Current Psychiatry Reports (2019) 21: 17
https://doi.org/10.1007/s11920-019-1003-6

ATTENTION-DEFICIT DISORDER (A ROSTAIN, SECTION EDITOR)

Attention-Deficit/Hyperactivity Disorder (ADHD) and Emotion Regulation Over the Life Span

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Published online: 2 March 2019

Abstract

Purpose of Review Emotional symptoms are common and persistent in youth and adults with attention-deficit/hyperactivity disorder (ADHD) and cause clinically significant impairments. We review recent neuropsychological, neurophysiological, and peripheral psychophysiological evidence for emotion and emotion regulation deficits in ADHD across youth and adults.

Recent Findings Central and autonomous nervous system correlates argue in favor of more general self-regulation deficits and also specific emotional deficits in ADHD. These include general performance deficits in executive functions, and structural as well as functional impairments in neuronal networks associated with top-down self-regulation. Specific deficits with bottom-up emotional activation in the amygdala and emotion evaluation associated with the orbitofrontal cortex have also been described. Furthermore, vagally mediated, high-frequency heart rate variability is associated with emotional self-regulation deficits throughout the life span. The current evidence is based on multilevel studies that assess associations of emotion regulation. However, further studies that adequately consider the processual recursive character of emotion generation and regulation may give important new insights into emotional regulation of ADHD.

Summary Emotion regulation deficits in ADHD are associated with specific as well as general self-regulation deficits traceable on the level of neuropsychological, neurophysiological, and psychophysiological assessments. The temporal dynamics of the interplay of those different systems need further study in order to optimize and personalize treatment of emotion regulation difficulties, including emotional reactivity, in patients with ADHD.

Keywords Attention-deficit/hyperactivity disorder (ADHD) · Emotion regulation · Children · Adults · Neuropsychology · Neurophysiology (fMRI EEG) · Peripheral psychophysiology (heart rate variability)

Introduction

Attention-deficit/hyperactivity disorder (ADHD) has an onset in childhood (before the age of 7 according to ICD-10 [1] and before the age of 12 years according to DSM-5 [2]) with impairing and age-inadequate levels of the three core symptoms (inattention, hyperactivity, and impulsivity) that present cross-situational (i.e., at home as well as at school or work). Originally conceptualized as a disorder of childhood, longitudinal studies demonstrate persistence rates of the disorder of about 20–80% [3]. According to prevalence studies, about 5% of youths [4, 5] and 2.5% of adults are affected [6], making ADHD one of the most prevalent disorders in childhood and adolescence and a still very prevalent disorder during adulthood [7, 8].

As the core symptoms do not sufficiently explain either the severity of the disorder nor functional impairments over the life span [9–11], emotion regulation deficits (ERDs) have been considered a translational factor relevant for ADHD [12–14, 15••], as ERDs are often observed in patients [9, 16–18, 19•, 20••, 21••], and are associated with significant functional impairments [22–24]. With high affect lability and emotional reactivity as well as reduced affect control, the ERD concept is already present in the Wender Utah criteria of adult ADHD [25], even though those are not explicitly considered in DSM-5 or ICD-10. A major inconsistency in

This article is part of the Topical Collection on *Attention-Deficit Disorder*

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the literature on emotion regulation (ER) is the large variety of terms used to describe those deficits in patients with ADHD (for instance, "emotional dysregulation" [25], "emotional impulsiveness" [26], and "emotional lability" [27]). Despite the expansion of empirical work in the field of emotion and ER, the field still lacks a consensual definition that describes ER with clarity, precision, and differentiation among constructs. To address this, we first give a short overview about emotions and their regulation and then review recent evidence of the association between ER, ADHD, and associated neuropsychological and physiological characteristics that may be compromised in ADHD.

Emotions and ER

Emotions are temporally limited, qualitative states that are associated with changes of feelings, expression, and physiology [28]. They differ from stress responses and mood in several ways, but the most prominent difference is that emotions are elicited by specific internal or external activating events. Emotions require that we direct our attention (consciously or preconsciously) to this activating event, and that we value or appraise it with regard to our goals. Finally, emotions promote relevant action urges (approach vs. withdrawal), physiological activation (central and peripheral), and expressive behaviors [29]. Thus, emotions unfold over time and are helpful when they appropriately guide sensory processing, enhance decision making, or provide information regarding the best course of action. Emotions are potentially harmful when they are of inappropriate intensity, duration, frequency, or type for a particular situation [29]. The fact that emotions unfold over time also implies that each emotion is characterized by dynamic features, such as latency of onset, rise gradient, maximal intensity of the response, duration or persistence of an emotion, and slope of recovery. Across response levels, those temporal features may differ, and they may also differ among emotions [30•].

When seeking a definition of emotion regulation, it is more or less consensual that ER refers to all efforts to influence the emotions we have, when we have them, and how we experience and express them [29]. As emotions have a strong temporal dimension, regulatory dynamic processes—no matter whether automatic or voluntarily used [31••]—interact to influence our emotional states flexibly so as to promote adaptive, goal-oriented behaviors [32].

ER strategies vary greatly (e.g., putting on a poker face, applying mediative breathing, taking a shower, trying to think differently about a situation), and they are traditionally classified into those that occur prior to the emotional response (antecedent-focused regulation; e.g., situation selection, situation modification, attentional deployment, and cognitive change) and regulation strategies that occur after the emotional response is already triggered (response-focused regulation; e.g., expressive suppression; see Fig. 1). The strategies also differ in their effectiveness to modify emotional outcomes as indexed by experiential, behavioral, and physiological measures [33]. For example, active thinking about a positive event that is unrelated to the evocative situation is an effective distraction strategy across all response levels. Cognitive change strategies such as perspective taking are effective in modifying experiential and behavioral outcomes, and response modulation may change behavior in the intended direction, while physiological states are not necessarily related to such strategies.

However, temporal dynamics have been spotlighted in recent theoretical debates. According to the extended model of emotion regulation by Gross [31••] (see Fig. 1 for details), more dynamic processes or regulatory stages have been introduced. To gain control of emotions, we enter a cyclical process whereby we identify the emotion needing to be regulated (identification), select (selection), and then execute an ER strategy (implementation). Finally, we monitor the regulatory effects so as to decide whether regulation was sufficient or not (stopping or maintaining regulatory engagement) or whether we should switch to another strategy (monitoring). For more details, see Sheppes et al. [34].

ADHD and ER

Elevated ERD is a rather consistent observation in ADHD across the life span. A recent meta-analysis ($k = 77$) reported that ADHD is strongly associated with both emotion reactivity ($d = .95$) and ERD ($d = .80$) [20••]. Prevalence studies on children, adolescents, and adults with ADHD show that a considerable portion of them present with elevated ERD. For the youngest group of patients, a population-based study by Overgaard and colleagues [35] reported that about 25% of preschoolers with ADHD show signs of emotional dysregulation, significantly more frequent than in control children. In a large community-based population sample, parents reported emotional lability for 6% of the children overall, whereas children with ADHD were found to have greater than a 12-fold increase in the odds ratio observed [36]. Other studies on youth with ADHD and ER report rates ranging from 21% [37] to 66% [38], with roughly similar rates observed for adults with ADHD [17, 24].

Given that ERDs are present in a variety of mental disorders, from both the externalizing and internalizing spectrum [36], it is important to note that ERD in ADHD is not simply explained by comorbidity [17, 18, 24]. It is also observed in non-comorbid ADHD cases [10, 19•]. A longitudinal study by Biederman and colleagues [39] found that ER problems are highly persistent within individuals. In their sample, 57% of youth with childhood ERD still had ERD 4 years later. ERDs in childhood were also positively related to the persistence of ADHD symptoms 4 years later. About 50% of youth with

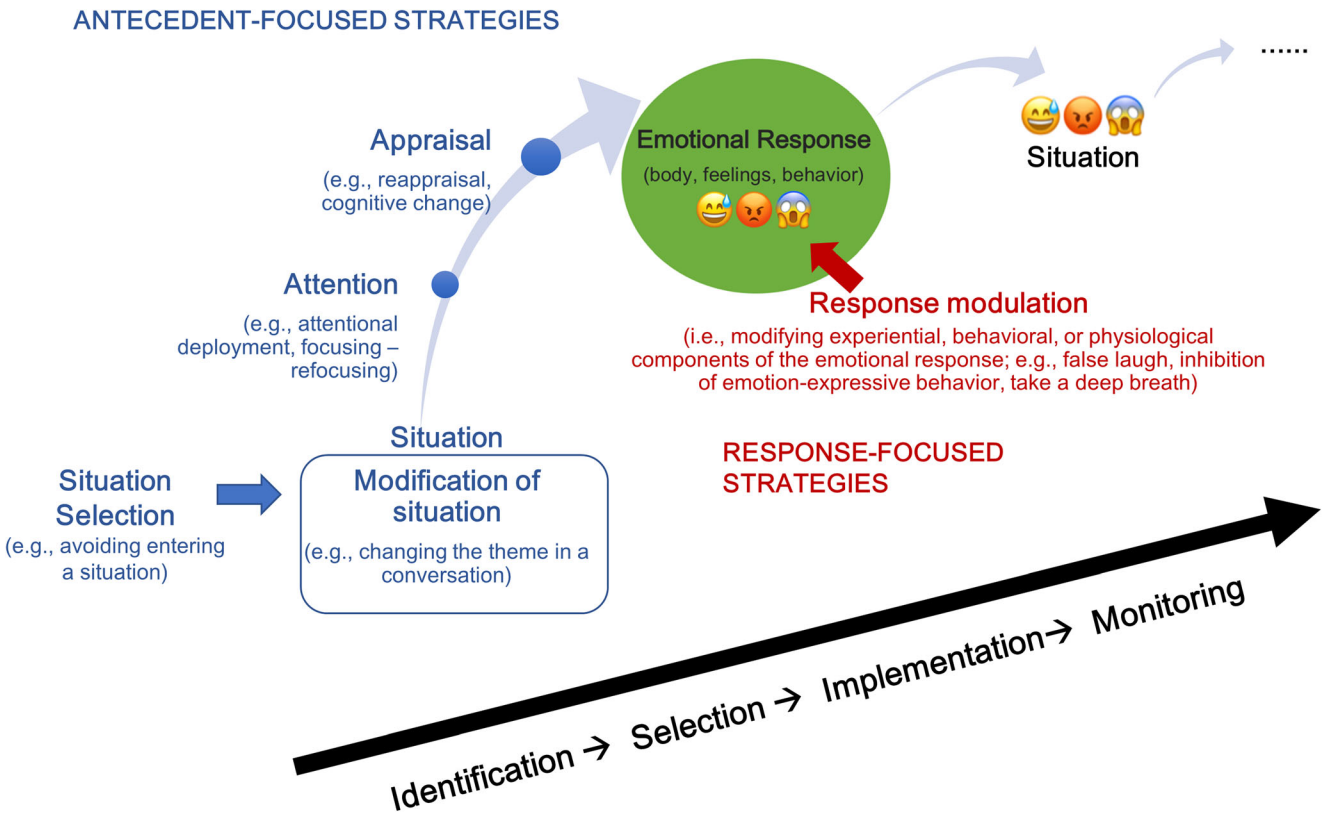

Fig. 1 Modified ER model based on Gross [31••]

persistent ADHD had ERD compared to none in the group of the non-persisters. This finding is corroborated by a recent longitudinal study showing an association of childhood ERD with the high persistent pattern of ADHD, but not with the ameliorating symptom trajectory [40]. Another recent prospective longitudinal study, in which a sample of 5-year-old children was followed up to age 13 [41•], found that poor ER and particularly poor positive ER were predictive of an increase in inattention symptoms, whereas good ER predicted a decrease of inattention symptoms across time [41•].

In sum, those findings underscore the importance of ERD in the diagnosis and treatment of ADHD. A recent comprehensive review concludes that emotional symptoms of ADHD are not simply epiphenomena of comorbidity, as they are common and persistent in youth and adults with ADHD, are present in non-comorbid cases, and are sufficiently specific for ADHD to function as diagnostic criteria [21••]. Nevertheless, the definition of the constructs presently used under the umbrella term of ER needs more precision, as does the association of the nature and pathophysiological mechanisms of the ER-ADHD association. With respect to the latter, Shaw et al. [15••] suggest three models to explain the overlap between ADHD and ER. According to *model 1*, ERDs are a core symptom of ADHD due to joint neurocognitive deficits in self-regulation that underlie cognitive and ER systems [42]. This joint deficit could be a common consequence of (i) the regulation of physiological arousal, (ii) inhibition deficits, (iii) attention regulation and cognitive flexibility, and (iv) action planning [24]. *Model 2* perceives ADHD with ERD as qualitatively different from *pure* ADHD. A basis for this model is genetic studies that demonstrate the co-occurrence of ADHD and ERD in families [16, 18]. This model thus implies a distinct etiological entity as well as a distinct course of ADHD + ERD. *Model 3* postulates ADHD and ERD as correlated, with overlapping circuits, but distinct neurocognitive deficits. Proof comes from studies with moderate correlations between ADHD symptoms and ERD [43].

Neurocognitive Correlates of ER in ADHD

ERD in ADHD is often hypothesized to result from the same or at least overlapping neurocognitive deficits in inhibition, working memory, and executive functions as core symptoms do (see models 1 and 3 of Shaw et al. [43]).

Children and Adolescents

In a seminal study by Walcott and Landau [44], boys aged 6 years to 11 years who participated in a frustrating peer competition, inhibitory control in a stop-signal task were predicted to have successful ER. Another large study of 424 children with ADHD (6–18 years of age) and their 564 healthy siblings assessed ER using aggregated parent and teacher ratings along neuropsychological functioning tests of inhibition, working

memory, and delay aversion [45]. Results suggest that a range of neuropsychological variables predicted ERD, but this association was completely mediated by severity of ADHD symptoms. Conversely, the association of ADHD symptom severity with neuropsychological variables was not mediated by ERD. This finding is supported by another study of ADHD children demonstrating that emotional cues did not impair interference control in an inhibition task [46], (but see, e.g., [47] for diverging results).

A neuroimaging study demonstrated a double dissociation between two neural circuits engaged in executive attention and control vs. ER [48]. Medication-naïve children with ADHD ($n = 22$) showed reduced connectivity in the executive attention circuit (major seed dorsolateral prefrontal cortex (PFC); connectivity with caudate, anterior cingulate) compared to controls ($n = 20$). This deviation was correlated with executive attention deficits, but *not* with emotional lability. By contrast, reduced connectivity in the ER circuit (major hub ventral striatum, connectivity among others with the orbitofrontal cortex (OFC) and amygdala) correlated with emotional lability, but *not* with executive attention deficits.

Adults

One of the first studies on ADHD and ER in adults compared male and female patients ($n = 39$) with gender- and IQ-matched control subjects ($n = 40$) on an emotional working memory task [49]. Compared to control subjects, patients with ADHD showed both a general working memory deficit and enhanced distractibility (lower performance accuracy). Controls showed impaired working memory performance for highly arousing negative background pictures, while patients with ADHD demonstrated decreased attention and a comparable decrement with low arousing pictures [49]. A large study on 325 adults with and without ADHD and with and without ER deficits revealed no differences in executive functions for the two groups with ADHD (that is ADHD + ER and ADHD only). Compared to adults without ADHD, both groups with ADHD demonstrated lower scores across several measures of executive function, suggesting that deficits in ER are independent of neuropsychological impairments [50].

In two studies by our group, we were able to demonstrate that ERDs are a core component in adult ADHD and that neuropsychological measures do not contribute significantly to subgroup formation in adults with ADHD and ERD. In our first study, apart from ADHD symptoms assessed with the Conners Adult ADHD Rating Scales (CAARS) [51], we separately assessed negative and positive affective states and ER skills in adults with ADHD to test a model that distinguished positive and negative affect, problems with self-concept, and ER skills as factors that were distinct, but most likely correlated with the symptom domains of inattention, hyperactivity, and impulsivity [52•]. Our sample consisted of 213 individuals newly diagnosed with adult ADHD who were all medication-naive. A seven-factor confirmatory model was supported by our data leading to the conclusion that deficits in ER are an integral part of adult ADHD. Our study contributes to a deeper understanding of the impairments related to adult ADHD and can be used for further differentiation of subgroups within this population. This was examined in more detail in a follow-up study applying a cluster analytic approach to subtype patients on the relative presence or absence of ERD [53]. We found two clusters, with cluster 2 in contrast to cluster 1 showing high deficits in ER that were associated with higher impairments in most clinical areas: ADHD core symptoms, depression, additional clinical symptoms, and symptoms of personality disorders. Women were overrepresented in this cluster. There were also higher rates of comorbidity in this cluster, such as somatoform disorders that were associated with deficits in ER. Of note, the presence of comorbidity did not significantly account for ERD in this sample. Neuropsychological variables like the Quantified Behavior Test Plus (Ob+) [54] also did not contribute significantly to cluster formation.

As those studies rely on self-reports and parent/observer reports and their associations with other variables, we now review experimental tasks using central and autonomous nervous system activity to follow up emotion and ER processes.

Neurophysiology and ER in ADHD

A brief review of underlying brain activity may be useful to differentiate emotion generation (*bottom-up* processes arising from related brain areas as amygdala, ventral striatum, and somatosensory areas) from emotion regulation (*top-down* processes associated with neuronal activity in *executive* neural networks including the central role of the prefrontal cortex).

Bottom-Up Emotion Generation

Two different paradigms in neuroscience are regularly used to study emotion generation: (1) functional MRI that confronts patients with affectively charged stimuli and (2) associations of structural MRI or resting state connectivity with trait measures of emotional reactivity. A representative example of the former is the work by Posner et al. [55]. They assessed emotional reactivity to subliminally presented fearful faces and detected abnormally high activity in the amygdala and stronger connectivity in the lateral prefrontal cortex in children with ADHD. Both these responses were attenuated with stimulant medication. Yu et al. [56] detected elevated emotional lability based on Conners' parent ratings in medication-naïve boys with ADHD as well as an association with reduced resting state functional connectivity among the bilateral superficial amygdala, the dorsolateral prefrontal, and the inferior parietal

areas. Superficial amygdala regions are connected to the OFC areas associated with emotional outcome evaluation. These detected circuits may form the core of an emotion network that is dysregulated in ADHD. The amygdala may also modulate the processing of fearful compared to neutral and joyful stimuli and may initiate visual attention orientation when processing fearful ones by establishing functional connectivity between the superficial amygdala, the visual cortex, and the superior parietal cortex [57]. Further, abnormally increased amygdala activity (and hypo-responsiveness in the ventral striatum) was evoked when anticipated reward was delayed, in a study with adults with ADHD [58]. As such, activity in the amygdala may serve as an important bottom-up processing hub that is probably impaired in ADHD.

Top-Down Regulation

A recent meta-analysis on human neuroimaging studies [42] divides top-down processes into three parts: (1) a common core system for self-regulation, encompassing the inferior frontal gyrus, the medial frontal cortex (MFC) with the anterior cingulate cortex (ACC), the supplementary motor area (SMA), and the right temporoparietal junction (TPJ); (2) a specific *action* regulation or response inhibition system (e.g., dorsal premotor cortices, intra-parietal sulci, thalamus); and (3) an ER system (e.g., more posterior parts of the TPJ and MFC) [42]. A number of studies revealed difficulties in patients with ADHD within the former two regulation systems; however, there are very few studies that elucidate emotional regulatory processes specifically.

Core Systems for General Self-Regulation and Action Regulation

It is well known that patients with ADHD show deficits in cognitive control, an important aspect of executive functions [59–62]. Such deficits have been demonstrated with a wide range of tasks such as tapping interference control (Stroop tests) or inhibition of (prepotent) responses (Go/NoGo and Stop tasks) [63–66]. Deficits typically appear as slower and more error-prone responses and as abnormal activity in brain regions associated with cognitive control. Patients with ADHD showed diminished brain activity during cognitive control in the dorsolateral prefrontal cortex (DLPFC), the ACC, and (pre-)SMA in studies using functional magnetic resonance imaging (fMRI) and in others using brain electrical activity. Moreover, error processing seems to be blunted in ADHD. For example, early error negativity assumed to indicate response conflict between required response and reinforcement learning mechanism (main sources: ACC, DLPFC) is less pronounced in ADHD compared to healthy controls [67, 68].

Interestingly, there is some evidence that performance in cognitive tasks and associated brain activity impairments may be ameliorated by either immediate rewards or ADHD medication [69–71]. Thus, it seems plausible to assume that even the "cold" self-regulation and action regulation systems are modulated by emotional bottom-up input. From an experimental psychology point of view, it seems indispensable to distinguish those emotionally colored cognitive control and action control processes from emotion regulation processes.

Core Systems of Emotion Regulation

There are few studies that address ER and ERD in patients with ADHD using emotional stimulation. Most of them rely implicitly on Shaw et al.'s model 1, namely that ER and cognitive deficits may be common consequences of a core deficit in self-regulation. The current evidence suggests that a lack of cognitive control in emotionally stimulating situations may lead to increased distraction and thus impaired ER in patients with ADHD. In line with this proposed general lack of cognitive control (see above), there is evidence from a study of boys with ADHD that revealed emotional distractors caused diminished performance in a digit categorization task. For boys with ADHD, this was accompanied by larger N2 amplitudes to emotional as compared to neutral distractors, indicating that these emotional stimuli may induce a larger conflict in patients with ADHD than in healthy controls [72]. On the other hand, another study with a Go/NoGo paradigm incorporating background affective pictures revealed enhanced NoGo-P3 amplitude and concurrent activity in the OFC sensitive to emotional context similar to that of controls (a potential compensatory increment of neuronal processing) [73].

There is also some evidence regarding the impact of ER strategies while processing affective pictures. Compared to controls, adult patients with ADHD showed increased late frontal positive potentials that in a subgroup of medication-naïve patients also extended towards centro-parietal locations. This is generally associated with conscious processing of stimulus' emotional intensity [69]. In a study by Shushakova et al. [74•], late frontal potentials were associated with ADHD symptom severity. Analyses of the visual N2pc to cues related to selective attention revealed an abnormal bias towards negative emotional background stimuli in patients with ADHD that was also related to diminished self-reported ER abilities [75].

Another study found diminished activity in the OFC during processing of an oddball task incorporating emotional facial expressions in children with ADHD [76]. This is in line with the finding of diminished OFC activity as reflected in lower error positivity amplitude, particularly in tasks where errors are rare and salient. Taken together, these findings of abnormal activity in the OFC may indicate difficulties with the

emotional evaluation of stimuli along with impaired response outcomes in ADHD.

Psychophysiology and ER in ADHD

Along with the central nervous system (CNS), the autonomic nervous system (ANS) is highly relevant for emotions and their regulation [77]. While emotion reactivity has been associated with sympathetic alterations, ER seems to be more closely related to parasympathetic regulation [78, 79, 80•]. As hypothesized by the neurovisceral integration theory [81], accumulating evidence suggests that measures of vagally mediated heart rate variability reflect PFC function and hence CNS substrates of ER [82–85]. This is corroborated by evidence demonstrating that resting state vagal tone, indexed by high-frequency heart rate variability (HF-HRV), predicts sustained attention, task engagement, and ER over the life span [86–89]. A recent meta-analysis reports a small, but relevant association between markers of self-control in laboratory tasks and vagally mediated resting state heart rate ($r = .15$) [90]. Finally, greater task-induced withdrawal in vagal tone has been associated with better attention [86, 91], social behavior [92, 93], impulse control [77, 94, 95], and fewer externalizing and internalizing problems among children (see [96] for a review).

Children and Adolescents

Among children and adolescents, several studies have investigated ADHD, ER, and autonomic parasympathetic/vagal markers such as HF-HRV, as well as markers related to sympathetic activity such as the pre-ejection period (PEP) [78, 79, 94, 97, 98]. Most evidence comes from a number of consecutive investigations from Musser et al. [78, 97], Karalunas et al. [79], and Musser and Nigg [80•] with overlapping samples. In their studies, boys and girls with ADHD watched emotional evocative film clips (positive and negative) and were instructed to empathize with the main character. They either had to mimic (induction task) or to suppress (suppression task) emotions. In their first study, the researchers did not find any group differences for resting state HF-HRV. However, across emotion tasks, healthy controls showed a systematic variation in HF-HRV depending on valence (reduced for positive emotion, increased for negative emotion) and task demand (suppression needed more activation than induction). Children with ADHD were observed to have variable HF-HRV in response to the emotional tasks overall, but without any significant modulation [78]. In later studies, the authors demonstrated that there are subsamples of patients with ADHD who have altered sympathetic-based emotion reactivity as well as regulatory differences in parasympathetic HF-HRV [97]. These results have been confirmed by other groups using different paradigms such as social exclusion [98].

The most recent study from this lab [80•] studied emotion systems coherence in 100 children aged 7–11 years. Children watched the same emotion evocative film clip as in the study of Musser et al. [78], and facial emotion display across both negative and positive emotion induction was coded in addition to HF-HRV and PEP, in order to examine time-linked coherence of facial display, autonomic reactivity, and regulation. Children with ADHD showed weaker coherence among autonomic activation and negative facial affect behavior during the induction of both negative and positive emotions when compared to typically developing children.

Taken together, studies on children and adolescents demonstrate specific physiological regulation patterns of ADHD and ER in certain contexts (i.e., social vs. cognitive tasks with frustration; positive vs. negative valence). Overall, the studies are indicative of an altered vagal regulation pattern—presumably reflecting PFC dysfunctions.

Adults

We are only aware of two studies on adult ADHD, ER, and physiological arousal [99, 100]. A study on stress reactivity [99] used the Trier social stress test—a standardized cognitive-psychosocial stressor, simulating a job interview in conjunction with mental arithmetics in front of an audience [101]. Subjective stress levels were higher in patients with ADHD compared to healthy controls, but HF-HRV did not differ between groups. Comparable results were obtained in a study with young adults with and without ADHD symptoms on a frustrating driving simulation [100]. Participants with high ADHD symptoms reported more anger and frustration, but differences for HF-HRV were not significant. The study by Lackschewitz and colleagues [99] followed a high diagnostic standard, but both studies lack power due to small sample sizes ($n < 25$). Thus, evidence for pathophysiologic differences in ADHD adults is currently very sparse, and interpretations and implications are consequently limited.

Conclusions and Future Research

ADHD is often considered a neurocognitive disorder, although emotional symptoms are highly prevalent and associated with higher impairment and undesired negative outcomes across life span. We know from the literature that emotional symptoms are only partially responsive to treatment [102]; thus, it is necessary to understand the association of ER and ERD in ADHD. This review demonstrates that current translational research suffers from major limitations resulting from a lack of conceptual clarity regarding ER and ERD in ADHD. The extant research summarized

clearly points to the relevance of ER and ERD in ADHD, but it ignores related processes such as the unfolding of emotions over time, and the time variation for different components of emotion such as subjective feelings, motivation, cognition, action tendencies, expressive behavior, and neuropsychophysiology [28]. To improve our understanding of ER and ERD in ADHD, the time dimension seems to be highly salient, and there is growing consensus that processual characteristics should be incorporated in studies of emotion and ER [21••, 30•]. This consensus also calls, at least partially, for a paradigmatic shift in research, as neuroscience methods such as fMRI have merits in testing structural assumptions but are less suited to investigate the time dimension compared to other techniques such as EEG, MEG, and peripheral psychophysiology. The research reviewed in the present article illustrates the current pitfalls and benefits of ERD in ADHD. An important pending research question in the field of emotion is whether, and if so, how, emotions can be induced in our labs to ensure research that is internally and externally valid and that is informative for the delineation of treatment options.

However, the aforementioned extended process-oriented framework by Gross [31••] specifies iterative regulatory stages, regulatory decisions, and potential failure points related to (1) whether or not to regulate (identification), (2) when to select a regulatory category and specific regulation tactic to use, (3) then how to execute this strategy with the aim of changing the emotion or its generation, and (4) how to monitor the regulatory effects that tell us whether ER is sufficient or not (stopping or maintaining regulatory engagement) or whether we should switch to another strategy. Those challenges and potential points of failure might differ among individual patients with ADHD and may suggest specific alternative treatments for different subgroups. Such disparities may explain why one treatment, e.g., pharmacological interventions, has limited effects on specific emotional symptoms.

In sum, the process model allows for studying ER in real time, to specifically track emotions and the associations among emotions, ER, and psychopathology. Beyond general self-regulation problems, ERDs are thus viewed as specific difficulties or configurations of difficulties arising from separate levels of emotion processing (e.g., difficulties due to a deficient set of strategies, rigidity in using only a small set of strategies, identification problems (when to regulate which emotion in a specific context), problems with implementing the strategy). This could reveal valuable information about treatment options in order to tailor personalized strategies to improve ER. However, to this end, we need more sophisticated experiments and research targeting the temporal dynamics of emotion and ER using multilevel approaches and techniques with high temporal resolution.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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