Borderline Personality Disorder and Emotion Dysregulation

RESEARCH

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Emotion dysregulation in adolescents is normalized by ADHD pharmacological treatment

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Abstract

Background Attention-deficit/hyperactivity disorder (ADHD) is associated with emotion dysregulation (ED) and in ADHD, beyond ADHD and comorbidity severity, ED confers increased risk for negative outcomes. First- and second-line ADHD pharmacotherapy is effective at ameliorating core symptoms and improving cognitive functioning and accumulating evidence indicates primairly in children and adults, active ADHD pharmacotherapy has beneficial effects on emotional symptoms. Gaps in knowledge remain about whether in adolescents, ADHD pharmacotherapy has beneficial effects on ED or about the extent to which effects are apparent for discontinued/ past ADHD pharmacotherapy.

Methods Examined, in $N = 297$ adolescents ($M_{age} = 15.77$ years, $SD = 1.06$; 39.06% girls; $n = 86$ classified as with ADHD), whether accounting for depression and oppositional symptoms, concurrent and 18-month prospective measures of parent- and self-reported ED (1) differ across adolescents without ADHD, medication-naïve adolescents with ADHD, and ever-medicated (currently or previously) adolescents with ADHD.

Results In case of parent-reported ED, ever medicated adolescents with ADHD exhibited a decline in ED over time whereas adolescents without ADHD and never medicated adolescents with ADHD exhibited no changes in ED over time. In case of self-reported ED, ever-medicated adolescents with ADHD exhibited lower ED than never medicated adolescents with ADHD and never medicated adolescents with ADHD exhibited greater ED than adolescents without ADHD. Currently and previously (but not currently) medicated adolescents did not differ in ED. Across parent- and self-reported findings, observed pattern of results held when analyses focused on adolescents who did not change medication status between baseline and follow-up.

Conclusions ADHD pharmacotherapy may have a boosting effect on longitudinal changes in parent-reported ED and a normalizing effect on concurrent measures of self-reported ED in adolescents.

Keywords ADHD, Emotion dysregulation, Pharmacotherapy, Adolescence, Longitudinal

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Introduction

Attention-deficit/hyperactivity disorder (ADHD) is an early-onset disorder diagnosed in 5% of girls and 10% of boys worldwide [1]. ADHD is associated with increased risk for a host of negative outcomes including academic problems, aggression and bullying/victimization, comorbidities, or risk-taking [2].

ADHD is also associated with emotion dysregulation (ED) [3–5]. In the context of ADHD, emotion regulation has been defined as the ability to modulate behavioral, experiential, and neurophysiological aspects of emotion escalation, intensity, and de-escalation in a manner that is consistent with adaptive functioning [3]. ED is difficulties with any or all aspects of emotion regulation to a degree that is inconsistent with adaptive functioning [3]. For example, in the context of a game, a child may become upset by her/his team missing a shot. In case of emotion regulation, the child may down-regulate the intensity of the negative emotion and speed up the de-escalation of that negative emotion by re-focusing her/his attention on the game, the goals thereof, and next steps. As a result, this child is able to continue participating without problems. In the same situation but in case of emotion dysregulation, the child may have difficulties with re-focusing their attention and may dwell on the negative emotion by retaining focus on the situation that elicited the negative emotion. The child begins crying and lashing out, feels upset and tense, has elevated heart rate. As a result, this child is unable to focus on next steps and is thus unable to continue participating.

ED is conceptually and empirically a mechanism of ADHD-related negative outcomes, including aggression, bullying/victimization, certain comorbidities, functional impairment, or risk-taking [3–6]. Yet, debate prevailed about the extent to which ED is a core or merely associated feature of ADHD [3]. Data indicate that relative to children with ADHD but no ED, ADHD polygenic risk scores (but not depression polygenic risk scores) are elevated in children with ADHD and ED [7]. Findings also show that ADHD is associated with ED, independent of cognitive functioning and comorbidities, including depression, conduct, and oppositional defiant (ODD) disorders [4, 8]. Further, in adolescents with ADHD, ED is associated with functional impairment above and beyond comorbidities, including depression and ODD [8, 9]. If ED is an aspect of ADHD presentation and is a mechanism of ADHD-associated negative outcomes, then it is an appropriate target for ADHD treatment.

Psychostimulants (typically, methylphenidate) and nonstimulants (typically, atomoxetine) are European Medicines Agency (EMA) and Food and Drug Administration (FDA) -approved first and second-line pharmacotherapies of ADHD [10–12]. Meta-analytic findings indicate efficacy of both in alleviating ADHD symptoms [11, 13] and in improving neurocognitive performance [14].

Stimulants and atomoxetine may also attenuate but possibly exacerbate severity of emotional and mood features. Although the location of action of stimulants and nonstimulants on ADHD symptoms is predominantly the prefrontal cortex, these drugs also diffuse to other brain regions. Dopamine (a stimulant target) also has a crucial role in the mesolimbic system implicated in regulating reward sensitivity. Norepinephrine (a nonstimulant target) is a key neurotransmitter in the brainstem and the limbic system, implicated in pairing affective, autonomic, and cognitive functions. Drugs acting on dopamine and norepinephrine may thus have effects on emotion and mood [15]. Yet, relative to core symptoms, less is known about the effects of ADHD pharmacotherapies on ADHD-associated ED.

In adults, meta-analytic data of double-blind randomized controlled trials indicate small-to-moderate effects of ADHD pharmacotherapies on *concurrent* ED (methylphenidate: SMD = 0.34, 95% CI = 0.23–0.45; atomoxetine: SMD = 0.24, 95% CI = 0.15–0.34; lisdexamfetamine: SMD = 0.50, 95% CI = 0.21–0.8) [16]. In children, data indicate beneficial effects of ADHD pharmacotherapy on emotional difficulties [17], including for methylphenidate on emotional lability/ problems [18, 19], for lisdexamfetamine (LDX) on emotional control [20] and, for atomoxetine (ATX) on emotional difficulties [21]. Although adolescence is an especially vulnerable developmental period with regard to acquisition of adaptive emotion regulation skills [22], relative to adults and children, less is known about the effects of ADHD pharmacotherapies on ADHD-associated ED in *adolescents* [15]. Available data are mixed insofar as those indicate elevated emotional lability as an adverse event in > 5% of patients in response to mixed amphetamine salts [23] and to LDX [24] but also elevated mood [25] and improved ED [26] in response to methylphenidate in adolescents with ADHD.

Further, gaps in knowledge remain about whether these the effects of ADHD pharmacotherapies on ADHD-associated ED are maintained even after discontinuation of pharmacotherapy. Studies on concurrent and studies on long-term effects [27] of ADHD pharmacotherapies on ED characteristically assessed outcomes when individuals were on active treatment. Yet, preliminary findings also show that ADHD pharmacotherapies may normalize ED even after long-term discontinuation of treatment. Relative to adults without ADHD, adults with ADHD who received methylphenidate treatment during childhood (but not as adults) exhibited comparable subgenual cingulate and ventral striatal response to affective stimuli (negative and positive images) to adults without ADHD [28]. Conversely, adults with ADHD who did not receive pharmacotherapy during childhood exhibited lower

response to affective stimuli relative to adults without ADHD [28]. Accordingly, in adults with ADHD, childhood methylphenidate treatment may normalize neural emotion processing in brain regions implicated in reward anticipation and emotion regulation. In the only available, longitudinal research with a medicated and a medication-naïve ADHD group, ED was indexed as emotional symptoms on the Strengths and Difficulties Questionnaire (five items on complaints of head and stomachache; fearfulness; nervousness; sadness; and worry) and differences between previous but not current and current medication were not assessed [27].

Current study

Aim was to examine whether relative to adolescents without ADHD, ADHD pharmacotherapy normalizes concurrent and prospective measures of ED in adolescents with ADHD. Multi-method and –informant measurement, especially of complex and heterogeneous characteristics such as emotion regulation, have long been recommended for research on child and adolescent psychopathology [29–31]. In case of emotion regulation, self-report is key, given the largely internal and subjective nature of emotions [3]. However, as children and adolescents with ADHD are often unreliable reporters of their behavior and functioning [32, 33], augmenting self-report with observer-report (e.g., parent-report) is advantageous for capturing the different, multi-faceted aspects of the phenomenon.

Accordingly, we examined whether accounting for depression and ODD symptoms, concurrent and prospective measures of parent- and self-reported ED (I) differ across adolescents without ADHD, medication-naïve adolescents with ADHD, and ever-medicated (currently or previously) adolescents with ADHD. Where a normalization effect was apparent, i.e. no difference between adolescents without ADHD and ever-medicated adolescents with ADHD was observed, we examined this question by differentiating between ever-medicated adolescents with ADHD who were currently medicated and ever-medicated adolescents with ADHD who were previously but not currently medicated. Specifically, we examined whether accounting for depression and ODD symptoms, concurrent and prospective measures of parent- and self-reported ED (II) differ across adolescents without ADHD, medication-naïve adolescents with ADHD, currently medicated adolescents with ADHD, and previously but not currently medicated adolescents with ADHD.

We hypothesized that (I) both concurrent and prospective measures of parent- and self-reported ED will differ across adolescents without ADHD, medication-naïve adolescents with ADHD, and ever-medicated (currently or previously) adolescents with ADHD such that adolescents without ADHD will not differ from ever-medicated (currently or previously) adolescents with ADHD but medication-naïve adolescents with ADHD will exhibit greater ED than adolescents without ADHD and than ever-medicated (currently or previously) adolescents with ADHD. (II) ever-medicated adolescents with ADHD who were currently medicated will not differ from ever-medicated adolescents with ADHD who were previously but not currently medicated.

Methods

Data analyzed in the current study were collected in a larger, 5-year longitudinal project, the (BLINDED) study, where adolescents were followed for 4 years and across three timepoints. In that project, adolescents were recruited from local high schools and, to oversample for ADHD, from local clinics and hospitals with departments of psychiatry. The data analyzed in the current study were obtained during the first two timepoints, at baseline, and at 18-month follow-up.

Exclusionary criteria were cognitive ability ≤ the percentile rank corresponding to a full-scale IQ score of 80 on abbreviated versions of age-appropriate versions of the Wechsler scales [34, 35]; meeting diagnostic criteria for bipolar, obsessive–compulsive or psychotic disorder on the SCID-5-CV; prior diagnosis of autism spectrum disorder (severity ≥ 2); neurological illness; and visual impairment (uncorrected, impaired vision < 50 cm).

Parents and participants provided written informed consent (and assent) and then participants underwent a series of tests, including assessment of cognitive ability and a clinical interview, followed by genetic sampling and questionnaires (first assessment session) and an EEG measurement and questionnaires (second assessment session) at baseline and including questionnaires (first assessment session) and an EEG measurement (second assessment session) at 18-month follow-up. Parents completed questionnaires using the Psytoolkit platform [36, 37] and the Qualtrics software, Version June 2020–May 2023 (Qualtrics, Provo, UT). This research was approved by the National Institute of Pharmacy and Nutrition (OGYÉI/17089-8/2019) and has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

ADHD classification was determined using parent-report on the ADHD Rating Scale-5 (ARS-5) [38]. To be classified as with ADHD, adolescents had to meet a total of ≥ 6 (youth < 17 years old) or 5 (youth ≥ 17 years old) of the DSM-5 ADHD inattentive (IA) or hyperactive/impulsive (H/I) symptoms and exhibit impairment (i.e., rating of ≥ 2 = moderate impairment) in ≥ 3 areas of functioning.

Participants

Participants were a community sample of $N = 297$ adolescents; at baseline, they were between the ages of 14–17 years (M_{age}=15.77 years, $SD = 1.06$; 39.06% girls); $n = 86$ (28.96%) were classified as with ADHD. At T2, data were available for $n = 226$ adolescents (23.9% attrition), of whom $n = 99$ (43.8%) were classified at baseline as at-risk for ADHD. At T2, adolescents were between the ages of 15–19 years (M_{age}=17.21 years, $SD = 1.06$; 39.82% girls).

Average cognitive ability was in the 61st percentile ($SD = 21.1$). Participants were from a slightly above-average socioeconomic background based on net household income per person, $t(297) = 2.354$, $p = .019$, Cohen's $d = 0.183$, with a sample average of 157 635 HUF ($SD = 77$ 863) vs. the average of approx. 147 000 HUF in Hungary in 2020 [39].

Of 86 adolescents with ADHD, $n = 46$ (53.49%) were medication-naïve, $n = 19$ (22.09%) were currently prescribed ADHD pharmacotherapy, and $n = 21$ (24.42%) were previously but not currently prescribed ADHD pharmacotherapy. Of those previously but not currently prescribed ADHD pharmacotherapy ($n = 22$), six indicated discontinuation length; two discontinued three years before baseline measurement, one discontinued two years before, one discontinued 1.5 years before, and one discontinued 8 months before baseline measurement.

Measures

Primary variables

ADHD ADHD was measured using the ADHD Rating Scale-5 (ARS 5) [38], a 30-item parent- and teacher-report measure of the past 6-month presence and severity of DSM-5 ADHD symptoms (9 inattentive symptom items and 9 hyperactivity/impulsivity symptom items) and functional impairment across six domains: relationship with significant others (family members for the home version), relationship with peers, academic functioning, behavioral functioning, homework performance and self-esteem (2 × 6 impairment items, with one set corresponding to inattention and one to hyperactivity/impulsivity). Parents and teachers rate items on a four-point scale ranging in case of symptoms from 0 (never or rarely) to 4 (very often) and in case of impairment from 0 ("no problem") to 3 ("severe problem"), with higher scores indicating more severe symptoms and impairment. The ARS-5 is comprised of two symptoms scales, Inattention and Hyperactivity-Impulsivity, and a Total Scale. The ARS-5 is suitable for ages 5–17 years, with separate forms for children (5–10 years) and adolescents (11—17 years) and age-appropriate and DSM-5 compatible descriptions of symptoms. In the current study, the adolescent home (i.e., parent-report) version was used. Prior findings indicate both the original (e.g., internal consistency and 6-week test-retest reliability; factor structure; concurrent validity and predictive validity) [38] and the Hungarian translation (internal consistency) [40, 41] have acceptable psychometric properties. In the current sample, the ARS-5 Total exhibited acceptable internal consistency ($\omega_{baseline} = 0.921$) and was used in analyses.

ED Self-reported ED was measured using the Difficulties in Emotion Regulation Scale (DERS; [42], a 36-item self-report measure of ED, comprised of six subscales, Nonacceptance of Emotional Responses (Nonacceptance, e.g., *When I'm upset, I become angry with myself for feeling that way*), Difficulties Engaging in Goal-Directed Behavior (Goals, e.g., *When I'm upset, I have difficulty concentrating*), Impulse Control Difficulties (Impulse, e.g., *When I'm upset, I become out of control*), Lack of Emotional Awareness (Awareness, e.g., *When I'm upset, I acknowledge my emotions*), Limited Access to Emotion Regulation Strategies (Strategies, e.g., *When I'm upset, I believe that wallowing in it is all I can do*), and Lack of Emotional Clarity (Clarity, e.g., *I have difficulty making sense out of my feelings*). Items are rated on a five-point Likert-type response format scale (1 – 'Almost Never' to 5 – 'Almost Always'), with higher scores indicating greater difficulty with emotion regulation. Prior findings indicate the DERS has acceptable psychometric properties, including good internal consistency, good test–retest reliability, and adequate construct and predictive validity in multiple adolescent samples [8, 9, 43–45]. In addition, the DERS exhibited robust correlations with psychological problems reflecting ED [45] and physiological measures of ED [44]. The Hungarian translation also demonstrated acceptable psychometric properties, including good internal consistency (all $\alpha s > 0.70$) as well as construct and convergent validity with the Zung Self-rated Depression Scale [46].

In the current sample, internal consistency of the subscales was acceptable to excellent, with McDonald's omega values as follows: Awareness = 0.775; Clarity = 0.806; Goals = 0.866; Impulse = 0.869; Nonacceptance = 0.825; Strategies = 0.859; Total DERS = 0.920. In case of Δ scores (derived as T2-baseline values), negative values indicate a decrease in ED from baseline to 18-month follow-up, positive values indicate an increase in ED from T1 to 18-month follow-up. In the current study, data from the Total DERS and Δ Total DERS were used in analyses.

Parent-reported ED was measured using the Difficulties in Emotion Regulation Scale-Parent Report (DERS-P) [47], a 29-item parent-report measure of child ED, comprised of four subscales, Attuned (e.g., *My child pays attention to how he/she feels*), Catastrophize (e.g., *When my child is upset, he/she believes that he/she will end up feeling very depressed*), Distracted (e.g., *When my child is upset, he/she has difficulty concentrating*), and Negative

Secondary (e.g., *When my child is upset, he/she feels ashamed with him/herself for feeling that way*). Items are rated on a five-point Likert-type response format scale (1 – 'Almost Never' to 5 – 'Almost Always'), with higher scores indicating greater difficulty with emotion regulation. Prior findings indicate the DERS-P has acceptable convergent, concurrent, and incremental validity as well as internal consistency [47]. In the current sample, the Hungarian translation of the DERS-P exhibited acceptable internal consistency ($\omega_{baseline} = 0.957$). In case of Δ scores (derived as 18-month follow-up-baseline values), negative values indicate a decrease in ED from baseline to 18-month follow-up, positive values indicate an increase in ED from baseline to 18-month follow-up. In the current study, data from the Total DERS-P and Δ Total DERS-P were used in analyses.

Medication status At baseline, parents were asked whether their child was ever diagnosed with an emotional or psychiatric problem. Those who indicated that their child was ever diagnosed with such a problem, are asked to indicate the diagnosis. Next, they are asked whether their child was ever prescribed medication for an emotional or psychiatric problem. Those who indicated that their child was ever prescribed medication for a such a problem were asked to provide details about the medication(s): (1) emotional or psychiatric problem for which the medication was prescribed; (2) medication name, recommended dosage, taken dosage, type [e.g. long- or short-acting], length of medication treatment; and, for each listed medication (3) whether it was prescribed and taken in the past and/or whether it was prescribed and taken currently. For medications prescribed in the past, parents had the option to indicate the last time the medication was taken. At the 18-month follow-up, parents were asked whether, since the last assessment (i.e. baseline), their child was diagnosed with an emotional or psychiatric problem, was prescribed medication for an emotional or psychiatric problem, and to indicate the above details about the medications. A child was considered as having been prescribed an ADHD medication if their parents reported they were prescribed a European Medicines Agency and/ or a National Institute of Pharmacy and Nutrition (OGYÉI) approved ADHD drug.

Covariates

Depression symptoms Depression was measured using the Youth Self-Report 11–18 (YSR) [48], a 112-item self-report questionnaire for adolescents (ages 11–18) assessing aspects of adaptive and impaired functioning. The YSR measures adaptive functioning through *competence scales*: academic performance, activities, and social competence and impaired functioning via *DSM-oriented scales*: anxiety problems, depressive problems, somatic problems, attention-deficit/ hyperactivity problems, oppositional defiant problems, and conduct problems; as well as *syndrome scales*: anxious/depressed, depressed/withdrawn, somatic complaints, attention problems, social problems, thought problems, aggressive behavior, rule-breaking behavior, externalizing problems and internalizing problems. Respondents rate items are rated on a 3-point scale (0 – 'Not True', 1 – 'Somewhat or Sometimes True', 2 – 'Very True or often True').

Prior findings indicate both the original (internal consistency, test-retest reliability [48]) and the Hungarian translation (test-retest reliability, convergent validity [41]) of the YSR has acceptable psychometric properties. In the current study, the Depression Problems subscale total score (possible min-max = 0–26) exhibited acceptable internal consistency ($\omega = 0.859$) and was used in analyses.

ODD symptoms ODD symptoms were measured using the Disruptive Behaviour Disorders Rating Scale (DBD-RS) [49], a 45-item parent- and teacher-report measure of the presence and severity of DSM-III-R ADHD symptoms (9 inattentive symptom items and 9 hyperactivity/impulsivity symptom items), ODD (8 items), and CD symptoms (15 items). Parents and teachers rate items on a four-point scale ranging 0 (not at all) to 3 (very much), with higher scores indicating more severe symptoms. In the current study, the parent-report form was used and the ODD items were of interest. Because items reflect DSM-III-R symptom wording, those were modified to match DSM-5 symptom wording [50]. Prior findings indicate both the original (e.g., factor structure and internal consistency [49, 51–53] and the Hungarian translation (internal consistency) [41] have acceptable psychometric properties. In the current sample, the ODD subscale exhibited acceptable internal consistency ($\omega = 0.915$) and was used in analyses.

Analytic plan

All analyses were conducted in RStudio (version 2023.09.1. Build 494, R version 4.3.2.).

Comparisons of groups

Across models, the dependent variable was residualized Total DERS, Δ Total DERS, Total DERS-P, and Δ Total DERS-P, with the effects of depression and ODD regressed out. Independent variable was medication status. In case of concurrent models, data were analyzed from adolescents with available data for baseline and in case of prospective models, data were analyzed from adolescents with available data for baseline and 18-month follow-up.

Comparisons involved differences across (I) adolescents without ADHD, medication-naïve adolescents with ADHD, and ever-medicated (currently or previously) adolescents with ADHD and, where a normalization effect was apparent, (II) adolescents without ADHD, medication-naïve adolescents with ADHD, currently medicated adolescents with ADHD, and previously but not currently medicated adolescents with ADHD. A normalization effect was defined as ever-medicated adolescents with ADHD differing from medication-naïve adolescents with ADHD (lower ED) but not from adolescents without ADHD, and medication-naïve adolescents with ADHD differing from adolescents without ADHD (higher ED).

Across models, distribution of ED scores at each level of the medication status variables was checked using normality tests (Anderson-Darling, Lilliefors-corrected Kolmogorov-Smirnov tests using the nortest package (v1.0-4) [54]) as well as visual inspection of diagnostic plots (histograms, density and Q-Q plots). Equality of variances of ED scores at each level of the medication status variables was checked using Bartlett's test. If assumptions were met, one-way ANOVAs were conducted to determine whether any of the medication groups differ with regard to residualized Total DERS, Δ Total DERS, Total DERS-P, and Δ Total DERS-P scores. In case of a significant omnibus test, follow-up pairwise t-tests were conducted and effect sizes (Cohen's *D*) were calculated, to determine which of the medication groups differ from each other with regard to residualized Total DERS, Δ Total DERS, Total DERS-P, and Δ Total DERS-P scores. If assumptions were violated, nonparametric alternatives to the ANOVA, t-test, and Cohen's *D* were conducted [55]; Kruskal-Wallis H tests (using the dplyr package (v1.1.4) [56]) with follow-up pairwise Wilcoxon rank-sum tests (using the dplyr package (v1.1.4) [56]), and calculation of effect size (Wilcoxon *r*) (using the rstatix package (v0.7.2) [57]).

Attrition

To determine whether attrition was at random, binary logistic regression analyses were conducted with age, sex, ADHD status, cognitive ability, socioeconomic status, baseline parent- and self-reported ED, and baseline medication scores (both the three and the four-level variable) as independent variables entered simultaneously and whether an adolescent had 18-month follow-up data as the dependent variable.

Availability of data and materials

The datasets analyzed and/or used during the current study are available from the corresponding author on reasonable request.

Results

For descriptive statistics across subsamples, see Table 1. European Medicines Agency and/ or a National Institute of Pharmacy and Nutrition (OGYÉI) approved ADHD drugs were Bitinex, Ritalin, and Strattera.

To compare groups on baseline parent-reported ADHD severity, Kruskal-Wallis H tests were conducted, with follow-up pairwise Wilcoxon signed-rank tests.

A Kruskal-Wallis H test showed a difference between ever-medicated, medication-naïve, and control groups in parent-reported ADHD severity, $\chi 2(2) = 200.46$, $p < .001$. Follow-up pairwise Wilcoxon signed-rank tests indicated adolescents without ADHD differed from ever-medicated adolescents with ADHD ($p < .001$) and from medication-naïve adolescents with ADHD ($p < .001$) but ever-medicated adolescents with ADHD did not differ from medication-naïve adolescents with ADHD ($p = .014$).

A Kruskal-Wallis H test showed a difference between currently medicated, previously but not currently medicated, medication-naïve, and control groups in parent-reported ADHD severity, $\chi 2(3) = 179.67$, $p < .001$. Follow-up pairwise Wilcoxon signed-rank test indicated adolescents without ADHD differed from currently medicated ($p < .001$), previously but not currently medicated ($p < .001$), and medication-naïve ($p < .001$) adolescents with ADHD but none of the ADHD groups differed from each other ($ps > 0.21$).

Attrition

The model for attrition analysis was nonsignificant: $\chi^2(10) = 6.315$, $p = .788$.

Concurrent ED

Self-reported ED

A Kruskal-Wallis H test showed a difference between ever-medicated, medication-naïve, and control groups in self-reported ED, $\chi 2(2) = 7.885$, $p = .019$. Follow-up pairwise Wilcoxon signed-rank tests indicated ever-medicated adolescents with ADHD differed from medication-naïve adolescents with ADHD ($p = .037$) but not from adolescents without ADHD ($p = .942$) and that medication-naïve adolescents with ADHD differed from adolescents without ADHD ($p = .005$). Ever-medicated adolescents with ADHD exhibited lower ED than medication-naïve adolescents with ADHD and medication-naïve adolescents with ADHD exhibited greater ED than adolescents without ADHD (Table 2; Fig. 1).

A Kruskal-Wallis H test showed a difference between currently medicated, previously but not currently medicated, medication-naïve, and control groups in self-reported ED, $\chi 2(3) = 8.064$, $p = .044$. Follow-up pairwise Wilcoxon signed-rank test indicated the only between-groups difference was between medication-naïve adolescents with ADHD and adolescents without ADHD

Table 1 Descriptive statistics across adolescents without ADHD, currently medicated, previously but not currently medicated, medication-naïve medication-naïve adolescents with ADHD

	Without ADHD (*n* = 165)	Previously but not currently medicated (*n* = 21)	Currently medicated (*n* = 19)	Ever medicated (*n* = 58)	Medication naïve (*n* = 46)
Age at baseline					
Mean (SD)	15.884 (1.035)	15.972 (1.158)	15.333 (1.071)	15.693 (1.101)	15.596 (1.090)
min-max	14.091–18.073	14.015–17.939	13.768–18.019	13.768–18.019	13.998–17.698
IQ at baseline					
Mean (SD)	64.165 (20.013)	48.595 (23.550)	53.474 (23.321)	54.810 (22.490)	55.012 (22.146)
min-max	15.500–99.650	17.500–96.500	11.500–94.500	11.500–96.500	8.500–94.500
Sex					
%female	49.09	14.29	26.32	20.69	28.26
ADHD severity* at baseline					
Mean (SD)	0.812 (1.172)	11.857 (2.920)	12.579 (2.950)	10.569 (3.565)	11.739 (3.409)
min-max	0.000–5.000	7.000–18.000	5.000–18.000	5.000–18.000	5.000–18.000
Depressive problems at baseline					
Mean (SD)	52.618 (5.012)	54.913 (5.815)	52.618 (5.012)	54.500 (7.467)	54.913 (5.815)
min-max	50.000–86.000	50.000–75.000	50.000–82.000	50.000–82.000	50.000–70.000
ODD severity* at baseline					
Mean (SD)	0.776 (1.508)	4.714 (2.390)	3.737 (2.281)	3.483 (2.487)	3.696 (2.289)
min-max	0.000–8.000	1.000–8.000	0.000–8.000	0.000–8.000	0.000–7.000
DERS total at baseline					
Mean (SD)	75.721 (18.554)	81.286 (22.782)	82.579 (24.923)	80.776 (23.355)	89.630 (21.965)
min-max	42.000–130.000	46.000–123.000	44.000–149.000	41.000–149.000	42.000–127.000
DERS-P total at baseline					
Mean (SD)	58.618 (20.839)	88.048 (19.153)	86.421 (13.418)	84.724 (17.332)	85.957 (17.464)
min-max	29.000–126.000	44.000–120.000	56.000–111.000	44.000–120.000	41.000–118.000
DERS total at 18-month follow-up					
Mean (SD)	81.724 (21.334)	78.353 (22.647)	86.643 (25.626)	81.636 (23.511)	90.781 (22.610)
min-max	41.000–146.000	46.000–115.000	45.000–127.000	42.000–128.000	59.000–135.000
DERS-P total at 18-month follow-up					
Mean (SD)	56.812 (19.807)	82.833 (17.198)	84.556 (10.584)	83.840 (14.141)	78.684 (16.885)
min-max	29.000–111.000	57.000–109.000	68.000–101.000	57.000–109.000	36.000–106.000

Notes ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder, DERS = difficulties in emotion regulation scale; DERS-P = difficulties in emotion regulation scale-parent report; *=number of symptoms

($p = .005$) (none of the other between-groups differences were significant, all $ps > 0.103$). Medication-naïve adolescents with ADHD exhibited greater ED than adolescents without ADHD (Table 2; Fig. 2).

Parent-reported ED

A Kruskal-Wallis H test showed a difference between ever-medicated, medication-naïve, and control groups in parent-reported ED, $\chi 2(2) = 18.497$, $p < .001$. Follow-up pairwise Wilcoxon signed-rank test indicated both ever-medicated ($p < .001$) and medication-naïve ($p = .001$) adolescents with ADHD differed from adolescents without ADHD but ever-medicated and medication-naïve adolescents with ADHD did not differ ($p = .880$). Ever-medicated and medication-naïve adolescents with ADHD both exhibited greater ED than adolescents without ADHD (Table 2; Fig. 3).

Prospective ED

Self-reported ED

A Kruskal-Wallis H test showed no difference between ever-medicated, medication-naïve, and control groups in self-reported ΔED, $\chi 2(2) = 1.994$, $p = .369$.

Of adolescents with 18-month follow-up self-reported DERS data, 9 changed medication status from baseline to 18-month follow-up, 6 discontinued and 3 newly started. Analyses were repeated without these 9 participants and findings were replicated, a Kruskal-Wallis H test showed no difference between ever-medicated, medication-naïve, and control groups in self-reported ΔED, $\chi 2(2) = 3.038$, $p = 0.218$.

Parent-reported ED

A one-way ANOVA showed a difference between ever-medicated, medication-naïve, and control groups in parent-reported ΔED $F(1, 201) = 5.811$, $p = .016$.

Table 2 Descriptive statistics and effect sizes for analytic groups and models

self-reported ED					
group1	group2	effsize[1]	*n*1	*n*2	magnitude
ever med	med naïve	0,204	58	46	small
ever med	control	0,00499	58	165	small
med naïve	control	0,194	46	165	small
group	count	*M*	*SD*	median	IQR
ever med	58	-1,49	24,2	-4,15	37,1
med naïve	46	6,86	20,8	5,39	26
control	165	-1,69	18,1	-5,44	21
self-reported ED					
group1	group2	effsize[1]	*n*1	*n*2	magnitude
control	past med	0,0265	165	21	small
control	curr med	0,00855	165	19	small
control	med naïve	0,194	165	46	small
past med	curr med	0,0321	21	19	small
past med	med naïve	0,2	21	46	small
curr med	med naïve	0,189	19	46	small
group	count	*M*	*SD*	median	IQR
control	165	-1,69	18,1	-5,44	21
past med	21	-3,28	24,6	-6,93	42,6
curr med	19	-0,345	25,9	-3,77	29,9
med naïve	46	6,86	20,8	5,39	26
parent-reported ED					
group1	group2	effsize[1]	*n*1	*n*2	magnitude
ever med	med naïve	0,0151	58	46	small
ever med	control	0,237	58	165	small
med naïve	control	0,221	46	165	small
group	count	*M*	*SD*	median	IQR
ever med	58	4,94	15,8	4,22	20,9
med naïve	46	4,64	15,2	5,18	19,3
control	165	-3,95	17,8	-4,23	27,7
parent-reported ΔED					
group1	group2	effsize	*n*1	*n*2	magnitude
ever med	med naïve	-0,27	44	32	small
ever med	control	-0,428	44	127	small
med naïve	control	-0,145	32	127	negligible
group	count	*M*	*SD*	median	IQR
ever med	44	-6,98	26	-4,57	36,8
med naïve	32	0,142	27	1,26	39
control	127	3,84	25	0,445	30,2

Notes ED = emotion dysregulation; [1] = Wilcoxon effect size (*r*), where 0.10 - < 0.3 (small effect), 0.30 - < 0.5 (moderate effect) and >= 0.5 (large effect). [2] = Cohen's D. IQR = interquartile range

Follow-up pairwise t-tests indicated the only between-groups difference was ever-medicated adolescents with ADHD and adolescents without ADHD ($p = .016$) (none of the other between-groups differences were significant, all $ps > 0.231$). Ever-medicated adolescents with ADHD exhibited a greater change – a reduction – in parent-reported ED over time than adolescents without ADHD, who exhibited no changes in parent-reported ED over time (Table 2; Fig. 4).

Of adolescents with 18-month follow-up parent-reported DERS data, 6 changed medication status from baseline to 18-month follow-up, all discontinued. Analyses were repeated without these 6 participants and findings were replicated, a one-way ANOVA showed a difference between ever-medicated, medication-naïve, and control groups in parent-reported ΔED $F(1, 195) = 6.385$, $p = .012$.

Follow-up pairwise t-tests indicated the only between-groups difference was ever-medicated adolescents with ADHD and adolescents without ADHD ($p = .008$) (none of the other between-groups differences were significant, all $ps > 0.085$). Ever-medicated adolescents with ADHD exhibited a greater change – a reduction – in parent-reported ED over time than adolescents without ADHD, who exhibited no changes in parent-reported ED over time (Fig. 5).

A one-way ANOVA showed no difference between currently medicated, previously but not currently medicated, medication-naïve, and control groups in parent-reported ΔED ($p = .302$).

Discussion

Aims in the current study were to examine whether accounting for depression and ODD as relevant confounds, ever medicated adolescents with ADHD, never medicated adolescents with ADHD, and adolescents without ADHD differ with regard to concurrent ED and changes in ED over time. To our knowledge, this is the first longitudinal study where the effects of current, no, and past ADHD pharmacotherapy is examined on ADHD-associated ED in adolescents.

Generally, findings indicated effects of ADHD pharmacotherapy on concurrent and prospective measures of ED, consistent with earlier results indicate beneficial concurrent effects in children [17–21], adolescents [25, 26] and adults [16] with ADHD. Further, our results are also consistent with earlier work focused on neural emotion processing [28] suggesting that the beneficial effects of ADHD pharmacotherapy on ED are apparent even after discontinuation of medication.

Specific findings differed depending on informant (parent- or self-report) and measurement time (concurrent or prospective). In case of parent-reported ED, for concurrent measurement, there was no normalizing effect of ADHD pharmacotherapy. Both ever and never medicated adolescents with ADHD exhibited greater ED than adolescents without ADHD. For prospective measurement, there was a boosting effect of ADHD pharmacotherapy as ever medicated adolescents with ADHD exhibited a greater change – a decline in ED over time relative to never medicated adolescents with ADHD and adolescents without ADHD who exhibited no change in ED over time. This pattern of results held when analyses

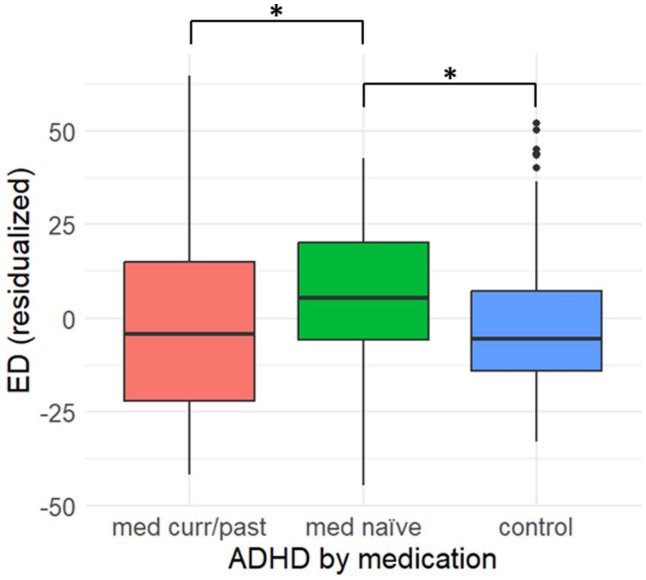

Fig. 1 Differences between ever-medicated adolescents with ADHD, medication-naïve adolescents with ADHD, and adolescents without ADHD on self-reported ED, accounting for depression and ODD symptoms

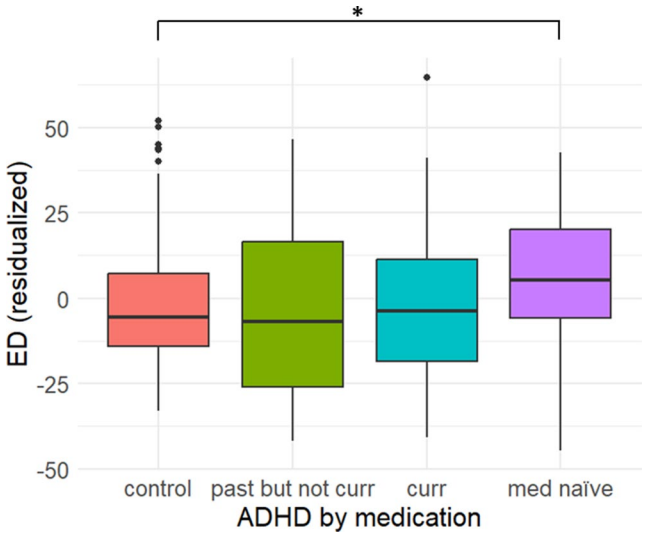

Fig. 2 Differences between currently medicated, previously but not currently medicated, medication-naïve, and control groups in self-reported ED, accounting for depression and ODD symptoms

focused on adolescents who did not change medication status between baseline and follow-up. In case of self-reported ED, for concurrent measurement, there was a normalizing effect of ADHD pharmacotherapy as ever-medicated adolescents with ADHD exhibited lower ED than medication-naïve adolescents with ADHD who exhibited greater ED than adolescents without ADHD. Further, findings indicated this normalizing effect was comparable for currently and for previously but not currently medicated adolescents. When comparing currently medicated, previously but not currently medicated, medication-naïve, and control groups, only never medicated adolescents differed from adolescents without ADHD but neither currently nor previously but not currently medicated adolescents differed from adolescents without ADHD. For prospective measurement, there was no boosting effect of ADHD pharmacotherapy, as ever medicated, never medicated and without ADHD groups did not differ in changes in ED over time.

The current design did not allow for exploration of the mechanisms of the apparent beneficial effects. It has been hypothesized that such effects may reflect a direct effect of ADHD pharmacotherapy on the dopamine system or an indirect effect of such therapy on emotional development [28]. As noted, adolescence is a critical developmental period for emergence of adaptive emotion

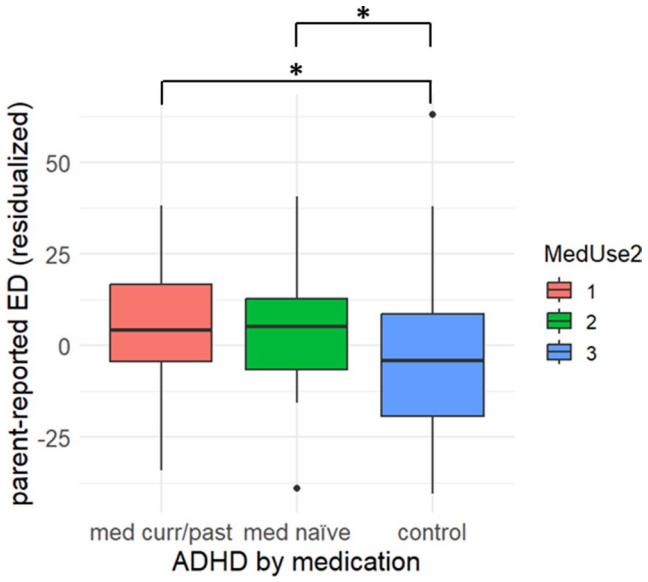

Fig. 3 Differences between ever-medicated adolescents with ADHD, medication-naïve adolescents with ADHD, and adolescents without ADHD on parent-reported ED, accounting for depression and ODD symptoms

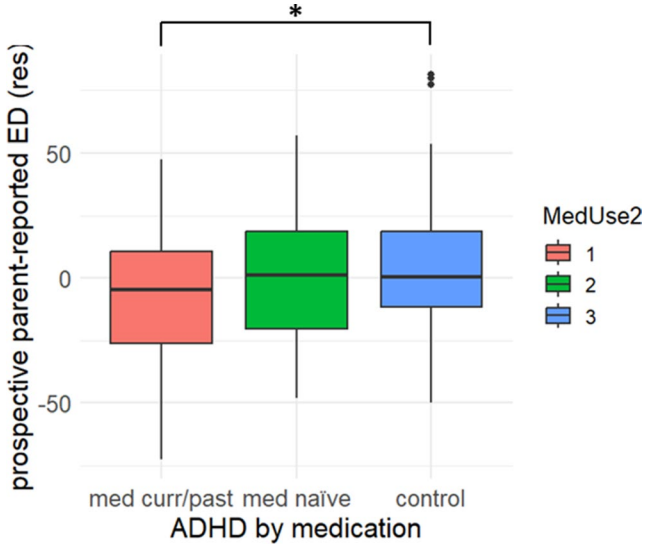

Fig. 4 Differences between ever-medicated adolescents with ADHD, medication-naïve adolescents with ADHD, and adolescents without ADHD on parent-reported ΔED across 18 months, accounting for depression and ODD symptoms

regulation skills [22]. ADHD pharmacotherapy may improve academic and social functioning and, given bidirectional relations between emotion regulation and social functioning [58] thereby facilitate age-appropriate emotional development and learning processes [28].

Clinical implications

Findings indicating that ED exacerbates [8] or explains [6, 9] negative behavioral and functional outcomes of ADHD would suggest that targeting ADHD symptoms may not be sufficient to ameliorate such outcomes and that ED also has to be potentially targeted to attenuate those. This depends, however, on whether treatments targeting ADHD symptoms also improve ED or additional, adjunct treatments are needed for such improvement. The current findings suggest that ADHD pharmacotherapy may be a parsimonious and sufficient approach to ameliorating not only ADHD symptoms but also ED. Next steps in this line of research are to determine the extent to which changes in ED are a mechanism of response to treatment (that is nonredundant with ADHD symptoms) as indexed by functional outcomes as well as the extent to findings generalize to psychotherapy.

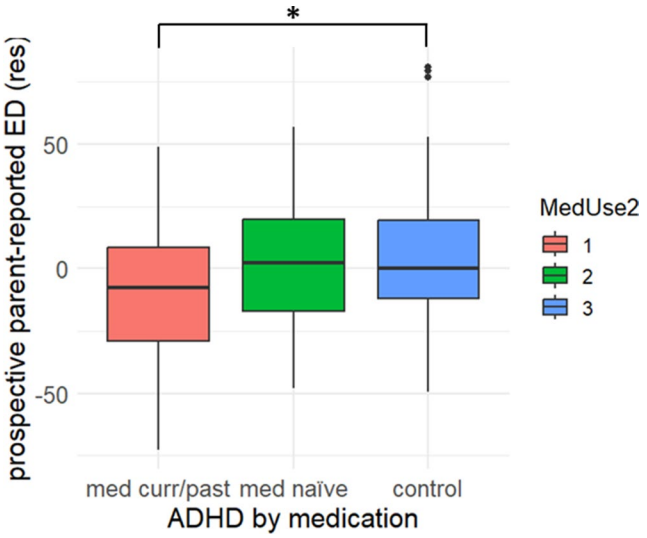

Fig. 5 Differences between ever-medicated adolescents with ADHD, medication-naïve adolescents with ADHD, and adolescents without ADHD on parent-reported ΔED across 18 months, accounting for depression and ODD symptoms, in a subsample of adolescents who did not change medication status from baseline to follow-up

Directions for future research and limitations

We note additional directions for future research as well as limitations of the current study. Although nuanced comparison of currently, previously but not currently, and never mediated groups and a without ADHD group is an advantage of the current research, allocation to groups was not random. We were able to rule out confounding-by-indication for ADHD severity as a *measured* baseline variable and to account for depression and ODD effects, but we were not able to rule out confounding-by-indication for *nonmeasured* differences. Future studies in this line of research may account for or examine the effects of such variables. Confounding may also have occurred during the study phase; e.g. behavioral treatment may have been more common in one group compared to the other. Both self-reported and parental evaluation of ED is subject to potential biases, given the open nature of the assessment. Blinded longitudinal studies are necessary to account for this potential effect.

Arguably, the reason for greater improvement in parent-reported ED over time or the greater benefits for concurrent self-reported ED for the ever medicated group of adolescents with ADHD could be explained by a ceiling effect such that those who are medicated are also more severe and as such, have greater room for improvement (by medication) than those who are not medicated and less severe. However, ever and never medicated groups did not differ on ADHD severity, indicating a ceiling effect cannot account for the herein observed results. Related, heterogeneity in ED [2] can also cause ceiling effects in findings. Some adolescents with ADHD may not had elevated have ED. Findings can benefit from replication in adolescents with ADHD and assessed moderate to high levels of ED at baseline.

We detected differential effects of ADHD pharmacotherapy across ever medicated, never medicated and control groups on longitudinal measures of parent-reported ED but not between currently medicated, previously but not currently medicated, never medicated, and control groups, likely because of insufficient power. In larger samples, the same pattern (of no difference between currently medicated and previously but not currently medicated groups) as for self-reported concurrent ED may also be apparent for parent-reported changes in ED over time.

Due to lack of information from participants or to lack of power, we could not assess the effects of medication dosage, medication type (nonstimulant vs. stimulant or long- or short-acting), length of treatment, or the effects of non-ADHD pharmacotherapy (e.g. antidepressants). Such effects may be observable, e.g. there may be differential effects of nonstimulants and stimulants across types on ED, given the distinct mechanisms of action and pharmacological properties [15]. In larger, subsequent samples, the herein assessed questions may also be examined with regard to these variables. Further, not all parents who indicated their child took ADHD medication previously but not currently stated discontinuation length and among those who did indicate discontinuation length, there was variability. In larger, subsequent samples the herein assessed differences may also be examined for effects of discontinuation length. Beyond depression and ODD, additional comorbidities are also relevant to the association between ADHD and ED (e.g. anxiety, callous-unemotional traits, or substance use) [3]

and may be examined in the future. Finally, given limited diversity of the sample, study findings need to be replicated in a more diverse sample for greater confidence in generalizability.

Conclusions

Taken together, the current findings indicate that in middle-late adolescents with ADHD, independent of depression and ODD, ADHD pharmacotherapy has beneficial – normalizing – effects on *concurrent, self-reported* ED and beneficial – boosting – effects on *prospective, parent-reported* ED. In case of effects on concurrent self-reported ED, these results are consistent with those observed with adults with ADHD [28] insofar as they indicate that pharmacotherapy effects may be apparent even after long-term discontinuation.

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Author contributions
KK: Conceptualization, Methodology, Software, Formal analysis, Data Curation, Writing - Original Draft. MT: Investigation, Data Curation, Writing - Review & Editing, Project administration. EP: Investigation, Data Curation, Writing - Review & Editing, Project administration. CSz: Investigation, Writing - Review & Editing, Project administration. TW: Software, Data Curation, Writing - Review & Editing, Project administration. JR: Writing - Review & Editing, Supervision. NB: Conceptualization, Methodology, Software, Validation, Formal analysis, Data Curation, Writing - Original Draft, Visualization, Supervision, Project administration, Funding acquisition.

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Data availability
The datasets analyzed and/or used during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This research was approved by the National Institute of Pharmacy and Nutrition (OGYÉI/17089-8/2019) and has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Parents and participants provided written informed consent (and assent) to participate.

Consent for publication
N/A.

Competing interests
The authors declare no competing interests.

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