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Annual Research Review: What processes are dysregulated among emotionally dysregulated youth? – a systematic review

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Proliferation of the term "emotion dysregulation" in child psychopathology parallels the growing interest in processes that influence negative emotional reactivity. While it commonly refers to a clinical phenotype where intense anger leads to behavioral dyscontrol, the term implies etiology because anything that is dysregulated requires an impaired regulatory mechanism. Many cognitive, affective, behavioral, neural, and social processes have been studied to improve understanding of emotion dysregulation. Nevertheless, the defective regulatory mechanism that might underlie it remains unclear. This systematic review of research on processes that affect emotion dysregulation endeavors to develop an integrative framework for the wide variety of factors investigated. It seeks to ascertain which, if any, constitutes an impaired regulatory mechanism. Based on this review, we propose a framework organizing emotion-relevant processes into categories pertaining to stimulus processing, response selection and control, emotion generation, closed- or open-loop feedback-based regulation, and experiential influences. Our review finds scant evidence for closed-loop (automatic) mechanisms to downregulate anger arousal rapidly. Open-loop (deliberate) regulatory strategies seem effective for low-to-moderate arousal. More extensive evidence supports roles for aspects of stimulus processing (sensory sensitivity, salience, appraisal, threat processing, and reward expectancy). Response control functions, such as inhibitory control, show robust associations with emotion dysregulation. Processes relating to emotion generation highlight aberrant features in autonomic, endocrine, reward functioning, and tonic mood states. A large literature on adverse childhood experiences and family interactions shows the unique and joint effects of interpersonal with child-level risks. We conclude that the defective closed-loop regulatory mechanisms that emotion dysregulation implies require further specification. Integrating research on emotion-relevant mechanisms along an axis from input factors through emotion generation to corrective feedback may promote research on (a) heterogeneity in pathogenesis, (b) interrelationships between these factors, and (c) the derivation of better-targeted treatments that address specific pathogenic processes of affected youth. **Keywords:** Emotional dysregulation; anger; frustration; hostility; child development.

Introduction

What is emotion dysregulation?

The term "emotion dysregulation" has proliferated in child psychology and psychiatry (Althoff & Ametti, 2021; Freitag et al., 2023). However, different emphases across – and within – subfields produce differences in its meaning. Summarizing its prevailing uses will provide grounding for this review of the processes that might underlie its phenomenology.

Perhaps the leading use of emotional dysregulation pertains to *emotion regulation*. This area of investigation focuses chiefly on the deliberate strategies through "which individuals influence which emotions they have, when they have them, and how they experience and express these emotions" (Gross, 1998). Several frameworks have been proposed for these regulatory strategies (e.g., D'Agostino, Covanti, Rossi Monti, & Starcevic, 2017; •Gratz & Roemer, 2004; Gross, 2015; McRae & Gross, 2020). They include modifying situations, attentional deployment, cognitive change such as reappraisal, and response modulation. Some accounts include automatic and implicit mechanisms, in addition to these deliberate ones (Braunstein, Gross, & Ochsner, 2017). However, these strategies may exist on a continuum from fully automatic to fully intentional (Gyurak & Etkin, 2014).

Emotional *dys*regulation, in this view, refers to ways these efforts might "go awry: emotion regulation failure (i.e., failing to regulate when it would be helpful to do so), emotion misregulation (i.e., regulating in a way that isn't well matched to the situation), and emotion regulation misexecution (i.e., using an appropriate strategy but failing to execute effectively)" (Gross, 2024, p. 10).

Child development and developmental psychopathology literatures place stronger emphases on additional concepts that are present but less prominent in adult-focused work. (a) Interpersonal influences, especially early ones, affect the development of emotion regulation (Eisenberg, 2020). (b) Its emergence is linked to the development of self-control functions more generally (Perry, Calkins,

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Dollar, Keane, & Shanahan, 2018). (c) The relationship between emotional dysregulation and common forms of childhood psychopathology can be bidirectional. That is, emotion dysregulation confers risk for psychopathology (e.g., Cole, Hall, & Hajal, 2017) and certain disorders can also engender emotion dysregulation (e.g., Walkup, Friedland, Peris, & Strawn, 2021). (d) Some view emotion dysregulation chiefly as the inadequate use of regulatory strategies. Others define it by the observable problematic behaviors from which one infers that intense emotional reactions are poorly controlled (Freitag et al., 2023).

Emotion dysregulation as a clinical phenotype

While high negative emotional reactivity might appear in numerous emotional states, literature in child clinical areas often refers to emotional dysregulation as a specific phenotype (Connor & Doerfler, 2021). This phenotype features high susceptibility to intense anger that leads to rageful behavioral dyscontrol, escape, or aggressive lashing out. These reactions typically arise in upsetting situations that unaffected age-mates handle with composure. Positive emotions can also be poorly modulated. However, unless sparked by mania or intoxication, the consequences are seldom as detrimental (Faraone et al., 2019).

This behavior pattern may arise within several disorders but is most prevalent in disruptive disorders, disruptive emotion dysregulation disorder (DMDD), and attention-deficit hyperactivity disorder (ADHD; Blader, 2021). It may also develop in other mood and anxiety disorders, following trauma, and in developmental syndromes, notably autism spectrum disorders (ASD). The specific symptoms that define these other conditions might have some regulatory disturbances at their core. However, in current usage, when one mentions "emotionally dysregulated children," it is this rageful presentation that is usually evoked. Furthermore, in most measures of emotion dysregulation among youth, items related to anger and its control are prominent (Freitag et al., 2023).

The clinical phenotype "emotional dysregulation" describes is hardly new (Connor & Doerfler, 2021). Over the years, different terms and diagnoses have designated it, including irritability, "borderline child," mood lability, multidimensionally impaired, inflexibility, bipolarity, DMDD, and "complex ADHD," among others. It is among the leading concerns in child and adolescent mental health. Its high prevalence is reflected in estimates that range from 1% to 7% (Althoff et al., 2016; Brotman, Kircanski, & Leibenluft, 2017; Copeland, Brotman, & Costello, 2015; Evans, Corteselli, Edelman, Scott, & Weisz, 2022). Its associated behavioral dyscontrol is among the chief reasons for emergency service presentation, psychiatric hospitalization, and antipsychotic medication use among youth. The risk for persistent mood, behavioral, substance use-related, and other psychiatric disturbances is high, as is the risk for peer victimization and suicidal ideation (Copeland et al., 2015; Damme, Norton, Briggs-Gowan, Wakschlag, & Mittal, 2022; Fogleman, Slaughter, Rosen, Leaberry, & Walerius, 2019).

Emotion dysregulation as an etiological concept

"Emotion dysregulation" goes beyond designating a difficulty children experience. It implies etiology because something that is dysregulated must have an impaired regulatory process. Compared with other terms for this phenotype, this usage goes further in committing to dysregulation as its cause. Research on factors that influence the development of emotion dysregulation spans a wide range of candidate processes using many investigative methods.

In a strict sense, regulation is how a system maintains outputs in a desirable range, in response to feedback about external conditions and current output (Khoo, 2018, p. 4). Literature on pathogenic processes in emotion dysregulation provides no consensus yet on what these faulty regulatory mechanisms might be. An integrative review of this research that considers if the processes studied reflect regulatory – or some other – functions is therefore timely.

Aims of this review

This review's main goal is to survey research on processes that may underlie clinically significant negative emotional reactivity, the essence of emotion dysregulation. We consider a range of methodologies and approaches that include rating scales, neuroimaging paradigms, psychophysiological research, observational methods, and other tools. Through doing so, we also endeavor (1) to determine which, if any, is a regulatory process, and (2) to develop a framework that integrates the diverse processes that have been studied to date.

One consideration about this approach is that users of "emotion regulation" might not really mean to imply a specifiable regulatory mechanism. Perhaps it is just a figure of speech to describe affective states that seem under-controlled, as if to say, "this is something that *should be* better regulated," whatever such regulation might entail. This more metaphorical use of dysregulation nonetheless reinforces the importance of establishing how problematic emotional experiences bypass the normal controls it implies.

Whether we interpret emotion dysregulation in the exact sense of a dysfunctional regulatory mechanism or a more colloquial one, a controlled-systems approach provides a useful integrative framework. First, such a framework is inherent in the concept of dysregulation. Second, most basic components of a controlled system that we detail below (e.g., inputs, controllers, response generators, feedback, regulators) have counterparts in models of emotion even as their characterizations differ (e.g., Carver, 2015; Lewis, 2005; Ochsner & Gross, 2014; Scherer & Moors, 2019). We should emphasize that this review is not predicated on the accuracy of a systems view like this. We offer it as a heuristic way to describe and organize the many processes thought to bear on emotion dysregulation without making strong assumptions about the true nature of emotion.

Accordingly, the first section of this review outlines (a) the concepts and components of a controlled, regulated system, (b) how emotion-related processes map onto these features, and (c) issues that are critical to the study of emotion dysregulation. The second section describes the method to systematically select and review the literature. The third presents the literature review itself. The fourth section discusses implications for clinical concepts, further research, and therapeutics.

Conceptual issues

What constitutes a regulatory process?

When a system produces outputs beyond a desirable range, two ways of correcting these deviations are "closed-loop" and "open-loop" mechanisms, the former is a cardinal feature of regulated systems. Regulated systems have other components besides these mechanisms. Therefore, briefly summarizing the basic components of a regulated system (Figure 1) in relation to emotion will help us to consider where the processes that generate and express emotion might exert their effects. Further details on the elements of a controlled system in physiology are available in Khoo (2018, see esp. pp. 4–10) and Hall and Hall (2021, esp. pp. 6–10). Åström and Murray (2021) focus on engineered systems with some biological examples. This area becomes more complex and mathematically rigorous as it considers specific systems whose functioning is well-understood. Fortunately, for this review we can stay at a high level of generality and many of these ideas will be familiar when put into an everyday context.

Inputs: Processing emotionally-relevant stimuli. "Inputs" are the external conditions to which a system mounts a response. As examples, current temperature is the input to a thermostat, and an automobile's wheel rotation speed is the input to cruise control. One input in the regulation of blood pressure is the stretch on baroreceptors located in the carotid arteries and aorta – higher arterial pressure triggers these neurons. In models of emotion, inputs are stimuli. Sensory stimuli undergo processing for identification and to extract relevance (i.e., salience), meaning, value (e.g., valence), and other attributes. For this review, literature on stimulus extraction and processing is considered under the general rubric of "input" factors. As we detail below, accounts of emotion dysregulation often point to anomalies in stimulus processing as important contributory factors. For instance, a person with a propensity to perceive situations as more threatening or frustrating than their peers is likely to repel or avoid things more forcefully and at lower thresholds than others who lack that propensity.

Controllers: Managers of outputs. Controllers use information from inputs to activate, deactivate, change, or maintain outputs. A simple example in building climate control is the switch that turns on or off the heating/cooling equipment, based on inputs (i.e., the desired temperature and the current one). In blood pressure regulation, baroreceptor signals are transmitted to the medulla, which controls vasoconstriction through the sympathetic nervous system (SNS; Hall & Hall, 2021, p. 7).

The neural architecture of the control of behavior and cognition is broadly understood; the functioning of neural networks in the context of affective arousal is also becoming clearer. For instance, networks subserving *impulse control* and *resisting interference* involve the dorsal caudate and inferior frontal cortex (Aron, Robbins, & Poldrack, 2014; Schmidt et al., 2020). *Higher-level executive functions*, anchored by prefrontal and anterior cingulate cortices underpin cognitive control and executive functions (Menon & D'Esposito, 2022). The latter includes processes that select and activate responses. If controllers are unable to properly weigh and direct the implementation of response alternatives or to constrain undesirable behavior, responses may default to reflexive, stimulus-driven options.

Outputs: Processes that generate emotional responses. If the mechanisms that produce an *output* are defective, undesirable results can occur even if input and control processes are intact (e.g., a furnace's broken fan will not circulate warm air even though the thermostat works).

For emotion, outputs include subjective emotional experience, behaviors, and physiological changes. As we will see, some of these have been studied in relation to the emotion dysregulation phenotype. They include phenomena like heart rate variability, endocrine function, and neural factors that underlie the capacity to experience pleasure. Much of this research does not settle whether anomalies in the control of, say, heart rate is necessarily a cause rather than a downstream effect of other emotion-related processes (such as inputs, controllers, etc.). But for conceptual and practical reasons it seems worthwhile to examine distinct factors that might underlie the production of emotion-related phenomena in its own rubric. There are many clinical examples of physiologic changes altering the substrate of behavior: for instance,

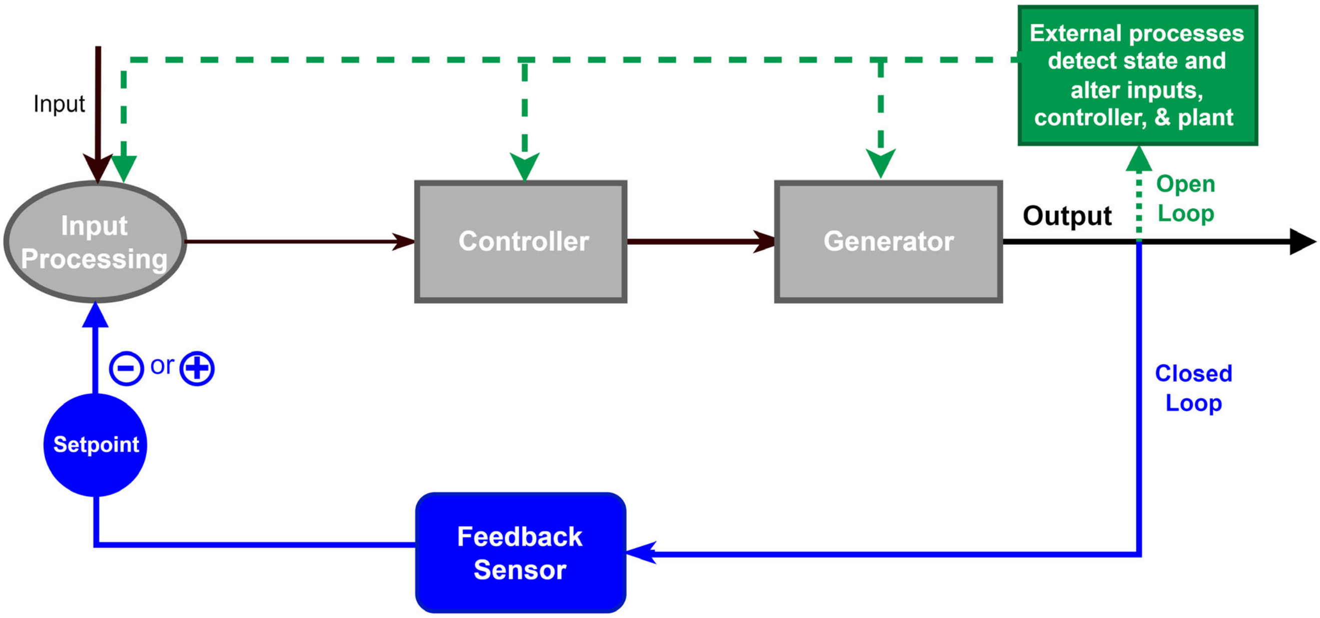

Figure 1 Components of a basic regulated system

hyperthyroidism, which alters metabolic rate, neurotransmitter synthesis, and sleep, can make some individuals appear manic (Feldman, Shrestha, & Hennessey, 2013). In other words, anomalies in processes that directly incite somatic or cognitive features of emotion might independently contribute to the emotion dysregulation phenotype.

Closed-loop regulation: Feedback, Setpoint, and intrinsic mechanisms. A *closed-loop regulation system*, like a thermostat or blood pressure, is self-correcting. In contrast, an *open-loop* system discussed next, can provide feedback but involves some process external to the system to make corrections. *Closed-loop regulation* uses feedback to compensate automatically when output deviates from the target value or *set point*. The closed-loop components appear in blue in Figure 1. They include a sensor that measures the output, a mechanism to compare it with a set point, and an intrinsic mechanism to correct deviations from it. Closed-loop processes are ubiquitous in physiology (Khoo, 2018). Most involve *negative feedback*, where deviations from a set point trigger an output correction in the opposite direction. A thermostat is a good example in an engineered system (e.g., if room temperature is higher than the set point, the air conditioning is turned on), and baroreceptors that control blood pressure (e.g., excessive stretch on the receptors indicate high pressure in the vessel and leads brain stem areas to cause vasodilation). *Positive feedback* systems respond to outputs beyond the set point by instigating measures that push output further away from it, further exacerbating the deviation from set point. Positive feedback loops tend to be destabilizing, but are useful in some cases (e.g., early contractions in childbirth stimulate more frequent and intense ones).

Open-loop regulation: Self-monitoring and deliberate regulatory action. In open-loop regulation, the output has no direct effect on the system's controller. Instead, getting the desired output requires intervention from outside the system (e.g., the room gets cold and someone puts logs on the fire). The brown dashed-line pathway at the top of Figure 1 illustrates open-loop control. The open-loop processes with counterparts in emotional control involve deliberate, rather than automatic, strategies to alter emotional experience and behavior, such as reappraisal, deep breathing, and effortful control (e.g., "counting to ten" before reacting). While deliberate regulatory strategies of this sort are prominent in the adult emotion regulation (e.g., Gross, 2015), their role for children is less clear. One issue is the development of the cognitive capabilities to implement strategies such as reappraisal, and the strengthening of top-down controls for such reasoning to be effective. Another issue discussed further below is that the emotion dysregulation phenotype in children often involves a rapid escalation of emotionally charged behavior. With short latencies for strong negative reactions to get underway, the restraining effects of deliberate, contemplative strategies may be reduced compared with less intense emotional states that "simmer below boil."

Issues in the study of emotional dysregulation

What distinguishes dysregulation from other sources of affective disturbances?. Even in a controlled system, not all disturbances reflect impaired regulation (Gross & Jazaieri, 2014). For example, we have mentioned how arterial blood pressure is regulated with closed-loop feedback mechanisms. However, dysfunction in those mechanisms is not the prevailing cause of hypertension, which more often results from high flow resistance in blood vessels. In these cases, the regulatory mechanism per se usually works within its expected capacity but has limited ability to compensate for high vascular resistance.

Applying the analogy to emotion, anomalies in processing emotion-eliciting stimuli may lead some individuals to experience a given stimulus as more aversive or intense. This heightened sensitivity may provoke a strong response against which some countervailing regulatory processes are ineffective, not because the regulatory process does not function but because the stimulus is perceived as so intense that it is not susceptible to the usual controls. Similarly, abnormalities in the processes that generate emotional "outputs" can contribute to excessive physiological responses. Regulatory mechanisms *might* moderate these outputs to more acceptable levels, but their shortcomings in doing so may not be the primary factors causing disorder.

Processes influencing negative emotional reactivity can therefore exert effects, regulatory or otherwise, at various points in the generation and management of emotional experience. We structured this review accordingly, by attempting to situate where in the chain of events the processes studied might have their primary impact.

Discordance among emotion response systems. Although convenient to refer to emotion as a unitary entity, emotional experience involves several response processes (Lange, Dalege, Borsboom, van Kleef, & Fischer, 2020; Scarantino, 2018; Scherer & Moors, 2019). Response components – subjective experience (including cognition), behavior, and physiological change – show on average weak-to-moderate agreement (Benoit Allen, Allen, Austin, Waldron, & Ollendick, 2015; Hollenstein & Lanteigne, 2014; •Lanteigne, Flynn, Eastabrook, & Hollenstein, 2014; Mauss & Robinson, 2009; Smith, Hubbard, & Laurenceau, 2011). This "desynchrony" limits inferences about one component based on the measurement of another. For instance, the person who yells louder is not necessarily angrier, and we hesitate to say that the crying mourner grieves more than the quiet one (Wu & Schulz, 2020). It is therefore possible that one response system may appear under-controlled while others are functioning within normal range. This has implications for measurement models and inferences in emotion research.

Is emotional dysregulation distinct from other self-control processes?. The capacity to modulate emotion develops in tandem with other aspects of self-regulation, especially attention and inhibitory motor control (Perry et al., 2018). ADHD is highly prevalent among youth with emotion dysregulation (Faraone et al., 2019; Shaw, Stringaris, Nigg, & Leibenluft, 2014), and some have suggested that ineffective control over emotional reactivity is as central to the condition as behavioral hyperactivity and inattention (Barkley, 2015; Faraone et al., 2019). Moreover, rigorously implemented pharmacotherapy for ADHD very often leads to remission of aggressive behavior and other difficulties associated with emotionally charged maladaptive behavior (Aman et al., 2014; Blader et al., 2021; Blader, Pliszka, Jensen, Schooler, & Kafantaris, 2010; Fernández de la Cruz et al., 2015). However, only a minority of those with ADHD exhibit emotion dysregulation symptoms, and not all emotionally overreactive individuals have ADHD (Karalunas et al., 2014). It is therefore important to consider that presentations of the emotion dysregulation phenotype may have heterogeneous etiologies. We might suspect that for youth with ADHD, maladaptive reactions in emotionally evocative situations might be another manifestation of generalized weak impulse control. For others, different processes influencing emotional reactivity may be more significant.

Temporal aspects of emotional experience. Time-dependent features of emotion, also referred to as "affective chronometry" or emotion dynamics (Davidson, 1998; Hollenstein, 2015; Lamm, Granic, Zelazo, & Lewis, 2011; Thompson, 1990; Trull, Lane, Koval, & Ebner-Priemer, 2015) are clinically important. For instance, a short latency to intense emotional reactions could outpace regulatory control. Other relevant temporal features include time to peak response intensity, overall duration of response, rate of change between emotional states, and recovery time. Few assessment devices (Carlson, Silver, & Klein, 2022; Stringaris, Goodman, et al., 2012) consider these time aspects.

In DMDD, behavioral outbursts punctuate a chronic, prevailing mood state of anger or irritability. Most youth with these kinds of outburst episodes, however, do not meet DMDD criteria because they are euthymic in between these episodes. Emotion research has sought to conceptualize transitions from one's typical baseline mood (Hollenstein, 2015; Sheeber et al., 2009). Measuring these features of emotional flux has so far gained little emphasis in child psychiatric literature. Their clinical relevance, though, is implied in time-dependent concepts like emotional impulsivity and lability (Factor, Reyes, & Rosen, 2014; Faraone et al., 2019).

Methods

Search strategy and initial identification

Figure 2 presents the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Page et al., 2021) flow chart for article selection.

We queried two bibliographic databases, MEDLINE (filtered to behavioral science journals) and PSYCInfo for publication years 1980 through 2022. The following Boolean expression was applied to search article titles, abstracts, and keywords with asterisks indicating truncation and quotes showing word adjacency:

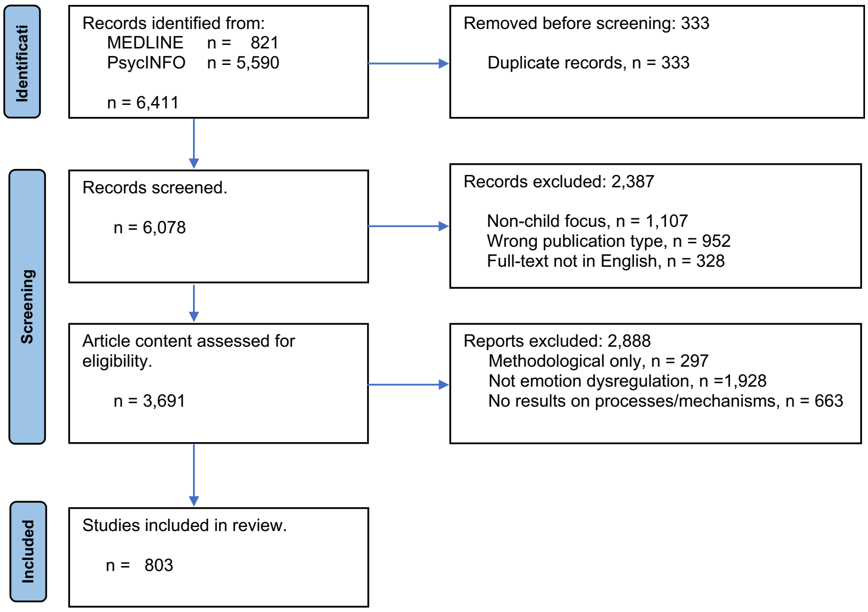

Figure 2 PRISMA flowchart for article selection

("emotion* *regula*" OR "emotion* reactivity" OR "irritability" OR "anger")

AND

("pathogenesis" OR "etiology" OR "mechanism" OR "process" OR "fmri" OR "reward sensitivity" OR "threat" OR "appraisal" OR "neur*al" OR "underl*" OR "*inhibit*" OR "setpoint")

The following limits were also applied: Age below 19, peer-reviewed, journal article, English language, and full-text available.

Records from this search were uploaded to the Rayyan online systematic review utility (Ouzzani, Hammady, Fedorowicz, & Elmagarmid, 2016; www.rayyan.ai) to facilitate further review of studies.

Screening and selection

Filtering at the identification stage eliminated duplicates. During the screening stage, case reports, other nonempirical publication types, articles not focused on children, and non-English reports were eliminated. A detailed review of article contents was conducted to determine if emotion dysregulation or a substantively related topic was examined and whether the results were relevant to pathogenesis or mechanism. Review papers and reports with a solely methodological focus (e.g., rating scale properties) were excluded.

Review and categorization

The 803 articles that met these criteria underwent review. To distinguish these items from other citations in this article used as background and explanatory material, they are prefixed with a bullet marker (•) in text and suffixed in the references list. From this literature, we identified several global aspects of emotional functioning that the processes examined in these studies reflected. These included (1) stimulus-related processes (further parsed into sensory sensitivity, salience, attention, appraisal, and valuation [notably threat and reward processing]), (2) behavioral control and response selection, (3) emotion generation and expression, (4) monitoring and corrective feedback processes (including open- and closed-loop regulation), and (5) experiential factors that influence the development and functioning of these processes. We organize the review sections accordingly, though we note where a process might be alternatively situated.

To evaluate the reliability of the categorization scheme, 200 (25%) of the included articles were independently assigned to categories by two authors (JB and AG) based on the *a priori* definitions laid out above. Concordance was high, with 13 (6.5%) assigned differently. For another 12 (6%), authors agreed on at least one category, but one author suggested an additional one. Final assignments for these 25 articles were done by review and consensus.

Limitations on the number of references prevent all publications meeting search criteria from appearing in the published bibliography. When there were many articles that converged on the same overall conclusions, we gave priority in citing to those that were more recent, used a quantitative measure of emotion dysregulation, and had larger sample sizes. The complete bibliography of the final search set for this review is available as Appendix S1, where the process assignment for each article is also shown.

Results: Summary of candidate processes in the emotion dysregulation phenotype

Figure 3 depicts an elaborated regulated system model that includes the processes identified in our review apportioned to the type of role they play. Our presentation of review findings is organized by these process categories.

Input processes

Sensory sensitivity. Individual differences in sensory sensitivity appear in early childhood. A low threshold to react to visual and auditory stimuli has been associated with a lower capacity for sustained attention and self-regulation over emotion (Calkins, Fox, & Marshall, 1996; •Ostlund et al., 2019). Preschoolers with "sensory over-responsivity symptoms" have a higher likelihood of significant

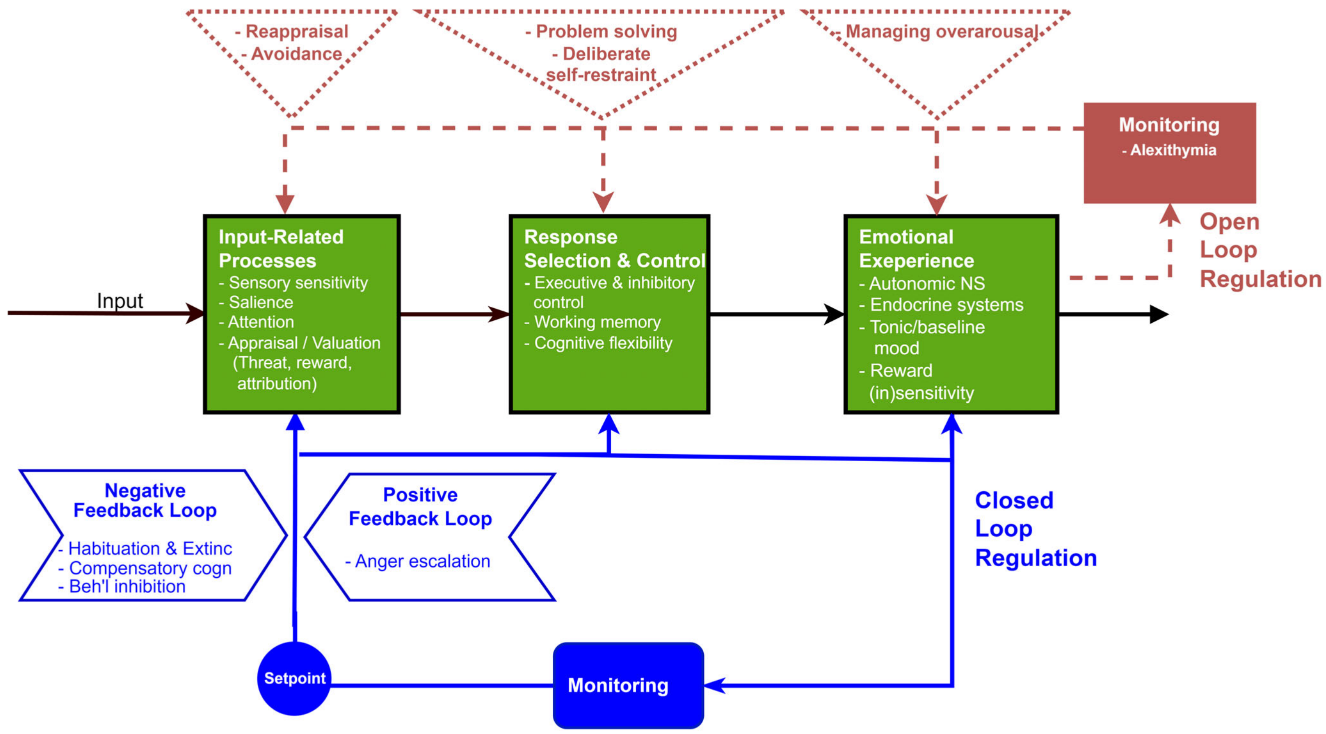

Figure 3 Proposed integrative framework for emotion-related processes involved in the emotion dysregulation phenotype among youth

irritability (•Carpenter et al., 2019) and other behavioral disturbances (•Ben-Sasson, Carter, & Briggs-Gowan, 2009). Children with high irritability also have a higher prevalence of sensory sensitivity compared with peers (•Benarous et al., 2020). •Karim and Perlman (2017) found that children high in irritability were more affected by stimulus luminance on brain activation measures.

Children on the autistic spectrum often experience as noxious certain stimuli that do not trouble others, especially in tactile and auditory modalities (Marco, Hinkley, Hill, & Nagarajan, 2011). Such stimulus sensitivities in ASD are associated with emotional dysregulation and irritability (•Ben-Sasson et al., 2009), but it is uncertain how much sensory anomalies contribute to emotional dysregulation since both may also be linked to overall autism severity. *Hypo*sensitivity and other aberrant reactions to stimuli are also common but do not seem as strongly associated with impairments in ASD (Ausderau et al., 2014).

Salience and attentional capture. Salience reflects the motivational significance given to stimuli (i.e., their capacity to signal potentially desirable or unfavorable environmental cues). The details of how salience perception and attention are temporally related have different viewpoints, but their conceptual and neurophysiological distinctiveness is generally accepted (Anderson, 2019; Parr & Friston, 2017). Salience is also distinguishable from valuation because perception of the impact of salience is separate from whether the stimulus is positive or negative (Kong & Zweifel, 2021).

The threshold for stimuli to signify salience that warrants engagement may vary between individuals. The anterior portion of the insular cortex “anchors” a salience network (Seeley, 2019; Uddin, 2015), where structural and functional anomalies have been reported among several phenotypes with emotion dysregulation (•Beauchaine, Sauder, Derbidge, & Uyeji, 2019; •Dougherty et al., 2018; •Hwang et al., 2016; •Jankowski et al., 2018; •Ross et al., 2021; •Tseng et al., 2016; •Vetter et al., 2018). Activations of medial prefrontal and anterior cingulate cortices during emotional challenge correlate with irritability (•Crum et al., 2021); these areas are thought to mediate determinations that stimuli are self-relevant. However, while neurofunctional data are suggestive, our search did not reveal studies addressing differences in salience perception directly in emotionally dysregulated youth. Methods that disentangle salience effects, attention processes, and stimulus valence have been developed (e.g., Bartra, McGuire, & Kable, 2013; Kim, Nanavaty, Ahmed, Mathur, & Anderson, 2021) and it seems worthwhile to adapt them for this clinical population.

Signals of potential harm are strong attractors of attention (•Lagattuta & Kramer, 2017; •LoBue, 2009). This effect may be greater in those prone to react forcefully to such stimuli (e.g., Alia-Klein et al., 2018). Research on the impact of harm signals on attention among youth indicates that those with emotional overreactivity show greater attentional capture by lower intensity harm-related stimuli.

Several studies report an association between emotional dysregulation and a stronger bias for attending to stimuli with negative content (•Cadesky, Mota, & Schachar, 2000; •Deveney et al., 2020; •Harrewijn et al., 2021; •Hommer et al., 2014; •Pavuluri, Passarotti, Harral, & Sweeney, 2009; •Salum et al., 2017). Aggressive behavior was associated with attentional bias for social threat-related stimuli only in the initial stages of stimulus processing (•Miller & Johnston, 2019). Experimentally induced frustration was also reported to elicit greater activation of neural circuits mediating attention among those with vs. without high parent-reported irritability (•Tseng et al., 2019).

It might be noted that similar relationships are reported in groups who show elevated symptoms other than irritability and emotional dysregulation, such as depression (•Petro, Tottenham, & Neta, 2021) and anxiety (Bar-Haim, Morag, & Glickman, 2011; but cf.; •Mullin et al., 2022; •Waters, Henry, Mogg, Bradley, & Pine, 2010).

Efforts to home in on the specific attentional processes associated with emotional dysregulation suggest less extensive initial stimulus processing (•Dennis & Hajcak, 2009; •Dennis, Malone, & Chen, 2009; •Deveney et al., 2020; •Kim et al., 2013). Infants with shorter periods of sustained attention were more likely to show impaired self-regulation in childhood, indicating that superficial processing may contribute to "false alarms" and overperception of personal salience (•Brandes-Aitken, Braren, Swingler, Voegtline, & Blair, 2019).

Appraisal and valuation. Threat processing: A tendency to overestimate an event's threats to one's safety, status, or prerogatives affects appraisal and valuation processes. These in turn influence emotion and can contribute to the heightened reactivity seen in emotion dysregulation (e.g., Blair, Veroude, & Buitelaar, 2018; Brotman, Kircanski, Stringaris, Pine, & Leibenluft, 2017). This tendency is found not only in those with emotion dysregulation but also in anxiety and depressive disorders (•Basile, Toplak, & Andrade, 2021; •Denham et al., 2002; •Erhart, Dmitrieva, Blair, & Kim, 2019; •Keil, Tuschen-Caffier, & Schmitz, 2022; •Köchel, Leutgeb, & Schienle, 2014; •Kohls et al., 2020; •Reeb-Sutherland et al., 2015; •Rich et al., 2008, 2011; •Sylvester, Petersen, Luby, & Barch, 2017; •Vidal-Ribas et al., 2018; •Waters et al., 2010; •Weiss, Rominger, Hofer, Fink, & Papousek, 2019; •Wiggins et al., 2016). Neurofunctional studies suggest aberrations in the processing of emotion-relevant stimuli, including those evocative of threat, among youth who are irritable, emotionally dysregulated, or display impulsive aggression (Aggensteiner et al., 2022; •Bertocci et al., 2019; •Brotman et al., 2010; •Crum et al., 2021; •Deveney et al., 2013; •Gaffrey, Barch, Luby, & Petersen, 2021; •Karim & Perlman, 2017; •Kircanski et al., 2018; •Kryza-Lacombe, Iturri, Monk, & Lee Wiggins, 2020; •Perlman et al., 2015; •Stoddard et al., 2017; •Thomas et al., 2012, 2014; •Tseng, Abend, Gold, & Brotman, 2021; •Tseng et al., 2016, 2019; •Wiggins et al., 2016). The most common paradigm is brief exposure to pictures of human faces that express fear, anger, happiness, etc. during fMRI scans. However, convergence on specific anomalies (specific brain regions, networks, hypo- vs. hyperactivation) has been elusive (Lee et al., 2022) and we summarize some of these diverging data.

Low "alarm thresholds" may reflect higher amygdalar engagement (•Petro et al., 2021). However, clinical sub-phenotypes, notably the presence or absence of anxiety along with irritability, may show different abnormalities in limbic system responsiveness (•Crum et al., 2021; •Kircanski et al., 2018; •Liuzzi et al., 2020; •Stoddard et al., 2017; •Thomas et al., 2012, 2014).

In adolescents with ADHD, fearful faces evoked greater amygdalar activation compared with controls (•Posner et al., 2011). However, among children with DMDD, greater irritability was associated with *diminished* amygdalar activation to moderately fearful faces while youth with bipolar disorder showed enhanced activation (•Wiggins et al., 2016). Other studies have also shown amygdala underresponsiveness in irritability (•Blair et al., 2020; •Brotman et al., 2010; •Deveney et al., 2013; •Sauder, Derbidge, & Beauchaine, 2016), reduced coactivation with other limbic structures (•Naim et al., 2022), mixed findings as a function of covariates (•Aggensteiner et al., 2022) or no differences from controls (•Tseng et al., 2016). While amygdalar hypoactivity may seem counterintuitive if one assumes that aversive stimuli trigger its activation, there is increasing recognition that the amygdala's basolateral nuclei have more complex roles in appetitive as well as aversive motivation and in mediating approach behavior (Berridge, 2019; Paré & Quirk, 2017).

Few studies have involved auditory stimuli, but in an event-related potential (ERP) paradigm those with conduct disorder, a group also high on emotion dysregulation, showed a response to voices evincing fear (compared with sadness) that was higher than controls (•Hung, Ahveninen, & Cheng, 2013).

The N170 is an ERP during the visual encoding of faces. Youth with ADHD showed an attenuated N170 response to fearful compared with neutral faces, while controls showed the more typical enhanced negativity to such stimuli (•Flegenheimer, Lugo-Candelas, Harvey, & McDermott, 2018). These findings suggest anomalies in valence encoding in a group at high risk for emotion dysregulation.

Biased assessments of threat may also influence how one perceives the emotional state of others. For example, overattributing anger to them increases the “expected value” of a forceful retaliatory response to neutralize that perceived threat. Numerous studies, but not all, implicate inaccurate labeling of others’ emotional expressions in the genesis of emotion dysregulation (•Basile et al., 2021; •Denham et al., 2002; •Erhart et al., 2019; •Köchel et al., 2014; •Kohls et al., 2020; •Rich et al., 2008, 2011; •Vidal-Ribas et al., 2018; •Weiss et al., 2019; •Wiggins et al., 2016). Misidentification by DMDD patients of happy faces as fearful correlates with future depression symptoms (•Rich et al., 2011). We elaborate below on the broader area of attributional biases, but the main point here is the relevance of this biase to threat perception.

Treatments that attempt to counteract a bias to overperceive threat in ambiguous situations have not yet, on their own, produced symptomatic change among ED youth. Further development that validates such approaches is important to pursue (•Haller et al., 2021).

Reward processing: One’s perception of the expected reward value that a given situation offers can influence behavior and mood (e.g., Belleau et al., 2021; Nielson et al., 2021; Sonuga-Barke & Fairchild, 2012; Watson, 1970). Mismatches between expectancies and perceived outcomes could influence negative emotional reactivity through several pathways.

First, a predisposition to perceive less value from seemingly positive events, or *reward insensitivity*, may contribute to emotion dysregulation. Reward insensitivity is found among emotionally dysregulated youth (•Bebko et al., 2014; Brotman, Kircanski, Stringaris, et al., 2017; •Perlman et al., 2015). It may dispose to chronic dissatisfaction, negative expectancies for daily events, and irritability. The neural basis for reward representation is thought to be a function of the ventral striatum, ventromedial PFC (vmPFC), and orbitofrontal cortex, among other regions. Related neurofunctional anomalies have been observed in irritability (•Ballard & Knutson, 2009; •Blair et al., 2020; •Deveney et al., 2013; •Dougherty et al., 2018; •Kryza-Lacombe et al., 2021; •Mao, Zuo, Ding, & Qiu, 2020; •Perlman et al., 2015; •Ross et al., 2021; •Sauder et al., 2016; •Tseng et al., 2019). Aberrant connectivity between the ventral striatum and other cortical areas characterizes irritability among preschoolers (•Dougherty et al., 2018).

Second, the absence of an expected rewarding event can prompt strong negative reactions. In a paradigm called *frustrative nonreward*, reinforcers that previously followed a learned response are withheld. In animals, this often elicits agitated and combative behavior, and in humans, it may elicit anger (•Ametti et al., 2022; Brotman, Kircanski, Stringaris, et al., 2017). Irritable preschoolers (•Grabell et al., 2022), older children (•Perlman et al., 2015), and adolescents (•Hodgdon et al., 2021) show neural and psychophysiological anomalies in these situations, notably underactivation of prefrontal and anterior cingulate cortical regions.

There is also likely to be a synergism between threat perception and nonreward (Brotman, Kircanski, Stringaris, et al., 2017) when one attributes nonreward, correctly or not, to the malice of another person rather than to chance, one’s own abilities, or other factors. In experimental paradigms that induce the perception of monetary loss because of another’s willful action, irritable youth show weaker coupling of the amygdala and vmPFC relative to controls (•White et al., 2016). As discussed below, erroneous attributions commonly occur among irritable individuals, so parsing the effects in children of frustrative nonreward and interpersonal antagonism is important.

Third, impulsive individuals, especially with disruptive behavioral disorders, are often unsatiated by the positive events that ordinary daily experience affords. They are impaired in learning the contingency effects of reward on behavior (Blair et al., 2018; de Zeeuw, Weusten, van Dijk, van Belle, & Durston, 2012; Plichta & Scheres, 2014; Tenenbaum et al., 2018; Tripp & Alsop, 1999; but cf. •Norman et al., 2018). One consequence may be limited tolerance for setbacks or reductions in what, for them, is already an unsatisfying level of positive stimulation. In youth with ADHD, functional connectivity between the ventral striatum and orbitofrontal cortex correlated negatively with emotional lability symptoms (•Posner et al., 2013); this effect was independent of functional connectivity abnormalities involving executive attention networks (•Posner et al., 2013). Similarly, network components more specific to reward processing showed less activation during reward anticipation as a function of ADHD symptoms (•Jia et al., 2020).

Fourth, situations that offer reward only after an extended wait are also strongly disfavored impulsive individuals, an effect called *delay aversion* (Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). Delay risks creating high frustration in the many situations where desired outcomes are deferred (•Blair et al., 2020). Relatedly, there is a normal tendency to prefer immediate acquisition of desirable things over getting them later, known as *temporal discounting* (Ballard & Knutson, 2009), because the longer wait interval discounts their reward value. Delayed receipt usually becomes more tolerable when the payoff for waiting is a larger reward. If larger enough, deferring may be the more advantageous strategy. However, some individuals are less likely to accept an enriched payoff for delayed receipt. They tend to be more impulsive (Costa Dias et al., 2013; Wilbertz et al., 2013; cf. Demurie, Roeyers, Wiersema, & Sonuga-Barke, 2013). This pattern, and perhaps a more general aversion to waiting (Coghill, Seth, & Matthews, 2014; Sørensen et al., 2017), could lead impulsive individuals to experience greater frustration when situations offer only the prospect of deferred reward.

Interpersonal attributional biases: Social information processing concerns how one perceives and interprets the behavior of other people and plans responses based on those determinations. Youth with impulsive aggressive behavior, which overlaps with the emotion dysregulation phenotype, have been shown to display a bias toward attributing hostile intentions to others in situations where age-mates are more likely to attribute behavior to accidental or other benign causes (•Dodge & Coie, 1987; Dodge & Crick, 1990). When asked to generate possible solutions to conflicts or frustrations involving peers, they are more likely than nonaggressive peers to mention retaliatory aggressive behaviors and to expect more favorable outcomes compared with more prosocial alternatives. A similar attribution bias is found in depressed children, although they do not have the same tendency to endorse aggressive, or, for that matter, even assertive behavior, as a suitable response (•Quiggle, Garber, Panak, & Dodge, 1992). This tendency is only modestly correlated with the threat-related attentional bias noted above, suggesting it is not entirely derivative of threat perception per se (•Miller & Johnston, 2019). Hostile intention bias effects on aggression appear to be stronger in emotionally engaging contexts, raising the question of whether rapid negative affective arousal influences cognition in addition to the supposed causal role of cognition on emotion and behavior (Verhoef, Alsem, Verhulp, & De Castro, 2019).

The attributions youth make for social rejection may also distinguish those prone to withdrawal from those likely to retaliate (•Zimmer-Gembeck, Nesdale, Webb, Khatibi, & Downey, 2016). Youth who blame themselves for rejection were more likely to withdraw, while those who blamed their peers were more prone to anger and aggression.

Attributing hostility to others serves to promote or disinhibit retaliatory behavior. Youth prone to retaliation show greater activation of threat-related circuitry and weaker functional connectivity between the vmPFC and amygdala, as well as deficient activation of inhibitory control (•Bubenzer-Busch et al., 2016; •Mathur et al., 2023; •White et al., 2016), and these effects correlated with overall ratings of aggressive behavior. These effects were strongest in those with low callous/unemotional (CU) traits, suggesting that the negative affective arousal that characterizes reactive aggression may have distinct determinants from planful and deliberate aggressive behavior. Hostile attributional bias, similarly, was reported only among aggressive boys with low CU traits (•Frick et al., 2003).

Behavioral control: Response selection and enactment

Individuals prone to anger outbursts often show deficits in several indices of self-control, notably response inhibition and performance of adaptive behavior. Weak inhibitory control strongly correlates with the emotion dysregulation phenotype, both cross-sectionally and longitudinally (Barkley, 2015; •Berzenski & Yates, 2021; •Cardinale et al., 2021; •Carlson & Wang, 2007; •Eisenberg et al., 2009; •Eyre et al., 2017; Faraone et al., 2019; •Galera et al., 2021; •Groves, Kofler, Wells, Day, & Chan, 2020; •Hudson & Jacques, 2014; •Hughes, White, Sharpen, & Dunn, 2000; •Karalunas, Gustafsson, Fair, Musser, & Nigg, 2019; •Kessel, Dougherty, et al., 2021; •Kochanska, Murray, & Harlan, 2000; •Liuzzi et al., 2020; •Miller, Hane, Degnan, Fox, & Chronis-Tuscano, 2019; •Seymour, Chronis-Tuscano, Iwamoto, Kurdziel, & MacPherson, 2014; •Shaw et al., 2014; •Walcott & Landau, 2004; •Whedon, Perry, Curtis, & Bell, 2021; •Wilson et al., 2021; •Yap et al., 2011).

Links between depressive disorders and general cognitive control have also led some to attribute emotional dysregulation in those conditions to inefficient overall self-control (e.g., •Loeffler et al., 2019). However, ADHD-associated gene profiles were more strongly linked with the emotion dysregulation phenotype than depression-associated ones (•Nigg et al., 2020; •Riglin et al., 2017).

High irritability is associated with greater activation in the superior frontal gyri while viewing emotionally negative videos, suggesting a higher degree of controller burden (•Karim & Perlman, 2017). Children with severe temper outbursts show greater disruption of intrinsic functional connectivity involving the anterior cingulate cortex (ACC), which is implicated in several higher order control functions, compared with both healthy controls and those with ADHD but no outbursts (•Roy et al., 2018). In a frustration paradigm, youth with emotion dysregulation show anomalous activation of regions implicated in evaluation and response selection (medial frontal gyri and ACC), while those who also had bipolar disorder showed more anomalous “bottom-up” activation involving the insular cortex and superior marginal gyrus (•Rich et al., 2011).

A high capacity for effortful control may partially offset the risk negative emotionality poses for behavioral disturbances (•Wilson et al., 2021). Nonetheless, irritability in the absence of ADHD is still associated with elevated mood disorder risk (•Eyre et al., 2019; •Stringaris, Zavos, Leibenluft, Maughan, & Eley, 2012; •Waldman, Rowe, Boylan, & Burke, 2021). Among irritable youth, those with lower cognitive flexibility and inhibitory control had distinct neural activation patterns during performance errors from those with higher scores of these functions (•Kryza-Lacombe, Palumbo, Wakschlag, Dougherty, & Wiggins, 2022).

In areas associated with cognitive control, gray matter volume is smaller and may thin in late adolescence to a greater degree among those with emotion dysregulation (•Bajaj et al., 2021; •Chaarani et al., 2020; •Dickstein et al., 2005; •Jirsaraie et al., 2019; •Seok et al., 2021). Among youth without psychiatric disorders, self-reported ability to diminish negative emotional arousal was associated with better differentiation between a neural network supporting self-control functions and one focused on limbic regions (•Guassi Moreira, McLaughlin, & Silvers, 2021; •Viering et al., 2021). Weaker distinctions between activations in established networks are generally associated with greater dysfunction (e.g., Tozzi et al., 2024).

ERP studies show a diminished P300 waveform while viewing faces displaying various emotions, among youth with reactive aggression (•Köchel et al., 2014; •Sun, Li, Niu, Zhang, & Chang, 2020). The P300 is associated with decision-making and response control.

Other cognitive processes relating to executive control show differences among those with emotion dysregulation compared with those without. Working memory may have an association with emotional dysregulation that is distinct from its correlation with inhibitory control (•Ametti et al., 2022; •Cauwenberge, Sonuga-Barke, Hoppenbrouwers, Leeuwen, & Wiersema, 2015; •Frick, Asherson, & Brocki, 2020; •Groves et al., 2020; •Jensen et al., 2018). Working memory involves simultaneous storage and manipulation of information and is important in maintaining focus on overarching goals while resisting momentary distractions and changing environmental contingencies.

Irritable youth show difficulty shifting behavior when previously rewarded responses result in nonreward, or "reversal learning" (e.g., Blair, 2012). Imaging studies implicate under-reactivity during these contingencies to differences in brain regions involved in inhibitory control (•Adleman et al., 2011). It may be one mechanism that promotes perseveration with disadvantageous response selection. Reversal learning problems might also reflect deficits in the timely revaluation of outcomes, potentially a type of feedback sensitivity impairment involved in emotion regulation (Braunstein et al., 2017) that we also consider below in the context of feedback processes.

High cognitive inflexibility and irritability are correlated among preschoolers as well as older youth (•Ametti et al., 2022; •Li, Grabell, Wakschlag, Huppert, & Perlman, 2017). In contrast, one study reported irritability was correlated only with ADHD symptoms but not with measures of executive function (•Colonna et al., 2022).

Figures 1 and 3 depict controllers directing outputs. However, weak attentional control in ADHD also correlates with neurofunctional anomalies in the *processing* of emotional information (•Hulvershorn et al., 2014; •Lugo-Candelas, Flegenheimer, Harvey, & McDermott, 2017; •Posner et al., 2011, 2013). Such effects suggest (a) dysfunction in feed-forward control in processing emotion-relevant stimuli, or (b) irritable youth with ADHD are affected by both attentional impairments that lead to insufficient processing of stimuli and poor response control.

Output generation and emotional expression

Abnormalities in the processes that generate outputs can cause undesirable results even if input processing, monitoring, and feedback system components are intact. Correspondingly, emotional responses may be affected by anomalies that lie chiefly in the physiological, cognitive, and behavioral processes that produce them. In practice, though, it can be difficult to distinguish abnormalities in emotion generation per se from other "upstream" processes like stimulus processing. Innovative experimental designs and longitudinal studies are important tools to disentangle the interdependence of these processes.

Autonomic nervous system function. Healthy people often show heart rate changes in which inter-beat intervals are longer during exhalation than during inhalation. A chief cause is respiratory-cycle-dependent shifts in parasympathetic nervous system (PNS) effects on the heart by the vagus nerve, which synapses on the sino-atrial node (Hall & Hall, 2021, p. 158). The phenomenon is therefore known as respiratory sinus arrhythmia (RSA). Low resting heart rate variability – attributed to smaller or absent RSA – has been reported in several conditions including depressive, anxiety, traumatic-stress, and disruptive behavior disorders (•Ametti et al., 2022; Beauchaine, 2015; •Beauchaine et al., 2013; •Bunford et al., 2017; •Byrd et al., 2022; •Kovacs et al., 2016; •Naim et al., 2021; •Sousa, Petrocchi, Gilbert, & Rijo, 2021). The inference is that the PNS has less modulatory "calming" influence than normal on emotion-related arousal, leading to excessive negative emotional reactivity in these conditions.

Efforts to link changes in RSA in response to experimentally induced emotional challenge, though, have yielded mixed findings (•Beauchaine, Bell, et al., 2019; •Deutz et al., 2019; •Morris et al., 2020; •Quiñones-Camacho & Davis, 2018; •Taskiran et al., 2018). Sex differences may influence these discrepancies (•Vidal-Ribas, Pickles, Tibu, Sharp, & Hill, 2017). Integration of RSA with other cardiorespiratory measures over longer time periods may further flesh out a more complete picture of PNS and SNS abnormalities in the emotion dysregulation phenotype (e.g., •Fiskum et al., 2017).

SNS reactivity may also affect emotion generation among children, particularly those who are more behaviorally active (•Stifter, Dollar, &

Cipriano, 2011). One consequence of SNS activation is increased sweat production. Increased moisture makes skin a better conductor of low-voltage electrical current. By passing such a low current via electrodes, the measurement of the skin conductance response (SCR) therefore serves as one gauge of emotional response. An elevated SCR may signal susceptibility to engagement of the "fight or flight" reactions that the SNS mediates. However, data are mixed. Lower SCR reactivity is often associated with behavioral disturbances in ADHD (•Bubier & Drabick, 2008; •Morris et al., 2020) and ASD (•Fenning et al., 2019). More generally, low sympathetic arousal is reported to predict antisocial conduct. This is attributed by some to the possibility that lower SNS reactivity makes one insensitive to the negative social consequences that ordinarily discourage such behavior (Ortiz & Raine, 2004). This is partially consistent with findings that high SCR predicts reactive aggression while it has no relationship – including no indication of dampened reactivity – with proactive aggression (•Hubbard et al., 2002). Allelic variation in the *mu* opioid receptor OPRM1 interacts with parenting style in influencing SCR reactivity (•Partington et al., 2018), suggesting genetic factors that could moderate susceptibility to environmental exposures on autonomic reactivity. Developing cognitive control may also affect SCR. In preschoolers, both reward and frustrative nonreward produce an SCR spike; recovery from this spike is usually associated with lateral PFC activation, but among highly irritable children, this association is absent (•Grabell et al., 2022).

The startle response occurs following sudden intense and aversive stimuli, such as loud sounds. In humans, electromyography captures the eyeblink reflex that results. Eyeblinks elicited by loud sounds are often more intense when accompanied by an unpleasant visual stimulus and attenuated in the presence of a pleasant one, an effect called "affective modulation." Anxious children have been reported to show larger startle responses than others during affective modulation (•Waters, Neumann, Henry, Craske, & Ornitz, 2008) but not children with high irritability (•Rich et al., 2005) and the effect itself has been inconsistently reported in normal youth (•Latham et al., 2017).

Inhalation of air enriched with carbon dioxide provokes heightened anxiety and panic-like phenomena more readily among individuals with a range of anxiety disorders and their offspring (•Roberson-Nay et al., 2010; •Vickers, Jafarpour, Mofidi, Rafat, & Woznica, 2012). In a non-psychiatric sample of youth, higher CO_2 exposure-related distress was associated with higher irritability, even more strongly than with higher depression and fearfulness (•Rappaport et al., 2017).

Endocrine systems. The hypothalamus receives neural inputs from many brain areas relevant to emotion, pain, and motivation, including limbic regions and brain stem. In response, the hypothalamus produces peptides that control the anterior pituitary's release of adrenocorticotropic hormone (ACTH). That in turn stimulates the adrenal gland release of glucocorticosteroids, notably cortisol and the adrenal androgens. Thus, cortisol is a marker of hypothalamically recognized stress whose dysregulation may produce susceptibility to negative emotional reactions.

ADHD and other externalizing behavior symptoms are more strongly associated with less diurnal variation and smaller stress-induced changes in cortisol production, while the opposite is found among youngsters with predominantly internalizing symptoms (•Kessel, Frost, et al., 2021; •Locke, Davidson, Kalin, & Goldsmith, 2009; •Miller et al., 2017; •Northover, Thapar, Langley, Fairchild, & van Goozen, 2016; •Taylor et al., 2020; •van Goozen et al., 1998). Proposed reasons for this pattern include diminished fearlessness in ADHD, poor early processing of stress signals, and circadian abnormalities that affect typical trends in cortisol levels. Some data, though, suggest that these associations may differ across cultural populations (•Grabell et al., 2015). Elevated levels of cortisol suppress its own production via feedback to the pituitary and hypothalamus that inhibits corticotropin-releasing hormone; sustained high cortisol may reflect abnormalities in this negative feedback mechanism (Gjerstad, Lightman, & Spiga, 2018).

Deficient neural responsivity to reward. Reward insensitivity was discussed above in the context of appraisal and expected value, but the evidence for diminished neural reactivity for positive events also suggests dysfunction in the generation of positive emotion. Diminished neural reactivity and connectivity related to the anticipation and receipt of reward have been reported in groups with high negative emotional reactivity (•Belden et al., 2016; •Blair et al., 2020; •Dougherty et al., 2018; •Fussner, Luebbe, & Bell, 2015; •Kryza-Lacombe et al., 2021; •Sauder et al., 2016).

Tonic or baseline mood; production of affect. Only 20%–40% of irritable youth have abnormalities of mood that endure most of the day (Blader et al., 2016; Copeland et al., 2015; •Eyre et al., 2017; •Roy et al., 2013; Vidal-Ribas & Stringaris, 2021). Chronic negative affect that persists through most of the day might itself represent an abnormality of emotional function against which regulatory processes must strain. More than just a roundabout way of saying "irritable people are easily irritated," baseline states and movements in and out of them have become a focus of work in psychopathology (e.g., Granic, 2005; Hollenstein, 2013; Rolls, 2021; Scheffer et al., 2024). Ongoing negative mood may constitute a maladaptive “basin” attractor state – evoking how a small ball placed in a basin will be attracted by gravity toward the bottom (Rolls, 2021; Scheffer et al., 2024). In this context, predominantly negative mood is an enduring disposition (see also Scherer, 2021) that hinders flexible adaptation to challenging circumstances. That is, the system requires more energy to generate a different (i.e., more hedonic, less affectively volatile) configuration. Accordingly, children with consistently high irritability from preschool through ages 7–12 had high risk for depression, independent of maternal depression and adversity (e.g., •Pagliaccio, Pine, Barch, Luby, & Leibenluft, 2018).

Another aspect of chronic mood disturbance is the tendency toward rumination that colors cognitive processes to maintain mood abnormalities (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Rumination that is sadness-themed vs. anger-themed may have different correlates. Sadness is resistant to updating based on new information while, in anger, attention-shifting and inhibitory control are more impaired (•Hamilton et al., 2017; •Harmon, Kistner, & Kofler, 2020; •Leigh, Lee, Brown, Pisano, & Stringaris, 2020). Rumination was associated with greater negative emotional reactivity during laboratory mood induction, while depression alone led to emotional blunting (•Somers, Borelli, & Hilt, 2020). Efforts to put more positive appraisals on situations are also less effective among depressed adolescents (•Stephanou, Davey, Kerestes, Whittle, & Harrison, 2017) and have less impact on amygdalar and vmPFC activity compared with controls. Rumination also hampers the effectiveness of reappraisal strategies in children with histories of depression (•Murphy, Barch, Pagliaccio, Luby, & Belden, 2016). Higher rates of successive periods with negative emotions, or “affective inertia,” was more likely among adolescents with a short allele of the serotonin transporter gene, even after controlling for depression severity (•van Roekel, Verhagen, Engels, & Kuppens, 2018).

Monitoring and corrective feedback processes

Monitoring processes. Problems identifying when emotional states need modulation could lead to failures to initiate regulatory processes. Alexithymia refers to difficulties in the identification, description, and recognition of emotion as manageable internal states. It is common in depression and anxiety disorders, ASD, adults with ADHD, as well as among adolescents who self-harm with or without suicidal intent (•Costa, Steffgen, & Vögele, 2019; •Demers et al., 2019; •Donges & Suslow, 2017) and it is associated with emotion dysregulation in youth (•Factor, Rosen, & Reyes, 2016; •Sfärlea, Dehning, Keller, & Schulte-Körne, 2019; •Venta, Hart, & Sharp, 2013). While one study found no direct association between adolescents’ emotional awareness and depression, emotional awareness was correlated with adaptive emotion regulation skills (•Van Beveren et al., 2019).

Errors on laboratory tasks may produce an ERP about 50 ms afterward that originates in the ACC, known as the error-related negativity (ERN). The ERN has been interpreted as a neural indicator of self and situational monitoring. Smaller amplitude ERNs at age 6 correlated with higher externalizing behavioral symptoms at age 9, while high-amplitude ERNs were associated with later internalizing symptoms (•Filippi et al., 2020; •Kessel et al., 2016) suggesting that both inadequate and excessive monitoring can be disadvantageous. Among children with anxiety disorders, ERN was more strongly associated with a similar ERP on *correct* trials (•Tan et al., 2022).

Closed-loop regulation. Closed negative feedback loops are central to most definitions of regulatory processes. Some studies report automatic negative feedback to low-level emotional arousal in nonclinical populations (Koole, Webb, & Sheeran, 2015). Similarly, university students exposed to experimental induction of social exclusion responded with a heightened, compensatory bias toward pleasurable memories and other cognitions, interpreted as automatic, rather than effortful, emotion regulation (DeWall et al., 2011).

However, strong negative stimuli may activate a *positive* feedback loop that intensifies, at least in the short term, anger and depression, producing a cascade that is harder for other processes to curtail (•Alia-Klein et al., 2020; Petro et al., 2021; Petro, Tong, Henley, & Neta, 2018). Adolescents low in trait anxiety were more likely to show negative feedback limiting arousal during a stressful task, while those higher in trait anxiety showed an escalatory, positive feedback one (•Yang, Ram, Lougheed, Molenaar, & Hollenstein, 2019). Following frustration, highly irritable youth were more likely to show enhanced, sustained activation in the dorsolateral prefrontal cortex, reflecting an attentional bias that is self-perpetuating rather than self-limiting among dysregulated individuals (•Tseng et al., 2019).

Habituation is the process through which repeated or sustained exposure to a stimulus produces a smaller response than it did initially. Extinction is the process by which a response associated with a stimulus or an outcome diminishes when the stimulus or outcome is withheld. These involuntary, automatic ways of dampening affective responses that are no longer useful may represent a form of closed-loop regulation. In experimental paradigms, diminished habituation of amygdalar reactivity was reported in nonclinical adolescents who reported higher trait anxiety (•Hare et al., 2008). More effective habituation of amygdalar reactivity in young children was related to the integrity of a white matter tract linking amygdala to frontal regions, the uncinate fasciculus (•Hein et al., 2018), consistent with that tract's possible role in vulnerability for mood disorders (•Weathers et al., 2018).

Slower habituation of psychophysiological reactions to acoustic elicitation of the startle reflex has been related to both fearfulness and aggressiveness, but habituation findings among anxious children have been less robust (•Blanch, Balada, & Aluja, 2014; Reeb-Sutherland et al., 2009; •Waters et al., 2008). Preadolescents exposed to trauma, though, showed equivalent decreases, compared with non-exposed children, on measures of extinction in a conditioning/reversal paradigm; however, simulated approach behavior retained greater avoidance (Marusak, Martin, Etkin, & Thomason, 2015). Along with other findings that specific response components in children may habituate at different rates (•Chen, Aksan, Anderson, Grafft, & Chapleau, 2014), these results highlight the need for multi-domain measurement. Overall, the fundamental idea that one can become less susceptible to anger-provoking stimuli with graded exposure is potentially significant, and treatments have been piloted on this concept (Linke et al., 2020; Naim et al., 2024).

Open-loop regulation. In contrast to the automatic measures that typify a closed-loop system, open-loop regulation involves the deliberate employment of steps to alter emotional states. Recent research has addressed several aspects of their development in children. By early elementary school, most children grasp that their experience of upsets can be changed internally, independent of the events that elicit them (•Crowell, 2021). This capability develops through caregiver guidance and cognitive control (•Camacho, Williams, Ding, & Perlman, 2021; •Grabell et al., 2019; •Kopp, 1989; •Perry et al., 2018).

Management of antecedents and stimulus inputs: Gaze aversion, exiting a situation, or diverting one's attention are rudimentary "input-focused" ways of limiting exposure to stimuli that provoke undesirable affect. In contrast, the cognitive process of *reappraisal* occurs when one tries to adopt a perspective on an emotion-eliciting situation that changes its meaning, attributions, implications, or significance. Reappraisal and related cognitive processes are central to influential models of emotion regulation (Gross, 2015) and are at the core of cognitive therapies for mood and anxiety disturbances (Storch, Abramowitz, & McKay, 2022).

The ability to reappraise in emotion-evocative situations is present in young children as well as other youth (•Gullone, Hughes, King, & Tonge, 2010; •Nook, Vidal Bustamante, Cho, & Somerville, 2020; •Padgaonkar, Phuong Uy, DePasque, Galván, & Peris, 2021). Preschoolers showed the diminished late positive potential ERP associated with reappraisal after they were exposed to negative pictures accompanied by verbal reframing (compared with a neutral narrative) intended to blunt their impact (•Hua, Han, & Zhou, 2015). Another study with older youth found the LPP to decrease as a function of age and low anxiety when negative stimuli were paired with benign descriptions of them (•DeCicco, O'Toole, & Dennis, 2014). Older age was associated with more successful use of reappraisal of emotional stimuli when instructed to do so (•Silvers et al., 2012; •Wessing et al., 2015). The involvement of ventrolateral prefrontal cortex in modulating amygdalar activation seems to increase with age (•Silvers et al., 2017). Linguistic analysis of overt reappraisals to negative pictures among children, adolescents, and young adults also showed that older individuals more often reinterpret *context* to make depicted events less troubling while younger people more often alter imagined *consequences* to make them more benign (•Nook et al., 2020).

Temporal distancing, in which one cognitively frames upsetting events as more remote, is a reappraisal strategy that can blunt their salience and impact. Adolescents with high reactive aggression showed less reduction of SCR when instructed to use temporal distancing than nonaggressive peers (•Ahmed, Somerville, & Sebastian, 2018). In a cohort of adolescents with non-suicidal self-injury, increasing use of reappraisal over several years of follow-up was associated with decreased severity of injuries, though not frequency of self-harm (•Voon, Hasking, & Martin, 2014).

Cognitive reappraisal may be most effective in down-regulating negative emotion when undertaken early and for less intense emotional arousal (Sheppes, Brady, & Samson, 2014). Depressed adolescents more effectively employed reappraisal with negative pictures when their attention was drawn to a non-emotional element of the display than when looking directly at more emotional sections (•Greimel, Piechaczek, Schulte-Rüther, Feldmann, & Schulte-Körne, 2020). Adolescents' use of cognitive regulatory strategies measured via ecological momentary assessment showed that strategy use varied with the intensity of negative emotion (•Lennarz, Hollenstein, Lichtwarck-Aschoff, Kuntsche, & Granic, 2019). Different emotions engage different strategies, and more intense arousal results in rumination, a frequently counterproductive approach to emotion management (•De France & Hollenstein, 2022). Regulatory strategies in the context of social stimuli were overall less effective than with nonsocial stimuli, underscoring that specific elicitors of emotion may interact with regulatory strategies in determining outcome (•Silvers et al., 2012).

Some investigators have localized the neural substrate of reappraisal to frontoparietal circuitry (•Wessing et al., 2015). Reappraisal tasks showed activation of the vmPFC, an effect that was larger among depressed participants than in controls;

however, reappraisal was less effective in curbing amygdala activation (•Stephanou et al., 2017).

Depressed adolescents performing a reappraisal task showed less connectivity than controls between (a) dorsolateral prefrontal cortex and anterior insula and (b) between dorsomedial PFC and anterior insula (•LeWinn et al., 2018). These results suggested diminished efficacy of top-down cognitive control on brain circuits processing salience and proprioception (•LeWinn et al., 2018). Following instructions to reappraise sad photographs, both depressed and nondepressed children showed heightened activation of the inferior frontal gyrus, as well as dampened amygdalar reactivity (•Belden, Luby, Pagliaccio, & Barch, 2014; •Belden, Pagliaccio, Murphy, Luby, & Barch, 2015). Research showing similar activation patterns in children asked to employ reappraisal strategies to attenuate disgust elicited by pictures (•Pitskel, Bolling, Kaiser, Crowley, & Pelphrey, 2011) suggests that there may be some commonalities in mechanisms across emotions, at least negative ones. The time course for increased prefrontal cortex activation and diminished amygdalar activation relates to successful reappraisal in children, drawing attention to temporal features of processing (•Pierce, Haque, & Neta, 2022).

Modulation of responses: In general, literature on emotion regulation emphasizes that efforts to *suppress* emotion-activated responses produce less satisfactory outcomes than other strategies to alter one's emotional state, such as the cognitive approaches of reappraisal, problem-solving, and perspective-taking (Brans, Koval, Verduyn, Lim, & Kuppens, 2013; •De France & Hollenstein, 2022; Gross & Jazaieri, 2014; Webb, Miles, & Sheeran, 2012). However, selective suppression of emotional *expression* can be advantageous, even if trying to suppress emotional *experience* is not (Calkins, Dollar, & Wideman, 2019; •Riley, Sullivan, Hinton, & Kliewer, 2019; Webb et al., 2012). This distinction is important in the context of children's emotion dysregulation, where dysfunction and adverse impact often stem from harmful or distressing behaviors.

Because deliberate emotion regulation involves explicit skills, it is important to consider whether youth with ED lack them. Skills emphasized in treatment are in the broad domains of attention management, somatic arousal, problem-solving, and prosocial alternatives to conflict escalation (e.g., Sukhodolsky & Scahill, 2012). A prerequisite to learning and using such behaviors may be the capacity for verbal mediation of problem-solving, or "private speech," which is linked to several aspects of self-regulation (Winsler, 2009). The association between private speech and inhibitory control is highest among young children high in anger reactivity, which suggests that verbal mediation of conflict plays a larger role in self-control when there is greater bottom-up pressure to do so (•Whedon et al., 2021).

However, even when youth know these skills, and can demonstrate them in role-play or other contrived practice situations, they often are unable to *perform* them reliably in emotionally evocative situations. This overall pattern, where skills are acquired and demonstrated under certain conditions but not performed effectively when needed, is a common finding among impulsive individuals (Aduen et al., 2018; de Boo & Prins, 2007). Novel, online delivery of training in emotion regulation strategies was associated with reduced non-suicidal self-injury among adolescents (•Bjureberg et al., 2023), and joining such approaches with prompting for use in vivo may be even more impactful.

Experiential factors

A major focus in emotion regulation research concerns the impact of early experiences. The number of studies on these factors warrants consideration of how they may influence emotion regulatory processes. (To avoid increasing its complexity, we do not include these factors in Figure 3, but this choice does not signal that they are less important.)

Maltreatment, trauma, psychosocial adversity, and exposure to violence. High emotional reactivity is among the many adverse long-term risks of childhood maltreatment (•Maughan & Cicchetti, 2002; •Robinson et al., 2009; •Shackman & Pollak, 2014; •Shields & Cicchetti, 1998). Alterations in the processing of threat-related stimuli and responses to them are well-documented (•Hart et al., 2018; •Marusak et al., 2015; •McCrory, Gerin, & Viding, 2017; •McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015; •Peverill, Sheridan, Busso, & McLaughlin, 2019; •Pollak, Klorman, Thatcher, & Cicchetti, 2001; •Shackman, Shackman, & Pollak, 2007). Maltreatment by others or chronic exposure to neighborhood violence may bias vigilance, attention, and threat-response toward overperceiving negative stimuli in situations that others perceive as more benign (•Maheu et al., 2010; •McCoy, Roy, & Raver, 2016; •Pagliaccio et al., 2015; •Pine et al., 2005; •Pollak, Messner, Kistler, & Cohn, 2009; •Shackman et al., 2007). Similar patterns of neural reactivity to threat stimuli have been reported among children in poverty who had not also been exposed to violence (•White et al., 2019) and among adolescents with high cumulative stressful life events (•Yuan et al., 2022).

The interpersonal and environmental unpredictability that often accompanies maltreatment may also undermine the formation of learned contingencies associated with reward. Maltreated youth, compared with peers, perceive predictable rewards as more random (•Hanson et al., 2017) and their decision-making patterns reflect difficulties adapting to changes in the outcome values of choices (•Weller & Fisher, 2013). They seem primed to show stronger irritability-related neural activation when frustrated (•Hodgdon et al., 2021), which is also consistent with high uncertainty about when gratification will be forthcoming. The adverse impacts that specific forms of ecological unpredictability have on children were recently reviewed by Davis and Glynn (2024).

Abuse and neglect experiences in childhood may affect distinct emotion regulation processes. Young adults who reported abuse as children showed amygdalar hyperreactivity in response to threat-relevant stimuli (Puetz et al., 2020) and larger ERP wave forms relating to emotional face processing (Fang, Wang, Liu, & Gong, 2019). However, those reporting neglect but not abuse demonstrated alterations in a more distributed fronto-parietal network that implicates valuation, response selection, self-monitoring, and behavioral restraint functions (Puetz et al., 2020). Research with adolescents suggests more specific associations between violence exposure and emotion dysregulation while deprivation correlates with lower cognitive control (•Lambert, King, Monahan, & McLaughlin, 2017).

Harassment and aggression from peers are associated with adverse emotional outcomes, including measures of emotion dysregulation. Victimization by peers was associated with stronger functional connectivity between the amygdala and right ventrolateral prefrontal cortex (•Rudolph et al., 2021). Inducing the belief that peers had rejected them yielded increased sensitivity to losses and diminished distress tolerance (•King, McLaughlin, Silk, & Monahan, 2018).

Some studies indicate that the effects of adverse experiences like maltreatment and exposure to family conflict are higher among those with low effortful behavioral and cognitive control, or, put another way, high effortful control can be protective (•Rodman, Jenness, Weissman, Pine, & McLaughlin, 2019; •Thompson, Davies, Hentges, Sturge-Apple, & Parry, 2020).

Other interpersonal influences. Emotional expression has interpersonal signaling functions. How others respond to these signals shapes one's emotion-related behavior, a process called "emotion socialization" (Eisenberg, 2020). Parental responses to child upsets are the most well-studied aspect of emotion socialization. Observational coding and self-report methods characterize them broadly as supportive (encouraging expression, validating, promoting problem-solving, relieving distress) and nonsupportive (dismissive, punitive, distressful; Fabes, Poulin, Eisenberg, & Madden-Derdich, 2002). The effects, often bidirectional, between these types of interactions and youth behavioral functioning have been examined extensively in children (e.g., •Berona et al., 2023; •Blair et al., 2014; •Calkins, Smith, Gill, & Johnson, 1998; •Chang, Schwartz, Dodge, & McBride-Chang, 2003; •Feng et al., 2008; •Halligan et al., 2013; •Perry, Dollar, Calkins, Keane, & Shanahan, 2020; •Shaffer, Suveg, Thomassin, & Bradbury, 2012; •Shewark et al., 2021; •Whalen et al., 2009) and adolescents (•Berla et al., 2022; •Herd, Brieant, King-Casas, & Kim-Spoon, 2022; •LoBraico, Brinberg, Ram, & Fosco, 2020; •Skripkauskaite et al., 2015; •Stocker, Richmond, Rhoades, & Kiang, 2007). Genetic factors may affect susceptibility to these influences (•Noroña et al., 2018).

Interpersonal influences on emotion processes may be observed only in specific dyadic interactions and are not necessarily a stable trait (•Crowell et al., 2014; •Ratliff et al., 2021). Innovative simultaneous neurofunctional assessment of parents and children while they are interacting suggests potential pathways that underlie cooperative behavior (•Cosgrove et al., 2022; •Reindl, Gerloff, Scharke, & Konrad, 2018).

Parents' own emotional dysregulation suggests a potential genetic and environmental basis for offsprings' dysregulation (•Han & Shaffer, 2014; •Mazursky-Horowitz et al., 2015; •Milojevich, Machlin, & Sheridan, 2020; •Shaffer & Obradović, 2017). In particular, harsh authoritarian parental behavior accounts for a large portion of intergenerational transmission (•Shaw & Starr, 2019) and children's social relations (•Maughan, Cicchetti, Toth, & Rogosch, 2007). Parents' biases in processing emotional information are echoed in their children's appraisal biases (•Bickett, Milich, & Brown, 1996; •Root & Jenkins, 2005). Parent's own threatening behavior was associated with a reduced capacity in their offspring for goal-directed coping under experimentally induced stress (•Trent et al., 2019). Susceptibility to negative arousal by children's challenging behavior – or "emotional flooding" – may promote affective overreactivity in parenting situations (•Mence et al., 2014; •Oddo et al., 2022).

Conclusions

Summary

We used a controlled, regulated systems framework to categorize how factors identified in the genesis of the childhood emotion dysregulation phenotype may exert their effects. We were motivated in part by a desire to determine which of them may reflect a regulatory mechanism, that is, one that uses feedback to moderate emotional expression. The framework does not necessarily portray a validated neurobiological or psychological model. Rather, it just makes explicit what the concept of emotion dysregulation implies when laid out in a conventional systems format.

Overall, there is evidence for dysfunction in the processing of stimuli, notably sensory sensitivities, aberrant salience, and attentional biases, as well as appraisal and valuation processes that elaborate on their threat and outcome potentials. A large literature supports the substantial impact of "controller" functions over response selection, execution, and inhibitory control in emotion contexts. Abnormalities in the generation of the emotional response itself, potentially distinct from antecedents and control factors, have been implicated, including autonomic nervous system, endocrine, and reward sensitivity functions.

In the context of emotion, there is less understood about the types of closed-loop, automatic, negative feedback processes that are usually the core of regulatory control mechanisms. The strongest candidate is perhaps inhibitory control that ordinarily may be activated to restrain negative emotional arousal from leading to problematic behavior, but that may be ineffective for those with impulse control deficits.

We found limited research showing automatic compensatory cognitive activity to modulate emotion in nonclinical, older adolescent groups. There is, however, evidence that some emotions, notably anger, demonstrate an initial escalatory *positive* feedback loop, whose temporal relationships with inhibition and automatic cognitive effects warrant further study. Further work on deficient habituation and extinction processes would be fruitful, especially given that they are key to several psychological treatments for problematic emotional arousal.

Open-loop, deliberate efforts to modulate emotional experience, such as reappraisal, have been studied extensively in emotion research. Acquisition of the underlying skills and the ability to deploy them are important developmental milestones, and their trajectory is an active area of investigation. Some evidence shows that deliberate cognitive strategies are more effective when used early in the response chain and for less intense affect. The childhood emotion dysregulation clinical phenotype involves rapid intensification of negative arousal and outward behavior, often among youngsters with more generalized limitations in impulse control. This combination hinders the swift efficacy of contemplative strategies like reappraisal.

Interpersonal and environmental influences on emotion-related experience and behavior have been examined extensively. The mechanisms by which these adverse exposures affect specific emotion processes remain an important area for further research.

Limitations

Because of this review's concern with research on processes that might account for emotion dysregulation, our search terms sought to target studies on mechanisms. We excluded studies in which dysregulation was an independent variable or predictor of something else with no data bearing on underlying processes. It is therefore possible that our search strategy misspecified the full range of potential factors that have a role in producing emotion dysregulation, and that excluded studies reflect on mechanisms in ways we failed to recognize.

Our organizing framework using the basic concepts of a controlled, regulated system is intended to stimulate consideration of how disparate findings bearing on emotional reactivity might be integrated. We recognize this may introduce at least three sources of error. First, the overall idea that emotion-related processes can be parceled in this manner may be wrong, and that some other scheme might be more appropriate. Second, even if it is broadly correct, our definitions and distinctions among them may not stand up to further research. Third, even if our framework is fundamentally sound, our assignments of studies to specific processes may include misspecifications. Mindful of these risks, we suggest they are offset in part by the desirability of encouraging an integrative approach to an area currently dominated by individual studies that are siloed in the aspect of emotion they consider and in the investigational methods they employ.

Implications and further directions

Modeling of emotion processes. The framework we described may help to organize research on emotion and its disturbances by helping (a) to specify which component(s) of emotion generation and expression a study addresses, (b) to encourage research into the effects processes have on each other, and (c) to keep the "big picture" in mind. A clinically useful account benefits when investigators are mindful that emotional experience entails multiple processes that warrant assessment. As we have seen, most studies examine the association of one or sometimes two processes, seeking to determine individual differences as a function of behavioral indicators of emotion dysregulation. One result appears to be a patchwork of findings that bear on a multitude of factors without an integrative account. As it stands, variability in methods, outcomes, and populations studied makes it almost impossible to quantify and compare their influences on the emotion dysregulation phenotype. Referring back to some of the conceptual issues from the introduction, we can take stock of how current research has addresses them and what important areas need further elaboration.

First, we noted that not all disturbances of a controlled system reflect impaired regulation per se. The research we reviewed has in fact focused largely on antecedents and their perception, response selection control, and emotion generation factors (such as physiology). While emotion regulation is often defined by processes that may moderate an emotional state once it emerges, such processes remain weakly defined for the childhood emotion dysregulation phenotype. It would be worthwhile to look at sources of disturbance or dysregulation *within* these processes to learn how they affect emotion generation and expression. Interventional studies can be useful. They can be crafted to target a specific process with experimental controls, complementing the large number of observational studies using correlational, case–control approaches.

Studies on emotional dysregulation have seldom addressed the desynchrony of the main response systems of physiology, behavior, and subjective experience. The large number of neurofunctional studies have been illuminating, but their downstream significance for these "effector" systems is unclear. It is uncertain, for instance, that amygdalar activation to threat necessarily promotes autonomic reactivity, such as startle reflexes, cognitive appraisals, or attack vs. avoidance behaviors. Greater use of paradigms that elicit responses in effector systems (e.g., White et al., 2016) as well as their neural substrates can contribute to filling this gap.

The large literature on family, experiential, and other social factors on child behavior has not been well integrated into mechanistic research on the emotion dysregulation phenotype. Incorporating these factors can offer several advantages. First, it is important to understand how these influences might (or might not) influence the processes already studied. For instance, there are indications that threat sensitivity is upregulated among those with traumatic exposures, but the extent that this finding mediates the relationship between exposure and emotionally dysregulated behavior is uncertain. Second, regardless how problematic behavior originates, we have seen that patterns of interpersonal behavior have a role in sustaining maladaptive behavior related to emotion dysregulation. Third, the corrosive impact of chronic conflict in families and other relationships that high irritability often promotes has a public health impact well beyond the child him or herself.

Disentangling processes. While mapping the emotional dysregulation concept onto this framework may have heuristic value, we recognize that the independence and separability of the components we covered remain uncertain. Even if they are, they are likely to interact in a bidirectional manner. It can be challenging to determine if, for example, the mechanisms producing weak response inhibition may, at least in some individuals, also cause inaccurate predictions of threat or reward. Similarly, lower parasympathetic tone may have a sensitizing effect to stimuli ordinarily perceived as only mildly noxious so that behavioral responses to them become excessive.

Established interventions can be used fruitfully, by examining more completely the changes in processes that accompany treatment effects. For instance, among dysregulated children with ADHD, stimulant medication often produces remission of behavioral symptoms such as aggression, irritability, and outbursts. Improvements in ADHD symptoms are also observed, but it is important to determine whether other anomalies such as those affecting reward and threat processing also normalize. With new technologies, innovative experimental designs, and sophisticated data analytic techniques disentangling of this sort will likely become feasible. Our hope is that the framework we outlined here might help to highlight the interactions between processes on which to focus.

Heterogeneity. The current emphasis on the transdiagnostic nature of many symptoms, such as emotion dysregulation stems from the recognitions that (a) psychiatric syndromes have high heterogeneity within diagnoses and (b) some disturbances appear in many supposedly distinct disorders. This review raises the possibility that the emotion dysregulation phenotype itself may not be unitary. It may encompass numerous sub-phenotypes with different underlying processes (e.g., primary disturbances of stimulus processing, response control, emotion generation, etc.) and trajectories (•Riglin et al., 2019). Understanding the variety of ways that severe negative emotional reactivity may arise is an important aim for psychopathology research. An emerging approach for parsing heterogeneity was demonstrated by the validation of "biotypes" in adults with depression and anxiety (Tozzi et al., 2024; Williams, 2017). Neurofunctional data from both task-free (resting state) and task-active conditions generated six clusters of patients. These clusters showed distinct associations with symptoms constellations and treatment outcomes. Given the range of potential sources of dysfunction that may contribute to the emotion dysregulation phenotype, this is a promising avenue for further research.

Therapeutics. Better specification of the processes that give rise to an individual's clinical disorder may spur targeted interventions that hasten meaningful improvement. Many irritable, behaviorally volatile youth with ADHD experience robust benefits from stimulant medication and family-based behavior therapy, while others do not even though ADHD symptoms improve (e.g., Blader et al., 2021). The sources of this variability in response are not known, but heterogeneity in underlying pathogenic processes is a plausible reason. Examining outcomes to treatments with discrete targets can promote more individualized, rational, and potentially safer, approaches to care.

Nomenclature. We introduced this review by noting the potential incongruity between the widespread use of the term "emotion dysregulation" and vagueness about the disrupted regulatory process its use implies. This review finds that there is a range of processes that might contribute to seemingly undercontrolled, emotionally charged behavior in children, but none yet that fits the strict definition of a feedback-based regulating process. Although the term is entrenched, one could question if its adoption is premature if we are uncertain about the disturbed regulatory process that causes it. In some contexts, descriptive terms that are agnostic about mechanisms, such as irritability or emotional overreactivity, may be preferable for now.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Complete bibliography of included articles (n = 803).

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Data availability statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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Key points

- The term "emotion dysregulation" in child psychopathology refers to a clinical phenotype in which intense anger leads to behavioral dyscontrol. It also implies etiology because it invokes a disturbed *regulatory* mechanism that curtails excessive arousal and the behavior that ensues. However, the nature of that regulatory mechanism is unclear.
- This review of research on mechanisms that might underlie the emotion dysregulation phenotype finds scant evidence for a closed-loop feedback-based automatic process whose malfunction causes poorly regulated emotions or behavior. Open-loop, deliberate regulatory strategies seem most relevant for low-to-moderate arousal.
- Stronger evidence supports roles for anomalies in stimulus processing, in response selection and control, and in factors that influence emotion generation (e.g., autonomic, endocrine, and reward functions) in the pathogenesis of the emotion dysregulation phenotype. Many studies show this phenotype's associations with adverse experiences and family interactions.
- This review suggests that the defective regulatory mechanism that emotion dysregulation implies needs further specification. A systems-control framework that integrates the wide range of emotion-relevant processes studied to date may improve understanding of their interrelationships, potential heterogeneity in etiology, and better-targeted treatments.

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