2016

Familiality of mood repair responses among youth with and without histories of depression.

Lauren M. Bylsma
Szeged University

Ilya Yaroslavsky
Cleveland State University

Johnathan Rottenberg
University of South Florida, rottenberg@usf.edu

Enikő Kiss
Szeged University

Krisztina Kapornai
Szeged University

See next page for additional authors

Authors
Lauren M. Bylsma, Ilya Yaroslavsky, Johnathan Rottenberg, Enikő Kiss, Krisztina Kapornai, Kitti Halas, Roberta Dochnal, Eszter Lefkovics, Ildikó Baji, Ágnes Vetró, and Maria Kovacs

HHS Public Access

Author manuscript

Cogn Emot. Author manuscript; available in PMC 2017 June 01.

Published in final edited form as:

Cogn Emot. 2016 June ; 30(4): 807–816. doi:10.1080/02699931.2015.1025707.

Familiality of mood repair responses among youth with and without histories of depression

Lauren M. Bylsma[1], **Ilya Yaroslavsky**[2], **Jonathan Rottenberg**[3], **Enikő Kiss**[4], **Krisztina Kapornai**[4], **Kitti Halas**[4], **Roberta Dochnal**[4], **Eszter Lefkovics**[4], **Ildikó Baji**[4], **Ágnes Vetró**[4], and **Maria Kovacs**[1]

[1]University of Pittsburgh, Pittsburgh, PA

[2]Cleveland State University, Cleveland, OH

[3]University of South Florida, Tampa, FL

[4]Szeged University, Szeged, Hungary

Abstract

Affect regulation skills develop in the context of the family environment, wherein youths are influenced by their parents', and possibly their siblings', regulatory responses and styles. Regulatory responses to sadness (mood repair) that exacerbate or prolong dysphoria (maladaptive mood repair) may represent one way in which depression is transmitted within families. We examined self-reported adaptive and maladaptive mood repair responses across cognitive, social, and behavioral domains in Hungarian 11–19 year old youth and their parents. Offspring included 214 probands with a history of childhood-onset depressive disorder, 200 never depressed siblings, and 161 control peers. Probands reported the most problematic mood repair responses, with siblings reporting more modest differences from controls. Mood repair responses of parents and their offspring, as well as within sib-pairs, were related, although results differed as a function of the regulatory response domain. Results demonstrate familiality of maladaptive and adaptive mood repair responses in multiple samples. These familial associations suggest that relationships with parents and siblings within families may impact the development of affect regulation in youth.

Keywords

Mood repair; affect regulation; familiality; youth; siblings

Mood repair refers to affect regulation responses that specifically entail the attenuation of sadness or dysphoria (e.g., Morris & Reilly, 1987). Affect regulation responses have been categorized as maladaptive or adaptive (see Aldao et al., 2010, for review). Adaptive strategies, including reappraisal, distraction, and problem solving, typically reduce the intensity or duration of dysphoric states such as sadness, and their use has been associated with emotional health. In contrast, maladaptive ways of responding to negative affect states,

Correspondence: Lauren M. Bylsma, Department of Psychiatry, University of Pittsburgh, Western Psychiatric Institute and Clinic, 3811 O'Hara St., Pittsburgh, PA 15213. BylsmaL@pitt.edu.

such as rumination, avoidance, and suppression, are usually associated with increased distress and psychopathology.

Affect regulation skills emerge over the course of development, in which family interactions are particularly important (e.g., Grusec, 2011; Kopp, 1989; Morris, Silk, Steinberg, Myers & Robinson, 2007). It is widely accepted that children's developing repertoires of affect regulation strategies reflect a combination of social modeling of parental repertoires, differential parental reinforcement of offspring responses, and related contextual influences (Grusec, 2011; Morris et al., 2007; Thompson, 1994). Presumably, then, parental and offspring regulatory strategies would overlap. Indeed, Stansbury and Sigman (2000) reported that affect regulation responses used by preschoolers (categorized as comforting, instrumental, distraction, or cognitive) were highly correlated with the types of responses that parents used to help their children during frustrating emotional episodes. Bariola, Hughes, and Gullone (2012) found that mothers' self-reported use of emotion suppression strategies in response to negative or positive emotion was significantly correlated with offspring's use of suppression strategies; however, there was no correspondence in the use of cognitive reappraisal. Saritas, Grusec and Gencoz (2013) extended this work, also finding a significant relationship between the affect regulation difficulties of mothers and their adolescent daughters. However, most work on transmission of affect regulation responses between parents and offspring has assessed only one or two specific regulatory responses, and individual differences have not been considered.

Another understudied area is the role of siblings in the transmission of affect regulation responses within families. While we are not aware of any published articles on this topic, related work clearly suggests that siblings influence each other's emotional development. For example, conduct problems in one child increases the risk that siblings will develop similar problems (Brody, Kim, Murry & Brown, 2003). Work on affect regulation in the context of sibling interactions has demonstrated that older siblings who ignored the distress of a younger sibling were more likely to display their own personal distress reactions (Volling, 2001). These results suggest that when older siblings have difficulty regulating their own emotions, they are less likely to help younger siblings to use appropriate affect regulation responses.

Individual differences in affect regulation strategy use have been viewed as critical for explaining variation in adaptation and risk for adaptive functioning and have been implicated in various psychiatric disorders, including depression. Mood repair is highly salient for understanding depression. Indeed, depressed children, children at high risk for depression, as well as depressed adults, have mood repair problems (Garber, Braafladt & Weiss, 1995; Kovacs, Rottenberg, & George, 2009; Kovacs & Lopez-Duran, 2010; Silk, Shaw, Skuban, Oland & Kovacs, 2006). Compared to controls, depression-prone individuals typically display a more limited repertoire of adaptive mood repair strategies that can alleviate sadness, alongside a more extensive repertoire of maladaptive responses that are likely to prolong or intensify distress (Kovacs et al., 2009). Thus, it is plausible that offspring of depressed parents, or the unaffected siblings of depressed youths, would both evidence mood repair patterns that parallel the mood repair patterns of their affected

relative. However, to our knowledge, there have been no investigations of mood repair problems in siblings unaffected by, but at high risk for depression.

In the present study, we examine the familiality of mood repair response domains in various pairs of relatives (parent and offspring; siblings). Our samples include youth with a history of childhood-onset depression (probands), their unaffected siblings, and emotionally healthy control peers, along with their parents. Importantly, our sample consists primarily of adolescents, a population that has been understudied in developmental affect regulation research (see Bariola, et al., 2011). Given previous findings, we hypothesized that: (1) depressed youth and the unaffected siblings of depressed youths will both evidence mood repair deficits relative to controls, (2) parents' mood repair responses will be related to their offspring's mood repair responses in all three groups of subjects, and (3) mood repair responses will be related among siblings. We also explored whether depression vulnerability (history of depression in a parent or sibling and youth's own prior history) moderated the degree of correspondence of maladaptive mood repair responses among the targeted relative pairs.

Method

Subjects

We examined 3 groups of youths along with their parents. One group included 214 probands whose histories of childhood-onset major depressive disorder (MDD) were established previously and were a subset of a larger sample gathered in Hungary for a prior study (henceforth designated as the *archival* study; see Kiss et al., 2007). Another group of youth included 200 high-risk siblings of probands, namely, siblings who had no history of depressive disorders (72 siblings who developed depressions were not included in the current study). The analyses presented here included probands and siblings from a total of 291 families. There were 214 probands, who had altogether 106 siblings (10 probands had 2 available siblings; 118 probands had no sibling in this study). There were also 94 siblings for whom data on the linked proband was not available (representing 77 families). Thus, the data included 96 probands who could be linked to a sibling (including 10 who could be matched to 2 siblings) and 34 siblings with no matching proband who could be linked to another sibling (i.e., 17 sibling pairs). The final group of youth also included 161 normal controls who never had any major psychiatric disorder.

Probands for the archival study had been recruited in multiple mental health and guidance facilities across Hungary if they had current or recent DSM-IV (APA, 2000) depressive disorder, were 7–14 years old at initial screen and not mentally retarded, and also met several other criteria. Six probands and 8 siblings with bipolar disorder family history were excluded from the present analyses. Control subjects (not part of the archival study) were identified in public elementary and secondary schools in the 3 cities where most of the proband in the current study resided. Controls were recruited to approximate the sex and age distribution of probands.

Ages of offspring ranged from 11- to 19-years (probands: M=16.99, SD=1.41; siblings: M=15.91, SD=2.16; controls: M=15.85, SD=2.14) and males constituted 64% of the

probands, 47% of the siblings, and 65% of the controls. Consistent with the racial distribution in Hungary, probands were 96% Caucasian, 2% biracial (or other), and 2% Roma; siblings were 96% Caucasian, 2% biracial (or other), and 3% Roma; controls were 99% Caucasian, and 1% biracial (or other).

At the diagnostic assessment for the current study, 59% of probands had one MDD episode, 32% had two episodes, and 10% had three or more episodes; 184 subjects were in full remission from their most recent MDD episode, while 30 (14%) were currently in a depressive episode. Mean age at onset of first MDD episode in the proband youth was 9.04 years (SD=1.89 years). Of the probands, 39% had co-morbid anxiety disorder and 37% had behavioral disorder. Among siblings, 7% had a current anxiety disorder and 6% had a behavioral disorder. Controls had no history of any psychiatric disorder.

Proband/sibling parents were aged 35.8 years, on average (SD=5.35) while control parents were aged 33.3 years, on average (SD=5.01). Most participating parents were mothers (controls: 89%, probands: 88%). Parents' racial distribution paralleled that of their offspring. Control parents were relatively more educated than were parents of probands and siblings: 52% of control parents had a college or higher degree (versus 15% of proband/sibling parents). Finally, 12% of control parents and 30% of proband parents had lifetime histories of a depressive disorder (including 1% of control parents and 9% of proband parents with a current depressive disorder).

Diagnostic Assessment Procedures

As described in detail (e.g., Kiss et al., 2007; Tamás et al., 2007), caseness for each proband was established during the archival study via a stringent procedure that included standardized psychiatric diagnostic evaluations (involving the youth and a parent informant) by different trained interviewers (child psychiatrists and psychologists), each of whom generated DSM-IV mood-disorder diagnoses, which were then subjected to a final best-estimate diagnostic procedure. The diagnoses were based on the Interview Schedule for Children and Adolescents: Diagnostic version (ISCA-D), a semi-structured tool, described in previous publications (e.g., Kiss et al., 2007), which covers all mood disorders and the most common non-affective disorders using DSM-IV criteria (APA, 2000). We have previously reported acceptable inter-rater reliability on the ISCA-D symptom ratings (kappas=.63–.92 for current MDD from child interviews and .65–.87 from parent interviews; Kiss et al., 2007). Parental depression history was determined by trained clinicians using the structured Mini-International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998), which yields diagnoses based on DSM-IV and ICD-10 criteria. A study using 4 clinicians yielded high inter reliability on the M.I.N.I for mood disorder diagnoses (kappa=.92).

This study was approved by the institutional review boards of the University of Pittsburgh and the Hungarian clinical research sites. Parents provided written informed consent, and youth provided either assent or consent (depending on their ages). All procedures and instruments used in this study were first developed in English, translated into Hungarian, and then back translated by bi-lingual child psychiatrists and clinical psychologists; discrepancies between versions were resolved using an iterative procedure.

Mood Repair Measures

Feelings and Me (FAM)—The self-rated Feelings and Me questionnaire for adults (FAM-A; Kovacs, Rottenberg, & George, 2009) and a parallel version with age-appropriate language for youth ages 7–17 (FAM-C; Tamás et al., 2007) were used to assess mood repair response repertoires. The FAM is a rationally derived questionnaire, which surveys the use of responses to depressed, dysphoric mood, focusing on those often reported in the literature to maintain or to attenuate those affects (see Tamás et al., 2007 regarding the development of the FAM). It yields an Adaptive and a Maladaptive total score (based on 32 and 22 items, respectively), each of which encompasses sub-scores for Cognitive, Behavioral, and Social regulatory responses. Respondents rate from "0=not true of me" to "2=many times true of me" the extent to which items characterize them when feeling sad or upset. Cognitive subscale items include "think about things being bad forever" (maladaptive) or "think of something fun" (adaptive). Behavioral subscale items include "pick my skin, pull my hair, or bite my fingers" (maladaptive) or "listen to fun music" (adaptive). Social subscales include items such as "yell or scream at my family" (maladaptive) or "look for a teacher or other adult to talk to" (adaptive).

The FAM demonstrated good psychometric properties in the present sample and in prior work with clinical and non-clinical samples in the US and in Hungary (Kovacs et al., 2009; Tamás et al., 2007). Both the adult (FAM-A) and child (FAM-C) total scores were highly internally consistent in the present study (α's=.85–.87), mirroring prior reports (Kovacs et al., 2009; Tamás et al., 2007). Internal consistency is adequate for most sub-scale scores for both the adult (Adaptive Behavioral α=.77, Cognitive α=.73, Social α=.74; Maladaptive Behavioral α=.59, Cognitive α=.84, Social α=.65) and child versions (Adaptive Behavioral α=.73, Cognitive α=.74, Social α=.71; Maladaptive Behavioral α=.58, Cognitive α=.80, Social α=.63). Test-retest reliability of total scores over one year has been satisfactory in youth (Tamás, et al., 2007) and adult samples (Kovacs et al., 2009).

Mirroring construct validity, FAM-C maladaptive scores were shown to correlate with depression symptoms (r=.64, p<.0001) and rumination (r=.71, p<.0001; Tamás, et al., 2007) in a large clinical sample of youths. Likewise, among adults, maladaptive FAM scores and rumination (rs =.74–.80, ps<.0001) and adaptive FAM scores and distraction (rs =.65–.67, ps<.001) are significantly correlated (Kovacs et al., 2009). A previously conducted confirmatory factor analysis using n=2,558 school-based youths supports the validity of the 3 subdomains: we obtained excellent fit for the Adaptive subscales (CFI=0.95, RMSEA=0.07) and adequate fit for the Maladaptive subscales (CFI=.91, RMSEA=.06). Further, among adults with and without a history of early onset depression in a longitudinal study, FAM Maladaptive score prospectively predicted a recurrent episode of major depression after controlling for other key predictors (Kovacs et al., 2009).

Statistical Analyses

Differences on FAM scores between probands, siblings, and controls were examined utilizing multilevel models to take into account within-family clustering. ICCs ranged from .06 to .20 for offspring Adaptive FAM scores, and from .03 to .11 for the Maladaptive FAM scores. Age and sex of offspring were included in these models, as we have previously

found these variables to relate to FAM scores. Significant group effects were examined using post-hoc tests that controlled for multiple comparisons. The first step in testing our hypothesis regarding correspondence between parent-offspring FAM scores was to conduct descriptive correlational analyses. Then, we conducted follow-up multilevel models incorporating the family clustering effect and other potential confounding variables: sex and age of parents and offspring, group membership, and offspring's current depression status. We also explored whether parental depression history, youth's own depression history (proband status), or being a sibling of an affected proband (sibling status) moderated the relationship between parent and offspring FAM scores by including the corresponding interaction terms in the models. To test our hypothesis of sibling-sibling correspondence in FAM scores, in families with multiple siblings, two were selected at random, irrespective of depression histories. We then followed up with regression analyses, controlling for the age, sex, and depression history of each sibling in the sibling pairs.

Results

Sex and age were significant predictors of FAM scores: females had higher Adaptive and Maladaptive scores than males ($p<.001$) and older subjects had lower Maladaptive scores than younger ones ($p<.05$). Importantly, the groups differed on all FAM scores as predicted (Table 1, $ps<.01$). Pairwise post-hoc LSD tests showed that probands reported lower Adaptive and higher Maladaptive FAM scores relative to controls across all mood repair response domains ($ps<.001$). Further, with few exceptions, siblings' mean FAM scores fell midway between the mean scores of controls and probands, with many of these comparisons being statistically significant (Table 1).

Relations Among Parents' and Offspring' Mood Repair Scores

We predicted that mood repair responses of parents and their offspring would be associated in all groups. To test this hypothesis, correlational analyses were first run across the entire sample on all scores. Overall, there were significant associations between parent and offspring Maladaptive ($r=.13$, $p<.01$) and Adaptive FAM Scores ($r=.13$, $p<.01$) and their respective Behavioral and Social subscales, with the exception of the Cognitive Subscales (Table 2).

To control for possible confounding variables that may impact the associations between mood repair of parents and their offspring, a series of multilevel regression models were performed with youths' FAM scores (Adaptive and Maladaptive scores and corresponding subscale scores) as the dependent variables. The predictor variables were: corresponding parental FAM score, parent's age, parent's sex (female=0, male=1), offspring's age, offspring's sex (female=0, male=1), proband status, (proband=1, not proband=0), sibling status (sibling=1, not sibling=0), and offspring's current depression status (yes=1, no=0). Results revealed that Parental Adaptive scores significantly predicted Offspring Adaptive scores (b=.09, t=2.28, p<.05), and Parental Maladaptive scores marginally predicted Offspring Maladaptive scores (b=.07, t=1.72, p=.09). Proband and sibling status, as well as offspring sex, continued to be significant predictors of mood repair ($ps<.05$) and mirrored the patterns described above (see Table 2). Offspring depression predicted higher

Maladaptive scores (b=7.67, t=6.57, p<.001), but was unrelated to Adaptive scores. Parent's sex, parent's age, and offspring's age were not significant predictors in the model (*p*s>.05) and were not considered further.

In follow-up analyses of parent-offspring associations across mood repair domains, parental Adaptive Behavioral and Social subscale scores continued to predict corresponding offspring Adaptive Behavioral (b=.10, t=2.31, p<.05) and Social (b=.11, t=2.78, p<.01) scale scores. Similarly, parental Maladaptive Behavioral scores predicted corresponding scores in their offspring (b=.10, t=2.13, p<.05).

Finally, in exploratory analyses with the Adaptive and Maladaptive overall scale scores, none of the interaction terms for depression vulnerability was significant (ps>.26). These results indicate that none of the risk variables (parental depression history, youth depression history, sibling status) affected the associations between parent and offspring FAM scores.

Relations Among Siblings' Mood Repair Scores

Overall, paired siblings reported similar Adaptive (r=.21, p<.01) but not Maladaptive (r=.12, p>.05) response total scores. Adaptive Cognitive (p<.001) and Behavioral (p<.05) subscales were correlated between paired siblings (Table 2), while the Social subscales were not (ps<.05; Table 2). Among the Maladaptive subscales, only the Cognitive subscales correlated between paired siblings (p<.05; Table 2).

Since siblings differed on sex, age, and depression histories, these were controlled in a series of follow-up analyses, with FAM scores of sibling 1 as independent variables and the corresponding FAM scores of sibling 2 as dependent variables. These analyses revealed significant sibling pair correspondences in Adaptive (β=.23, t=2.87, p<.01) but not Maladaptive total scores (p>.05) and for various mood repair domains. Namely, Adaptive Cognitive (β =.27, t=3.59, p<.001) and Behavioral (β=.20, t=2.52, p<.05) subscale scores of sibling 1 significantly predicted the same scale scores for sibling 2, and Maladaptive Cognitive subscale scores of sibling 1 significantly predicted the same scale scores for sibling 2 (β=.19, t=2.32, p<.05).

Discussion

Consistent with previous reports of mood repair difficulties among depressed and high-risk individuals (Kovacs, et al., 2009; Kovacs & Lopez-Duran, 2010; Silk, et al., 2006), we found that youth probands with depression histories reported higher rates of maladaptive and lower rates of adaptive mood repair responses compared to controls. Additionally, currently depressed probands were characterized by higher rates of maladaptive mood repair responses than probands with remitted depression. Finally, to our knowledge, this is the first report of impaired mood repair among never depressed, high-risk siblings (of youth probands with depression histories). Possibly, greater use of maladaptive mood repair responses and reduced use of adaptive mood repair may contribute to risk for future depression.

Our study comprehensively tested whether mood repair responses are familial by assessing cognitive, behavioral, and social regulatory responses, using different types of relatedness (parent-offspring; sibling pairs), and employing various samples of youth (youths with a history of childhood-onset depression, their high-risk siblings, and control peers). Consistent with our hypotheses, mood repair responses showed significant associations between parents and offspring, even after controlling for possible confounding variables that previous studies have not addressed (e.g., age and sex). In other words, parents with extensive repertoires of adaptive mood repair responses were likely to have children who also endorsed a host of adaptive ways of responding to their own sadness or distress. Likewise, there was a relationship between parents' and offsprings' reports of maladaptive mood repair responses. Therefore, our results are consistent with findings that demonstrated relationships between parents' and offspring's affect regulation responses (e.g., Bariola et al., 2012; Stansbury & Sigman, 2000) and the proposition that parental affect regulation strategies have an impact on how offspring respond to their own affect experiences (e.g., Grusec, 2011; Morris et al., 2007; Thompson, 1994).

The extent of parent-offspring correspondence in mood repair responding differed as a function of the regulatory response domain. Namely, while parents and their children demonstrated correspondence in their social and behavioral regulatory responses to sadness, this was not the case for cognitive mood repair responses. Possibly, social and behavioral mood repair responses (which are typically observable) are more easily modeled by offspring. Another possible explanation is that youth are less likely to use cognitive regulatory strategies than are adults (e.g., Garnefski & Kraaij, 2006). This developmental phenomenon would result in a stronger association of cognitive responses among siblings (with both youth reporting relatively low use of cognitive strategies) than among parent-offspring pairs. Although no prior study has examined the familiality of cognitive mood repair, it has been reported that cognitive explanatory styles are familial (e.g., Pearson et al., 2013). However, our results suggest that when a range of cognitive mood repair response is examined, no familial relationships are observed.

Our study is the first to directly examine the proposition that siblings play important roles in the development of mood repair (Brody et al. 2003; Brody, 2004). We found that siblings were similar with respect to adaptive cognitive and behavioral mood repair response use. The fact that siblings differed in the adaptive use of social agents for mood repair could be age-related: For example, a 10-year-old proband and her 13-year-old sibling are likely to differ in the nature, scope, and accessibility of social networks. Notably, siblings also differed with respect to their overall maladaptive mood repair response repertoires. This would suggest that social modeling/learning across youth siblings is not a key mechanism of maladaptive mood repair regulatory response acquisition, or that the adverse impact of problematic mood repair in youths on their siblings is mitigated by some resilience of the sibling.

Since parental history of depression has been associated with difficulties in modeling and training appropriate affect regulation responses to offspring (e.g., Goodman & Gotlib, 1999), and depressed children themselves show mood repair difficulties (e.g., Garber, Braafladt & Weiss, 1995), we explored whether personal history of depression might affect

the correspondence between parents' and offspring's mood repair. We found, however, that depression history, or risk status, did not alter parent-child associations in mood repair responses.

In summary, using self-reports, we confirmed across multiple samples that adaptive and maladaptive mood repair responses are familial, but that the extent of the association differs as a function of type of relatedness and the regulatory response domain. The fact that adaptive as well as maladaptive response repertoires showed transmission from parent to child underscores the importance of parents in the developmental unfolding of their children's responses to sadness. Our study was the first to demonstrate that siblings share adaptive mood repair styles, underscoring the potential influence of siblings in the development of competent affective regulation. Our study was also the first to show comparable familiality of mood repair responses across samples of youth at low- and high-risk for depression.

Results of the present study have implications for early prevention efforts among high-risk families. For example, the findings suggest that reducing parental maladaptive mood repair responses and increasing adaptive ones, particularly in the social and behavioral domains, may benefit their children and thereby reduce their risk for psychopathology. Quite possibly, improving youths' mood repair response repertoires may additionally benefit their parents' mood repair. There may also be an advantage to targeting high-risk siblings in the same family with interventions aimed to improve adaptive mood repair responses, as our results point to siblings' evident influence on each other in this domain.

Our findings should be viewed in light of the study's limitations. Since most participating parents were mothers, we were unable to examine if parent-child mood repair concordance differs as a function of parent sex, an important goal for future research. Although our results were relatively consistent across mood repair, the effect sizes were quite modest, and it is likely that there are many other factors that contribute to the development of mood repair. Also, while the present study did not address genetic contributors to heritability of mood repair, there is some evidence that genetic factors likely play a role as well (Moore et al., 2013). Also, given that our sibling analyses are based on a sample of children with a family loading for depression (as we did not recruit siblings for controls), findings may not generalize to typical families. In addition, it would be useful for future studies to examine observable mood repair behaviors, not just self-reports, particularly because our effect sizes were quite modest. Future research would also benefit from examining the regulation of other negative emotions as well as positive emotions. Further, given that our study was cross-sectional, it could not establish temporal or causal relationships among the key variables. Undoubtedly, multiple familial and contextual factors shape the development of mood repair in offspring, which underscores the need build more complex models of the familial transmission of mood repair responses.

Acknowledgement

This research was supported by NIH Grants MH-084938 and MH-056193,

References

1. 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]
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders-Text Revision. 4th ed.. Washington, DC: Author; 2000.
3. Bariola E, Gullone E, Hughes EK. Child and adolescent emotion regulation: The role of parental emotion regulation and expression. Clinical Child and Family Psychology Review. 2011; 14:198–122. [PubMed: 21424275]
4. Bariola E, Hughes EK, Gullone E. Relationships between parent and child emotion regulation strategy use: A brief report. Journal of Child Family Studies. 2012; 21:443–448.
5. Brody GH. Siblings' direct and indirect contributions to child development. Current Directions in Psychological Science. 2004; 13(3):124–126.
6. Brody GH, Kim S, Murry VM, Brown AC. Longitudinal direct and indirect pathways linking older sibling competence to the development of younger sibling competence. Developmental Psychology. 2003; 39(3):618. [PubMed: 12760528]
7. Garber J, Braafladt N, Weiss B. Affect regulation in depressed and nondepressed children and young adolescents. Development and Psychopathology. 1995; 7(01):93–115.
8. Garnefski N, Kraaij V. Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. Personality and Individual Differences. 2006; 40(8):1659–1669.
9. Goodman SH, Gotlib IH. Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. Psychological Review. 1999; 106(3):458–490. [PubMed: 10467895]
10. Grusec JE. Socialization processes in the family: Social and emotional development. Annual Review of Psychology. 2011; 62:243–269.
11. Kiss E, Gentzler AM, George C, Kapornai K, Tamás Z, Kovacs M, Vetró Á. Factors influencing mother–child reports of depressive symptoms and agreement among clinically referred depressed youngsters in Hungary. Journal of Affective Disorders. 2007; 100(1):143–151. [PubMed: 17125844]
12. Kopp CB. Regulation of distress and negative emotions: A developmental view. Developmental Psychology. 1989; 25(3):343.
13. Kovacs M, Lopez-Duren N. Prodromal symptoms and atypical affectivity as predictors of major depression in juveniles: Implications for prevention. Journal of Child Psychology and Psychiatry. 2010; 51(4):472–496. [PubMed: 20202041]
14. 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]
15. Moore MN, Salk RH, Van Hulle CA, Abramson LY, Hyde JS, Lemery-Chalfant K, Hill H. Genetic and environmental influences on rumination, distraction, and depressed mood in adolescence. Clinical Psychological Science. 2013; 1(3):316–322. [PubMed: 23956956]
16. Morris WN, Reilly NP. Toward the self-regulation of mood: Theory and research. Motivation and Emotion. 1987; 11(3):215–249.
17. Morris AS, Silk JS, Steinberg L, Myers SS, Robinson LR. The role of the family context in the development of emotion regulation. Social Development. 2007; 16(2):361–388. [PubMed: 19756175]
18. Pearson RM, Fernyhough C, Bentall R, Evans J, Heron J, Joinson C, Stein AL, Lewis G. Association between maternal depressogenic cognitive style during pregnancy and offspring cognitive style 18 years later. American Journal of Psychiatry. 2013; 170:434–441. [PubMed: 23318526]
19. Saritaş D, Grusec JE, Gençöz T. Warm and harsh parenting as mediators of the relation between maternal and adolescent emotion regulation. Journal of Adolescence. 2013; 36(6):1093–1101. [PubMed: 24215956]

20. Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, Dunbar GC. The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. Journal of Clinical Psychiatry. 1998; 59:22–33. [PubMed: 9881538]

21. Silk JS, Shaw DS, Skuban EM, Oland AA, Kovacs M. Emotion regulation strategies in offspring of childhood-onset depressed mothers. Journal of Child Psychology and Psychiatry. 2006; 47(1): 69–78. [PubMed: 16405643]

22. Stansbury K, Sigman M. Responses of preschoolers in two frustrating episodes: Emergence of complex strategies for emotion regulation. The Journal of Genetic Psychology. 2000; 161(2):182–202. [PubMed: 10851681]

23. Tamás Z, Kovacs M, Gentzler AL, Tepper P, Gádoros J, Kiss E, 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(4):640–652. [PubMed: 17530394]

24. Thompson RA. Emotion regulation: A theme in search of definition. Monographs of the Society for Research in Child Development. 1994; 59(2/3):25–52. [PubMed: 7984164]

25. Volling BL. Early attachment relationships as predictors of preschool children's emotion regulation with a distressed sibling. Early Education and Development. 2001; 12(2):185–207.

Table 1

Offspring's self-report Feelings and Me (FAM) Score means (SDs) by group.

FAM Score	Probands	Siblings	Controls	*F* (*ps*<.*01*)	Pairwise LSD Comparison (*ps*<.05)
Adaptive Total	**19.52 (8.64)**	**22.79 (8.80)**	**24.16 (8.73)**	**13.38**	**Control > Sibling > Proband**
Adaptive Cognitive	6.63 (3.38)	7.89 (3.37)	8.65 (3.38)	16.60	Control > Sibling > Proband
Adaptive Social	3.44 (2.61)	4.20 (2.70)	4.46 (3.14)	7.57	Control, Sibling > Proband
Adaptive Behavioral	9.43 (4.69)	10.70 (4.54)	11.05 (4.24)	5.57	Control, Sibling > Proband
Maladaptive Total	**10.59 (7.09)**	**9.88 (6.26)**	**7.52 (5.07)**	**13.78**	**Control < Sibling < Proband**
Maladaptive Cognitive	4.07 (3.58)	3.88 (3.11)	2.89 (2.49)	8.14	Control < Sibling, Proband
Maladaptive Social	2.72 (2.14)	2.47 (1.93)	1.85 (1.72)	11.07	Control < Sibling < Proband
Maladaptive Behavioral	3.31 (2.76)	3.51 (2.29)	2.78 (2.08)	10.01	Control, Sibling < Proband

Note: F-statistics are provided for the group fixed effects. All effects are adjusted for age and sex.

Table 2

Associations among Cognitive, Social, and Behavioral Adaptive and Maladaptive Mood Repair Scores for Parent-Offspring (top) and Sibling-Sibling pairs (bottom).

Adaptive	**Parent Cognitive**	**Parent Social**	**Parent Behavioral**
Offspring Cognitive	0.06	0.11*	0.10*
Offspring Social	0.05	0.13**	0.07
Offspring Behavioral	0.08	0.09*	0.11**
Maladaptive	**Parent Cognitive**	**Parent Social**	**Parent Behavioral**
Offspring Cognitive	0.06	0.05	0.08
Offspring Social	0.15***	0.11**	0.11**
Offspring Behavioral	0.10*	0.09*	0.10*
Adaptive	**Sibling 1 Cognitive**	**Sibling 1 Social**	**Sibling 1 Behavioral**
Sibling 2 Cognitive	0.28***	0.09	0.17*
Sibling 2 Social	0.05	0.08	0.07
Sibling 2 Behavioral	0.09	0.16*	0.19*
Maladaptive	**Sibling 1 Cognitive**	**Sibling 1 Social**	**Sibling 1 Behavioral**
Sibling 2 Cognitive	0.18*	0.02	0.05
Sibling 2 Social	0.09	−0.01	0.04
Sibling 2 Behavioral	0.11	0.01	0.14

Note:

* p<.05,

** p<.01,

*** p<.001