HHS Public Access

Author manuscript

J Abnorm Child Psychol. Author manuscript; available in PMC 2017 August 11.

Published in final edited form as:

J Abnorm Child Psychol. 2011 April ; 39(3): 463–474. doi:10.1007/s10802-010-9473-3.

Expression of anger in depressed adolescents: The role of the family environment

Jennifer Jackson,
University of Melbourne

Peter Kuppens,
University of Leuven and University of Melbourne

Lisa B. Sheeber, and
Oregon Research Institute

Nicholas B. Allen
University of Melbourne

Abstract

The expression of anger is considered to be abnormal in depression, yet its role is only poorly understood. In the present study we sought to clarify this role by examining the moderating influence of the family environment on overall levels of anger expression and anger reactivity in depressed and non-depressed adolescents during conflictual interactions with their parents. One hundred and forty one depressed and non-depressed adolescent participants engaged in a problem-solving task with their parents during which their behavioral expression of anger and heart rate were recorded. The results demonstrate that general levels of parental anger in the family environment (as indicated by the overall level of expressed anger by the parents during the interactions) strongly moderates how depressed differ from non-depressed adolescents in terms of their anger, heart rate and reactivity. Overall, the findings suggest that in depressed adolescents anger is much less adaptively attuned to the environment, consistent with models that predict dysfunction in the regulation of anger that prevents depressed individuals responding adaptively to their social environment.

Keywords

Depression; Anger; Adolescence; Parent-child interactions; Heart rate; Behavioral observation

Anger plays an important role in the experience, onset, and maintenance of depressive symptoms and disorder (Ingram, Trenary, Odom, Berry, & Nelson, 2007; Sheeber et al., 2009; Wenze, Gunthert, & Forand, 2009). Indeed, the sustained presence of irritability is part of the diagnostic criteria for depression in children and adolescents (APA, 2000). The relationship between anger and depression is complex, however, and not well understood. Findings regarding the experience of anger in depressed and non-depressed persons have

Please direct correspondence to Nicholas B. Allen, ORYGEN Youth Health Research Centre and Department of Psychological Sciences, University of Melbourne, Victoria 3010, Australia; nba@unimelb.edu.au.

been inconsistent, with depression being associated with both greater and lesser average levels of anger, as well with both greater and lesser reactivity to anger eliciting events (e.g., Brody, Haaga, Kirk, & Solomon, 1999; Harmon-Jones et al., 2002; Luutonen, 2007; Newman, Gray, & Fuqua, 1999; Painuly, Sharan, & Mattoo, 2007; Sigmon & Nelson-Gray, 1992).

Consideration of the social environment, particularly the family environment, may help to shed light on the inconsistencies in the relationship between depression and the experience and expression of anger. In particular, the emotional climate of the family – such as the overall level of expressed anger by the parents – may moderate an adolescent's expression of anger. Based on recent thinking about the broader social function and adaptive value of depressive behaviors, we present hypotheses regarding how the family environment may moderate differences between depressed and non-depressed youth in both their overall level of anger and in the likelihood of reacting angrily to specific eliciting events. We then present empirical evidence about how the overall level of parental aggression differentially relates to adolescent anger during conflictual interactions with their parents as a function of depressive status. We examine two key indices of adolescent anger during the course of such parent-adolescent interactions: 1) overall levels of adolescent anger; and 2) angry behavior in reaction to angry parental behavior (i.e., anger reactivity).

The moderating role of the family environment in overall levels of adolescent anger

Parents and children tend to mirror each other's affective behavior during the course of conflict (Forgatch, 1989; Kobak, Cole, Ferenz-Gillies & Fleming, 1993; Sheeber, Allen, Davis, & Sorensen, 2000). Given this, adolescents whose parents display high levels of anger would be expected to do the same. However, based on evolutionary theories regarding the function of depression, we postulate that the association between parental and adolescent anger is likely to be different for depressed and non-depressed adolescents. Evolutionary theories of depression suggest that depressed states evolved as adaptations to reduce risk in contexts in which individuals perceive themselves to be subject to attack as a function of lowered social status (Price & Solomon, 1987; Gilbert, 1989, 1992) or in which they are vulnerable to subsequent loss of status (Allen & Badcock, 2003). In these situations, individuals tend to behave submissively (Gilbert, 2000) and to resort to interpersonal behaviors with more certain and less variable outcomes (Allen & Badcock, 2003). These behaviors are adaptive because it is not in a subordinate's interest to instigate or escalate conflicts, or make claims on resources they cannot win or defend.

These changes in social perception and behavior during depression (i.e., submissive behavior and less risky social investments) imply a potential mechanism by which social environments, including the family environment, may influence the experience of anger differentially for depressed and non-depressed adolescents. In family environments where parents express higher levels of anger and hostility, depressed, relative to non-depressed, individuals would be predicted to *suppress* anger as a means to decrease social risk and avoid conflict. Conversely, in family environments characterized by relatively lower levels of

parental anger, depressed adolescents would be expected to express more anger than non-depressed adolescents. This is predicted on the basis that depression is associated with increased feelings of anger (Wenze et al. 2009), and this anger may be expressed more frequently in family environments in which adolescents feel safer and in which conflict is more likely to be resolved successfully. Accordingly, we hypothesize that although parental anger is associated with higher levels of adolescent anger overall, depressed adolescents will display relatively less anger in families with high levels of expressed parental anger, and relatively more anger in families with low expressed parental anger, when compared to non-depressed adolescents.

The moderating role of the family environment on the adolescent's anger reactivity

The family environment may play an important role in moderating the adolescent's reactions to their parents' angry behaviors (i.e., anger reactivity). Importantly, this influence may differ for depressed and non-depressed youth. In general, evidence suggests that it is normative for children to more likely express their emotions in environments that are responsive and predictable (Eisenberg, Cumberland, & Spinrad, 1998; Morris, Silk, Steinberg, Myers, & Robinson, 2007). Thus, in less aggressive family climates, non-depressed adolescents may feel free to reciprocate and express their anger, whereas such reciprocation may be inhibited in more aggressive climates given the potential negative consequences of aggressive reciprocation.

Yet, the concept of "emotion context insensitivity" helps to explain how the family environment may differently moderate anger reactivity in depressed youth. Evidence suggests that depressed persons are less responsive to both positive and negative stimuli, a characteristic referred to as emotion context insensitivity (ECI; Bylsma, Morris & Rottenberg, 2008; Rottenberg, Gross & Gotlib, 2005). The ECI hypothesis is based on an evolutionary model that suggests that depressed states promote a defensive disengagement from one's environment (Nesse, 2000). Depressive symptoms such as a loss of interest in the environment are thought to prevent depressed individuals from taking ill-considered actions (Rottenberg et al. 2005). The ECI hypothesis can, therefore, be likened to the social rank theory and social risk hypothesis (Allen & Badcock, 2003; Price & Soloman, 1987; Gilbert, 1992) in suggesting that depressed states encourage disengagement from one's environment so as to reduce the likelihood of behaviors that may result in social defeat or rejection. Therefore, based on the ECI model, we predict that the responses of depressed youth to parental angry behavior may be less influenced by the environmental context (i.e., their family environment) than are those of non-depressed youth. The interaction between depressive status and environmental attunement and context may therefore explain the inconsistent findings with regard to between group differences in anger reactivity.

The Present Study

The aim of the present study was to better understand how the experience of anger differs as a function of adolescent depressive status and levels of parental angry behavior. The study design has a number of strengths. First, because research on emotional reactivity is enhanced

by using idiographic stimuli that capture the type of emotion-eliciting events encountered in daily life (Rottenberg et al. 2005), parental and adolescent anger were assessed during parent-adolescent problem-solving interactions, which elicit personally relevant conflict behavior. Second, adolescent anger was assessed using two objective indices, observational coding of angry behavior and heart rate, both of which capture dynamic aspects of emotional responding as they occur in real-time. Moreover, behavioral and physiological measures provide complementary information. Behavioral observations tap overt expression of anger, while physiological data can potentially reveal instances of emotional processing not otherwise observed due to behavioral inhibition of emotional expression (Gross, 1998; Gross & Levenson, 1993, 1997). In terms of physiology, we focused on heart rate as an indicator of anger arousal. The stress response, of which anger can be considered a part, generally involves a significant heart rate increase (Cacioppo, Berntson, Laresen, Poehlmnn, & Ito 1993; Haddy & Clover, 2001). This acceleration of cardiac activity likely reflects anger's close association with the "fight" response system (Frijda, 1986), which requires quick and efficient energy mobilisation (Levenson, 1992). Hence, we anticipate that heart rate will increase as a function of anger concordanct with the increases in behavioral expression of anger. Nonetheless, physiological processes, including heart rate, are multiply determined such that there is not a one-to-one relationship between behavioural expressions of anger and increased heart rate. As such, examining both physiology and behavior is advantageous in that parallel findings across these indicators will enable stronger inferences to be drawn regarding the meaning of between group differences, while places where the findings diverge may provide insight into the disparate functions of these different components of emotion.

Our predictions lay out how the family environment may differentially affect the experience of anger in depressed and non-depressed adolescents. We hypothesize that in family environments characterized by higher levels of expressed parent anger, depressed adolescents will display less angry behavior and a slower average heart rate than will non-depressed adolescents. In contrast, in less angry family climates, depressed adolescents will display more angry behaviors, and a faster average heart rate than non-depressed adolescents. Furthermore, depressed adolescents will show a consistently low level of behavioral and physiological reactivity to their parents' angry behaviors, irrespective of the overall level of parental anger in the family. In contrast, non-depressed adolescents will show higher levels of anger reactivity in less angry family environments and lower levels of reactivity in more angry environments.

Method

Participants

From an initial sample of 152 adolescents, 11 were excluded for the present study due to missing physiological data, and another 45 were excluded because only one parent attended the laboratory interaction session. The remaining sample consisted of 96 participants, aged 14 to 18 (Mean = 16 years; 37 boys), and their parents. To be included in the investigation, adolescenthad to meet research criteria for placement in one of two groups (Depressed, n = 43, of which 17 boys; or Healthy, n= 53, of which 20 were boys; gender and depressive

status were unrelated, $\chi^2(1) = 0.03$, $p = .86$). Depressed adolescents met DSM IV (APA, 2000) diagnostic criteria for a current unipolar depressive disorder. Consistent with DSM guidelines for establishing the offset of depressive episodes, a diagnosis was considered current if it was ongoing or had an offset within two months preceding the diagnostic interview(APA, 2000); using this definition, two of the depressed adolescents were in partial remission at the time of the assessment. Healthy adolescents had no lifetime history of psychopathology or mental health treatment. Adolescents were excluded if they evidenced comorbid externalizing or substance dependence disorders or were taking medications with known cardiac effects. Eight participants reported taking other forms of medication (all from the depressed group), and 17 were undergoing treatment during the time of the study (of which 16 were from the depression group).

Recruitment and Assessment Procedures—Families wererecruited using a two-gate procedure consisting of an in-school screening and an in-home diagnostic interview. Inorder to facilitate recruitment of a representative sample of students, we used a combined passive parental consent and active student assent protocol for the school screening (Biglan & Ary, 1990; Severson & Ary, 1983). A letter describing the purpose, procedures, risks, and benefits of completing the questionnaire was mailed to the parents of each student two weeks before the screening. Parents who wished to decline participation returned a stamped and pre-addressed postcard to the school. Additionally, on the day of the assessment, this information was provided verbally and in writing to the students, who signed the written information form if they chose to assent. Students who did not assent, whose parents declined permission, or for whom letters to parents were returned as undeliverable, were excluded. Active parent consent and adolescent assent for the full assessment were obtained prior to the diagnostic interview.

School screening: Students (N=4182) from area high schools completed the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977) and a demographic form during class. Approximately 70% of eligible students participated, 12% declined or had parents decline their participation and 18% were absent or off campus on the day of the assessment. CES-D cut-off scores for sample selection were based on the distribution of scores obtained in an earlier screening of high school students in the same area (N= 4495; Sheeber, Davis, Leve, Hops & Tidesley, 2007). Relatively high scores (≥ 31 for males and ≥ 38 for females) were selected to maximize the positive predictive power to identify adolescents experiencing depressive disorder. The pool for the healthy group was defined as students not more than ½ SD above the mean of the earlier sample (< 21 for males and < 24 for females).

Diagnostic assessment: Interviewers conducted the Schedule of Affective Disorders and Schizophrenia-Children's Version (K-SADS, Orvaschel & Puig-Antich, 1994) with adolescents who had elevated CES-D scores. Subsequent to the interviews, families of adolescents who met criteria for MDD were invited to participate in the lab-based assessment. After each adolescent in the depressed group completed the lab assessment, a healthy, demographically matched comparison participant was recruited from the pool of students who scored within the normal range on the CES-D. Of families invited to

participate, approximately 26% declined. Rates of decline did not vary as a function of pre-interview group status (i.e., elevated or healthy CES-D score), age, or race, though more males than females declined (31.6% vs. 23%), χ^2 (1, n=498)=4.57, p<.05. Of adolescents with elevated CES-D scores who participated in the interview, 38% met criteria for MDD. Of adolescents with CES-D scores in the healthy range, approximately 76% met criteria for inclusion.

Lab assessment: Families who met criteria for the investigation after the diagnostic interview were invited to participate in the lab assessment. Approximately 4% of families declined. The decline rate did not vary as a function of group status, age, race, or gender. In approximately 93% of two-parent families, both parents participated. Participants were instructed to abstain from alcohol and illicit drugs on the day of the assessment. Compliance with this instruction was confirmed on the day of the assessment via self report. The lab assessment included an 18-minute parent-adolescent Problem-Solving Interaction (PSI) in which families were asked to discuss and resolve two areas of conflict identified based on parent and adolescent responses on the Issues Checklist (Prinz, Foster, Kent, & O'Leary, 1979). The PSI was videotaped for behavioural coding. Adolescent electrocardiograph activity was measured during the interaction.

Measures

Depression Screener—The CES-D is a widely used self-report measure of depressive symptoms that has acceptable psychometric properties for use with adolescents (e.g., Roberts, Andrews, Lewinsohn, & Hops, 1990; Radloff, 1977). It has a well-established record of use as a screener in adolescent samples (e.g. Asarnow et al., 2005; Sheeber et al., 2007).

Diagnostic Interview—The K-SADS was conducted with the adolescents in order to obtain current and lifetime diagnoses. Interviewers, who were bachelor and masters level research staff, participated in a rigorous training program and demonstrated agreement with a senior interviewer ($\kappa \geq .80$) on at least two interviews before conducting independent interviews. All interview-derived diagnoses were confirmed by supervisors who reviewed both item endorsement and interviewers' notes. Questions regarding the accuracy of diagnoses were resolved based upon discussion with the interviewer and review of the audiotaped interview as needed. Based on a random selection of 20% of the interviews, inter-rater reliability was $\kappa = .94$.

Behavioral Coding—The Living in Family Environments Coding System (LIFE; Hops, Biglan, Tolman, Arthur, & Longoria, 1995) was used to code the adolescent's and each parent's behavior (i.e., mothers and fathers coded separately) during over the entire PSI. The LIFE is an event-based, microanalytic coding system in which a new code is entered each time there is a change in a participant's verbal content or affective behavior. Each entry is comprised of several components which identify the: a) target (i.e. whose behavior is being coded); b) verbal content; and c) nonverbal (or para-verbal) affect. Data analysis is done at the level of constructs, which are operationalized as particular combinations of content and affect codes. Observers, blind to diagnostic status, coded family members' nonverbal affect

and verbal content in real time. A construct reflecting angry behavior was derived from individual affect and content codes. Angry behavior included aggressive (e.g., raised voice; clenched teeth) or contemptuous (e.g., eye rolling; sneering) nonverbal behavior and cruel (e.g., mocking; insults; threats) or provoking (e.g., taunts; dares) statements. Approximately 25% of the videos were coded by an additional observer for reliability. Inter-rater reliability (κ) for angry behavior equaled .73. The validity of the LIFE system as a measure of family processes has been established in prior studies of adolescent depression (e.g., Katz & Hunter, 2007; Sheeber et al., 2007).

Physiology Recording—Heart rate data were acquired using software and equipment from the James Long Company (www.jameslong.net). The ECG signals were recorded using Ag-AgCl electrodes. To record the ECG signal, a three-lead system was used to maximize the r-wave amplitude, and to minimize movement artifact and t-wave amplitude. The positive electrode was placed directly under the left armpit about 4 to 6 inches down, at heart elevation. The negative electrode was placed directly under the right armpit at the same elevation. The ground electrode was placed on the sternum, halfway between the positive and negative electrodes. The ECG signals were input to an isolated bioelectric amplifier, with a gain of 250 and bandpass of frequencies between 0.1 – 1000 Hz. Heart rate was calculated based on the time (in milliseconds) between successive r-waves (R-R intervals) on the ECG. The ECGRWAVE program identified r-waves from the ECG signal with an automated, multiple-pass, self-scaling algorithm. These signals were then visually inspected. Missed or misrepresented r-waves were manually corrected and sections of movement, noise, or flat line artifact were removed.

Data Analysis—The data were analyzed using autocorrelation-crosscorrelation regression models in a multilevel framework. The multilevel regression framework takes into account dependencies in the data resulting from the nested structure (second-by-second observations nested in individual participants), and separates within from between person variance (Bryk & Raudenbush, 1992; Snijders & Bosker, 1999). The autocorrelation-crosscorrelation approach enables examination of the influence of one variable on *change* in a second variable, and is the closest means by which to infer a causal relationship using multivariate time series data (see Gottman 1990; Granger, 1969).

Multi-level regression models were estimated separately for each of the dependent variables (i.e., adolescent angry behavior and heart rate). To model the physiological data, normal multilevel regression models were used. However, to model the adolescents' behavioral data, a logistic transformation function was added (resulting in logistic multilevel regression) to accommodate for their binary nature.

At level one of the models, the dependent variable (e.g. the adolescent's anger display orheart rate) was predicted firstly by an *intercept.* The intercept reflects the average level of expressed anger or heart rate, which was allowed to vary across participants (due to the multilevel modelling approach). The intercept is thus relevant to the research questions relating to average levels of anger in the adolescent. Additionally, the dependent variable was also predicted by maternal and paternal anger displayed 5 seconds earlier (the cross correlation) while controlling for the dependent variable 5 seconds earlier (the

autocorrelation). All predictor variables were group-mean centered (Enders & Tafoghi, 2007). The *slopes* of the predictor variables also varied across individuals and reflect the adolescents' behavioral and physiological reactivity in response to their parents' display of anger. A 5 second lag has been used successfully in our prior work with these measures (Allen, Kuppens, & Sheeber, submitted; Kuppens, Allen, & Sheeber, 2010).

At level two of the models, both the person-specific intercept and slope values were modeled as a function of adolescent depression (binary coded as a 1 for depressed, and 0 for non-depressed), the average level of observed parental anger during the interaction (calculated as the average level of angry behaviours demonstrated by the adolescent's parents throughout all interactions, collapsed across mothers and fathers and centered; *Mean* before centering = 0.07, *SD* = 0.07) and the interaction of depression and parental anger. Their effects on the *intercept* reflect how these factors relate to the adolescents overall level of anger during the interaction. The effect of these factors on the *slope* reflects how these factors relate to adolescent anger reactivity (see Appendix for more detail on the used model).

Results

Preliminary analyses

As the addition of level 1 predictor variables changes the interpretation of the intercept, a preliminary analysis was performed to determine whether levels of overall anger expression and heart rate were significantly different between depressed and non-depressed adolescents, without additionally including predictors at level 1 of the model. In two random intercept models, adolescent angry behavior and heart rate were predicted by a random intercept at level one, which at level two, was modeled as a function of depression only. The results of these analysis indicated that depressed youth expressed more anger on average than non-depressed adolescents, $B = -2.123$, $SE = 0.169$, $p = .000$, and had a higher heart rate, $B = 75.206$, $SE = 1.178$, $p = .000$, during the interactions (see also Sheeber et al., 2009; Allen et al., 2010; Byrne et al., 2010). Following this, the main analyses described above were conducted. The findings for anger behavior and heart rate are reported in Table 1 and Table 2, respectively.

In addition, for comparison, unconditional models were estimated predicting adolescent anger and heart rate (including only a random intercept term without level 2 predictors). For adolescent anger, the average intercept value equalled −1.70, T-ratio = −10.71, $p < .001$; variance = 2.42. For heart rate, the average intercept equalled 77.80, T-ratio = 78.20, $p < .001$; variance = 95.98.

Average adolescent anger and heart rate (intercept)

Adolescent anger behavior—There was a significant main effect of overall parental anger, collapsed across mothers and fathers, on the average level of adolescent angry behaviors (Table 1). Adolescents whose parents displayed greater levels of angry behavior overall, displayed more angry behavior themselves. This effect was significant for both depressed, $\chi^2(1) = 6.73$, $p = .009$, and non- depressed, $\chi^2(1) = 20.59$, $p = .000$, adolescents. The interaction between depression and parental anger was also significant, however,

indicating that differences between depressed and non-depressed adolescents varied as a function of levels of parental angry behavior. As can be seen in Figure 1a, in families with low parental expressed anger, depressed adolescents exhibited significantly more angry behavior than did non-depressed adolescents, $\chi^2(1)= 12.99$, $p = .0006$, while no between group differences emerged in families with high parental expressed anger, $\chi^2(1)= 0.10$, $p > .500$.[1]

Adolescent heart rate—The results regarding the intercept in Table 2 again show that depressed adolescents had a higher heart rate throughout the interactions when compared to non-depressed adolescents, regardless of family climate. There was, however, again a significant interaction between depression and family climate, which is graphically portrayed in Figure 1b. In families with low parental expressed anger depressed adolescents had a higher average heart rate than non-depressed adolescents, $\chi^2(1)= 12.34$, $p = .001$, but this was not the case in families with higher parental expressed anger, $\chi^2(1)= 0.00$, $p > .500$. Moreover, although aggressive family climate was marginally associated with increased levels of heart rate overall (see Table 2), the results presented in Figure 1b show that this trend only held for non-depressed adolescents, $\chi^2(1)= 2.96$, $p = .081$, whereas for depressed adolescents this difference was non-significant, $\chi^2(1)= 1.54$, $p = .212$.

Adolescent anger reactivity (slope)

Adolescent anger behavior—In response to displays of anger by the mother, there was a significant main effect of overall parental anger on adolescent behavioral anger reactivity whereby adolescents in environments characterized by lower levels of parental angry behavior were more reactive to parental anger (see results with respect to slopes in Table 1). However, this main effect was moderated by depressive status as displayed in Figure 2a. As can be seen, the effect of overall parental anger occurred predominantly for non-depressed adolescents, $\chi^2(1)= 6.91$, $p = .008$. Overall levels of parental anger did not significantly influence the way in which depressed adolescents responded to maternal anger, $\chi^2(1)= 0.18$, $p > .500$.

Hence, within less angry family climates, non-depressed adolescents showed greater behavioral reactivity than depressed adolescents, $\chi^2(1)= 4.33$, $p = .035$, whereas no such difference was found in more angry families, $\chi^2(1)= 0.68$, $p > .500$. No significant main effects, interaction effects, or significant contrasts were found for behavioral adolescent anger reactivity in response to paternal angry behavior (see Table 2, and Figure 2b).

[1]It should be noted that in the full analysis reported in Table 1, the effect of depression on average levels of adolescent anger became only marginally significant in contrast to the previously reported preliminary random-intercept model. Follow-up analyses revealed that this change in significance was *not* due to the inclusion of parental anger and the interaction term in predicting the intercept. Random intercept analyses which included depression, anger in the family environment, and their interaction, showed that all three variables significantly contributed to the intercept (Depression: $B = .461$, $SE = .204$, $p = .025$; parental anger: $B = .14.97$, $SE = 2.332$, $p = .000$; interaction: $B = -7.385$, $SE = 2.845$, $p = .011$), demonstrating that the effect of depression on average adolescent anger was not mediated by these other factors. Instead, the change in significance was a result of the inclusion of the level 1 group-mean centered predictor variables (autocorrelation and cross-correlations) and their level 2 moderators (Snijders & Bosker, 1999). It can therefore be safely concluded that depressed adolescents indeed displayed more anger on average compared to non-depressed adolescents in the present study, despite the lack of significance of depression reported in Table 1.

Adolescent Heart rate—Physiologically, a similar pattern emerged. There was a significant main effect of overall parental anger as well as a significant interaction effect with depression on adolescent heart rate reactivity in response to mothers' angry behaviour (Table 2). As can be seen in Figure 2c, the heart rate of non-depressed adolescents increased in response to mothers' anger in more angry family environments, while in less angry environments, it decreased, $\chi^2(1)= 7.06$, $p = .008$). There were no differences for depressed adolescents, $\chi^2(1)= 1.53$, $p = .213$. Within less angry family environments, contrasts furthermore revealed significant differences between depressed and non-depressed adolescents, $\chi^2(1)= 6.16$, $p = .013$. Non-depressed adolescents showed heart rate decrease, while depressed youth experienced heart rate increase. In more angry environments, group differences were not significant, $\chi^2(1)= 2.02$, $p = .152$.

Interestingly, despite the absence of behavioral differences, there was a main effect of depression on the physiological reactivity of adolescents in response to fathers' angry behaviors. Depressed adolescents consistently showed heart rate increase while non-depressed adolescents showed decrease in response to fathers' anger, regardless of the affective climate of the family environment. Contrasts revealed that group differences in this pattern of reactivity was more pronounced within angrier family environments, $\chi^2(1)= 6.28$, $p = .012$, as opposed to less angry environments, $\chi^2(1)= 2.22$, $p = .132$. These findings are graphically represented in Figure 2d.

The same series of analyses, including gender (dummy coded with 1 = male and 0 = female) as a level-2 predictor above the other predictors, was run as well. The results and conclusions remained the same, save from additionally showing that girls on average expressed more anger during the interactions than boys, B = −0.38; *T*-ratio = −2.99, $p = .004$, and had marginally significant higher heart rate overall, B = −3.50; *T*-ratio = −1.68, $p = .095$. There were no significant gender differences in anger reactivity (in response to mother: B = −0.08; *T*-ratio = −1.22, $p = .23$; in response to father: B = −0.09; *T*-ratio = −1.12, $p = .27$) or in heart rate reactivity (in response to mother: B = −0.08; *T*-ratio = −0.212, $p = .83$; in response to father: B = 0.50; *T*-ratio = 1.20, $p = .23$).

Discussion

Depression is most typically associated dysphoric mood, yet research suggests that the emotional dysfunction evident in depression also extends to the domain of anger. Previous research has yielded inconsistencies with regard to differences in the nature and mechanism of anger regulation and expression between depressed and nondepressed persons.

In the present paper, we wanted to shed light on this issue by examining how the family climate may differentially moderate anger in depressed and non-depressed adolescents during conflictual interactions with their parents. The overall pattern that emerged (see Figures), illustrates that non-depressed adolescents' levels of angry behavior and physiology mirror that of their parents' behavior, whereas this is significantly less so (in terms of behavior) or even reversed (in terms of physiology) for depressed adolescents. In other words, the finding that children's regulation of anger mirrors that of their parents, as well as that parents and children tend to show a matching of affect and behavior during the course of

conflict (Forgatch, 1989; Kobak et al. 1993), seems to apply less to depressed than to non-depressed adolescents. As such, the findings from this study indicate that in contrast to non-depressed adolescents, depressed adolescents' anger expression is not as contingent upon usual cues in their social environment. This overall pattern was apparent in data on both heart rate and anger behavior, both in terms of overall level as well as reactivity, and expressed itself in significant interaction effects between depressed status and overall family anger expression style on these parameters (with effects sizes ranging from .22 to .28 for the reported interaction effects).

Being less in tune with their social environment when expressing anger may be particularly problematic for depressed adolescents, given that, consistent with reports that depressed persons experience greater levels of anger than non-depressed persons (Fava & Rosenbaum, 1998; Ingram et al. 2007; Sheeber et al., 2000; Wenze et al. 2009), we found that depressed adolescents demonstrated both more angry behavior and higher heart rates than did non-depressed adolescents. However, we argued that because depressed mood has been hypothesised to have evolved to serve socially adaptive functions, consideration of the social context of anger would facilitate a better understanding of how the experience of anger differs between depressed and non-depressed adolescents. The findings indeed demonstrated that the emotional climate of the family significantly moderates the relationship between depression and the expression of anger. Depressed adolescents displayed higher overall levels of anger expression and heart rate in families characterized by lower levels of parental anger, whereas this difference was not present in families characterized by high levels of parental anger. In line with our hypothesis, this finding provides support for the notion that increased anger expression by depressed individuals occurs in environments that pose a lower risk of social defeat or rejection. Moreover, these findings add specificity to Joiner's (2000) theory of interpersonal conflict avoidance by highlighting boundary conditions under which such avoidance may occur. That is, while interpersonal conflict avoidance (i.e., low assertiveness, social withdrawal, avoidance and shyness) may indeed prolong and exacerbate symptoms of depression in social environments whereby the individual is at greater risk of social loss (e.g. environments where increased anger is expressed by others), in less angry social environments, it may in fact be the depressed individuals' poorer attunement to social contingencies that exacerbate their depressive symptoms.