescent psychopathology.

Figure 2



Simplified life span developmental model of emotion dysregulation across biological, behavioral, and contextual domains (See refs. [47–51\*,61,63–67,69,71,73–78,81,83,84,87–89\*,92,99–116]).

## Adolescence

The consequences of ED extend beyond home. Over time, responses to conflict become entrenched and are characterized by physiological dysregulation and labile emotions [73]. This affects social functioning and peer relationships. For example, ED mediates the relationship between adolescent ADHD and social/friendship problems [74]. Peer difficulties also increase risk for depression and anxiety [75,76]. Additionally, adolescents with ADHD who experience peer rejection are more likely to progress along externalizing risk trajectories, including tobacco use, conduct problems, and delinquency [77,78].

Adolescence is also a period of biological change in systems that underlie self-regulation, including improved prefrontal modulation of subcortical brain structures [79]. However, neuromaturation development of subcortical structures (e.g., the amygdala and ventral striatum) outpaces development of cortical regions involved in regulation (e.g., prefrontal cortex [80]). Vulnerable adolescents are also more likely to engage in high-reward risky behaviors (e.g., substance use, promiscuity), further disrupting neurodevelopment maturation [7]. During late adolescence and young adulthood, affiliation with deviant peer groups may mediate associations between ED and externalizing behaviors [78]. Deviancy training is a process by which deviant behaviors are modeled and reinforced by group members, leading to academic decline, psychopathology, and health-risk behaviors [81–83].

## Adulthood

Dynamic associations between individual dysregulation and deviant peer groups increase risk into young adulthood via school drop-out [84], delinquency and incarceration [85,86], and limited prospects for employment [87]. Over time the effects of dysregulation can accumulate, resulting in immune deficiencies, cardiovascular disease, and early mortality [88]. Accumulating evidence suggests that ED affects inflammatory pathways [89,90] via increased allostatic load [91,92]. Furthermore, dysregulated adults are more likely to experience social stressors in relationships and work [93,94], which place acute strain on physiological systems [95,96]. Psychopathology is also associated with obesity, metabolic syndrome, diabetes onset, and mortality through complex interactions between emotional, behavioral, and neuroendocrine systems [97–99].

## Summary and future directions

The human tendency to catalog has produced numerous psychiatric and medical diagnoses. This obscures connections within and across disciplines, creating artificial boundaries between scholars. Child development researchers have argued that the origins of distress begin at conception and are exacerbated by social inequity, especially inadequate access to mental and physical health resources, discrimination, and poverty. Support

for parents is especially limited (e.g., parental leave, high-quality daycare, affordable housing) and high-risk families are disproportionately affected. When parents are stressed, resources for socializing healthy emotion regulation are depleted and ED is more likely to be transmitted across generations.

In this review, we have highlighted evidence that ED is shaped and maintained through close relationships, leading to severe problems among those who are most vulnerable. Thus, ongoing interventions targeting healthy emotional development in children and families have potential to reduce risk, public health costs, and severe distress. Future research should continue to examine emotional underpinnings of physical health conditions, with the objective of preventing disease and speeding recovery among the afflicted. Additional research and resources should also be directed toward prevention and rapid intervention following childhood trauma, especially interpersonal traumas such as sexual or physical abuse by a caregiver. When physical and emotional health are conceptualized jointly, the public health implication are apparent. Early intervention and prevention have the potential to improve wellbeing and reduce burden on a taxed healthcare system.

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