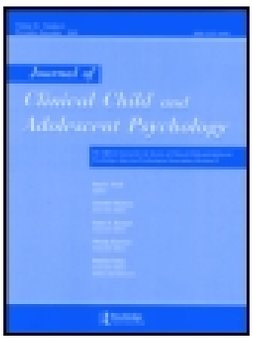

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Journal of Clinical Child & Adolescent Psychology

ISSN: 1537-4416 (Print) 1537-4424 (Online) Journal homepage: http://www.tandfonline.com/loi/hcap20

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To cite this article: Maria Kovacs, Nestor L. Lopez-Duran, Charles George, László Mayer, Idiko Baji, Enikö Kiss, Ágnes Vetró & Krisztina Kapornai (2017): The Development of Mood Repair Response Repertories: I. Age-Related Changes Among 7- to 14-Year-Old Depressed and Control Children and Adolescents, Journal of Clinical Child & Adolescent Psychology, DOI: 10.1080/15374416.2017.1399399

To link to this article: https://doi.org/10.1080/15374416.2017.1399399

Published online: 18 Dec 2017.

Journal of Clinical Child & Adolescent Psychology, 00(00), 1–10, 2017

ISSN: 1537-4416 print/1537-4424 online
DOI: https://doi.org/10.1080/15374416.2017.1399399

The Development of Mood Repair Response Repertories: I. Age-Related Changes Among 7- to 14-Year-Old Depressed and Control Children and Adolescents

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The purpose of this study was to test developmentally informed hypotheses about regulatory responses to sadness that attenuate versus exacerbate it (adaptive versus maladaptive mood repair responses, respectively) across late childhood, early adolescence, and mid-adolescence. In a multi-site study in Hungary, clinic-based, 7- to 14-year-olds with *Diagnostic and Statistical Manual of Mental Disorders*' (4th ed., text rev.) depressive disorders (N = 697; 55% male) and age/sex matched (at 1:2) nondepressed, school-based controls (N = 1,394) reported on their usual responses to sadness/dysphoria; parental reports were obtained separately. Adaptive and maladaptive response repertoire scores were compared across ages within and across subject groups, and by informant, controlling for confounds. Contrary to Hypothesis 1, older (vs. younger) youths in both groups reported fewer *adaptive* regulatory responses. *Maladaptive* response repertoires were unrelated to age among controls but significantly increased with age among depressed youths, particularly the girls. Partially supporting Hypothesis 2, subject groups differed in age-related trajectories of mood repair repertories, but not as expected (e.g., younger depressed children reported larger adaptive response repertoires than did controls). Parental reports revealed no developmental changes in offspring's mood repair repertories. Parent-offspring reports were most discordant for younger (vs. older) offspring, tended to converge around age 11, and were consistently and significantly larger in the depressed sample. Self-reported *adaptive* mood repair repertories appear to have been laid down by late childhood and then undergo "trimming" across ages 7–14 years. The extensive *maladaptive* mood repair response repertoires of depressed youths, which increased with age, distinguish them primarily from controls. Therefore, reducing maladaptive regulatory responses to sadness should be a priority when treating depressed youths.

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The key role of problematic emotion regulation in depressive disorders (e.g. Kovacs, Joormann, & Gotlib, 2008) has prompted considerable research on the responses that facilitate (or hinder) the regulatory process. There is now compelling evidence that responses to sadness/dysphoria that *maintain* or *exacerbate* it (e.g., ruminating about one's sadness, suppressing its overt expression) generally are functionally maladaptive and are associated with psychopathology, whereas responses that *attenuate* or downregulate dysphoria (e.g., neutral/positive reappraisal of the experience, refocusing attention on non-dysphoric matters) typically are adaptive and signal good functioning (Aldao, Nolen-Hoeksema, & Schweizer, 2010).

The process of attenuating or recovering from sadness has also been called *mood repair* (Josephson, Singer, & Salovey, 1996). Not surprisingly, depressed individuals report mood repair difficulties in daily life, which can linger even after the depression has remitted (Brockmeyer et al., 2012; Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008; Kovacs, Rottenberg, & George, 2009). Mood repair difficulties partly reflect reliance on certain responses to depressogenic triggers, such as rumination (Aldao et al., 2010), which maintain or exacerbate the experience of distress.

How does development affect the availability of mood repair responses and strategies? It is known that the types of responses and the extent of response repertoires are developmentally mediated. However, the evidence concerns mostly toddlers and very young children (e.g., Fox, 1994), with some data on adolescents (e.g., Morris, Silk, Steinberg, Myers, & Robinson, 2007), whereas the transition from late childhood to midadolescence has been generally neglected. Indeed, there is scant empirical information about the unfolding of mood repair repertoires during this age span, although late childhood marks key shifts in functioning (Del Giudice, 2014), and early to midadolescence is a "particularly critical developmental window" for affect regulatory processes (Silvers et al., 2012),

Based on developmental theory, and as a function of children's growing cognitive, social and executive function skills (Eisenberg & Zhou, 2000), the progression from late childhood to mid-adolescence among typical youths should be associated with *expanding repertoires* of adaptive regulatory responses and *attenuated repertoires* of maladaptive responses (e.g., Garnefski & Kraaij, 2006; Gullone, Hughes, King, & Tonge, 2010; Waters & Thompson, 2014). However, these expectations have inconsistent empirical support. For example, when kindergarteners to eighth graders (N = 275) were presented with sad scenarios and various response options (Garber, Braafladt, & Weiss, 1995), only one response selection was influenced by age: As expected, children were more likely than were adolescents to choose a maladaptive response (behavioral avoidance). But in a study of 9- to 15-year-olds (n = 1,128), older (vs. younger) students reported significantly *less frequent* use of an adaptive response to sadness (cognitive reappraisal) and continued to do so across a 2-year follow-up, whereas, as predicted, younger (vs. older) students reported higher rates of a maladaptive response (expressive suppression) but decreasingly so over the follow-up (Gullone et al., 2010).

Studies that included wider age spans also yielded equivocal findings. Among the younger cohorts in a sample of 11- to 50-year-olds (n = 1,303), "adaptive regulation [of] sadness" (which included several responses) *declined* from age 11 to age 15 and then significantly increased by age 19; use of social support to manage sadness also declined from age 11 to midadolescence, rebounded by age 17, and declined again in later years; rumination decreased stepwise with age, whereas expressive suppression of sadness had no age-related pattern (Zimmermann & Iwanski, 2014). In contrast, among 12- to 18-year-olds (N = 1,761), adaptive *and* maladaptive cognitive strategies both were used increasingly as a function of age (Garnefski & Kraaij, 2006), and yet other studies of juveniles had null results regarding the impact of age on adaptive and maladaptive regulatory response use (Lantrip, Isquith, Koven, Welsh, & Roth, 2016; Silk, Steinberg, & Morris, 2003). Overall, therefore, whereas the normative developmental trajectories of *adaptive* mood repair responding are unclear, older age in community-based youths does signal *decreasing* deployment of *maladaptive* responses to sadness.

The information is likewise sparse and contradictory about the unfolding of affect regulatory repertoires in the context of depression. It has been reported, for example, that age does not alter the effects of depression on emotion regulation strategy use among youths (Garber et al., 1995) but also that depression across early adolescence signals increasing use of adaptive and maladaptive regulatory responses *both* (Garnefski & Kraaij, 2006). In contrast, age was negatively related to maladaptive mood repair responses among 11- to 19-year-olds with histories of major depression and healthy controls (Bylsma et al., 2016). The sparse information on mood repair development among depressed youths is surprising. Juvenile-onset depression is highly recurrent (Kovacs, Obrosky, & George, 2016), more impairing than adult-onset depression (Zisook et al., 2007), and has posed a treatment challenge (Weersing, Jeffreys, Do, Schwartz, & Bolano, 2017). If depressed youths evidence atypical emotion regulatory developmental trajectories, such trajectories could contribute to the morbidity of their conditions and may thereby represent targets for intervention.

THE CURRENT STUDY

To examine the interface of age and mood repair repertoires among depressed youths and matched controls, we hypothesized that (a) with increasing age, adaptive mood repair response repertoires will expand but maladaptive repertories

will diminish in both groups and (b) clinically depressed youths (vs. controls) will display less pronounced age-related expansion of adaptive mood repair repertoires and less pronounced diminution of maladaptive mood repair repertoires. The hypotheses were tested using youths' self-reports. However, we also examined whether parents' reports of their offsprings' mood repair responses reveal age-related changes, and if a youth's group status (depressed or control) affects parent-offspring report agreement.

METHOD

Clinical Sample

We report on 697 depressed patients (DEP) from a prior study with the needed mood repair data. As described previously (Tamás et al., 2007), youths were recruited through 23 psychiatric facilities in several cities across Hungary. The process included symptom- and demography-based prescreening and diagnostic evaluations (see next and Tamás et al., 2007). Entry criteria included ages 7 to 14, a *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV*; American Psychiatric Association, 2000) mood disorder diagnosis, which was primarily major depressive disorder (MDD), no major systemic medical condition, and availability of at least one biological parent.

Consistent with the sex distribution associated with childhood-onset depression, this group (Table 1) is predominantly male (55%). Ethnic distribution (not in Table 1) was mostly Caucasian (95%), with a minority of Roma (3%) and Africans (2%), representative of the population of Hungary. Using parental education as an index of socioeconomic status (SES), the clinical sample was mostly of middle or lower SES (Table 1).

Mean age at first MDD onset was 10.9 years (SD = 2.1): 76.6%, 20.8%, and 1.4% were, respectively, in their first, second, and third or more MDD episodes. At study entry, about 90% were clinically depressed, whereas 10.9% were in remission from a recent MDD. Anxiety disorders (35.9%) and externalizing disorders (24.4%) were the most common psychiatric comorbidities.

Control Sample

Controls (CONT) were from nine elementary schools (Grades 1–8 in Hungary) in several cities. Information packets, distributed in the schools, included an invitation for the family to participate in a study of children's emotions, along with several questionnaires. If multiple children from a family attended a school, the parent was asked to consider only the youngest offspring. Of 5,224 packets, 3,538 (67.7%) were returned but 830 had incorrect age and lost/incomplete data and thus were not considered.

TABLE 1
Sociodemographic and Treatment History Variables for the Depressed (DEP) and School-Based Control (CONT) Samples

	CONT[a]	*DEP*[b]	*Statistic*
Sex, *n (%) Female*	626 (44.9)	313 (44.9)	$X^2_1 = 0.00$
Age, in Years, *M (SD) [Range]*	11.59 (2.01) [7.15–14.95]	11.63 (2.03) [7.26–14.97]	$t(2089) = -0.43$
Household Size, *M (SD) [Range]*	3.95 (1.01) [2–10]	4.64 (1.20) [2–11]	$t(1192) = -12.86$***
Highest Level of Parental Education (either parent), *n* (%)			$X^2_3 = 297.8$***
Elementary School (up to Grade 8)	35 (2.51)	132 (18.94)	
Vocational School	201 (14.42)	185 (27.98)	
High School	518 (37.16)	241 (34.58)	
College or Higher	640 (45.91)	129 (18.51)	
Parent-Rated Financial Situation, Compared to Other People, *n (%)*			$X^2_4 = 148.7$***
Much Worse	46 (3.33)	72 (10.33)	
Worse	168 (12.17)	142 (20.37)	
About Average	755 (54.67)	418 (59.97)	
Better	361 (26.14)	60 (8.61)	
Much Better	51 (3.69)	5 (0.72)	
Psychiatric Treatment of Youth			
Inpatient	0	201 (28.84)	$X^2_1 > 400$***
Outpatient	0	515 (73.89)	$X^2_1 > 1000$***
Inpatient or Outpatient, *n (%)*	0	553 (79.34)	$X^2_1 > 1500$***
History of Psychotropic Medication for Youth, *n (%)*	0	433 (62.12)	$X^2_1 > 1000$***

[a] $n = 1{,}394$.
[b] $n = 697$.
***$p < .001$.

Further deletions were made due to mental health treatment histories (n = 97), parental failure to answer this question (n = 54), scoring at or above the sample's 94th percentile (nine for girls, eight for boys) on the short Children's Depression Inventory (CDI; Kovacs & MHS Staff, 2011; n = 186), and missing parental education (n = 172), leaving 2,199. Using a 2:1 age- and sex-matching procedure, anchored in the clinical sample, the final control sample was 1,394. Controls had higher SES than the clinical sample, although comparable portions of both groups rated their financial situation as "about average" (Table 1).

Ethical Considerations

For the clinical sample, we obtained written informed consent from parents and assent from younger participants, as approved by the Institutional Review Board of the University of Pittsburgh and a national Institutional Review Board in Hungary. For school-based controls, only fully deidentified data were collected: Permission was obtained from school principals, in accordance with local rules. Parents, who consented for themselves and on behalf of their offspring, returned the questionnaires to the schools in sealed envelopes.

Procedures

Diagnoses were ascertained via a semistructured psychiatric interview (the Interview Schedule for Children and Adolescents: Diagnostic version), administered by trained clinicians in separate interviews with the clinic youths about themselves and the parents of the youths about their offspring (Tamás et al., 2007). Pairs of senior psychiatrists then rendered best estimate *DSM-IV* diagnoses (American Psychiatric Association, 2000). Parents provided demographic information via fully structured questionnaires; parents and youths also completed self-rated inventories. Control data were gathered through questionnaires self-administered by parents at home; control youths completed questionnaires in the schools, supervised by a home room teacher, including the CDI (Kovacs & MHS Staff, 2011), which quantifies depressive symptoms, and a questionnaire about mood repair (see next). Deidentified questionnaires for a parent–child pair shared a unique ID to facilitate analyses.

Assessment of Mood Repair Response Repertoires

Youths reported on their usual responses to sadness/dysphoria via the Feelings and Me (FAM) questionnaire; parents reported on their offspring via the Feelings and My Child questionnaire. These parallel FAM versions list behavioral/instrumental (e.g., self-soothing, planned action), cognitive (e.g., reappraisal, attention refocusing), and interpersonal strategies (asking for emotional support, seeking physical comforting). Items start with the stem, "*When I feel sad or down, I ...*" or "*When my child feels sad and down, he/she ...*" followed by statements rated from 0 (*not true of me [or my child]*) to 2 (*many times true of me [or my child]*).

Adaptive responses (e.g., "I try to get busy with some project," "I talk to my mom/dad") are associated with lowered negative affect and better functioning; *maladaptive* responses (e.g., "I think about how everything is my fault," "I try to hide") are associated with maintaining or exacerbating negative affect and psychopathology (Aldao et al., 2010). The Adaptive Regulatory Response (ADAPT) score (range = 0–60) includes 30 items; the Maladaptive Regulatory Response (MALADAPT) score (range = 0–48) includes 24 items. Higher scores indicate larger repertoires of the given response type, and that the response type is more characteristic of the individual.

The FAM was empirically derived, starting with a pool of items that were gathered from the literature, which a panel of judges then categorized as mirroring generally adaptive or maladaptive responses to sadness: the FAM's psychometric properties have been reported (Bylsma et al., 2016; Kovacs et al., 2009; Tamás et al., 2007). In the current study, ADAPT and MALADAPT scores showed high internal consistencies (Cronbach's α = .80–.89). Long-term (9–15 months) test–retest reliability in several of our samples ranged from .41 to .53. Concurrent validity is documented by significant correlations between FAM MALADAPT and Rumination Scale scores (.52–.70) in several of our samples. FAM scores discriminate offspring at variable risk for depression (Bylsma et al., 2016) and predict new depression episodes (Kovacs et al., 2009).

Statistical Analyses

An analysis of covariance (ANCOVA) served to model the targeted FAM score on subject group (depressed vs. control) and age (continuous). Based on preliminary analyses, we adjusted for sex (girls typically had higher FAM scores than did boys) and highest level of parental education (typically associated with more favorable offspring scores). Because main effects in models with Class × Continuous Variable interactions are influenced by the continuous variable's scale and location (Engqvist, 2005), we started with main-effects-only models. Then, we added interactions terms, including a Group × Age term to determine if age trends differed by subject group. Significant interactions were probed via post hoc analyses of age (in discrete years) and sliced by group. Effect sizes are reported as ω^2.

Concordance of parent–youth reports was examined via correlation coefficients. It also was modeled through multivariate analysis of covariance via a mixed effect model, which specified an unstructured within-subject correlation matrix for each subject group. To determine if offspring's age affected parent–child agreement, we

examined the Informant × Group × Age interaction, sliced by age and group. Effect sizes are reported as R^2_β.

RESULTS

We present youths' self-reported FAM scores by age group in the samples, followed by parents' reports of their offsprings' responses to sadness, and then the agreement between these sets of informants. To facilitate across-group visual comparisons, Figures 1 and 2 were based on *t* scores ($M = 50$, $SD = 10$).

Youths' Mood Repair Repertories by Self-Report

Modeling *adaptive mood repair* responses, the main-effects ANCOVA was significant for age, $F(1, 2084) = 47.77$, $p < .001$, $\omega^2 = .02$, and group, $F(1, 2084) = 27.25$, $p < .001$, $\omega^2 = .01$. Added to the model, the Group × Age interaction also was significant, $F(1, 2083) = 7.40$, $p = .007$, $\omega^2 < .01$. However, contrary to Hypothesis 1, adaptive mood repair repertoires *decreased* in size across CONTs, $F(7, 2071) = 3.22$, $p = .002$, $\omega^2 = .01$, and DEP age groups, $F(7, 2071) = 5.77$, $p < .001$, $\omega^2 = .02$. Further, through age 11, younger DEP children reported significantly more ADAPT strategies than did CONTs (all $F > 4.7$, all $p \leq .03$, $\omega^2 \leq .01$), a tendency that fades by later ages (Figure 1).

Modeling *maladaptive mood repair scores* (Figure 1), the main-effects ANCOVA was significant for age, $F(1, 2084) = 8.14$, $p = .004$, $\omega^2 < .01$, and group, $F(1, 2084) = 556.19$, $p < .001$, $\omega^2 = .21$. The added Group × Age interaction was significant, $F(1, 2083) = 23.87$, $p < .001$, $\omega^2 = .01$. Contrary to Hypothesis 1, maladaptive repertories increased with age in the DEP group, $F(7, 1944) = 7.64$, $p < .001$, $\omega^2 = .02$, but remained stable among CONTs, $F(7, 1944) = 1.37$, $p > .21$, $\omega^2 < .01$. Further, CONT youths reported significantly smaller maladaptive mood repair repertoires at each age than those depressed, Fs > 41, $p < .001$, $\omega^2 = .02$–.07).[1]

Youths' Mood Repair Repertories by Parental Reports

Based on parental reports, neither the ADAPT ($F = 0.09$, $p > .7$, $\omega^2 = .00$) nor the MALADAPT scores of youths ($F = 3.55$, $p = .060$, $\omega^2 < .01$) showed age effects (Figure 2); Group × Age interactions likewise were not significant (Fs < 1.2, ps > .2, ω^2s < .001). However, parents of DEP youths, compared to parents of CONT youths, characterized their offspring as evidencing strikingly more extensive *maladaptive* response repertoires at every age (main effect $F = 1197.4$, $p < .001$, $\omega^2 = .38$) and somewhat smaller adaptive repertories (main effect $F = 19.95$, $p < .001$, $\omega^2 = .01$).

Parent-Youth Agreement on Youths' Mood Repair Repertories

Fitting a multivariate analysis of covariance on ADAPT scores (Figure 3, mirroring raw scores) revealed a significant effect of informant, $F(1, 303) = 189$, $p < .001$, $R^2_\beta = .38$; no effect of group, $F(1, 248 = 1.27$, $p > .2$, $R^2_\beta = .01$; and a Group × Informant, $F(1, 280) = 44.3$, $p < .001$, $R^2_\beta = .14$, but no Informant × Group × Age interaction, $F(7, 370) = 1.93$, $p = .06$, $R^2_\beta = .04$.

CONT parents rated their offspring somewhat less favorably (lower ADAPT scores) than the offspring rated themselves (score $M_{\text{CONTROL, parent}} = 18.8$, $M_{\text{CONTROL, child}} = 20.6$; ps < .03, R^2_βs = .02–.25), but CONT parent-youth ADAPT reports begin to converge at age 11 (ps = .003–.6, R^2_βs = .00–.03). Parents of DEP youths reported much smaller adaptive response repertories for their offspring than the offspring themselves reported ($M_{\text{DEP,}}$

[1] As articulated by Kraemer et al. (2000), cross-sectional comparisons of age groups will lead to erroneous developmental conclusions if the age groups differ in attributes that are known (or can be expected) to affect the outcome variable. Based on prior work, sex, depression severity, and the presence of comorbid anxiety disorder can influence FAM responses. Therefore, we examined the distribution of these variables across the various ages and their effects on the results. We confirmed that, overall, age is a significant predictor of FAM scores. Although the dramatic increase with age in MALADAPT scores in the DEP group appears to reflect primarily the impact of older female individuals, depression symptom severity and anxiety comorbidity make little or no contribution to this posited developmental trend.Namely, the sex ratio (in both groups) shifted from about 60% male in the 7- to 12-year-old subset to about 60% female in the 13- to 14-year-old subset. Adding Sex × Group × Age interaction terms when modeling MALADAPT scores, we found a significant main effect of sex ($F = 56.60$, $p < .001$) and a Group × Sex interaction ($F = 17.76$, $p < .001$): Girls scored higher than boys, and the difference is greater in the DEP sample. Further significant interactions include Sex × Age ($F = 12.66$, $p < .001$) and Group × Age ($F = 17.98$, $p < .001$): According to estimated slopes, MALADAPT scores significantly increased with age in DEP girls ($b = 1.14$, $SE = 0.19$, $p < .001$), not significantly in DEP boys ($b = 0.25$, $SE = 0.18$, $p = .2$), but did not change in control girls ($b = 0.14$, $SE = 0.14$, $p = .3$) and boys ($b = –0.12$, $SE = 0.13$, $p = .3$).Focusing on DEP subjects, depression severity (quantified via the CDI) significantly correlated with age and FAM scores ($r_{\text{CDI-Age}} = .13$, $r_{\text{CDI-MALADAPT}} = .63$, $r_{\text{CDI-ADAPT}} = –.13$, all ps ≤ .001). Depression severity was significant when added to the model of MALADAPT that included age, sex, and Age × Sex ($F = 359.9$, $p < .001$), but age *continued to remain significant* ($F = 10.47$, $p = .001$). In the entire DEP sample, 36% had a history of anxiety disorder. However, the rate of anxiety disorder did not vary significantly across the ages ($\chi^2_{\text{MH}} = 2.6$, $p = .1$) and was not analyzed further.Modeling FAM ADAPT scores, we found a significant main effect of sex ($F = 4.34$, $p = .037$) and significant Group × Age ($F = 6.88$, $p = .009$) and Sex × Group × Age ($F = 7.15$, $p = .008$) interactions. Estimated slopes showed that ADAPT scores decreased significantly with age in DEP girls ($b = –1.53$, $SE = 0.27$, $p < .001$) and boys ($b = –0.77$, $SE = 0.26$, $p = .003$); in CONTs, the decrease was significant in boys ($b = –0.78$, $SE = 0.18$, $p < .001$), but not in girls ($b = –0.31$, $SE = 0.20$, $p = .1$). Although depressive symptoms (CDI scores) also affected ADAPT scores ($F = 6.91$, $p = .009$), age continued to remain significant ($F = 26.31$, $p < .001$).

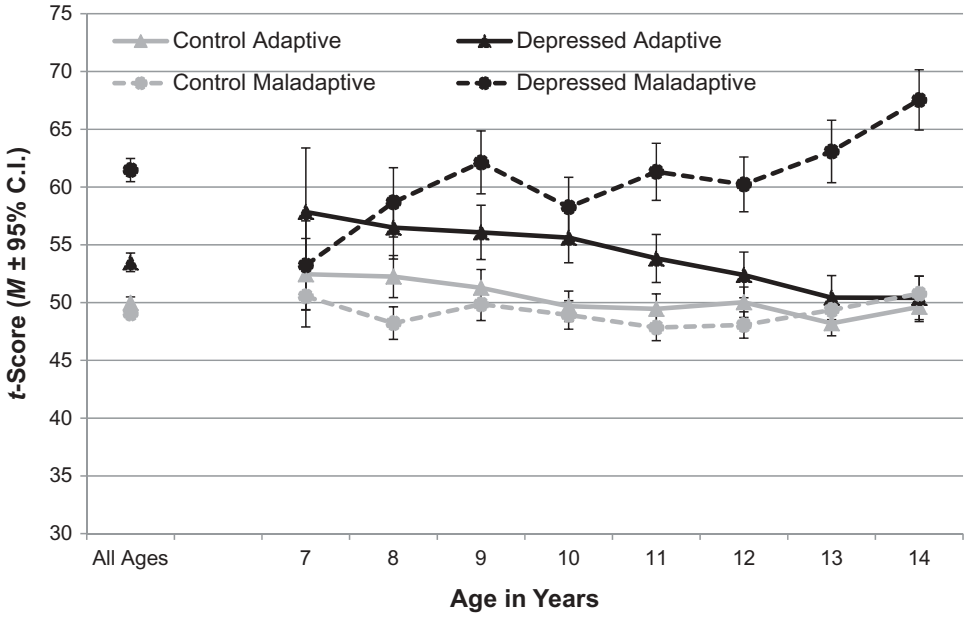

FIGURE 1 Self-reported adaptive and maladaptive mood repair response repertoires by age among depressed (N = 697) and control (N = 1,394) youths. *Note*. C.I. = confidence interval; ANCOVA = analysis of covariance.

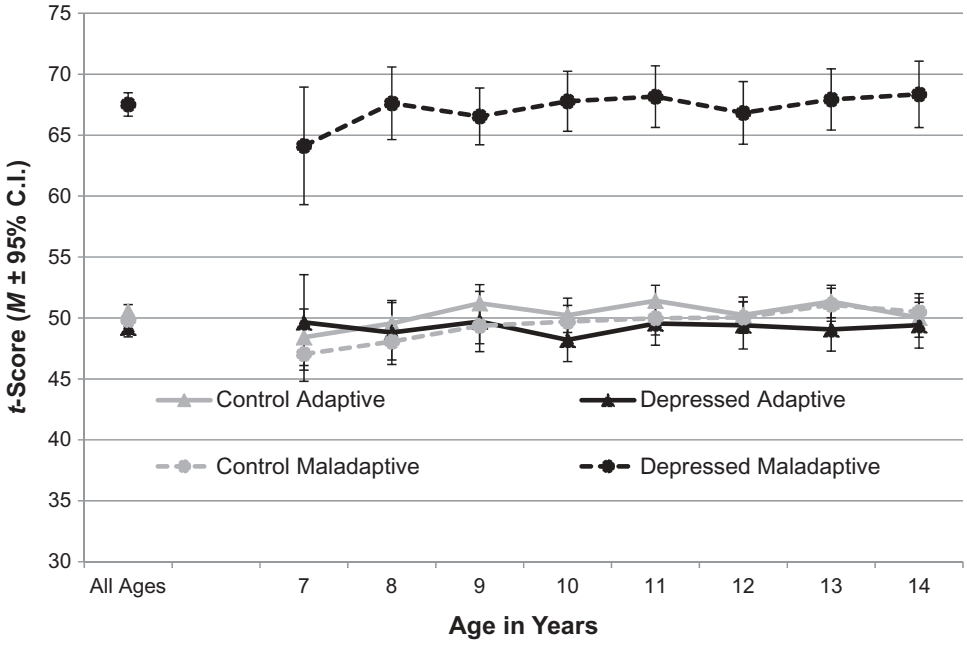

FIGURE 2 Parent-reported adaptive and maladaptive mood repair response repertoires by age among depressed (N = 697) and control (N = 1,394) youths. *Note*. C.I. = confidence interval; ANCOVA = analysis of covariance.

$_{\text{Parent}}$ = 17.6, $M_{\text{DEP,Child}}$ = 24.1; ps < .01, R^2_{β}s = .08–.39; Figure 3). Although parent-youth ADAPT scores become more similar among older youths, the differences remain significant (R^2_{β}s = .39–.08).

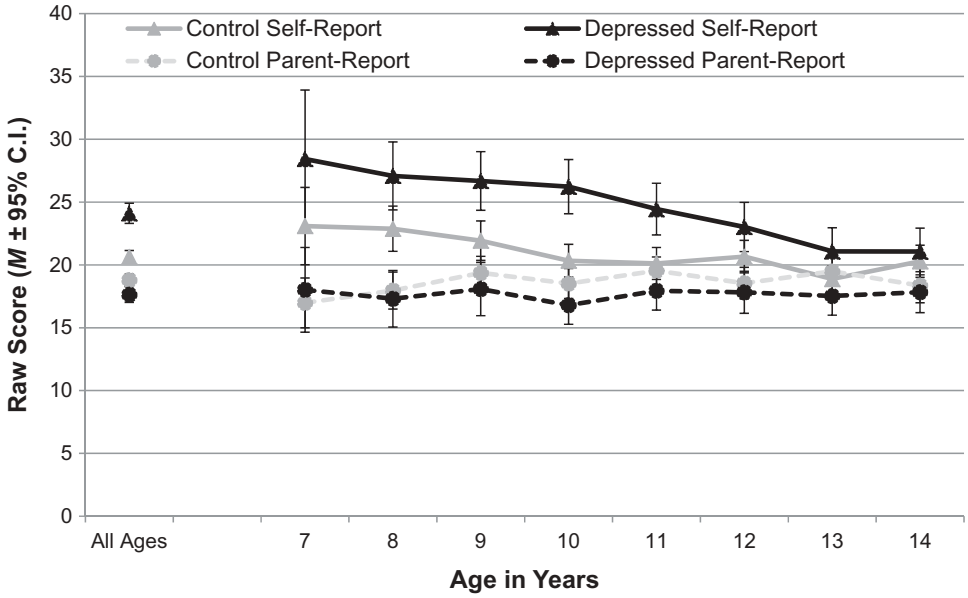

FIGURE 3 Parents' and offsprings' reports of offsprings' adaptive mood repair response repertoires by age among depressed (N = 697) and control (N = 1,394) youths. *Note*. C.I. = confidence interval; MANCOVA = multivariate analysis of covariance.

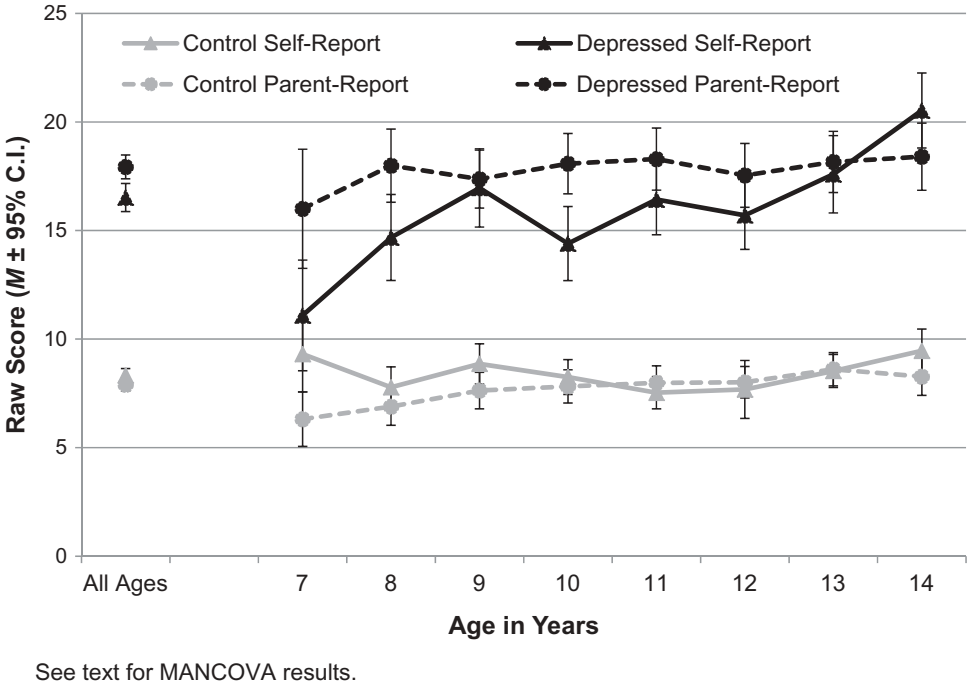

FIGURE 4 Parents' and offsprings' reports of offspring's maladaptive mood repair response repertoires by age among depressed (N = 697) and control (N = 1,394) youths. *Note*. C.I. = confidence interval; MANCOVA = multivariate analysis of covariance.

MALADAPT scores (Figure 4, mirroring raw scores) revealed a significant main effect of group, $F(1, 502) = 944$, $p < .001$, $R^2_\beta = .65$; no informant effect, $F(1, 374) = 0.09$, $p > .7$, $R^2_\beta < .01$; but significant Group × Informant, $F(1, 342) = 42.26$, $p < .001$, $R^2_\beta = .11$; and Group × Informant × Age, $F(7, 313) = 3.73$, $p < .001$, $R^2_\beta = .08$, interactions. CONT parents rated their children as having somewhat less extensive maladaptive response repertories than suggested by offspring's ratings ($ps <$

.01, R^2_β = .02–.23), but their reports start to converge around age 11 (Figure 4). In contrast, parents of DEP youths characterized offspring up to age 11 as having significantly larger MALADAPT response repertoires than the offspring reported (*p*s < .08, R^2_βs = .03–.26), with an inconsistent disagreement pattern across ages 12–14.

DISCUSSION

Using cross-sectional data from late childhood to midadolescence, we tested two hypotheses about age-related changes in the mood repair response repertoires of depressed and emotionally typical youths. The hypotheses were based on the development of skills related to mood repair. According to Hypothesis 1, older youths should have more extensive adaptive mood repair repertoires than younger ones. We failed to support this hypothesis among our 7- to 14-year-old subjects, which suggests that the *initial overall scope* of these repertoires already had been laid down or set prior to age 7.[2] This interpretation is supported by (a) parental reports, which revealed no age related increments in offspring's adaptive repertoires for either subject group, and (b) the generally negative/null age-related findings in the literature (noted earlier). However, late adolescence and emerging adulthood may prompt the acquisition of additional adaptive ways to respond to sadness as youths encounter novel contexts and new opportunities for social modeling. Indeed, findings suggest that the developmental trajectory of adaptive mood repair is quadratic rather than linear in form starting in early adolescence and across the second decade of life (Zimmermann & Iwanski, 2014).

Contrary to Hypothesis 1, y*ouths' self-reports* revealed that age and adaptive mood repair response repertoires were negatively related: Older youths reported fewer adaptive strategies than did younger ones. This pattern, evident in both subject groups, suggests that normative mood repair development from late childhood to midadolescence includes trimming or pruning of *adaptive mood repair* responses. Other investigators likewise have reported higher use of adaptive regulatory responses among children than adolescents (e.g., Gullone et al., 2010) and among younger than older adolescents (Zimmermann & Iwanski, 2014), possibly reflecting that children overestimate the effectiveness of regulatory strategies (Waters & Thompson, 2014).

However, developmental conclusions based on cross-sectional age comparisons can be erroneous (Kraemer, Yesavage, Taylor, & Kupfer, 2000). Therefore, we note that we have confirmed that trimming of adaptive responses occurs across the ages of 7 to 14 years, using longitudinal data on similarly aged, independent U.S. samples of control and high-risk subjects (Kovacs, Yaroslavsky, & George, 2017). Thus, the findings suggest that younger children explore many potentially helpful regulatory responses to sadness before they settle (possibly through trial-and-error) on a more reduced response set.

Although Zimmermann and Iwanski (2014) also found that the use of adaptive mood repair strategies declined with age across adolescence, and thus may reflect a true developmental phenomenon, other interpretations are possible. For example, the high rates of adaptive response deployment reported by young children could reflect the questionable validity of self-reports at that age. However, self-reports of maladaptive mood repair strategies did not indicate response trimming as a function of age, making it difficult to argue that younger children answer questionnaires indiscriminately and invalidly.

Hypothesis 1 also proposed that *maladaptive* mood repair response use will be more prevalent at younger ages (e.g., Gullone et al., 2010). However, control youths reported comparable maladaptive repertoire sizes at each age, whereas older depressed youths reported significantly larger maladaptive repertoires than did their younger counterparts. Because sex, depression severity, and comorbid anxiety can impact FAM scores in the context of depression, our secondary analyses examined their distributions at each age; if any of these variables is more prevalent at some age than at other ages, that variable (rather than age) may account for the results. Only sex was unevenly distributed across the age groups: Starting at ages 9–11, there were more girls than boys at each age, and depressed girls mostly account for the significant age-related increase in maladaptive FAM scores (although age continues to contribute). The preponderance of girls in the depressed sample after age 11 mirrors epidemiologic data on the sex-linked rise of depression in mid/late adolescence (Merikangas et al., 2010).

In partial support of Hypothesis 2, our subject groups did differ in age-related changes in mood repair repertoires, although not in the expected directions. Although it has been suggested that a lean adaptive regulatory repertoire increases the risk of psychopathology (e.g., Zimmermann & Iwanski, 2014), the depressed children in our study reported significantly *larger* adaptive response repertories than did similarly aged controls up until about age 11. Garnefski and Kraaij (2006) also found a positive association between depressive symptoms and adaptive cognitive strategies (e.g., positive

[2] Although the initial overall "scope" of mood repair responses appears to have been set, there are age-related changes in the content of mood repair repertoires. For example, examining the group of FAM items that reflect the use of interpersonal processes for mood repair ("I talk to my parents," "I call my friends"), we find that these scores in CONTs manifest significant linear, $F(1, 1375) = 14.10$, $p < .001$, $\omega^2 = .01$) and quadratic, $F(1, 1375) = 10.33$, $p = .001$, $\omega^2 = .01$, functions. Specifically, interpersonal strategy use declines around age 11 and then picks up again around age 14. Zimmermann and Iwanski (2014) reported similar findings. These trends are likely to mirror a developmental transition from relying on parents for interpersonal support to increasing use of peers to aid in combating sad affect.

refocusing) among 12- to 15-year-olds (but not among older youths). Thus, at least up until midadolescence, depressed youths reportedly deploy a wider scope of adaptive mood repair responses (than controls do), possibly because their default responses did not relieve sadness. Indeed, the presence of a depression diagnosis suggests that whatever adaptive strategies had been tried did not result in improved mood. Thus, the scope of adaptive mood repair responses prior to age 11 is not a reliable indicator of emotional health or depressive psychopathology. Although this finding needs to be confirmed, it is in line with the ambiguous prognostic value of adaptive emotion regulation in older samples (Aldao et al., 2010).

Contrary to Hypothesis 2, maladaptive repertoires increased with age in the depressed sample but remained stable among controls. (Recall that the MALADAPT scores of 14-year-old depressed youths were twice as high as the scores of controls.) Overall, therefore, depression in youths is characterized by an abundance of maladaptive responses to sadness, rather than a poverty of adaptive regulatory responses. In other words, although depressed youths have many responses at their disposal that can attenuate sadness, they have markedly more strategies that maintain or exacerbate sad affect. This finding suggests that interventions for depressed youths should focus on eliminating maladaptive mood repair responses rather than increasing the repertoire of adaptive regulatory strategies.

Parents' reports of their offspring's mood repair responses were not affected by offsprings' ages. Parent–youth FAM score discrepancies were detectable in both subject groups and mostly at younger ages, but with small to moderate effect sizes. However, the parent–youth discrepancies were notably wider in the depressed sample, and parental ratings showed the offspring in a less favorable light (higher MALADAPT and lower ADAPT scores) than the offspring perceived themselves. Discrepant parent–youth reports, particularly at younger ages, are likely to reflect, in part, the relative immaturity of children's cognitive and self-evaluative skills compared to the evaluative skills of their parents. This interpretation is supported by the finding that it was typically the offspring's report that came to resemble the parent's report, rather than the other way around (see Figures 3 and 4).

Along with its strength, our study has limitations. Most important, although we used a cross-sectional design to identify potential developmental trends, only longitudinal studies can determine whether development can account for the results and assess if group-level trends obscure individual differences in mood repair trajectories. The results also suggest the need for a more nuanced conceptualization of mood repair development after late childhood. Relatedly, our results may not generalize to emotions other than sadness. Further, our control parents were better educated than the general population, similar to a study in the Netherlands (Larsen et al., 2013). Higher SES may signal greater willingness to participate in offspring research, the impact of which should be examined. Because Hungary is ethnically more homogenous than is the United States, we could not examine the effect of ethnicity on mood repair development. Overall, however, the strengths of our study outweigh its limitations, and the results confirm that late childhood to midadolescence provides a "critical developmental window" on the unfolding of affect regulatory skills (Silvers et al., 2012, p. 1244).

FUNDING

This study was supported by National Institute of Mental Health (NIMH) Program Project grant PO 1 MH056193; preparation of this article was supported by NIMH grants MH085722 and MH084938 to Dr. Kovacs. The CDI is published by MHS Inc., from which Dr. Kovacs receives royalties. The other authors report no conflict of interest.

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