Published in final edited form as:

J Child Psychol Psychiatry. 2015 October ; 56(10): 1108–1117. doi:10.1111/jcpp.12376.

Mood repair via attention refocusing or recall of positive autobiographical memories by adolescents with pediatric onset major depression

Maria Kovacs[1], **Ilya Yaroslavsky**[2], **Jonathan Rottenberg**[3], **Charles J. George**[1], **Ildikó Baji**[4], **István Benák**[4], **Roberta Dochnal**[4], **Kitti Halas**[4], **Enikő Kiss**[4], **Ágnes Vetró**[4], and **Krisztina Kapornai**[4]

[1]Department of Psychiatry, University of Pittsburgh School of Medicine, PA, USA

[2]Cleveland State University, Cleveland, OH, USA

[3]Department of Psychology, University of South Florida, Tampa, FL, USA

[4]Szeged University Medical School, Szeged, Hungary

Abstract

Background—Impaired emotion regulation is increasingly recognized as a core feature of depressive disorders. Indeed, currently and previously depressed adults both report greater problems in attenuating sadness (mood repair) in daily life than healthy controls. In contrast, studies of various strategies to attenuate sad affect have mostly found that currently or previously depressed adults and controls were similarly successful at mood repair in the laboratory. But few studies have examined mood repair among depression-prone youths or the effects of trait characteristics on mood repair outcomes in the laboratory.

Methods—Adolescents, whose first episode of major depressive disorder (MDD) had onset at age 9, on average (probands), and were either in remission or depressed, and control peers, watched a sad film clip. Then, they were instructed to engage in re-focusing attention (distraction) or recalling happy memories. Using affect ratings provided by the youths, we tested two developmentally informed hypotheses about whether the subject groups would be similarly able to attenuate sadness via the two mood repair strategies. We also explored if self-reported habitual (trait) mood repair influenced laboratory performance.

Results—Contrary to expectations, attention re-focusing and recall of happy memories led to comparable mood benefits across subjects. Control adolescents reported significantly greater reductions in sadness than did depressed (Cohen's d=.48) or remitted (Cohen's d=.32) probands, regardless of mood repair strategy, while currently depressed probands remained the saddest after mood repair. Habitual mood repair styles moderated the effects of instructed (state) mood repair in the laboratory.

Correspondence: Maria Kovacs, WPIC, 3811 O'Hara Street, Pittsburgh, PA, 15213 USA; kovacs@pitt.edu.

Supporting Information

Additional Supporting Information is provided along with the online version of this article.

Conflicts of interest statement: The other authors have declared that they have no potential or competing conflicts of interest.

Conclusions—Whether depressed or in remission, adolescents with MDD histories are not as efficient at mood repair in the laboratory as controls. But proband-control group differences in mood repair outcomes were modest in scope, suggesting that the abilities that subserve affect regulation have been preserved in probands to some degree. Further information about the nature of mood repair problems among youths with depression histories would help to better understand the clinical course of MDD and to design personalized interventions for depression.

Keywords

mood repair; emotion regulation; depression; adolescents; attention; positive memories

Introduction

When a person feels sad or dysphoric, there is a natural inclination to behave in ways that will help to feel better (Morris & Reilly, 1987). For example, a young adolescent who becomes downcast because he did not do well on a school examination may seek out his mother for comfort as soon as he gets home, while another teenager in the same situation may march into his room and immerse himself in a martial arts videogame. All other things being equal, both strategies are likely to ameliorate the youngsters' dysphoric mood. This process of attenuating sadness, called *mood repair* (Isen, 1985; Josephson, Singer, & Salovey, 1996), is now recognized as an integral component of adaptive functioning and effective self-regulation (e.g., Joormann, Siemer & Gotlib, 2007).

Concomitantly, impaired mood repair has been increasingly conceptualized as a core feature of clinical depression (e.g., Gross & Muñoz, 1995; Tomarken & Keener, 1998). Indeed, adults suffering from major depressive disorder (MDD) tend to report mostly maladaptive mood repair styles and responses, which are more likely to exacerbate sadness than to attenuate it (Brockmeyer, Bents, Holtforth, Pfeiffer, Herzog, & Friederich, 2012; D'Avanzato, Joormann, Siemer & Gotlib, 2013; Donaldson & Lam, 2004; Joormann & Gotlib, 2010; Kovacs, Rottenberg & George, 2009). Importantly, even after the depression has remitted, individuals continue to report problematic mood repair (D'Avanzato et al., 2013; Ehring, Fischer, Schnülle, Bösterling & Tuschen-Caffier, 2008; Kanske, Heissler, Schönfelder & Wessa, 2012; Kovacs et al., 2009). The persistence of mood repair problems in daily life raises the possibility that impaired mood repair is a trait (vulnerability) marker of clinical depression (Kanske et al., 2012; Werner-Seidler & Moulds, 2012). Mood repair problems may reflect impairment in the skills that subserve adaptive responses, poor or sub-standard quality of the responses themselves, or excessive reliance on maladaptive responses to dysphoria that maintain or exacerbate that mood (Aldao, Nolen-Hoeksema, & Schweizer, 2010).

Laboratory studies have shed some light on the nature of mood repair problems among depressed and depression-prone individuals (those with prior episodes of depression). Although depressed adults may find it hard to execute a specific adaptive mood repair response as instructed (Beauregard, Paquette & Levesque, 2006) or sometimes perform more poorly than controls (Greening, Osuch, Williamson & Mitchell, 2013), the bulk of evidence shows that they attenuate sadness in the laboratory as well as controls. Specifically,

instructed mood repair via cognitive reappraisal or distraction was similarly successful for adults with ongoing or remitted MDD and controls in several neuroimaging (Beauregard et al., 2006; Erk et al., 2010; Kanske et al., 2012; Perlman et al., 2012) and non-imaging studies (Donaldson & Lam, 2004; Ehring, Tuschen-Caffier, Schnülle, Fischer & Gross, 2010). Joormann et al. (2007) also reported that the use of distraction led to comparable mood repair benefits regardless of subjects' histories, while another strategy--recalling positive memories to alleviate sadness--helped controls, did not help subjects with past depression, and worsened the mood of currently depressed cases. But, another experimental study that examined recalling positive personal memories for mood repair found that depressed and recovered depressed individuals evidenced similar mood benefits (Werner-Seidler & Moulds, 2012).

Thus, experimental evidence mostly suggests that the mood repair problems reported by depressed and depression prone adults are unlikely to reflect a deficit or impairment of regulatory ability or skill, or compromised quality of responses. As summarized above, when they are given instructions under controlled conditions, adults with current or past depression are usually able to benefit as much as control peers from various mood repair responses. Therefore, the problematic mood repair associated with depression in adults appears to reflect, at least to some extent, the excessive use of maladaptive responses to sadness, rather than the inability to execute adaptive responses.

Do the findings on mood repair and clinical depression hold up in younger cohorts? Although depression can onset as early as childhood (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984), to our knowledge, there has been only one investigation of mood repair in clinic-referred depressed youths. That neuroimaging study of 13- to 17-year olds with MDD (n=14) and controls (n=14) found that the two groups did not differ in self-rated magnitude of distress after using cognitive reappraisal to minimize experimentally induced sadness (Perlman et al., 2012). Another neuroimaging study focused on offspring at high familial risk for depression: it was found that 20 never depressed, high-risk girls, aged 9- to 14-years and 27 control peers reaped similar subjective benefits from recalling positive personal memories to alleviate experimentally induced sadness (Joormann, Cooney, Henry & Gotlib, 2012). While results of these two studies of youths echo the findings on adult samples, they alone cannot be taken as definitive. Indeed, given that depression that onsets in the juvenile years appears to have more impairing long term consequences than depression that starts in adulthood (Zisook et al., 2007), it is not clear to what extent the mood repair findings on adults can be generalized to younger ages. Thus, one goal of our study was to extend the investigation of instructed mood repair and clinical depression to adolescents with childhood-onset MDD.

Further, few mood repair responses have been examined experimentally, especially ones that have been shown to be fundamental in the development of competent mood regulation. One such response is attention re-focusing (often called distraction in the literature), which has been identified by 6-months of age (e.g., Harman, Rothbart, & Posner, 1997). Shifting attention to something innocuous is one of the developmentally earliest and most effective ways to attenuate distress and has been documented across the age span (e.g., Erber & Tesser, 1992; Joormann & Siemer, 2004; Reijntjes, Stegge, Terwogt, Kamphuis, & Telch,

2006; Van Dillen & Koole, 2007; reviewed by Augustine & Hemenover, 2009). The developmentally early emergence of attention refocusing for affect regulation provides ample time for its practice: Consequently, it should be one of the best rehearsed and most accessible responses in individuals' mood repair repertoires, and possibly the most robust in the face of later psychopathology. However, the few studies of attention refocusing and clinical depression included only adults (Donaldson & Lam, 2004; Joormann et al., 2007); thus, its examination in depression-prone adolescents was also a goal of our project.

Recalling positive autobiographical memories (PAM) is another mood repair response that is developmentally relevant to our study population. PAM, or turning to the remembrance of positive events in order to alleviate negative affect, has long been identified as an important mood repair strategy (Isen, 1985; Josephson et al., 1996), with indications that this response is often automatic (Parrott & Sabini, 1990). While the process of recalling happy memories after exposure to a distressing stimulus also involves attention refocusing, PAM additionally requires the ability to access positive affect. But the capacity for positive affect is attenuated in currently and previously depressed youths (for a review, see Gilbert, 2012), and this deficit has been detected in offspring at high-risk for depression as early as the toddler and pre-school years (Durbin, Klein, Hayden, Buckley, & Moerk, 2005; Olino et al., 2011). Decreasing sadness via PAM therefore may be more difficult for depressed youngsters than controls, echoing some findings on adults (Joormann et al., 2007). Examination of this possibility was another goal of our study.

Finally, while laboratory studies offer experimental control of various confounds, there has been increasing interest in contextual factors that are likely to shape mood repair outcomes. One contextual factor is the individual's habitual way of responding to his or her own sad mood (Aldao, 2013). Thus, we also sought to explore the impact of habitual mood repair styles on subjects' mood repair performance in the laboratory.

In the present study, we compared adolescents, previously identified with childhood-onset MDD, who were either currently depressed or remitted (probands), and emotionally healthy peers, regarding how well they repaired sad mood in the laboratory. We tested two regulatory strategies: attention refocusing and recall of positive autobiographic memories. Our design and hypotheses were similar to that of Joormann et al. (2007), albeit they were informed by the developmental literature as noted above. We expected that all subjects would reap similar mood benefits from attention re-focusing (hypothesis 1), but that PAM will be more effective for controls than either depressed or remitted probands (hypothesis 2). Because there is scant information on the putative effects of habitual mood repair styles on laboratory performance, our analysis of the impact of trait mood repair on laboratory-based mood repair outcomes was exploratory and inspired by Aldao (2013).

Method

Subjects

The overall sample includes youngsters with previously established childhood-onset MDD (probands), and control peers, who never had any major psychiatric disorder. Probands derive from a larger national sample of clinically referred youths with depressive disorders

in Hungary, who were identified in a prior investigation. As described in detail (Baji et al., 2009; Tamas et al., 2007), probands were recruited from multiple clinical sites and had to meet various criteria to enter the original study, including having a DSM-IV depressive disorder and be aged 7- to 14-years at initial screen: caseness was established via standardized psychiatric diagnostic evaluations and best estimate consensus diagnoses.

The current investigation enrolled 210 probands with MDD histories (6 with bipolar disorder were excluded): 133 boys (63%) and 77 girls, 11- to 19-years old, with a mean age of 17.00 years (SD=1.41 years), and with 72% of them in the 11- to 17-year old age range. Eleven percent of the mothers and 10% of the fathers had more than secondary school education (used as an indicator of socioeconomic status). Mean age at onset of first MDD episode in these adolescents was 9.07 years (SD= 1.89 years). At the time of the diagnostic assessment for the current study, 56 % had one MDD episode, 30% had 2 episodes, and 13% had 3 or more episodes of MDD; 180 subjects were in remission from their most recent MDD episode, while 30 (14%) were amidst a depressive episode. Altogether 71% had comorbid psychiatric disorders including some anxiety disorder (39%) and some behavior disorder (38%).

Controls (n=161) for the present study were selected from elementary and secondary schools in the probands' cities in Hungary. Research staff made presentations at parents' meetings and handed out pre-screen questionnaires. After reviewing pre-screen information, potential subjects and parents participated in the diagnostic evaluation (see below). Controls, (recruited to approximate the sex and age distribution of probands) include 104 boys (65%) and 57 girls with a mean age of 15.85 years (SD=2.14 years), with no history of any major DSM-IV psychiatric disorder. Fifty-two percent of the mothers and 44% of the fathers had more than secondary school education.

Diagnostic assessment

Both during the archival and current studies, diagnoses were based on semi-structured clinical interviews via the Interview Schedule for Children and Adolescents: Diagnostic version (ISCA-D), which was derived from the ISCA (Sherrill & Kovacs, 2000). The ISCA-D (Baji et al., 2009; Tamas et al., 2007) covers all mood disorders and the most common non-affective (e.g., anxiety and behavior) disorders using DSM-IV criteria (American Psychiatric Association, 2000). The ISCA-D was first administered to the parent about the youth, and then separately to the youth about him or herself. All diagnoses were reviewed by pairs of training supervisors and senior investigators, and final diagnoses were by consensus. We used operational definitions of onset and recovery for a disorder episode (Kovacs et al., 1984). Evaluations were conducted by child psychiatrists and psychologists, who met predefined ISCA-D symptom rating goals and reliability standards. We have reported acceptable inter-rater reliability on the ISCA-D symptom ratings, ranging from .63 to .92 for current MDD from child interviews and .65 to .87 from parent interviews (Baji et al., 2009).

The current study was approved by the institutional review boards of the University of Pittsburgh and the Hungarian clinical research sites. Parents provided written consent, and young subjects provided either assent or consent. All procedures were developed in English, translated into Hungarian, and then back translated by bi-lingual child psychiatrists and

clinical psychologists. Discrepancies between original and back-translated versions were resolved via an iterative procedure.

Procedures

Each youth completed a battery of self-rated questionnaires, including measures of depression symptoms and trait mood repair, participated in an experimental protocol (lasting about one hour) that included probes of physiological and psychological reactions to a series of stimuli, and then participated in a semi-structured psychiatric interview conducted by a trained clinician. The main physiological variable of interest was respiratory sinus arrhythmia (RSA), which is derived from the standard electrocardiogram. The physiological probes included holding an ice pack to the forehead and completion of a handgrip task, which are believed to elicit differing RSA reactions. Psychological stimuli and tasks included a neutral and a happy film clip in addition to a sad film clip, a series of solvable and unsolvable computerized puzzles, instructed mood repair subsequent to negative affect stimuli, and receiving a desired prize. During inter-task intervals, youths provided mood ratings. The order of the tasks was randomized; there were 8 different task sequences.

In this article, we report on the part of the protocol that involved mood induction via a sad film clip and subsequent mood repair. For half of the sample, the sad film clip and the accompanying mood repair were in the first 30-minutes of the protocol, while for the rest, these tasks occurred during the second half hour of the protocol. After mood induction, 50% of the subjects were randomized to mood repair via attention refocusing and the other 50% to mood repair via positive autobiographical memory (see below). To assure that mood induction did not have undue adverse effects, the happy film clip was always the final task. Further, the succeeding psychiatric interviews were conducted by trained clinicians, who could provide intervention if needed, although in no case was that indicated.

Questionnaires and Affect Ratings

The *Children's Depression Inventory* (CDI-2) is a widely used and reliable, self-rated measure of depression symptoms in the prior 2 weeks (Kovacs & MHS Staff, 2011). It served as an index of depression symptom severity.

The *Feelings and Me-Child version (FAM-C)* served as the measure of habitual mood repair responses. The FAM-C surveys behavioral, cognitive, and interpersonal responses to sadness/distress by listing 30 adaptive and 24 maladaptive strategies, which have been enumerated in the literature (e.g., Fox, 1994; Morris & Riley, 1987; Thayer, Newman, & McClain, 1994). Adaptive responses (e.g., attention refocusing) have been associated with attenuation of sadness/dysphoria, while maladaptive responses (e.g., depressive rumination) have been associated with onset or exacerbation of sadness. Items are endorsed on a scale from "0=not true of me" to "2=many times true of me" and yield a summary score for Adaptive Responses and a separate score for Maladaptive Responses. Adaptive responses when feeling sad include: "I think of projects to do," "I get my mom to give me a hug." Maladaptive responses when feeling sad include: "I go and hide," "I think of all the bad things that ever happened to me." The initial psychometric properties of the FAM-C, including high reliability and satisfactory construct and concurrent validity, have been

reported (Tamas et al., 2007), along with additional indices of validity (Bylsma et al., under review). In the current study, internal consistency across all subjects in the analyses was .84 for the Adaptive Responses score and .83 for the Maladaptive Responses score. Adaptive and Maladaptive Responses scores were not related to subjects' ages (r= -.11 and r=.02, respectively, both ns), and the two scores were unrelated to one another in both the proband (r=.04, ns) and control (r=.09, ns) groups. One-year test-retest with 110 probands yielded reliability (intraclass) coefficients of .68 for the Maladaptive Responses score and .58 for the Adaptive Responses score.

Affect Self-Ratings—Subjects rated on 8-point Likert scales, from 0 (not at all) to 7 (very much), the extent to which several feeling states characterized them at baseline, after mood induction, and after the mood repair task. Paralleling others (e.g., Joormann et al., 2007), the items of interest (feeling 'sad' and 'blue'), were interspersed with items such as happy, interested, upset, and angry to obscure the focus on sad affect. The index of dysphoria for each case at the given time point is the average rating of the items of 'sad' and 'blue.'

Mood Induction

We used a 163-second clip from the *Champ* (commercially synchronized in Hungarian), which depicts a boy's immediate reactions to the death of a loved one. This film has been used by others (e.g., Gross & Levenson, 1995) and received extensive validation with Hungarian samples (see online Appendix S1).

Mood repair tasks

The mood repair tasks were introduced to subjects as the next 'thing to do' in the protocol. The attention re-focusing task (2.5 minutes) was adapted from a pediatric study (Carlson, Broome, & Vessey, 2000). A commercially available kaleidoscope was used (15 cm in length; 4 cm in diameter). After the sad film, the youngster was handed a kaleidoscope as the next task and was instructed to look into it and start rotating it. To facilitate task engagement, experimenters posed a standardized set of questions: "*What shapes do you see? What are the different colors? What is the strongest color you see? Which shape do you like the best? Do you see your favorite color in there*?"

Recall of positive autobiographical memories (2 minutes) has been studied with adults (e.g., Joormann et al., 2007) and high-risk adolescents (Joormann et al., 2012). After the sad film clip, it was introduced as the next task, with the recall period restricted to the past year. After the subject had identified a memory, he/she wrote down a word as a reminder (Memory 1); the procedure was then repeated (Memory 2). Then the experimenter returned to each memory clue and asked the child to recount it (1 minute maximum for each). Given the age range of the subjects, experimenters used general, standardized prompts, namely: *"Can you tell me more about this?"* "*Is there anything else you can tell me about ___[this memory]?*"

Results

Manipulation Check

As expected, a repeated measures ANOVA revealed that the magnitude of sad/blue ratings increased in the overall sample after watching the sad film clip (Time effect $F[1,368] = 51.40$, $p < .001$, *partial* $\eta^2 = .12$). However, examination of the raw scores identified a subset of subjects with 0 post-film sad/blue ratings (20% of controls, n=33; 39% of remitted probands, n=70; and 23% of depressed probands, n=7; $\chi^2[2] = 14.41$, $p < .001$). These subjects were excluded from the analyses because some sad mood must be present in order to examine mood repair. The cases who were retained included 128 controls, 110 remitted probands, and 23 depressed probands; they were balanced across mood repair conditions ($n_{\text{Attention Refocussing}} = 126$, $n_{\text{Positive Autobiographical Memory}} = 135$) and did not significantly differ in affect ratings after mood induction (Group effect $F[2,258] = 1.02$, n.s.).

Compared to the rest of the sample, excluded subjects were 6 months older ($t[369]=2.67$, $p<.01$) and tended to be males (71% vs 61%, $\chi^2[1] = 3.35$, $p < .07$). They did not differ from the retained cases in rates of racial minorities (4% vs. 3%, $\chi^2[1] = 0.25$, n.s.) or self-rated depressive symptom severity ($t[360]= .52$, p = n.s.), although they had lower mean Adaptive and Maladaptive responses scores on the FAM-C ($t[367]= 2.76$, $p<.01$; $t[367]= 2.51$, $p<.05$, respectively).

Characteristics of Subject Groups

Table 1 displays selected characteristics of the subjects that were retained in the analyses. Both proband groups were somewhat older than controls ($F[2, 258] = 12.61$, $p < .001$), but the 3 groups had similar sex distributions. Overall, ethnic composition was 97% Caucasian, 2% multiracial, and 1% Roma, representative of the population of Hungary; minorities constituted 1% of controls, 4% of remitted, and 9% of depressed subjects ($\chi^2[2] = 5.34$, $p<.07$). Age and minority status did not significantly correlate with baseline, post-film, or post-mood repair affect ratings (r between -.01 and .12) and were therefore not considered further. Additionally, affect ratings after mood induction were not affected by task order, nor by group membership (as noted above) Thus, post-mood induction affect ratings and task order were not considered further as covariates.

The three subject groups differed in CDI-2 rated depression severity ($F[2,251] = 66.57$, $p < .001$, all post-hoc comparisons $p < .001$) (Table 1). Further, probands had lower Adaptive mood repair response scores than controls ($F[2, 257] = 12.98$, $p<.001$), with the differences reflecting moderate effect sizes (Cohen, 1992; control vs. depressed $d = .65$, control vs. remitted $d = .62$). Maladaptive mood repair response scores also differed across the groups ($F[2,257] = 28.16$, $p<.001$), with moderate to large effect sizes (Table 1): controls had the lowest maladaptive mood repair scores (control vs. depressed $d = 1.64$, control vs. remitted $d = .50$), depressed probands had the highest scores, and remitted probands scored in between (remitted vs. depressed $d = 1.15$).

Mood Repair

Table 2 displays self-ratings of dysphoria (sad/blue) at baseline, after negative mood induction, and after mood repair for the three groups of subjects (raw means; SEs).

We had hypothesized that probands and controls would report similarly reduced sadness after attention refocusing, but that probands would benefit less than controls from recalling positive memories. This expectation was tested via a 3-way, repeated measures analysis of variance (ANOVA), in which we modeled sad/blue ratings as a function of group membership (control vs. remitted vs. depressed), mood repair strategy (Attention Refocusing vs. Positive Autobiographical Memory), and time (post-film vs. post-repair). The ANOVA yielded a significant effect for time ($F[1,255] = 178.26$, $p < .001$, *partial* $\eta^2 = .41$) indicating decrements in sad/blue ratings, and a significant group-by-time interaction ($F[2,255] = 4.10$, $p = .02$, *partial* $\eta^2 = .03$) indicating that the 3 subject groups differed in how much their mood ratings changed from baseline. The effect size for time indicates that implementation of the mood repair response explains more of the variance in the outcome than does group membership. However, there was no main effect for type of mood repair strategy, and mood repair strategy did not significantly interact with group membership, time, or group-by-time (all $Fs \leq 1.50$, n.s.). Thus, the findings failed to support our expectation that the PAM strategy would be less effective for adolescents with childhood-onset MDD than for controls. Therefore, the two mood repair strategies were collapsed in subsequent planned comparisons. Figure 1 displays the results for the overall sample. [1]

Pair-wise contrasts of mood ratings revealed that controls experienced greater sadness reduction than remitted and depressed probands after having implemented either mood repair strategy (change in *sad/blue* ratings: $M_{control} = 1.44$, $M_{remitted} = 1.13$, $M_{depressed} = .91$, *ps* <.05; Cohen's ds: control vs. depressed = .48, control vs. remitted = .32). However, remitted and depressed probands did not significantly differ in the extent of mood repair (Cohen's d: remitted vs. depressed = .16). While the extent of mood repair (mirrored by each slope in Figure 1) differentiates controls from both proband groups, Figure 1 also shows that the post-repair sad/blue raw score is notably higher for depressed cases than the rest. Indeed, in a univariate ANOVA of post-repair ratings, group membership was significant ($F[2,255] = 5.36$, $p < .01$, *partial* $\eta^2 = .04$): Depressed probands reported greater sadness at the end than controls and remitted probands (post-repair *sad/blue* ratings: $M_{control} = 0.29$, $M_{remitted} = 0.40$, $M_{depressed} = 0.83$, *ps* <.05; Cohen's ds: control vs. depressed = .66, remitted vs. depressed = .51), while remitted probands and controls did not significantly differ (Cohen's ds: control vs. remitted = .12).

To explore whether habitual mood repair styles moderate the effects of instructed mood repair, two interaction terms (trait adaptive mood repair-by time, and trait maladaptive mood repair-by time) were added to the 3-way repeated measures ANOVA (group-by-time-by-mood repair) described above. As before, the dependent variable was sad/blue rating. The

[1] In response to one reviewer's suggestion, we examined parental education level (a surrogate for socio-economic status, which differed across probands vs. controls at p< .001) as a potential confound. Correlation analyses showed that parental education was unrelated to affect ratings at any assessment point (r between -.05 and .06). Further, adding parental education to the repeated measures ANOVA revealed that it did not significantly predict mood repair (F[2, 251] = .07-.93, n.s.).

effects of time (F[1,252]=4.27, p<.05, *partial* η^2 = .02) and group-by-time interaction (F[2,252]=4.21, p<.05, *partial* η^2 = .03) remained significant. In addition, we found a significant main effect for habitual maladaptive mood repair (F[1,252] = 13.81, p < .001, *partial* η^2 = .05) and its interaction with time (F[1,252] = 5.27, p < .05, *partial* η^2 = .02). The main effect of trait adaptive mood repair was not significant (F[1,252]=2.13, *partial* η^2 = .01), but there was a significant trait adaptive mood repair-by-time interaction (F[1,252] = 5.05, p < .05, *partial* η^2 = .02). Thus, habitual maladaptive and adaptive mood repair each independently increased the benefits of instructed mood repair ($\beta_{maladaptive}$ = .16, $\beta_{adaptive}$ = .14, *ps* <.05).

Discussion

Impaired mood repair has been receiving increasing attention as a key feature of depressive disorders with mounting evidence of lingering mood regulatory problems in daily life even after remission of depression. However, the self-regulation of sadness in the context of depressive disorders has been studied almost exclusively in adults. Exploring this problem area in youths has public health implications because depression that onsets in the juvenile years prognosticates worse outcomes later in life than does adult-onset depression (Zisook et al., 2007) and contributes significantly to the overall burden of depression in the general population (e.g., Kessler, Petukhova, Sampson, Zaslavsky & Wittchen, 2012). Characterizing mood repair in depressed and depression-prone adolescents also can inform prevention and early intervention, which is especially important during the teen years that are known to signal a rapid rise in rates of clinical depression (e.g., Kessler & Walters, 1998).

In the present study, we examined whether adolescents with histories of MDD (either currently depressed or in remission) differ from never depressed peers in how well they can repair experimentally induced sadness. Contrary to our hypotheses that the two mood repair strategies (attention refocusing and PAM) would result in differential mood benefits across the groups of subjects, we found that, regardless of the type of strategy, controls were more successful at attenuating sadness than either currently depressed or remitted probands. In other words, given two, previously researched, adaptive mood repair strategies, probands (regardless of depression status) benefited less than did controls from instructed mood repair. Whereas most studies of diagnosed adults and the sole investigation of mood repair in adolescents with MDD (Perlman et al., 2012) reported that current or past depression does not attenuate the ability to repair depressed mood *in the laboratory*, our results echo other reports, which noted some affect regulatory impairment in the context of depression (e.g. Greening et al., 2013; Joormann et al., 2007). Overall, it therefore appears that the typically competent mood repair performance of depressed adults in laboratory settings may not mirror the performance of adolescents with depression histories. But, our findings reinforce existing literature that juvenile-onset depressive disorders have more severe clinical and functional consequences than depressions that onset later along the life course.

The end-point affect self-ratings in Figure 1 indicate that currently depressed probands were sadder after mood repair than controls and remitted probands, while the latter two groups appear similar. This seems to contradict the results of the repeated measures ANOVA,

which revealed that controls were significantly more successful (or adept) at mood repair than either (remitted or currently depressed) proband group. However, there is no contradiction: the repeated measures ANOVA results reflect differences in the groups' mood rating *slopes and* take into account the magnitude of sad/blue ratings after mood induction. Thus, those results mirror the extent of mood *change*, given each youth's start level of feeling sad/blue. In contrast, the simple ANOVA on post-repair scores does not control for post mood induction levels of sadness and reflects only the end-point mood rating. Overall, however, there is a gradient of sadness severity after mood repair as mirrored by subjects' raw scores (Table 2), with depressed probands the saddest, controls the least sad, and remitted depressed cases scoring between the other groups on sadness.

We expected attention refocusing to yield similar mood benefits for probands and controls. This expectation was based on experimental findings on adults (e.g., Joormann et al., 2007) and the fact that the use of attention refocusing for mood repair emerges in infancy (e.g., Kopp, 1989) and thus, considerably earlier than the age at which our probands had their first MDD episode. We reasoned that our probands would have had the opportunity to learn and deploy this mood repair strategy before they ever became depressed and would be able to use it efficiently. However, the results suggest that even those affect regulatory responses that emerge early in the course of development are not immune to the deleterious effects of subsequent psychopathology.

Further, we expected probands to gain less mood repair benefits than controls from the recall of positive memories. This expectation, which was confirmed, was likewise based on experimental findings with adults (Joormann et al., 2007) and reports that a) depressed as well as remitted youths evidence lower levels of positive affect than control peers (Gilbert, 2012) and b) offspring at high-risk for depression display attenuated positive affectivity even prior to school-age (Durbin et al., 2005; Olino et al., 2011). Because depression prone youths may thus have lower than typical levels of 'hedonic reserve,' we reasoned that they would benefit less than controls from a mood repair response that requires access to positive emotions. Although, as reported elsewhere (Begovic et al., under review), probands' memories were less detailed, less positive, and required more experimenter's prompts than the memories of controls, these aspects of memory functioning explained negligible unique variance (2%) in post-repair negative mood, and thus cannot account for the present findings. While confirmation of our second hypothesis could therefore indicate that youths prone to early-onset depression indeed have less access to positive affect or lower hedonic capacity than normally developing youths, this explanation will have to be tested in future studies.

We found that current as well as remitted major depression compromised the ability of probands to benefit as much as controls from instructed mood repair. While this finding may imply that MDD is causally related to less efficient mood repair, it also is possible that probands had atypical mood repair characteristics even before the onset of their depressions, which should be examined in future studies. All in all, compromised mood repair may be a trait marker of depression that onsets in the juvenile years. Further, the extensive repertoires of maladaptive mood repair responses (see Table 2) and some degree of impairment in

deploying adaptive responses (as evidenced in the laboratory) together may partly explain the mood repair difficulties of depressed and depression prone youths in daily life.

It is important to note, however, that the post-repair proband-control group differences in levels of dysphoria (e.g., Cohen's *d* .51 - .66) were rather modest, especially when considered in light of probands' extensive maladaptive mood repair repertoires (probands vs. controls Cohen's *ds* 1.15 - 1.64). This can be interpreted to mean that, even in the context of maladaptive mood repair tendencies, the abilities that sub-serve mood repair have been preserved in probands to some extent. Thus, mood repair should be considered as a viable therapeutic target in prevention and intervention programs for depressed adolescents: such youngsters may benefit from specific instructions about (and practice) of adaptive ways to respond to experiences of sadness.

The results confirm that trait characteristics of the individual (Aldao, 2013) influence mood repair outcomes. Recall that our indexes of habitual maladaptive and adaptive mood repair responses were not significantly correlated. Thus, for example, it was possible for a subject to have a very high maladaptive mood repair score along with an average adaptive mood repair score. The finding that trait maladaptive mood repair was directly related to laboratory mood repair performance may reflect that adolescents with sub-optimal mood repair styles are particularly likely to benefit from instructions about how to respond to sad affect. Not surprisingly, youths who were characterized by robust repertoires of adaptive trait mood repair in daily life also exhibited better mood repair outcomes in the laboratory than youths with lower levels of trait adaptive mood repair.

Our results also raise reporting and methodological concerns. Using the customary mood induction manipulation checks, we found the expected mean score change: the pre- post-induction differences in sad mood amounted to moderate effect sizes. The change scores masked that many subjects reported no sadness at all after negative mood induction. Non-responders to mood induction were more likely to be probands, were about 6 months older, and were more likely to be male than the rest of the youths. However, the implications of these findings are unclear because current publications typically do *not* report response rates to mood induction. The exceptions include two studies of remitted depressed adults, in which 18% (Singer & Dobson, 2009) and 14% (Singer & Dobson, 2007) failed to exhibit the expected level of response to negative mood induction, another study in which 17% of depressed and 6% of remitted adults were non-responders to mood induction (Werner-Seidler & Moulds, 2012), and a study of normal adolescents, in which 62% failed to meet criteria for a 'marked deterioration in state mood' after mood induction (Reijntjes et al., 2006). The latter finding and results of the current study could indicate that it may be particularly challenging to elicit sadness in some groups of adolescents. However, as Gerrards-Hesse, Spies, and Hesse's (1994) noted in their review, success rates of depression mood induction strategies have varied from 30% to 93% across samples. Because mean change scores may provide misleading information about subjects' affect states, current reporting practices of the outcomes of mood induction should be altered.

The present study had notable strengths, including the stringent diagnostic evaluation of participants, large sample size, randomization of subjects to experimental conditions, pilot

testing of mood induction procedures, and the restriction of analyses to participants who had dysphoric mood to repair. Further, we examined emotion regulation strategies that can be anchored in a developmental framework. That said, our results should also be interpreted in the context of limitations. First, as noted, the sad film did not evoke the expected affect for a notable portion of the subjects, particularly the remitted depressed probands. It is unclear if that limitation is particular to the present study or if applies to adolescent age groups. Second, the size of our currently depressed sample was comparatively small. This resulted in reduced power to detect statistically significant differences between remitted and depressed cases in some analyses, and also precluded an examination of the potential effects of comorbid psychiatric disorders on mood repair outcomes. Indeed, because pediatric depression is typically comorbid with a range of internalizing and externalizing disorders, the effects of various comorbidities on mood repair performance warrant further study. Third, we lacked 'quality control' procedures for the attention re-focusing condition, which precluded any comparisons with behavioral indices derived for autobiographical recall (Begovic et al., under review). Fourth, the ethnic composition of the sample was less diverse than what is typical of samples in the USA. Further, like most experiments in this area, we asked subjects to implement cognitively-based mood repair strategies. It is uncertain to what extent our results may generalize to other responses that can serve mood regulatory goals, including seeking the company of a parent, getting involved in a sport, or listening to uplifting music (e.g., Marroquín, 2011; Thayer et al., 1994). Clearly, further research is needed to better understand the developmental psychopathology of mood repair and its role in clinical depression.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by NIH Grants MH-084938 and MH-056193. M.K. receives royalties for The Children's Depression Inventory, published by Multi-Health Systems, Inc.

References

Aldao A. The future of emotion regulation research: Capturing context. Perspectives on Psychological Science. 2013; 8:155–172. [PubMed: 26172497]

Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across psychopathology: A meta-analytic review. Clinical Psychology Review. 2010; 30:217–237. [PubMed: 20015584]

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 2000. Text Revision

Augustine AA, Hemenover SH. On the relative effectiveness of affect regulation strategies: A meta-analysis. Cognition and Emotion. 2009; 23:1181–1220.

Baji I, Lopez-Duran NL, Kovacs M, George CJ, Mayer L, Kapornai K, Kiss E, Gadoros J, Vetro A. Age and sex analyses of somatic complaints and symptom presentation of childhood depression in a Hungarian clinical sample. Journal of Clinical Psychiatry. 2009; 70:1467–1472. [PubMed: 19709501]

Beauregard M, Paquette V, Levesque J. Dysfunction in the neural circuitry of emotional self-regulation in major depressive disorder. NeuroReport. 2006; 17:843–846. [PubMed: 16708026]

Brockmeyer T, Bents H, Holtforth MG, Pfeiffer N, Herzog W, Friederich HC. Specific emotion regulations impairments in major depression and anorexia nervosa. Psychiatry Research. 2012; 200:550–553. [PubMed: 22910477]

Begovic E, Panaite V, Bylsma L, George C, Kovacs M, Yaroslavsky I, Baji I, Benak I, Dochnal R, Halas K, Kiss E, Vetro Á, Kapornai K, Rottenberg J. Manuscript under review. Positive Autobiographical Memory Deficits Among Youth with Depression History and Their Never Depressed Siblings.

Bylsma LM, Yaroslavsky I, Rottenberg J, Kiss E, Kapornai K, Halas K, Dochnal R, Lefkovics E, Baji I, Vetró Á, Kovacs M. Manuscript under review. Familiality of Mood Repair Responses Among Youth with and without Histories of Depression.

Carlson KL, Broome M, Vessey JA. Using distraction to reduce reported pain, fear, and behaviroal distress in children and adolescents: A multisite study. Journal of the Society of Pediatric Nurses. 2000; 5(2):75–85. [PubMed: 10879362]

Cohen J. A power primer. Psychological Bulletin. 1992; 112:155–159. [PubMed: 19565683]

D'Avanzato C, Joormann J, Siemer M, Gotlib IH. Emotion regulation in depression and anxiety: Examining diagnostic specificity and stability of strategy use. Cognitive Therapy and Research. 201310.1007/s10608-013-9537-0

Donaldson C, Lam D. Rumination, mood and social problem-solving in major depression. Psychological Medicine. 2004; 34:1309–1318. [PubMed: 15697057]

Durbin CE, Klein DN, Hayden EP, Buckley ME, Moerk KC. Temperamental emotionality in preschoolers and parental mood disorders. Journal of Abnormal Psychology. 2005; 114:28–37. [PubMed: 15709809]

Ehring T, Fischer S, Schnulle J, Bosterling A, Tuschen-Caffier B. Characteristics of emotion regulation in recovered depressed versus never depressed individuals. Personality and Individual Differences. 2008; 44:1574–1584.

Ehring T, Tuschen-Caffier B, Schnulle J, Fischer S, Gross JJ. Emotion regulation and vulnerability to depression: Spontaneous versus instructed use of emotion suppression and reappraisal. Emotion. 2010; 10:563–572. [PubMed: 20677873]

Erber R, Tesser A. Task effort and the regulation of mood: The absorption hypothesis. Journal of Experimental Social Psychology. 1992; 28:339–359.

Erk S, Mikschl A, Stier S, Ciaramidaro A, Gapp V, Weber B, Walter H. Acute and sustained effects of cognitive emotion regulation in major depression. Journal of Neuroscience. 2010; 30:15726–15734. [PubMed: 21106812]

Fox NA. The development of emotion regulation: Biological and behavioral considerations. Monographs of the Society for Research in Child Development. 1994; 59(2-3, Serial No. 240)

Gerrards-Hesse A, Spies K, Hesse FW. Experimental inductions of emotional states and their effectiveness: A review. British Journal of Psychology. 1994; 85:55–78.

Gilbert KE. The neglected role of positive emotion in adolescent psychopathology. Clinical Psychology Review. 2012; 32:467–481. [PubMed: 22710138]

Greening SG, Osuch EA, Williamson PC, Mitchell DGV. The neural correlates of regulating positive and negative emotions in medication-free major depression. Social Cognitive and Affective Neuroscience. 201310.1093/scan/nst027

Gross JJ, Levenson RW. Emotion elicitation using films. Cognitive Emotion. 1995; 9:87–108.

Gross JJ, Muñoz RF. Emotion regulation and mental health. Clinical Psychology. 1995; 2:151–164.

Harman C, Rothbart MK, Posner MI. Distress and attention interactions in early infancy. Motivation and Emotion. 1997; 21:27–43.

Isen AM. Asymmetry of happiness and sadness in effects on memory in normal college students: Comment on Hasher, Rose, Zacks, Sanft, and Doren. Journal of Experimental Psychology: General. 1985; 114:388–391.

Joormann J, Cooney RE, Henry ML, Gotlib IH. Neural correlates of automatic mood regulation in girls at high risk for depression. Journal of Abnormal Psychology. 2012; 121:61–72. [PubMed: 21895344]

Joormann J, Gotlib IH. Emotion regulation in depression: Relation to cognitive inhibition. Cognition and Emotion. 2010; 24:281–298. [PubMed: 20300538]

Joormann J, Siemer M. Memory accessibility, mood regulation, and dysphoria: Difficulties in repairing sad mood with happy memories? Journal of Abnormal Psychology. 2004; 113(2):179–188. [PubMed: 15122938]

Joormann J, Siemer M, Gotlib IH. Mood regulation in depression: Differential effects of distraction and recall of happy memories on sad mood. Journal of Abnormal Psychology. 2007; 116:484–490. [PubMed: 17696704]

Josephson BR, Singer JA, Salovey P. Mood regulation and memory: Repairing sad moods with happy memories. Cognition and Emotion. 1996; 10:437–444.

Kanske P, Heissler J, Schonfelder S, Wessa M. Neural correlates of emotion regulation deficits in remitted depression: The influence of regulation strategy, habitual regulation use, and emotional valence. NeuroImage. 2012; 61:686–693. [PubMed: 22613776]

Kessler RC, Petukhova M, Sampson NA, Zaslavsky AM, Wittchen HU. Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. International Journal of Methods in Psychiatric Research. 2012; 21:169–184. [PubMed: 22865617]

Kessler RC, Walters EE. Epidemiology of DSM-III-R major depression and minor depression among adolescents and young adults in the National Comorbidity Survey. Depression and Anxiety. 1998; 7:3–14. [PubMed: 9592628]

Kopp CB. Regulation of distress and negative emotions: A developmental view. Developmental Psychology. 1989; 25:343–354.

Kovacs M, Feinberg TL, Crouse-Novak MA, Paulauskas SL, Finkelstein R. Depressive disorders in childhood: I. A longitudinal prospective study of characteristics and recovery. Archives of General Psychiatry. 1984; 41:229–237. [PubMed: 6367688]

Kovacs, M.; Staff, MHS. Children's Depression Inventory (CDI 2), 2nd Edition: Technical Manual. North Tonawanda, NY: Multi-Health Systems; 2011.

Kovacs M, Rottenberg J, George C. Maladaptive mood repair responses distinguish young adults with early onset depressive disorders and predict future depression outcomes. Psychological Medicine. 2009; 39:1841–1854. [PubMed: 19379534]

Marroquín B. Interpersonal emotion regulation as a mechanism of social support in depression. Clinical Psychology Review. 2011:1276–1290. [PubMed: 21983267]

Morris WN, Reilly NP. Toward the self-regulation of mood: Theory and research. Motivation and Emotion. 1987; 11:215–249.

Olino TM, Lopez-Duran NL, Kovacs M, George CJ, Gentzler AL, Shaw DS. Developmental trajectories of positive and negative affect in children at high and low familial risk for depressive disorder. Journal of Child Psychology and Psychiatry. 2011; 52:792–799. [PubMed: 21039488]

Parrott WG, Sabini J. Mood and memory under natural conditions: evidence for mood-incongruent recall. Journal of Personality and Social Psychology. 1990; 59:321–336.

Perlman G, Simmons AN, Wu J, Hahn KS, Tapert SF, Max JE, Paulus MP, Brown GG, Frank GK, Campbell-Sills L, Yang TT. Amygdala response and functional connectivity during emotion regulation: A study of 14 depressed adolescents. Journal of Affective Disorders. 2012; 139:75–84. [PubMed: 22401827]

Reijntjes A, Stegge H, Terwogt MM, Kamphuis JH, Telch MJ. Emotion regulation and its effects on mood improvement in response to an in vivo peer rejection challenge. Emotion. 2006; 6:543–552. [PubMed: 17144746]

Sherrill JT, Kovacs M. Interview Schedule for Children and Adolescents (ISCA). Journal of the American Academy Child and Adolescent Psychiatry. 2000; 39(1):67–75.

Singer AR, Dobson KS. An experimental investigation of the cognitive vulnerability to depression. Behaviour Research and Therapy. 2007; 45:563–575. [PubMed: 16797484]

Singer AR, Dobson KS. The effect of the cognitive style of acceptance on negative mood in a recovered depressed sample. Depression and Anxiety. 2009; 26:471–479. [PubMed: 19067318]

Tamas Z, Kovacs M, Gentzler AL, Tepper P, Gádoros J, Kiss E, Kapornai K, Vetró Á. The relations of temperament and emotion self-regulation with suicidal behaviors in a clinical sample of depressed children in Hungary. Journal of Abnormal Child Psychology. 2007; 35:640–652. [PubMed: 17530394]

Thayer RE, Newman JR, McClain TM. Self-regulation of mood: Strategies for changing a bad mood, raising energy, and reducing tension. Journal of Personality and Social Psychology. 1994; 67:910–925. [PubMed: 7983582]

Tomarken AJ, Keener AM. Frontal brain asymmetry and depression: A self-regulatory perspective. Cognition and Emotion. 1998; 12:387–420.

Van Dillen LF, Koole SL. Clearing the mind: A working memory model of distraction from negative mood. Emotion. 2007; 7:715–723. [PubMed: 18039038]

Werner-Seidler A, Moulds ML. Mood repair and processing mode in depression. Emotion. 2012; 12:470–478. [PubMed: 22023367]

Zisook S, Lesser I, Stewart J, Wisniewski SR, Balasubramani GK, Fava M, Gilmer WS. Effect of age at onset on the course of major depressive disorder. American Journal of Psychiatry. 2007; 164:1539–154. [PubMed: 17898345]

Key Points

- It has been shown that depressed and depression-prone adults report problems in attenuating sad mood in daily life, but in the laboratory, they typically perform as well as never depressed controls under conditions of instructed mood repair.
- This study found that adolescents with histories of childhood-onset MDD also are likely to have problems in attenuating sadness in daily life, but in addition, are less effective than control peers in mood repair performance in the laboratory.
- Although it appears that childhood-onset depression has lasting adverse effects on mood repair, the modest extent of performance impairment among such adolescents suggests that the requisite abilities have been preserved to some extent. This finding can inform prevention and treatment efforts for youths with MDD.

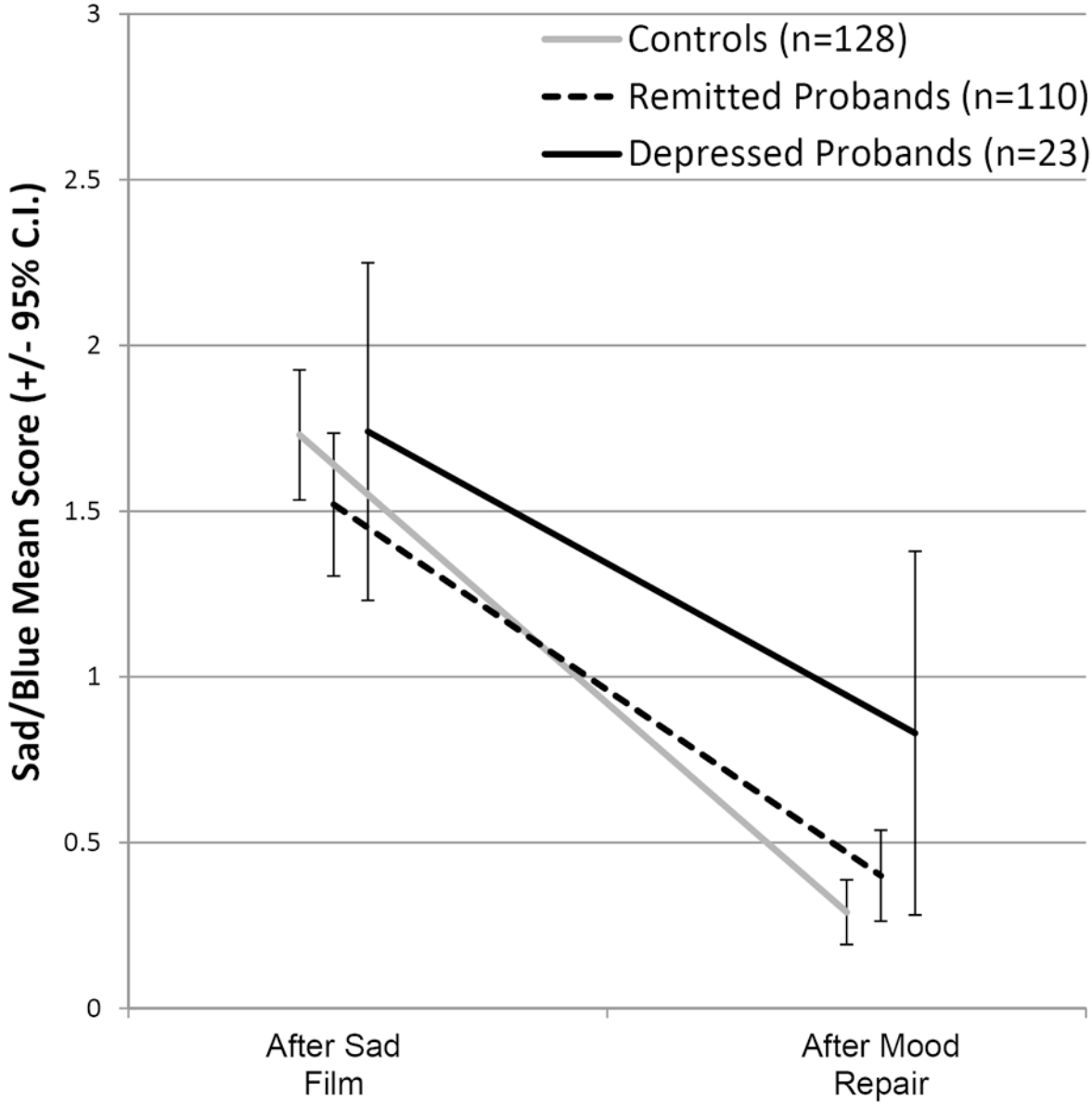

Figure 1.
Mood repair pooled results for both conditions.

Table 1

Selected Characteristics of Subject Groups

	Subject Group		
Variable	**Controls (N=128)**	**Remitted Probands (N=110)**	**Depressed Probands (N=23)**
Sex, Male n (%)	80 (63)	64 (58)	15 (65)
Age in Years	15.79_b (2.15)	16.98_a (1.41)	16.24_a (1.59)
# of MDD Episodes	--	1.52_b (0.67)	1.74_a (0.62)
Age at First MDD Episode (years)	-.-	8.87_a (1.80)	9.25_a (2.51)
CDI-2	4.78_c (4.24)	10.08_b (6.40)	18.33_a (7.12)
FAM Adaptive Mood Repair Score	25.03_b (8.68)	19.84_a (7.82)	19.48_a (8.56)
FAM Maladaptive Mood Repair Score	7.80_c (5.15)	10.71_b (6.32)	17.59_a (7.22)

Values are Mean (SD), unless otherwise noted.

Means with the same subscript within rows are not significantly different at $p < .05$ in planned contrasts.

MDD = Major Depressive Disorder; CDI-2 = Children's Depression Inventory–2; FAM= Feelings and Me questionnaire

Table 2

Ratings of Feeling Sad/Blue (M; S.E.) by Subjects At Three Time Points

Mood Repair Strategy	Time Point	Subject Group		
		Controls	Remitted Probands	Depressed Probands
Attention		(n=68)	(n=48)	(n=10)
Refocusing	Baseline	0.47 ± 0.10	0.70 ± 0.17	1.10 ± 0.50
	Post-Mood Induction	1.79 ± 0.14	1.31 ± 0.13	2.00 ± 0.47
	Post-Mood Repair	0.32 ± 0.07	0.38 ± 0.10	0.90 ± 0.41
Positive		(n=60)	(n=62)	(n=13)
Autobiographical	Baseline	0.28 ± 0.07	0.60 ± 0.13	1.46 ± 0.44
Memory	Post-Mood Induction	1.66 ± 0.13	1.69 ± 0.17	1.54 ± 0.30
	Post-Mood Repair	0.24 ± 0.08	0.41 ± 0.09	0.77 ± 0.39
Combined		(n=128)	(n=110)	(n=23)
Sample	Baseline	0.38 ± 0.07	0.65 ± 0.11	1.30 ± 0.33
	Post-Mood Induction	1.73 ± 0.10	1.52 ± 0.11	1.74 ± 0.26
	Post-Mood Repair	0.29 ± 0.05	0.40 ± 0.07	0.83 ± 0.28