Biological Psychology 90 (2012) 80–87

Contents lists available at SciVerse ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

Heart rate responses to parental behavior in depressed adolescents

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ARTICLE INFO

Article history:
Received 10 December 2010
Accepted 19 February 2012
Available online xxx

Keywords:
Depression
Adolescence
Parent–child interactions
Psychophysiology
Behavioral observation

ABSTRACT

In order to more fully understand the abnormalities in emotional responding associated with adolescent depression we examined clinically depressed and non-depressed adolescents' physiological responses to their parents' negative emotional behavior, as indexed by their heart rate responses to parental angry and dysphoric behavior during laboratory-based interactions. Maternal angry and dysphoric behavior predicted heart rate deceleration amongst non-depressed adolescents, a response that was not observed in depressed adolescents. Fathers' angry behavior predicted significant heart rate acceleration in depressed (but not non-depressed) adolescents, whereas fathers' dysphoric behavior predicted heart rate deceleration amongst depressed but not amongst non-depressed adolescents. These findings are interpreted within the framework of orienting and defense cardiac responses, and suggest that reactivity in adolescent depression is characterized by the absence of a normative orienting response toward aversive maternal behaviors, and a defensive physiological response to aggressive paternal behavior.

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1. Introduction

The prevalence of depressive symptoms and disorder increases greatly during adolescence, a developmental period of marked changes in affective functioning (Allen and Sheeber, 2008). Indeed, depression is frequently conceptualized as a disorder of affect or affect regulation (Cole and Kaslow, 1988; Davidson et al., 2002; Gross and Muñoz, 1995), which presumably has considerable significance for the way depressed people respond to interpersonal environments. Specifically, depressed persons experience mood states characterized by low levels of positive affects (PA) and high levels of negative affects (NA; see Watson, 2000), and demonstrate poor regulation of emotions as assessed at neurological, physiological, and behavioral levels (Garber et al., 2007; Gross and Muñoz, 1995; Johnstone et al., 2007; Tomarken and Keener, 1998). Notably these abnormalities also characterize depressive disorders during childhood and adolescence (Cole and Kaslow, 1988; Joiner et al., 1996; Sheeber et al., 2000; Silk et al., 2003). However, one critical issue is the relationship between heightened depressed *mood* and alterations in *reactivity* to social and other affective stimuli, especially during adolescence when developmental changes in affective functioning can interact with personal vulnerabilities to result in the emergence of depressive disorders (Allen and Sheeber, 2008).

The conventional view is that mood states potentiate similar emotional reactions, and inhibit incompatible ones. In the case of depression, this would suggest that those with high levels of depressed mood are likely to have stronger emotional reactions to sadness-inducing stimuli, and weaker emotional reactions to pleasant stimuli. Some studies in our labs (though not all, cf. Sheeber et al., 2000) have demonstrated that higher levels of adolescent depressive symptoms are associated with greater adolescent reciprocity of mothers' dysphoric and aggressive affect (Yap et al., 2011), supporting the notion that depression is associated with greater reactivity to aversive social stimuli.

Conversely, other recent studies have provided evidence of depressive hypo-reactivity to sadness-inducing stimuli. Rottenberg (2005), synthesizing results from a series of studies demonstrating that depressed individuals were *less* emotionally reactive to sadness-inducing stimuli, proposed that clinical depression is characterized by *Emotion Context Insensitivity* (ECI). The ECI model predicts that, despite the high levels of negative mood experienced by depressed persons, emotional reactivity (including responses to normatively sadness-inducing stimuli) are blunted during clinically depressed states. Indeed, the ECI model has been supported by a meta-analysis that concluded that depressed individuals evidenced reduced emotional reactivity to both positive and depressive stimuli (Bylsma et al., 2008).

Most functional or evolutionary views of depression have conjectured that depressed states are defensive reactions to threats to social wellbeing, such as defeat and humiliation (Price et al., 1994) or rejection (Ingram et al., 1998; see Allen and Badcock,

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doi:10.1016/j.biopsycho.2012.02.013

2003, for a review). These views suggest that depressed states will be associated with enhanced reactivity to personally relevant stimuli associated with social threats (e.g., interpersonal conflict or rejection), but not to stimuli depicting threats that are non-social in nature (e.g., physical threats), or even social threats that are not directed at the self (e.g., conflict or rejection directed at another person). As such, studies that have used either putatively "depression-relevant" stimuli that depict sadness (such as movies portraying people grieving the loss of a loved one; e.g., Rottenberg et al., 2002), or stimuli depicting physical threats (e.g., Allen et al., 1999; Dichter et al., 2004) may not be utilizing the types of affective stimuli to which depressed states most strongly alter reactivity.

1.1. The present study

In this investigation, we examined adolescent reactivity in response to aversive (i.e., dysphoric and angry) interpersonal behaviors displayed by the adolescents' parents during parent–adolescent interactions. Several considerations lead us to select aversive parent behavior as exemplars of the type of stimuli to which depressed persons may be differentially reactive. First, family conflict peaks during adolescence (Paikoff and Brooks-Gunn, 1991), with parent–child disagreements being not only more frequent than before, but also more intense than those that adolescents experience in peer relationships (Laursen and Collins, 1994). Second, threats to social well-being are richly represented in the types of family environments known to play an important role in the etiology and maintenance of depressed states (Butzlaff and Hooley, 1998; Sheeber et al., 1997; Stice et al., 2004). In particular, family environments characterized by harsh and conflictual family interactions are associated with depressive symptoms and disorder during adolescence (e.g., Ge et al., 1994; Patton et al., 2008; Sheeber et al., 2007). It is notable in this regard that family relationships appear to be more predictive of depressive symptoms in adolescence than are peer relationships (Barrera and Garrison-Jones, 1992; Stice et al., 2004). The family environment thus appears to provide the type of personally relevant stimulus context in which instances of altered emotional reactivity may be observed in depressed persons.

1.2. Heart rate as an index of reactivity to the interpersonal environment

In order to track adolescents' reactivity to the interpersonal environment in real time we examined their psychophysiological responses during interactions with their parents. Though we often use behavioral data to index emotional reactivity (e.g., Yap et al., 2011; Sheeber et al., 2000), psychophysiological data have important advantages. In contrast to behavioral expression, which may be regulated or suppressed due to social constraints and display rules (e.g., Gross, 1998), psychophysiological responses can provide a more direct index of reactivity that may bypass the regulatory controls exerted on overt behavior. Physiological indices, moreover, are able to capture more subtle or fleeting changes that may escape behavioral observation.

We selected heart rate change as the specific psychophysiological measure to examine in this investigation as it can be measured unobtrusively during interactions, allowing a relatively natural range of movement and non-verbal behavior. Also, the psychological concomitants of changes in heart rate are relatively well understood. In particular, deceleratory and acceleratory heart rate responses have been explicated within the theoretical framework of orienting (OR) and defense (DR) responses, respectively (Cook and Turpin, 1997).

The OR, or "what is it?" response, involves shifting attention toward a salient stimulus. It is distinguishable from the DR, also referred to as the "self-defense reflex", which involves heightened emotional arousal and bodily shifts that turn receptor organs away from the stimulus (Sokolov and Cacioppo, 1997). In general, HR deceleration is understood to be related to information intake and orienting, while HR acceleration is associated with information rejection and defense (Berntson et al., 1992; Cook and Turpin, 1997; Graham, 1992). As such, phasic changes in heart rate in response to changing circumstances during social interactions can be used to index a person's responses in terms of attentional orienting and information intake versus heightened emotional arousal geared toward self-defense, with consequent information rejection.

We hypothesized that in the personally relevant context of family interactions, depressed adolescents' responses to their parents' negative interpersonal behavior, as indexed by their heart rate responses to parental angry and dysphoric behavior during interactions, will be different from those of their non-depressed peers. Specifically, we hypothesized that depressed adolescents' heart rate responses to parental anger would be more likely to reflect a defensive response, consistent with the (socially) threatening nature of such behavior. In contrast, we predicted that non-depressed adolescents would be more likely to show orienting responses to their parent's angry behaviors, consistent with an adaptive recognition of the potential salience of these behaviors, and a non-defensive attending to the information that these behaviors might convey. In other words, we predicted that the depressed adolescents would experience greater heart rate acceleration, indicative of heightened emotional arousal and defensive reactions to angry parental behavior, whereas non-depressed adolescents would show greater heart rate deceleration, indicative of non-defensive orienting. Further we hypothesized that these differences would not be observed in reaction to parental dysphoria, as dysphoric behavior does not convey threats to the adolescent's own social well being as directly as does parental aggression. Accordingly we predicted that both depressed and non-depressed adolescents would evidence a deceleratory heart rate response to these behaviors, suggesting non-defensive attending.

Finally, one of the strengths of the current data set is the inclusion of both fathers and mothers in the majority of the interactions. Given that few studies have examined differential responses to fathers and mothers within these interactions, we primarily examined this issue in an exploratory fashion. Nevertheless, we were guided by previous findings demonstrating that harsh parenting by fathers is more strongly associated with adolescent depression than is harsh parenting by mothers (Sheeber et al., 2007), and that dysphoric behavior by depressed adolescents appears to be negatively reinforced by its ability to suppress fathers' aggressive behavior (Sheeber et al., 1998). Accordingly we tentatively hypothesized that fathers' anger would be especially salient and distressing to depressed adolescents, and would be associated with a defensive cardiac acceleration in this group.

2. Methods

2.1. Participants

Participants were 141 adolescents (47 boys), aged 14–18, and their parents. To be included in the investigation, adolescents had to live with at least one parent or permanent guardian, and meet research criteria for placement in one of two groups. Depressed adolescents met DSM IV (APA, 1994) diagnostic criteria for a current unipolar depressive disorder during a diagnostic interview. Consistent with 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, 1994); using this definition, three of the depressed adolescents were in partial remission at the time of the assessment and were excluded from further analyses, leaving a final sample size of 138 (Depressed, $n = 69$ or Healthy, $n = 69$). Healthy adolescents had no current or lifetime history of psychopathology, and no history of mental health treatment. Adolescents were excluded if they evidenced comorbid externalizing or substance dependence disorders, were taking medications with known cardiac effects, or reported regular nicotine use.

2.2. Recruitment and assessment measures and procedures

Families were recruited using a two-gate procedure consisting of an in-school screening and an in-home diagnostic interview. In order 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 and Ary, 1990; Severson and Ary, 1983). Active parent consent and adolescent assent for the full assessment were obtained prior to the diagnostic interview.

2.2.1. 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 et al., 1990). It has a well-established record of use as a screener for depressive symptoms in adolescent samples (e.g., Asarnow et al., 2005; Sheeber et al., 2007). As described above the CES-D was used as the initial gate of a two-stage recruitment and screening procedure.

2.2.2. Diagnostic interview

The K-SADS interview was conducted with the adolescents 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. Reliability ratings were obtained on approximately 20% of the interviews, chosen at random. The average agreement was $\kappa = 94$.

2.2.3. School screening

Students from area high schools completed the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977) and a demographic data form during class. Approximately 70% of enrolled students who were living with at least one parent or permanent guardian participated (4182 of 5975), 12% ($n = 695$) declined or had parents decline their participation and 18% ($n = 1098$) were absent or off campus on the day of the assessment. CES-D cut-off scores for selecting potential participants were based on the distribution of scores obtained in an earlier screening of high school students ($N = 4495$) in the same area (Sheeber et al., 2007). Relatively high scores ($\geq$31 for males and $\geq$38 for females) were selected to maximize the positive predictive power to identify adolescents experiencing depressive disorder. Approximately 8% ($n = 372$) of the students scored above these cut-offs. The pool for the healthy group was defined as students not more than 1/2 SD above the mean in the earlier sample (<21 for males and <24 for females).

2.2.4. Diagnostic assessment

Interviewers conducted the Schedule of Affective Disorders and Schizophrenia-Children's Version (K-SADS, Orvaschel and 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 laboratory assessment, a healthy, demographically matched comparison participant was recruited from the pool of students who scored within the normal range on the CES-D.

Approximately 9% ($n = 52$) of families contacted by phone were not eligible to participate as per the criteria described above (e.g., due to medicine regimen; moved out of family home). Of families invited to participate, approximately 26% ($n = 131$) 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% versus 23%), χ^2 (1, $n = 498$) = 4.57, $p < .05$. Of adolescents with elevated CES-D scores who participated in the interview, 38% ($n = 81$) met criteria for MDD. Of these, 13.9% ($n = 10$) had a comorbid anxiety disorder and were retained in the MDD group given the high rate of comorbidity between mood and anxiety disorders. Five individuals were excluded due to psychotic diagnoses (mania or schizophrenia). Of adolescents with CES-D scores in the healthy range, approximately 76% ($n = 84$) met criteria for inclusion.

2.2.5. Lab assessment

Families who met criteria for the investigation after the diagnostic interview were invited to participate in the lab assessment. Approximately 4% ($n = 7$) of families declined. The decline rate did not vary as a function of group status, age, race, or gender. Additionally, 11 participants were excluded from this report due to missing physiological data. In approximately 93% of two-parent families, both parents participated in the assessments. The average time between the diagnostic assessment and the lab assessment was 33.2 days (SD = 20.1; no between group differences).

The lab assessment included two family interaction tasks. Each task lasted 18 min, evenly divided across two discussions. The first task consisted of two consecutive problem-solving interactions in which families were asked to discuss and resolve two areas of conflict. In the second task, families were asked to discuss two areas of family life; one focused on identifying and describing the best and most difficult years the adolescent had experienced, and the other focused on the most challenging and most rewarding aspects of parenting the adolescent. Interactions were video recorded for subsequent behavioral coding. Although we have found that these tasks do preferentially elicit angry and dysphoric interactive behavior, respectively (Sheeber et al., in press), each task has the capacity to elicit a wide range of affective behaviors, and as such data from the two tasks were combined in the present analyses in order to allow greater number of examples of each affective behavior to be included in the analyses.

In addition, measures of adolescent cardiac physiology were obtained during the interactions. 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.

2.3. Physiological measures and procedures

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, we used a three-lead system to maximize the r-wave amplitude and minimize movement artifact and t-wave amplitude. The positive electrode was placed directly under the left armpit about 4–6 in. 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 custom, which amplified the signals with a gain of 250 and bandpass frequencies of 0.1–1000 Hz.

The ECGRWAVE program from the James Long Company identified r-waves from the ECG signal with an automated, multiple-pass, self-scaling algorithm. These signals were then visually inspected and manually corrected for missed or misrepresented r-waves, and sections of movement or noise artifact were removed. Overall, this accounted for only 0.5% (in s) of the total data that had to be marked and removed as artifact. Mean heart rate values were derived for each one-second epoch, based on a weighted average of the heart rate values associated with each of the inter-beat intervals (IBI) to fall either fully or partially within the epoch, with each of these intervals being weighted according to the time proportion of the epoch that each IBI constituted.

2.4. Behavioral observations

The Living in Family Environments coding system (LIFE; Hops et al., 1995) was used to code parent behavior during the video-recorded family interactions. 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. As such, duration of each behavior can be calculated as the time between onset of one code and onset of the next code, for each person in the interaction. 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 paraverbal) affect. These micro-level data are then combined into mutually exclusive constructs, which are operationalized as particular combinations of content and affect codes (Hops et al., 1995). Three binary constructs, angry, dysphoric, and happy were derived from individual affect and content codes (with 1 indicating presence of the behavior and 0 otherwise). The angry and dysphoric constructs were used in this report. 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. Dysphoric behavior was defined by sad nonverbal behavior (e.g., tearfulness, sighing) or complaining statements.

Extensively trained observers, with over a decade of experience, coded the parents' nonverbal affect and verbal content of verbal statements. Observers were blind to diagnostic status. Approximately 25% of the videos were coded by an additional observer for reliability Kappas for mothers and fathers angry and dysphoric behavior ranged from .60 to .64, which reflect good agreement (Fleiss, 1981; Landis and Koch, 1977). The validity of the LIFE system as a measure of family processes has been established in numerous studies of adolescent depression (e.g., Katz and Hunter, 2007; Sheeber et al., 2007).

2.5. Data analysis

The data involve concurrent information on depressed or non-depressed adolescents' heart rate and their parents' behavior across time. The data require an analytical strategy that takes into account the dependencies in the data created by the nested structure (second-by-second observations nested in persons) and that allows examination of between group differences in the influence of parental behavior on adolescent heart rate (HR). To accomplish this, we used a crosscorrelation–autocorrelation approach embedded in a multilevel modeling framework (for a similar approach, see e.g., Gottman, 1990; Moberly and Watkins, 2008), using HLM software (Bryk and Raudenbush, 1992). The crosscorrelation–autocorrelation examines how *change* in one variable can be explained by another variable, controlling for the variable's autocorrelation, and has been proposed to be the closest one can come to inferring causal relationships based on multivariate time series data (see e.g., Gottman, 1990; Granger, 1969). The multilevel or hierarchical linear modeling framework takes into account the nested data structure and facilitates the examination of the moderating role of depression

Table 1
Average adolescent heart rate and average proportion, frequency, and duration of maternal and paternal angry and dysphoric behavior during the interaction as a function of adolescent depressive status.

Variable	Depressed mean (SD)	Non-depressed mean (SD)	$t(df)$	p
Adolescent heart rate	78.80 (10.42)	75.16 (10.22)	2.07 (136)	.040
Proportion of duration of parental behavior				
Mother anger	0.12 (0.09)	0.07 (0.08)	3.29 (132)	.001
Mother dysphoria	0.15 (0.13)	0.17 (0.15)	−0.99 (132)	.33
Father anger	0.11 (0.15)	0.04 (0.06)	3.04 (97)	.003
Father dysphoria	0.16 (0.12)	0.15 (0.14)	0.39 (97)	.69
Frequency of parental behavior				
Mother anger	26.94 (16.47)	18.48 (14.84)	3.11 (131)	.002
Mother dysphoria	36.54 (16.36)	41.19 (16.11)	−1.61 (131)	.11
Father anger	18.14 (17.76)	10.41 (11.52)	2.57 (94)	.01
Father dysphoria	40.77 (16.08)	37.11 (16.28)	1.10 (94)	.27
Duration in seconds				
Mother anger	7.5 (4.1)	5.9 (3.4)	2.36 (131)	.02
Mother dysphoria	8.4 (5.0)	8.5 (5.4)	−0.18 (131)	.86
Father anger	7.7 (6.9)	4.8 (3.8)	2.59 (94)	.01
Father dysphoria	8.6 (4.5)	9.5 (8.0)	−0.58 (94)	.56

(e.g., Bryk and Raudenbush, 1992; Snijders and Bosker, 1999). Specifically, we performed analyses in which at level 1 of the model adolescents' HR at time t was predicted by the mother's or father's dysphoric and angry behavior at time t-5 s, controlling for the adolescents' HR at time t-5 s. For instance, when using maternal behavior as predictor, the level-1 model equation was:

$$HR_{t,i} = \pi_{0i} + \pi_{1i}(HR_{t\text{-}5\,s,i}) + \pi_{2i}(Anger\ mother_{t\text{-}5\,s,i}) + \pi_{3i}(Dysphoria\ mother_{t-5s,i}) + e_{ti}$$

with i indicating participant. All level 1 predictors were group-mean centered, and intercepts and slopes were allowed to vary randomly across participants (given by the index i). The crucial parameters in the equation are π_{2i} and π_{3i}, reflecting individual differences in reactivity of the adolescent's HR toward angry and dysphoric behavior by the mother. As can be seen from the equations, the intercept and slope values in the equation are allowed to vary randomly across participants. At level two, we examined how these parameters differed for depressed and non-depressed adolescents by including two binary variables, one indicating depression and one indicating non-depression, omitting the intercept:

$$\pi_{0i} = \beta_{00}\,Depressed_i + \beta_{01}\,NonDepressed_i + \varepsilon_0$$
$$\pi_{1i} = \beta_{10}\,Depressed_i + \beta_{11}\,NonDepressed_i + \varepsilon_1$$
$$\pi_{2i} = \beta_{20}\,Depressed_i + \beta_{21}\,NonDepressed_i + \varepsilon_2$$
$$\pi_{3i} = \beta_{30}\,Depressed_i + \beta_{31}\,NonDepressed_i + \varepsilon_3$$

Of primary interest in these equations, β_{20} and β_{21} reflect HR reactivity of depressed and non-depressed participants toward angry behavior by the mother, and β_{30} and β_{31} reflect HR reactivity of depressed and non-depressed participants toward dysphoric behavior by the mother. χ^2 hypothesis tests were used to determine the difference between depressed and non-depressed.[1] Because both parents were not always present during the interactions (see below), separate analyses for mothers' and fathers' behavior were performed.[2]

The crosscorrelation–autocorrelation approach thus allowed to examine how a parents' behavior at one point in time is associated with a *change* in the adolescent's physiology at a following point in time, and how this association was moderated by depressive status. We used a 5-s time-lag as previous research has documented that the difference between the orienting (deceleratory) and defense (acceleratory) HR response is most strongly observed in this time frame following the eliciting stimulus (e.g., Sokolov and Cacioppo, 1997).

3. Results

Table 1 reports the average adolescent HR, average proportion of time that each maternal and paternal behavior was displayed during the interaction, the average frequency of each parental behavior, and the average duration that each behavior lasted, as a function of adolescent depressive status. The results show that the average HR of depressed adolescents was higher than that of non-depressed adolescents during the interactions. Moreover, it showed that both fathers and mothers in depressed families displayed more, more frequent, and longer angry (but not dysphoric) behavior during the interactions.

3.1. Responses to maternal behavior

In the first analysis, we examined the role of the mothers' behavior (Table 1). This analysis was based on 134 participants for whom the mother participated in the interaction task.[3] The first two lines of Table 2 contain the intercepts (average HR) for depressed and non-depressed and show that depressed adolescents demonstrate elevated average HR relative to non-depressed adolescents during the interactions (as we have shown in a previous report, Byrne et al., 2010). Lines 3–4 show that HR is positively and similarly autocorrelated for all participants.

Findings central to our research question, are presented in the bottom half of the table, and indicate significantly different patterns of reactivity between depressed and non-depressed adolescents. In particular, both angry and dysphoric behaviors by the mother were associated with a decrease in the HR of non-depressed adolescents only (the difference between non-depressed participants' HR response to angry and dysphoric behavior was non-significant, $\chi^2 = 0.83$, $p > .50$). As an indication of effect size, the reported coefficients indicate that a 1 SD increase in maternal negative behavior elicited close to half an SD decrease in HR (a change of approx. 4 bpm). In contrast, depressed adolescents showed no change in heart rate in response to maternal behavior, which was moreover significantly different from the response of non-depressed adolescents; the difference between depressed participants' HR response to angry and dysphoric behavior was non-significant, $\chi^2 = 0.34$, $p > .50$.

3.2. Response to paternal behavior

Second, we evaluated the role of the fathers' behavior in a parallel model, based on 99 families in which the father participated

[1] This is not the case when formulating a model that includes an intercept term and only one dummy variable, which yields more difficult to interpret relative regression weights. Both models are mathematically equivalent, however, produce the same results, and can be used to extract identical information.

[2] Because both father and mother were not always present during the interaction tasks, combined analyses including both maternal and paternal behavior results in a large loss of participants. For this reason, separate analyses were performed and reported. It is important to note, however, that a combined analyses (based on 95 participants for which both father and mother were present) yields similar results and supports similar conclusions as the separate results reported here.

[3] The mother was absent for 3 of the depressed and 1 of the non-depressed participants (the difference being non-significant, $\chi^2(df = 1) = 1.03$, $p > .05$), leaving a sample of 66 depressed and 68 non-depressed adolescents whose mother participated. The families with and without mothers did not differ with respect to adolescent HR ($t(df = 136) = -0.43$, $p > .05$), paternal anger ($t(df = 97) = -0.48$, $p > .05$), or paternal dysphoria ($t(df = 97) = -0.26$, $p > .05$).

Table 2
Hierarchical model predicting adolescent HR by dysphoric and anger behavior of the mother at *t*-5 s controlling for HR autocorrelation at *t*-5 s (level 1) as a function of depression (level 2).

Level 1	Level 2					
	Group	*B* (fixed effect)	SE	*T*-ratio	$\chi^2_{D\text{-}ND}$	Random effect (parameter SD)
Intercept	D	78.97	1.32	59.61***	5.14*	10.64***
	ND	74.83	1.26	59.54***		
HR *t*-5 s	D	0.22	0.02	9.25***	0.52	0.19***
	ND	0.20	0.02	8.78***		
Anger mother *t*-5 s	D	0.17	0.18	0.93	4.74*	1.31***
	ND	−0.42	0.20	−2.11*		
Dysphoria mother *t*-5 s	D	−0.06	0.17	−0.32	5.54*	1.12***
	ND	−0.56	0.13	−4.39***		

Note. $\chi^2_{D\text{-}ND}$: χ^2-test of difference between depressed and non-depressed. Level 1 SD = 7.59.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

Table 3
Hierarchical model predicting adolescent HR by dysphoric and anger behavior of the father at *t*-5 s controlling for HR autocorrelation at *t*-5 s (level 1) as a function of depression (level 2).

Level 1	Level 2					
	Group	*B* (fixed effect)	SE	*T*-ratio	$\chi^2_{D\text{-}ND}$	Random effect (parameter SD)
Intercept	D	80.41	1.50	53.75***	7.91***	9.87***
	ND	74.84	1.30	57.65		
HR *t*-5 s	D	0.25	0.03	8.69***	0.94	0.19***
	ND	0.21	0.03	8.12***		
Anger father *t*-5 s	D	0.64	0.29	2.18*	6.88***	1.58***
	ND	−0.40	0.27	−1.50		
Dysphoria father *t*-5 s	D	−0.59	0.16	−3.68***	4.08*	0.96***
	ND	−0.15	0.15	−0.98		

Note. $\chi^2_{D\text{-}ND}$: χ^2-test of difference between depressed and non-depressed. Level 1 SD = 7.36.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

in the interaction task.[4] The results, shown in Table 3, again indicate that depressed adolescents showed elevated average levels of HR relative to non-depressed adolescents. Heart rate was again positively and similarly autocorrelated for all participants.

Concerning our central research question, significant between group differences in HR reactivity to fathers' negative affective behavior were observed. Depressed adolescents displayed an increase in HR in response to fathers' angry behavior, and a decrease in HR in response fathers' dysphoric behavior (the difference between depressed participants' HR response to angry and dysphoric behavior being significant, $\chi^2 = 16.90$, $p < .001$). In terms of effect size, the reported coefficients indicate that a change of 1 SD in paternal angry behavior elicited on average a 0.64 SD (approx. 5 bpm) increase in HR, while dysphoric behavior elicited a similar decrease in HR. Conversely, non-depressed adolescents showed no change in heart rate in response to either type of paternal behavior (the difference between non-depressed participants' response to the two behaviors being non-significant, $\chi^2 = 0.71$, $p > .50$).[5]

4. Discussion

These findings demonstrate that clinically depressed and non-depressed adolescents show different patterns of heart-rate reactivity to parental dysphoric and angry behavior. Consistent with predictions, both maternal angry and dysphoric behavior were associated with heart-rate deceleration amongst non-depressed adolescents. Importantly, this was not observed in depressed adolescents. Neither, however, did we observe the hypothesized acceleration amongst depressed adolescents to angry maternal affective behavior. Responses to paternal affective behavior were more variable. Fathers' angry behavior predicted significant heart-rate acceleration in depressed (but not non-depressed) adolescents. By contrast, paternal dysphoric behavior predicted heart-rate deceleration amongst depressed (but not amongst non-depressed) adolescents.

Viewed in light of the literature on the determinants of heart rate acceleration and deceleration in response to stimuli (Berntson et al., 1992; Cook and Turpin, 1997; Graham, 1992; Vila et al., 2007), these findings suggest that both dysphoric and angry maternal behaviors are salient interpersonal cues that strongly elicit an orienting response in non-depressed adolescents. This is consistent with other recent research that has demonstrated cardiac deceleration to social cues in non-clinical groups (e.g., social

[4] The father was absent for 24 of the depressed and 15 of the non-depressed participants (the difference being non-significant, $\chi^2(df = 1) = 2.90$, $p > .05$), leaving a sample of 45 depressed and 54 non-depressed participants whose father participated. The families with and without fathers did not differ with respect to adolescent HR ($t(df = 136) = -1.03$, $p > .05$), maternal anger ($t(df = 132) = 0.36$, $p > .05$), or maternal dysphoria ($t(df = 132) = -1.06$, $p > .05$).

[5] We also performed all analyses adding gender (next to depressive status) as a predictor of HR reactivity at level 2 of the models, and analyses adding gender, depressive status and the interaction between both as predictors of HR reactivity. The findings showed that none of the gender main effects nor gender × depressive status interaction effects on the reactivity of HR to parental behavior were significant (all *ps* > .05).

rejection; Gunther Moor et al., 2010). However, this response was absent in the depressed group. Whether this absence of response represents the habituation of a defensive response, as has been shown in studies of repeated presentation of aversive stimuli in high fear participants (Klorman et al., 1977), or the overlapping influence of simultaneous orienting and defense responses (i.e., with different modes of cardiac control essentially canceling each other out; Vila et al., 1997) remains to be clarified. One argument, against the habituation explanation, however, is that parallel findings were obtained for reactivity to dysphoric and angry behaviors, though there were no group differences in the amount of dysphoric behavior displayed by mothers. We should note, however, that as the LIFE coding system does not capture intensity of behavior, it is feasible that the groups differed on the intensity of maternal dysphoric behavior, which could contribute to differences in adolescent reactivity. Although another possible explanation for the blunted cardiac response patterns observed in the depressed group is reduced autonomic flexibility, which has previously been proposed to be a feature of anxiety and depressive disorders (Friedman, 2007), we consider this less likely as reduced heart rate variability and vagal tone, the proposed mechanism for this reduced autonomic flexibility, does not differentiate the depressed and non-depressed groups in this sample (Byrne et al., 2010).

The finding with regard to reactivity to paternal angry affect was consistent with our hypothesis that depressed participants would respond with heart-rate acceleration, indicating increased emotional arousal and a defensive stress response. It is intriguing that this increased reactivity was specific to angry behaviors displayed by fathers. Although both angry and dysphoric behaviors are potentially aversive to others, as noted above, they have somewhat different interpersonal implications and elicit different responses (Biglan et al., 1989). In particular, angry behavior reflects a clear threat signal (Blanchard and Blanchard, 2009), whereas this is not the case for dysphoric behavior.

Given the higher probability of physical aggression in men (Archer, 2004), it is therefore possible that paternal anger (albeit in this context reflecting verbal and expressive aggression rather than physical aggression) is more immediately threatening compared to such behavior by the mother. Indeed, the appearance of a threat response to parental angry behavior amongst depressed teenagers is interesting in light of evidence that harsh parenting by fathers is more strongly associated with adolescent depressive status than is harsh parenting by mothers (Sheeber et al., 2007). As noted earlier, fathers of depressed adolescents displayed more angry behavior and it is possible that depressed adolescents have been sensitized to the greater extent or intensity (which we did not directly examine here) of paternal anger. Furthermore, in previous work, we have reported that dysphoric behavior by depressed adolescents appears to be negatively reinforced by its ability to suppress fathers' aggressive behavior (Sheeber et al., 1998; but cf. Slesnick and Waldron, 1997). To the extent that dysphoric behavior is, in part, a self-protective response to perceived threat in the father–child relationship, heart-rate acceleration may represent a psychophysiological concomitant of this behavioral response.

In turn, the finding of heart deceleration in depressed (but not non-depressed) adolescents in response to fathers' dysphoric behavior was unexpected, and suggests that depressed participants were orienting more to this behavior than were the non-depressed participants. Combined, the results thus indicate that dysphoric paternal behavior elicits emotional orienting in depressed adolescents, whereas paternal angry behavior elicits defensive responding, with both these responses absent in non-depressed adolescents.

The differential elicitation of orienting and defense responses by the same aversive stimuli in different types of individuals has also been observed in studies of individual differences in fear. For example, Klorman et al. (1977) found that participants reporting low mutilation fear showed a deceleratory cardiac response to mutilation pictures (i.e., an orienting response), whereas participant high in mutilation fear showed an acceleratory response (i.e., a defense response). This finding, along with similar ones in the literature (see Cook and Turpin, 1997 for a review) suggests that individual differences in how fear prone an individual is to the specific class of aversive stimuli strongly moderates cardiac response patterns.

The findings of the current study are consistent with the interpretation that non-depressed adolescents respond to aversive (i.e., both angry and dysphoric) social behavior by mothers with attentional focus and information intake, whereas the depressed group lack this response. Moreover, in responding to paternal anger depressed adolescents show signs of a defensive physiological response that suggests autonomic arousal and preparation for action (e.g., fight or flight responses) more than attentional engagement. Further extending these ideas suggests that non-depressed participants might be thought of as processing aversive maternal behavior in a way that is analogous to the way that persons who have a low level of fearfulness process a potentially fear relevant stimulus (i.e., with an orienting response), whereas depressed participants responded to paternal anger in a way analogous to persons with a high fear response (cardiac acceleration; see Cook and Turpin, 1997; Klorman et al., 1977).

Given the importance of successfully negotiating aversive behaviors in interpersonal contexts, these findings may not only provide evidence of enhanced reactivity to personally relevant stimuli, but may also point toward processes that may contribute to dysfunctional family relationships amongst the families of depressed adolescents. If depressed adolescents are unable to process important interpersonal cues adequately, this may result in maladaptive behavioral responses that serve to escalate, rather than deescalate, conflict within family environments. Importantly, this study documents findings that are both consistent with the ECI model of emotion in depression (Rottenberg, 2005), as well as some potentially important exceptions. The lack of reactivity to maternal behavior observed in depressed adolescents is consistent with the notion that they are under-reactive to some aversive interpersonal behaviors, whereas the response to angry paternal behavior suggests that they may be over reactive to other such behaviors. This latter finding suggests, consistent with some evolutionary and interpersonal models of depression (e.g., Allen and Badcock, 2003; Price et al., 1994), that given the significance of interpersonal defeat and rejection for depressed states, affective reactivity in depression is especially enhanced to interpersonally aggressive rather than sadness inducing stimuli per se.

Although this study had a number of significant strengths, including the integration of behavioral and physiological data within personally and etiologically relevant social interactions, and the selection of a large clinically depressed cohort, there are also some important limitations that need to be considered. The reliance on behavioral and physiological data alone requires us to speculate as to the way in which participants interpreted environmental stimuli. Although self-report of such interpretations also suffers from limitations, and is difficult to implement in the context of dynamic naturalistic interactions, there are methodologies such as video mediated recall (Gottman and Levenson, 1985; Lorber, 2007) that can be useful in this regard. Indeed, we have recently have recently published findings using this procedure with a different cohort indicating that depressed adolescents do over-report parental aggressive affect and underreport parental happy and neutral affects when compared to neutral observers (Ehrmantrout et al., 2011). The integration of such techniques into future studies using behavioral and physiological data may help to provide an extra dimension of interpretation to the response

patterns observed in this study. This is also important in light of the fact that heart rate acceleration cannot be unambiguously interpreted as a DR given that it has also been shown to associated with positive incentives (Fowles et al., 1982), active coping (Light, 1981) and challenge (as compared to threat; Blascovich et al., 2001). Although the context in which cardiac acceleration was observed in this study (i.e., in response to angry paternal behavior) renders an appetitive interpretation of the response unlikely, nevertheless some other form of data with which to confirm the interpretation that this response likely reflects a DR would enhance confidence in the inferences made.

Another important caveat is that the absence of a psychiatric control group means that we cannot be certain the extent to which these responses characterize depression specifically, as opposed to emotional disorders, or psychopathology, in general. Indeed, we did allow comorbidity with anxiety disorder in the depressed group, although only 10 depressed participants (13.9%) had a comorbid anxiety disorder, making it very unlikely that these participants alone were responsible for the observed effects. Furthermore, separate analyses of this group would have very low power. Nevertheless, given the well established fact that depressed samples have higher levels of anxious symptoms than healthy groups, establishing the differential association of anxious and depressive symptoms with effects observed here remains an important avenue for future research. Another limitation is that although most of interactions examined were triadic (i.e., mother, father and adolescent were all present) we have not examined the influence of the joint pattern of mothers' and fathers' behavior on adolescents (e.g., whether consistent or inconsistent affective behavior between the parents moderates the adolescents' responses). Analysis of these issues goes beyond the scope of the current manuscript but remains an important topic for future research. Finally, given the delay between the diagnostic assessment and the laboratory assessment, it is possible that some depressed participants, despite still meeting diagnostic criteria for a depressive episode, may have experienced a reduction in the severity of their depressive symptoms. Future studies would do well to re-assess depressive symptoms on the day of the laboratory assessment to address this caveat.

In sum, our findings suggest that the cardiac reactivity of depressed adolescents to interpersonal behavior during family interactions is primarily characterized by the absence of a normative orienting response toward aversive maternal behaviors, along with some indications of defensive physiological responses to angry aversive paternal behavior specifically. This pattern combines both the idea of increased reactivity to socially threatening stimuli (in particular, paternal anger) with the possibility of underreactivity (lack of orienting toward salient maternal behavior) in personally relevant stimulus contexts. As this is a cross-sectional study, we cannot determine the extent to which differences in depressed adolescents' reactivity have developed consequent to differences in family interactional patterns associated with adolescent depression (e.g., Sheeber et al., 2007). To our knowledge, though, this study is among the first to provide such a detailed and direct examination of interpersonal reactivity in parent–child relationships in adolescent depression. Although more research is needed to pinpoint the precise processes that underlie the observed differential reactivity to paternal and maternal behavior, our findings emphasize the importance of taking into account differences between reactivity to the father and mother and the type (i.e., dysphoric versus angry) of their respective aversive behavior.

Acknowledgments

This research was supported by NIMH grant R01 MH65340 (PI Sheeber) and KULeuven Research Council Grant GOA/05/04 and OT/11/031. Nicholas Allen is partially supported by a grant from the Colonial Foundation. We gratefully acknowledge the assistance of Michelle Byrne in the processing of the psychophysiological data presented in this study.

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