Original article

Proper Emotion Recognition, Dysfunctional Emotion Regulation

The Mystery of Affective Dysregulation in Adolescent Psychiatric Inpatients

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Abstract: *Objective:* A considerable number of adolescents exhibit severe self-regulation deficits in affect and behavior, which are referred to as affective dysregulation (AD). AD may be conceptualized as a dimensional trait that, in its extreme form, resembles the diagnostic categories of severe mood dysregulation (SMD) or disruptive mood dysregulation disorder (DMDD). Assuming a shared pathway of psychopathology in AD and SMD, similar underlying dysfunctional mechanisms in emotion processing, particularly emotion recognition (RECOG) and regulation (REGUL), may be postulated. *Method:* Adolescent inpatients with AD (CAD, *N* = 35), without AD (CCG, *N* = 28), and nonclinical controls (NCG; *N* = 28) were administered a morphed facial recognition task (RECOG). REGUL abilities, levels of irritability as well as depressive symptoms were also assessed. *Results:* We found no significant group differences in accuracy and thresholds for RECOG abilities. Patients with AD reported more dysfunctional REGUL strategies than did CCG and NCG. Both depression and AD, but not irritability, influenced the overall degree of maladaptive REGUL. *Conclusion:* The broad phenotype of AD does not involve the deficits in RECOG reported for adolescents with a narrow phenotype (SMD); regarding REGUL strategies, AD seems to be associated with specific impairments.

Zusammenfassung: *Fragestellung:* Eine nicht unerhebliche Zahl Jugendlicher berichtet Symptome affektiver und behavioraler Dysregulation (AD). AD kann als dimensionaler Trait konzeptualisiert werden und ähnelt in seiner extremsten Ausprägung den diagnostischen Kategorien der Schweren Störung der Stimmungsregulation (SMD) oder der Disruptiven Affektregulationsstörung (DMDD). Davon ausgehend, dass AD und SMD dieselben psychopathologischen Entwicklungspfade aufweisen, nehmen wir an, dass auch bei AD zugrundeliegende dysfunktionale Mechanismen hinsichtlich der Prozessierung emotionaler Reize, vor allem der Emotionserkennung (RECOG) und -regulation (REGUL) vorhanden sind. *Methodik:* Adoleszente stationäre Patienten mit AD (CAD, *N* = 35), ohne AD (CCG, *N* = 28), und nicht-klinische Kontrollen (NCG; *N* = 28) führten einen gemorphten Emotionserkennungstest (RECOG) durch. REGUL Fähigkeiten, Ausmaß von Irritabilität als auch depressive Symptome wurden zusätzlich erfasst. *Ergebnisse:* Wir fanden keine Unterschiede hinsichtlich Erkennensgenauigkeit und Intensitätsausmaß (Schwelle) zwischen den Gruppen. Patienten mit AD berichteten mehr dysfunktionale REGUL Strategien im Vergleich zu CCG und NCG. Das Ausmaß von maladaptiver REGUL wurde sowohl von Depressivität und AD vorhergesagt, aber nicht durch das Ausmaß von Irritabilität beeinflusst. *Schlussfolgerung:* Der breite Phänotyp AD scheint nicht mit Defiziten in RECOG einherzugehen wie sie für Jugendliche mit dem engen Phänotyp (SMD) berichtet wurden; hinsichtlich der REGUL Strategien scheint AD mit spezifischen Beeinträchtigungen assoziiert zu sein.

Introduction

A considerable number of children and adolescents report symptoms such as chronic irritability, aggressive outbursts, hyperactivity, and mood swings (Leibenluft, 2011). These symptoms are difficult to capture within the existing diagnostic categories and are mostly categorized as either attention deficit hyperactivity disorder (ADHD) with an added mood disorder or as pediatric bipolar disorder (PBD, Grimmer, Hohmann, Banaschewski, & Holtmann, 2010). Recently, various efforts have been made to better characterize children with the described symptoms of affective dysregulation (AD) and to establish distinct diagnostic criteria.

Leibenluft and colleagues (Leibenluft, Charney, Towbin, Bhangoo, & Pine, 2003) suggested the label severe mood dysregulation (SMD) to identify children with *chronic* irritability and severe behavioral outbursts, as opposed to *episodic* irritability and mood swings as found in pediatric bipolar disorder (PBD). Only recently, the Dia-

gnostic and Statistical Manual of Mental Disorders in its fifth revision (DSM-5; American Psychiatric Association, 2013) established a diagnosis largely equivalent to SMD, i.e., namely, disruptive mood dysregulation disorder (DMDD). While a diagnosis of SMD requires symptoms of hyperarousal, DMDD does not, the rationale being that clinicians can also assign a diagnosis of comorbid ADHD if warranted.

To date, most researchers have used questionnaires such as the Child Behavior Checklist (CBCL; Achenbach, 1991) or the Strengths and Difficulties Questionnaire (SDQ; Goodman, Meltzer, & Bailey, 1998) to capture a phenotype of AD that is probably broader than the categorical diagnoses of SMD and DMDD. It has been hypothesized that the questionnaire-based phenotype of AD may be more of a dimensional trait (Shaw, Stringaris, Nigg, & Leibenluft, 2014), which is not unique to a specific diagnosis (e.g., Ayer et al., 2009) and which is observed in 6-7% of children in psychiatric clinical samples (Holtmann, Goth, Wöckel, Poustka, & Bölte, 2008). However, in its extreme form, AD may correspond to the diagnostic categories of SMD or DMDD.

The AD profiles measured by the CBCL or SDQ show high intercorrelations (Holtmann, Becker, Banaschewski, Rothenberger, & Roessner, 2011) and have proved to be associated with psychosocial impairments (Holtmann et al., 2008; Juksch et al., 2011; Legenbauer, Heiler, Holtmann, Fricke-Oerkermann &, Lehmkuhl, 2012) as well as genetic (Hudziak, Althoff, Derks, Faraone, & Boomsma, 2005) and biological correlates (e.g., Holtmann et al., 2013). By contrast, less is known about the possible mechanisms underlying AD, such as emotion regulation (REGUL). Given that REGUL is related to social functioning (Eisenberg et al., 2009) and the quality of social interaction (Lopes, Salovey, Côté, Beers, & Petty, 2005), disturbances in the regulation of emotions may explain some of the core symptoms of AD.

REGUL refers to the ability to modulate an emotional state in a way that facilitates adaptive and purposeful behaviors, and comprises complex processes, e.g., "... to select, attend to and appraise emotionally arousing stimuli and to do so flexibly" (see Shaw et al., 2014, p. 1). A basic, neuropsychological process that affects REGUL is the rapid and accurate recognition of emotions (RECOG) in human faces (Shaw et al., 2014). Misperception of emotional facial expressions may lead to aberrant emotional responses and hence aggravate dysfunctional REGUL and problems in social interaction. Recently, several studies investigated the link between RECOG, REGUL, and SMD. Initial evidence indicates worse RECOG and higher thresholds for the identification of the correct emotion in patients fulfilling criteria for SMD compared to nonclinical controls, in particular for a specific subset of emotions (surprise, anxiety, happiness, and disgust; Rich et al., 2008). Furthermore, these studies also showed that both patients with SMD and PBD performed worse than nonclinical controls (Guyer et al., 2007; Kim et al., 2013; Rich et al., 2008). Similar RECOG deficits were reported in patients meeting criteria for ADHD (Shaw et al., 2014; Yuill & Lyon, 2007). Evidence regarding disturbances in REGUL is scarce. A recent study showed deficits in RECOG and REGUL among patients with ADHD, with parents reporting that their children had difficulties in regulating specific emotions such as anger, fear, sadness, or happiness/exuberance. Interestingly, deficits in RECOG and REGUL did not necessarily occur together (Sjöwall, Roth, Lindquist, & Thorell, 2013).

In sum, there is evidence for specific deficits in RECOG and REGUL processing in the categorical disorders SMD, PBD, and ADHD, indicating shared psychopathological deficits between the disorders. Furthermore, these disorders show a symptom overlap with the broader AD phenotype assessed by questionnaires. Hence, these RECOG and REGUL deficits may be associated with comorbid symptoms of AD as assessed by the questionnaire-based dysregulation profile, independent of the diagnostic categories. To our knowledge, no studies so far have investigated mechanisms of RECOG and REGUL in psychiatric patients who show symptoms of AD as assessed by the questionnaire-based broad dysregulation profile but who do not fulfil the more stringent diagnostic criteria for SMD or DMDD. The present study aims to gain a better understanding of deficits in RECOG and REGUL in patients with AD as identified via the Strengths and Difficulties Questionnaire – Dysregulation Profile (SDQ-DP) self-report.

Methods

Participants

In total, 91 adolescents (mean age = 13.98 years, *SD* = 1.31) were included in this study (27 males (29.7%), 64 females (70.3%). 87 participants reported being of Central European ethnicity, and three participants had two or more ethnic influences. The average intelligence quotient (IQ) was 104.24 (*SD* = 13.31), and the mean BMI percentile was 63.20 (*SD* = 26.79). Of the total sample, 63 adolescents were consecutively recruited inpatients at the LWL University Hospital Hamm (Germany). Of these, 35 showed a score of 5 or above on the SDQ-DP and were assigned to the clinical group with AD (CAD); 28 patients showed SDQ-DP scores from 0 to 4 and were assigned to the clini-

cal control group without AD (CCG). A nonclinical control group (NCG; $n = 28$) was recruited from volunteers at a local grammar school. Nonclinical controls also performed the SDQ and were included if they had an SDQ-DP of 4 or less. Details on patient flow and recruitment of the nonclinical control group are provided in the Appendix of the online-only version (Figure 1 and 2).

Procedures and Materials

The present study is part of a larger study on emotional dysregulation in AD. Participants and their primary caregivers were given extensive information on the study and provided verbal and written consent. The study was approved by the local medical ethics committee of the Ruhr University Bochum (Germany). After providing informed consent, the participants completed the questionnaire in a diagnostic session, with the exception of NCG questionnaires, which were sent to the participants' homes. All participants performed the Expressed emotion multimorph task (EEMT) as well as one other experimental task in a 90-minute session. The KSADs (schedule for affective disorders and schizophrenia for school-age children) interviews with parents were conducted separately from the experimental session.

Strengths and Difficulties Questionnaire (SDQ). The SDQ is a self-assessment inventory allowing the assessment of a dysregulation profile (SDQ-DP). It has shown high reliability in identifying patients with AD if a cutoff value of 5 or above is used (Holtmann et al., 2011).

Kiddie-Schedule for Affective Disorders and Schizophrenia for School-Age Children (KSADS) – SMD Module. The SMD module of this semistructured diagnostic interview assesses mood, tantrums, and irritability, and has shown a high interrater reliability of $k \geq 0.9$ (Leibenluft et al., 2003). The interview was used to classify participants as patients with SMD or without SMD. The prevalence of DMDD was examined using a checklist based on a German translation of DMS-5 criteria (Falkai & Wittchen, 2014). All interviewers in this study were graduates, and either Master- or doctoral-level clinicians.

Expressed emotion multimorph task (EEMT). This task, originally developed by Blair and colleagues (Blair, Colledge, Murray, L., & Mitchell, 2001), was modified by Rich and colleagues (2008) to measure the ability to label facial emotions and the required thresholds. It consists of short video sequences (duration = 3.6 seconds) that display 40 stages of emotional expression from 100% neutrality to 100% intensity (anger, anxiety, disgust, sadness, happiness, and surprise). More details regarding the test can be found in Rich et al. (2008). We assessed thresholds by counting the morphs until a correct answer was given, and measured accuracy by counting the percentage of correct answers given by stage 40. These indices were measured separately for each of the six emotions.

Beck Depression Inventory (BDI). The BDI is a well-established self-assessment inventory for the quantification of symptoms related to depression. A total score is computed, which provides an indication of the severity of the participant's depressive symptoms (e.g., 20–28: moderate depression; 29–63 severe depression; Hautzinger, Keller, & Kühner, 2009).

Affective Reactivity Index (ARI). The ARI quantifies the subject's threshold for anger, frequency of anger, and duration of anger in a self-assessment inventory comprising seven statements. A total score is computed, with higher values corresponding to higher irritability. A recent study showed high internal consistency, and indicated that patients with AD score high on this scale (Stringaris et al., 2012).

Questionnaire Assessing Emotion Regulation Strategies (FEEL-KJ). The FEEL-KJ is a multidimensional questionnaire for the assessment of REGUL strategies for anger, anxiety, and sadness in children and adolescents aged from 10 to 19 years (Grob, 2005). Both adaptive and maladaptive strategies are analyzed for these three emotions, and an overall adaptive and maladaptive score is computed. *T*-values above 40 for adaptive strategies and up to 60 for maladaptive strategies represent REGUL strategies within the normative range.

Results

Baseline Characteristics

We compared the groups regarding age, IQ , and BMI percentile using ANOVAs with Bonferroni correction, and regarding ethnicity, sex, and psychiatric comorbidities using Fisher's exact test. We found no significant group differences for age and IQ between the three groups. No difference was found between CAD and CCG with respect to quality and quantity of psychiatric comorbidity. Psychiatric comorbidity was not assessed in the NCG. However, the ANOVA revealed significant differences between the groups for BMI percentiles. Posthoc analyses showed that the NCG had a significantly lower BMI percentile than the CAD ($p = .018$) and the CCG ($p = .047$). As all means were within the normal range (25th to 90th BMI percentile), the BMI was not controlled for in the subsequent analyses.

To further describe the sample, depressive symptoms were quantified with the BDI, and the degree of chronic irritability was measured with the ARI. Posthoc ANOVA

with Bonferroni correction for the BDI sum score indicated a significant difference between the groups. Posthoc comparisons revealed that CAD scored significantly higher than CCG (p = .002) and NCG (p <.001). CCG participants scored significantly higher than NCG (p = .002). In addition, ANOVA with Bonferroni correction for the ARI sum score showed a significant group effect, with CAD and CCG scoring significantly higher than NCG, CAD: p < .001; CCG: p = .037, but no significant difference between the two clinical groups (p = .131). All means and test statistics for the reported variables are displayed in Table 1.

One patient in the CCG and three patients in the CAD fulfilled criteria for SMD when the KSADS-SMD module was applied. None of the participants fulfilled DSM-5 criteria for DMDD. The most frequently reported diagnoses in the two clinical groups were depression (F32/33), anxiety disorders (F93, F40/41), conduct disorders (F91), disorders of conduct and emotions (F92), hyperkinetic disorders (F90) and adjustment disorders (F43). Because of their low prevalence, somatoform disorders, eating disorders, and obsessive-compulsive disorder were summarized as "other disorders."

Emotion Recognition (EEMT)

First, an ANOVA with Bonferroni correction was conducted to test for performance differences between the three groups in terms of overall *accuracy*, i.e., the ability to identify the presented emotions correctly. All three groups performed similarly well regarding overall accuracy, $F(2, 88) = 0.686$, p = .506. To further investigate performance and to check for emotion-specific effects, a repeated-measures ANOVA with the percentage of correct responses for the six emotions was calculated. Again, no significant effect of Group could be identified, $F(2, 88) = 0.447$, p = .641, and there was no Emotion × Group interaction, $F(8, 344) = 0.941$, p = .481. However, results showed a significant main effect of Emotion, $F(4, 344) = 59.50$, p < .001, indicating that the number of correct responses differed depending on the kind of emotion. Posthoc analyses revealed that happiness was identified better than all other emotions (all ps <.001). Accuracy rates for surprise and anger did not differ significantly, but were worse than those for happiness (all ps <.001) and better than those for disgust, sadness, and anxiety (all ps <.001) The latter three were significantly less accurately labeled than surprise, anger, and happiness (all ps <.001).

Further analyses concerned *thresholds*, i.e., the mean number of stages needed to correctly identify the emotion. First, we performed an ANOVA for mean threshold for all emotions. No significant difference between the groups could be identified, $F(2, 88) = 0.036$, p = .964. Second, to investigate an emotion-specific effect, we applied repeated-measures ANOVA to examine differences regarding mean thresholds separately for all six emotions. Again, no significant difference emerged between the groups, $F(2, 83) = .066$, p = .936), and the interaction effect Threshold *

Table 1. Demographics and clinical characteristics of inpatients with AD (CAD), inpatients without AD (CCG), and nonclinical controls (NCG)

Characteristics	Clinical groups						
	CAD (n = 35)	CCG (n = 28)	NCG (n = 28)	*df*	F/Fischer	η²/Φ	*p*
Age $_b$	14.17 ± 1.34	14.14 ± 1.24)	13.57 ± 1.29	2, 88	2.00	.044	.141
Sex $_a$ (female:male)	26:9	15:13	23:5		5.61	.255	.053
BMI perc. $_b$	69.60 ± 25.98	67.8 ± 27.13	51.22 ± 20.33	2, 82	4.68	.102	.012
IQ $_b$	101.74 ± 12.88	102.52 ± 11.49	108.68 ± 14.68	2, 83	2.40	.055	.096
SMD diagnosis $_a$ (yes)	3	1	0		*2.49*	.322	.381
SDQ-DP $_b$	5.71 ± 1.02	2.79 ± 1.13	1.86 ± 1.21	2, 88	104.68	.704	<.001
BDI $_b$	30.06 ± 14.34	18.35 ± 15.41	5.93 ± 6.95	*2, 86*	27.41	.389	< .001
ARI $_b$	5.15 ± 3.89	3.52 ± 3.01	1.39 ± 1.75	*2, 86*	11.385	.209	< .001

Note. CAD = clinical patients with affective dysregulation; CCG = clinical control group; NCG = nonclinical control group. BMI= body mass index in kg/m², IQ = intelligence quotient based on clinical report and for CCG assessed with the Zahlen-Verbindungstest, SMD= severe mood dysregulation diagnoses based on KSADS Module, SDQ-DP = Strengths and Difficulties Questionnaire – Dysregulation Profile based on self-report, BDI = Beck Depression Inventory, ARI = Affective Reactivity Index, *df* = degrees of freedom, F = F-value, η² = effect size, Φ = Phi-value, p = level of probability. $_a$ Group comparisons were conducted by means of analyses of variance (ANOVAs) with Bonferroni correction; $_b$ Group comparisons were conducted by means of Fisher's exact test.

Group, $F(9, 360) = 0.904$, $p = .519$, failed to reach statistical significance. However, repeated-measures ANOVA showed a significant main effect of Emotion, $F(4, 360) = 88.812$, $p < .001$, indicating that thresholds varied depending on the kind of emotion. Posthoc analyses showed that the mean threshold for identifying happiness was significantly lower than for all other emotions (all $p < .001$). The mean threshold for surprise was significantly higher than for happiness ($p < .001$), but lower than for the other four emotions (all ps = .001). With the exception of anger and disgust ($p < .001$), there were no relevant differences among the emotions anger, anxiety, sadness, and disgust. Details are presented in Table 2.

Emotion Regulation

To investigate differences between the groups regarding REGUL strategies, ANOVAs were performed with the mean standardized scores (t-scores) on subscales of the FEEL-KJ. The results indicate a significant group effect insofar as NCG reported significantly better REGUL strategies compared to CCG and CAD. Moreover, CAD reported fewer adaptive strategies and more maladaptive strategies compared to CCG. In addition, we also determined the percentage of abnormal t-values for "below normal range" (<40) in adaptive REGUL and for "above normal range" (>60) in maladaptive REGUL. Approximately 56% of patients with AD showed t-values below the normal range for total adaptive REGUL, and 73.5% of AD reported t-values above the normal range for maladaptive REGUL. Details can be found in Table 3.

Influence of AD on Emotion Recognition and Emotion Regulation Deficits

We hypothesized that AD is of predictive value for the deficits in RECOG and REGUL. In order to examine this, we performed linear regression analyses with (a) accuracy and

Table 2. Primary and secondary outcome variables of EEMT

	Clinical groups		
	CAD	CCG	NCG
Characteristics	$n = 35$	$n = 28$	$n = 28$
Overall accuracy [a]	77.1%	77.4%	79.9%
Specific accuracies [b]			
Happiness	98.6%	98.2%	100%
Surprise	84.8%	84.5%	88.1%
Anger	84.3%	85.7%	88.3%
Anxiety	67.1%	66.7%	75.0%
Sadness	71.0%	66.7%	63.1%
Disgust	56.7%	62.5%	63.7%
Overall thresholds [a]	28.87± 5.12	29.13 ± 4.93	28.80 ± 3.94
Specific thresholds [b]			
Happiness	22.49 ± 7.98	22.82 ± 7.85	20.52 ± 6.77
Surprise	28.61 ± 6.68	28.42 ± 6.64	28.41 ± 5.09
Anger	31.17 ± 5.36	29.77 ± 5.12	31.43 ± 4.14
Anxiety	31.40 ± 6.31	32.70 ± 5.52	31.86 ± 4.83
Sadness	32.33 ± 6.16	31.84 ± 5.30	31.92 ± 4.98
Disgust	33.77 ± 5.41	33.30 ± 5.37	32.97 ± 6.40

Note. Accuracy is reported as the percentage of correctly identified emotions by stage 40. Thresholds are reported as the mean number of stages needed until a correct response is given. CAD = clinical patients with affective dysregulation; CCG = clinical control group; NCG = nonclinical control group.

Table 3. Differences in emotion regulation, depression and irritability levels between the groups

	Clinical groups						
	CAD	CCG	NCG				
	n = 35*	*n* = 28	*n* = 28**	*df*	*F*/Fisher	*p*	η²/Φ
Feel KJ Adaptive scores							
Total	37.29 ±11.24	41.54 ± 14.95	49.35 ± 11.43	2, 85	6.81	.002	.138
	(55.9%)	(50.0%)	(17.9%)		9.00	.010	.317
Anger	38.68 ±10.00	42.68 ± 13.18	48.62 ± 10.83	2, 85	5.67	.005	.118
	(47.1%)	(42.9%)	(10.7%)		9.78	.007	.323
Anxiety	42.06 ±12.38	42.00 ± 13.60	51.77 ± 10.54	2, 85	5.76	.005	.119
	(58.8%)	(42.9%)	(23.1%)		7.67	.022	.295
Sadness	37.38 ± 10.93	42.82 ± 13.76	47.23 ± 12.29	2, 85	4.82	.010	.102
	(61.8%)	(42.9%)	(34.6%)		4.67	.098	.232
Feel KJ Maladaptive scores							
Total	67.38 ± 10.34	55.54 ± 15.65	47.58 ± 13.06	2, 85	17.66	< .001	.294
	(73.5%)	(35.7%)	(15.4%)		21.86	< .001	.493
Anger	65.35 ± 11.10	55.07 ± 11.64	49.19 ± 12.63	2, 85	14.71	< .001	.257
	(64.7%)	(32.1%)	(15.4%)		15.97	< .001	.426
Anxiety	61.79 ± 12.57	54.11 ± 16.94	45.54 ± 10.47	2, 85	10.56	< .001	.199
	(52.9%)	(35.7%)	(3.8%)		18.13	< .001	.429
Sadness	66.32 ± 9.32	54.43 ± 14.20	57.41 ± 14.65	2, 85	14.73	< .001	.257
	(73.5%)	(35.7%)	(23.1%)		17.10	< .001	.440

Note. CAD = clinical patients with affective dysregulation; CCG = clinical control group; NCG = nonclinical control group; FEEL KJ = Questionnaire assessing emotion regulation strategies, *t*-values are presented; normal range for adaptive strategies $t \geq 40$, normal range for maladaptive strategies $t \leq 60$. Rates of abnormal values per group are reported as percentages in parentheses. df = degrees of freedom, *F* = *F*-value, η^2 = effect size, Φ = Phi-value, *p* = level of probability. [a] Group comparisons were conducted by means of analyses of variance (ANOVAs) with Bonferroni correction; [b] Group comparisons were conducted by means of Fisher's exact test.*For CAD 34 data sets were completed; ** For NCG 26 data sets were completed.

thresholds and (b) FEEL-KJ REGUL strategies as dependent variables, and the SDQ-DP score as independent predictor. We also entered BDI and ARI sum scores as independent variables, because the groups showed significant differences in relation to depression and irritability. The models for RECOG deficits were not statistically significant, and ARI, BDI, and SDQ-DP were not significant predictors. However, the degree of maladaptive REGUL strategies was significantly influenced by the SDQ-DP score (p = .028) and by the BDI sum score indicating the level of depressive symptoms (p <.001). The regression model was statistically significant, $F(3, 83) = 35.19$, p <.001, explaining about 55% of the variance. Regarding the prediction of adaptive strategies, only the degree of depressive symptoms as assessed with the BDI was of predictive value (p = .018), whereas the degree of AD assessed with the SDQ-DP failed to reach statistical significance as a predictor (p = .075). The total model explained 30.7% of the variance, $F(3, 83) = 13.26$, p <.001. Details are provided in Table 4.

Discussion

This study investigated RECOG and REGUL deficits in patients with AD. Because this phenotypic pattern includes symptoms similar to SMD, such as irritability, depressive symptoms, and tantrums, we expected to find similar deficits in patients with AD when comparing them to a clinical control group without symptoms of AD and nonclinical

Table 4. Results of linear regression analyses

Prediction of emotion recognition								
	Accuracy				Threshold			
Variable	*B*	*SE B*	β	*p*	*B*	*SE*	β	*p*
ARI	−.001	.004	−.042	.747	.241	.169	.183	.158
BDI	.000	.001	.070	.650	−.046	.042	−.164	.281
SDQ-DP score	−.003	.008	−.058	.707	−.149	.337	−.067	.660
Prediction of emotion regulation (FEEL-KJ, Subscales)								
	Adaptive strategies				Maladaptive strategies			
Variable	*B*	*SE B*	β	*p*	*B*	*SE*	β	*p*
ARI	−.495	.409	−.131	.229	.269	.382	.061	.483
BDI	−.247	.102	−.309	.018	.506	.096	.544	<.001
SDQ-DP score	−.1511	.838	−.23	.075	1.759	.784	.231	.028

Note. ARI = Affective reactivity index, BDI = Beck Depression Inventory, SDQ-DP = Strengths and Difficulties Questionnaire Dysregulation Profile sum score, *B* = unstandardized regression coefficient, *SE* = standard error; β = standardized regression coefficient; *p* = level of probability.

controls. The results indicated that patients with AD performed equally well compared to CCG and NCG in the facial emotion-labeling task, but reported worse REGUL strategies than both CCG and NCG. Moreover, maladaptive REGUL strategies were influenced by the degree of depressive symptoms and the degree of AD, but not by the degree of chronic irritability.

These results require further consideration: First, contradicting previous studies that showed deficits in overall RECOG as a specific feature of patients with SMD, PBD, and ADHD (e.g., Kim et al., 2013; Rich et al., 2008; Yuill & Lyon, 2007), the present findings indicate that deficits in RECOG are not a general characteristic of psychiatric patients (Guyer et al., 2007) or patients who meet the broad AD phenotype. There is evidence that deficits in RECOG have neural correlates, e.g., impaired or aberrant activity in the amygdala or the orbitofrontal cortex (Rich et al., 2008). These regions are also affected in patients with SMD, PBD, and ADHD, which might explain the corresponding deficits in RECOG in these distinct neurodevelopmental disorders. It may also explain the lack of these deficits in AD, if AD is considered as a dimensional trait with less intense symptoms and less impairment in terms of basic neuropsychological mechanisms. However, it is possible that patients with AD simply do not show the same neural correlates as those who meet the criteria for SMD; in this case, AD would have to be considered as a distinct entity that co-occurs with SMD but is not an integral part of it. Future studies should therefore include both patients reporting AD and patients fulfilling criteria for SMD, and if possible apply imaging techniques to further explain the postulated associations and differences.

Nevertheless, there are also some methodological issues that may explain the contradictory findings. First, our sample included various psychiatric diagnoses, though ADHD was less common in our sample compared to previous studies (e.g., Kim et al., 2013; Rich et al., 2008). Furthermore, we cannot rule out the possibility that emotion-specific effects remained undetected because of the mixed sample of patients and the naturalistic approach of the study. Moreover, the present sample differed from previous studies applying the same face-labeling task with regard to the sex distribution (e.g., Kim et al., 2013; Rich et al., 2008). Because males were reported to show slower response rates and less accuracy for RECOG (e.g., McClure, 2000), the higher portion of females among the present samples might have contributed to the differences between findings.

Although we were unable to demonstrate RECOG deficits in patients with AD, participants with AD reported more maladaptive REGUL strategies compared to clinical and nonclinical controls, and they showed severe impairments regarding core symptoms of AD such as irritability and depression. This has several implications: (a) It emphasizes the fact that facial RECOG and REGUL are two distinct features that may co-occur, but are not necessarily related (e.g., Cole, Martin, & Dennis, 2004). (b) It points to impairments within the REGUL process in patients with AD which might contribute to their specific symptomatology. In particular, maladaptive, but not adaptive regulation strategies, are specifically influenced by the level of AD.

Research focusing on REGUL strategies among ADHD, PBD, and SMD is still scarce. However, some studies indicate higher degrees of maladaptive strategies in patients meeting criteria for ADHD compared to nonclinical controls (e.g., Schmitt, Gold, & Rauch, 2012; Sjöwall et al., 2013). The recent study by Schmitt and colleagues (Schmitt et al., 2012) applied the same questionnaire as that used in the present study in a sample of 10–13-year-old outpatients with ADHD and showed negative associations between the level of adaptive strategies and the SDQ subscales *emotional problems* and *conduct problems* as well as the *total score*. Moreover, they failed to show differences between nonclinical controls and ADHD children regarding the level of maladaptive strategies. However, these contradictory findings might be attributable to age and diagnostic issues, and can thus be neglected with regard to our results. Future studies need to further explore REGUL abilities in a more objective way, e.g., by using more ecological or experimental paradigms in addition to self-reports. Thus, it would be of interest to further explore, within such a paradigm, how these patients modulate their emotional arousal.

Finally, the results show that the SDQ-DP validly identifies patients suffering from impairments who share core psychopathological symptoms with patients fulfilling criteria for SMD such as irritability and depressive symptoms. However, when we applied the KSADS – SMD module to assess SMD via parent report and interview, barely any of the participants fulfilled the diagnostic criteria. This supports the assumption that AD cannot be put on the same level as a diagnosis of SMD or DMDD. The question of whether it lies on the same continuum as the diagnostic categories of SMD or DMDD still needs clarification.

Limitations

The following additional limiting factors need to be mentioned: (a) Because our sample consists of inpatients at a child and adolescent psychiatric clinic, the study is naturalistic and observational in nature. Therefore, differences in IQ and age between nonclinical controls and the clinical sample exist. However, we tried to control for these differences in our analyses by stratifying for age. Nevertheless, sex remained an unsolved issue, with more female participants than in previous studies, thus potentially limiting the comparability of the results. Furthermore, because the nonclinical control group did not undergo a thorough clinical assessment, we cannot rule out the presence of any psychiatric problems within this group. (b) We used self-report rather than parent-report assessments to determine AD, which might also impact the generalizability of the present findings. However, there is evidence that parent-report and self-report in adolescents correspond only moderately, and that parents underestimate the symptom burden of their children. As a consequence, it is recommended to apply self-report when assessing clinical symptoms in children and adolescents (e.g., Arman, Amel, & Maracy, 2013; Van der Ende, Verhulst, Tiemeier, 2012). (c) Our study design did not control for acute medication or physical impairments. Only a few patients reported physical problems (e.g., sprains) at some time during treatment, and it is not possible to say whether this had an influence on the reported results. None of the patients reported any thyroid issues or other physical impairments that – to our knowledge – might interfere with attention. We determined that the inpatients and also nonclinical controls were not impaired by any acute physical illness (e.g., fever, headaches, etc.) while performing the experimental task. However, we cannot exclude the possibility that there might be any effect on the results reported.

Conclusion

Our results suggest that a broad phenotype of AD is captured with the common CBCL or SDQ DP which does not correspond very well with the distinct entities of SMD or DMDD. Future research needs to answer the question whether AD (a) is an integral feature of patients with various psychiatric disorders, in particular SMD, PBD, and ADHD, and may also be a diagnostic feature of these; or (b) is a distinct dimension that co-occurs with these disorders and explains some of the shared psychopathological deficits in REGUL, but also brings with it distinct impairments that are specific to AD. Our results emphasize the need to carefully describe samples and methods used to identify patients with SMD, DMDD, or AD in order to further disentangle the associations and differences among these disorders. Future research investigating AD needs to include patients with SMD in order to better understand similarities and differences between these patient groups.

Ethical Standards

The authors confirm that the study protocol was approved by the appropriate ethics committee. The study was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments. All persons gave their informed consent prior to inclusion in the study. Details of the procedure are given in the text.

Conflict of Interest

Conflicts of interest are reported by Martin Holtmann (advisory role for Lilly & Shire, public speaking for Bristol-Myers Squibb, Lilly, Medice, Neuroconn, Novartis, Shire), Tanja Legenbauer (travel expenses covered by Lilly), and Benjamin Pniewski (public speaking for Lilly & Novartis). Jan Hübner, Anna Ball, and Marlies Pinnow report no conflicts of interest.

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Electronic Supplementary Material

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ESM 1. Figure.
Patient flow for inpatient psychiatric juveniles

ESM 2. Figure.
Recruitment Procedure School

ESM 3. Table.
Diagnoses for clinical groups on day of hospital discharge

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