Citation for published version (APA):
Anxious parents show higher physiological synchrony with their infants


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Abstract (205 words)

**Background.** Interpersonal processes influence our physiological states and associated affect. Physiological arousal dysregulation, a core feature of anxiety disorders, has been identified in children of parents with elevated anxiety. But little is understood about how parent-infant interpersonal regulatory processes differ when the dyad features a more anxious parent.

**Methods.** We investigated moment-to-moment fluctuations in arousal within parent-infant dyads using miniaturised microphones and autonomic monitors. We continually recorded arousal and vocalisations in infants and parents in naturalistic home settings across day-long data segments.

**Results.** Our results indicated that physiological synchrony across the day was stronger in dyads featuring more rather than less anxious mothers. Across the whole recording epoch, less anxious mothers showed responsivity that was limited to ‘peak’ moments in their child’s arousal. In contrast, more anxious mothers showed greater reactivity to small-scale fluctuations. Less anxious mothers also showed behaviours akin to ‘stress buffering’ – downregulating their arousal when the overall arousal level of the dyad was high. These behaviours were absent in more anxious mothers.

**Conclusions.** Our findings have implications for understanding the differential processes of physiological co-regulation in partnerships where a partner has anxiety, and for the use of this understanding in informing intervention strategies for dyads needing support for elevated levels of anxiety.
Keywords: infant, parenting, stress regulation, affect matching, anxiety, synchrony, physiology

Abbreviations: CCF: Cross-correlation function; ECG: Electrocardiogram; GAD-7: Generalized Anxiety Disorder 7-item; GPS: Global Positioning System; IQR: Inter-quartile range; RR interval - beat-to-beat interval (R is the peak of the QRS complex of the ECG wave).
Introduction

Research has shown continuity of lifetime anxiety disorders from parents to children: multiple anxiety disorders pose a significant risk of anxiety in offspring (Lawrence, Murayama, & Creswell, 2019). However, while anxiety disorders aggregate in families, the reasons for this are still not yet understood (Murray, Creswell, & Cooper, 2009). Genes associated with an underlying liability towards current anxiety symptoms across the population are largely shared with those predisposing individuals to professionally-diagnosed lifetime anxiety disorder (Purves et al., 2019), yet evidence acknowledges the key role of environmental influences in the development of anxiety (Eley et al., 2015). Early childhood has been found to be a crucial period for identifying environmental risk factors for anxiety disorder (Möller, Nikolić, Majdandžić, & Bögels, 2016), including the potential for early identification of high risk individuals, and for preventative, early interventions. The present study examines, therefore, how anxious symptoms in parents relate to affect co-regulation in parent-infant dyads.

In both anxious and non-anxious families, there is considerable evidence that parents play a positive role in regulating children’s physiological, behavioural and affective states (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Reddy, Hay, Murray, & Trevarthen, 1997). Behavioural studies have, for example, identified sensitive parenting behaviours that mediate the relationship between household chaos and infant self-regulatory skills (Vernon-Feagans, Willoughby, Garrett-Peters, & The Family Life Project Key Investigators, 2016), and parental encouragement mediates the relationship between parent anxiety and anxiety symptoms in early childhood (Murray et al., 2009, 2008). Physiological studies examining how autonomic arousal co-fluctuates in infant-
parent dyads have traditionally concentrated on physiological synchrony, defined as ‘any interdependent or associated activity [...] in the physiological process of two or more individuals’ (Davis, West, Bilms, Morelen, & Suveg, 2018). Previous research has suggested that the benefits of synchrony are bidirectional (Feldman, 2007): the parent, by adapting to the child, helps by responding contingently to the child’s needs (Feldman, 2009); the child, by adapting to the parent, gains both self-control, and self-awareness (Feldman, Greenbaum, & Yirmiya, 1999). Previous research has identified synchronous patterns of change in physiological arousal in the lab following the administration of experimental stressors (Ham & Tronick, 2009). However, recent research that recorded naturalistic arousal co-fluctuations in infant-parent dyads found that, in fact, synchronous patterns of co-fluctuating arousal were not observed across all arousal states: rather, that short-term increases in parent-child synchrony were triggered in response to ‘peak’ instances of physiological arousal in the infant, but that synchrony at other times was not observed (Wass et al., in press).

There is also substantive evidence that anxious parenting can lead to the dysregulation of behavioural and physiological states in children (Nikolić, Vente, Colonnesi, & Bögels, 2016). Behavioural studies examining tabletop play between anxious parents and their infants found evidence for an ‘overloaded, highly stimulating’ behavioural profile in anxious mothers (Feldman, 2007), along with higher levels of behavioural synchrony (Beebe et al., 2011; Granat, Gadassi, Gilboa-Schechtman, & Feldman, 2017). Experimental investigations have also shown overactive regulatory responses from infants of anxious mothers, particularly following the onset of positive social stimuli (Granat et al., 2017). Lab-based physiological studies have found evidence for ‘stress contagion’, whereby increases in autonomic activity in the mother are reflected in increases in the
infant following emotionally-valenced experimental tasks (Waters, West, Karnilowicz, & Mendes, 2017; Waters, West, & Mendes, 2014). However, investigations of physiological synchrony between infants and parents with anxiety are minimal.

Overall, studies of maternal anxiety and physiological dysregulation in early childhood remain scant. Arousal dysregulation is a core feature of anxiety in adulthood (Ottaviani et al., 2016; Thayer, Friedman, & Borkovec, 1996) and middle childhood (Dieleman et al., 2015; Koszycki, Taljaard, Bialejew, Gow, & Bradwejn, 2019), but the majority of research on this topic focuses on children aged 6 or over (Siess, Blechert, & Schmitz, 2014). In addition, these findings represent analyses of short periods (~<10 minutes) of ‘best behaviour’ lab-based interaction. No previous research has examined whether fluctuations in a child and parent’s biological and behavioural systems associate with one another in naturalistic, day-to-day settings, assessing how these relationships differ between more or less anxious parents.

To address this, we developed new techniques, including miniaturised microphones, video cameras, electro-cardiograms, and actigraphs that could be worn concurrently by infants and parents for a day at a time at home. We recorded both partners’ autonomic fluctuations during the day, by measuring electrocardiography (RR intervals), heart rate variability, and actigraphy (Cacioppo, Tassinary, & Bernston, 2007). We also recorded the auditory environment, and coded the vocalisations spoken by the infant, and those directed to the infant by the parent.

The goal of the current study was to examine differential physiological profiles for infant-parent dyads with higher or lower measures of maternal anxiety. Given previous evidence (Wass et al.,
in press; Beebe et al., 2011), we hypothesised that: (1) infants of more anxious parents would show event-related physiological hyperarousal; (2) co-fluctuation of physiological arousal between infants and more anxious parents would differ from infants with less anxious parents, and (3) inter-dyadic influences in arousal would vary contingent on the arousal level of parent and child, considered separately.

**Method**

*Experimental participant details*

The project was approved by the Research Ethics Committee at the University of East London. Participants were recruited from the London, Essex, Hertfordshire and Cambridge regions of the UK. In total, 91 infant-parent dyads were recruited to participate in the study, of whom usable autonomic data were recorded from 82. Of these, usable paired autonomic data (from both parent and child) were obtained from 74 participants. 68 of these participants also completed the full anxiety screening questionnaire. Further details, including exclusion criteria, and detailed demographic details on the sample, are given in Table 1 and SM section 1.1. Of note, we excluded families in which the primary day-time care was performed by the male parent, because the numbers were insufficient to provide an adequately gender-matched sample. All participating parents were, therefore, female.
Parent screening

To screen parents for maternal anxiety, participants filled out the GAD-7, which assesses anxiety symptoms over the past two weeks (Spitzer, Kroenke, Williams, & Löwe, 2006). Responses were given on a 4-point scale ranging from 0 (not at all) to 3 (nearly every day). Validity for this 7-item questionnaire has been provided by studies with clinical and non-clinical populations, with scores above 6 representing moderate anxiety (Löwe et al., 2008). The internal consistency of the scale was $\alpha = .89$.

Experimental method details

Participating parents were invited to select a day during which they would be spending the entire day with their child but which was otherwise, as far as possible, typical for them and their child. The researcher visited the participants’ homes in the morning (c. 7.30 - 10am) to fit the equipment, and returned later (c. 4 - 7pm) to pick it up. The mean (std) recording time per day was 7.3 (1.4) hours.

The equipment consisted of two wearable layers, for both infant and parent (see Figure 1). For the infant, a specially designed baby-grow was worn next to the skin, which contained a built-in Electrocardiogram (ECG) recording device (recording at 250Hz), accelerometer (30Hz), Global Positioning System (GPS) (1Hz), and microphone (11.6kHz). A T-shirt, worn on top of the device, contained a pocket to hold the microphone and a miniature video camera (a commercially available Narrative Clip 2 camera). For the parent, a specially designed chest strap was also worn next to
the skin, containing the same equipment. A cardigan, worn as a top layer, contained the microphone and video camera. The clothes were comfortable when worn and, other than a request to keep the equipment dry, participants were encouraged to behave exactly as they would do on a normal day. To ensure good quality recordings, the ECG device was attached using standard AgCl electrodes, placed in a modified lead II position.

Figure 1 – left: illustration of parent and child wearing the equipment; right: the equipment used for home monitoring.

Quantification and statistical analysis

Autonomic data parsing and calculation of the autonomic composite measure. Further details on the parsing of the heart rate, heart rate variability, and actigraphy are given in the SM (section 1.2). In the SM (section 1.3) we also present further details on our motivation for collapsing these three measures into a single composite measure of autonomic arousal.
Affect coding. The microphone recorded a 5-second snapshot of the auditory environment every 60 seconds. Post hoc, coders identified samples in which the infant was vocalising, and coded them for vocal affect on a scale from 1 (fussy and difficult) to 9 (happy and engaged). In order to assess inter-rater reliability, 24% of the sample was double coded; Cohen’s kappa was 0.60, which is considered acceptable (McHugh, 2012). All coders were blind to intended analyses. Negative affect vocalisations were defined as all vocalisations coded as 4 or less; positive affect vocalisations included all vocalisations coded on 6 or more; neutral affect vocalisations include vocalisations coded 5.

Home/Awake coding. Our analyses only examine segments of the data in which the dyad was at home, and the infant was awake. This is because our preliminary analyses suggested that infants tended to be strapped in to either a buggy or car seat for much of the time that they were outdoors, which strongly influenced their autonomic data. Further details for how these home/awake segments were identified are given in the SM (section 1.4).

Further details on the permutation-based temporal clustering analyses, and the cross-correlation analyses, used in the Results section are given in the SM (sections 1.5 and 1.6).

Results

The results section is in six parts. In analysis 1 we present raw data and descriptive statistics. In analysis 2 we examine how the high and low parental anxiety groups differed in mean physiological arousal in infant and parent, considered separately. Then, across the four subsequent analyses, we examine how arousal levels co-fluctuate across the dyad during the day. In analysis 3
we examine overall synchrony in infant-parent arousal. In analysis 4 we examine how associations between parent and infant arousal vary contingent on the arousal level of parent and child, considered independently. Analysis 5 examines how adults react to naturally occurring ‘peak’ moments in infant arousal, and Analysis 6 examines how differences in adult reactivity associate with child recovery.

Analysis 1 – raw data and descriptives

![Graph showing infant and adult arousal composites, infant vocal affect, and sample frames from the data recorded from the camera.](image)

Figure 2 – raw data sample. A sample day’s data from a single dyad is shown. Time (from 10am to 5pm is shown on the x axis. From top to bottom: the home/awake coding; the infant and parent arousal composites (see SM section 1.1); infant vocal affect; sample frames from the data recorded from the camera. All measures are calculated as described in the Methods.

The mean (std) (range) of scores obtained on the GAD-7 was 3.4 (3.9) (0-17). A median split was performed to differentiate between high and low anxiety groups. The mean (std) GAD-7 score was 0.76 (0.85) for the low anxiety group and 6.16 (3.96) for the high anxiety group, indicating moderate anxiety.
Table 1 shows demographic data for the sample, subdivided by low/high GAD-7 scores. Independent samples t-tests were conducted for all dimensional variables (i.e. with the exception of ethnicity) to assess whether significant group differences were observed. No significant differences were identified (all ps>.15).

Table 1: Demographic data split by low/high parental GAD-7 scores.

<table>
<thead>
<tr>
<th></th>
<th>Low anxiety</th>
<th>High anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant age (days) – mean (SD)</td>
<td>349 (39)</td>
<td>370 (41)</td>
</tr>
<tr>
<td>Gender (N (%) male)</td>
<td>14 (42)</td>
<td>13 (39)</td>
</tr>
<tr>
<td>Infant Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White British</td>
<td>17 (51)</td>
<td>16 (47)</td>
</tr>
<tr>
<td>Other white</td>
<td>1 (4)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Afro-Caribbean</td>
<td>4 (11)</td>
<td>3 (9)</td>
</tr>
<tr>
<td>Asian, Indian &amp; Pakistani</td>
<td>2 (7)</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Mixed - White/Afro-Caribbean</td>
<td>1 (4)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Mixed - White/Asian</td>
<td>0 (0)</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Other mixed</td>
<td>3 (9)</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Household Income (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under £16k</td>
<td>9 (27)</td>
<td>10 (31)</td>
</tr>
<tr>
<td>£16-£25k</td>
<td>8 (24)</td>
<td>10 (31)</td>
</tr>
<tr>
<td>£26-£35k</td>
<td>7 (20)</td>
<td>3 (9)</td>
</tr>
</tbody>
</table>
We examined how the anxious/non-anxious groups differed on mean arousal levels across the day. This analysis was based on the raw autonomic data included in the arousal composite, prior to the calculation of z scores on a per-participant basis for the composite measure. When considering just samples in which the dyad was at home, and the infant was awake, t-tests indicated no differences between the lower/higher anxiety groups on any of the variables included in the z-scored composite, namely mean waking heart rate, sleeping heart rate, waking or sleeping heart rate variability, for either infants or parents (all ps>.27).
Analysis 3 – Synchrony between infant and parent arousal – differences contingent on parental anxiety

Analysis 2 established that no differences in mean arousal levels were observed between the lower and higher parental anxiety groups. Next, we examined how arousal levels co-fluctuate across the parent-child dyad. To do this, we examined the cross-correlation between infant and parental arousal. Further details on the cross-correlation analyses are given in the SM (section 1.6). In brief, the Time lag 0 cross-correlation indexes whether, at times during the day when the infant’s arousal is higher, the adult’s arousal also tends to be higher, and the cross-correlation at other time lags (e.g. +10 mins) indicates whether the adult’s arousal significantly forward-predicts the infant’s arousal 10 minutes after that moment. Prior to conducting the t-test group comparisons described below, two outliers (one from each group) were excluded using the >2 IQR criterion.

In previous research we used an identical analysis to show that, across all parents, no significant temporal co-fluctuation in infant and parental autonomic arousal levels is observed (Wass et al., under review). When results are subdivided by parental anxiety, however, a significant zero-lagged cross-correlation between infant and parent arousal is observed in the anxious group (t-test vs chance value of 0 (t(32)=4.2, p<.001) but not the non-anxious group (t(32)=1.03, p=.32 (Figure 3a). Group comparisons indicated higher zero-lagged cross-correlations in Group 1 vs Group 2: t(64)=2.16, p=.035. These results indicate that, when considering all home-awake segments of the day, there is significant co-fluctuation between parent and child arousal in the anxious but not the non-anxious group.
Further details and interpretation of the cross-correlation function are given in the SM section 2.1.

Figure 3: a) scatterplot showing the zero-lagged cross-correlation between parent and child arousal, subdivided by maternal anxiety (i.e. low and high GAD-7). * indicates the results of the t-tests conducted as described in the main text p<.05. b) cross-correlation function (CCF) between parent and child arousal, subdivided by low and high parental anxiety. Shaded areas indicate the standard error of the means. * p<.05 following correction for multiple comparisons using permutation-based temporal clustering analyses (see SM section 1.5).

Analysis 4 – Vector plots

Analysis 3 examined differences in arousal synchrony across all data collected while the dyad was at home and the infant was awake. In addition, we also wished to examine how associations
between parent and infant arousal vary contingent on the fluctuating arousal level of parent and child, considered independently. To examine this, we calculated a Vector plot.

Prior to calculating the vector plot, all infant and adult arousal data were averaged separately into 60-second epochs. The arousal data were binned into six equally sized bins, individually for each participant (infant and adult). Then, each possible combination of bins was separated (e.g. [1,1] in which both the infant and adult were in bin 1, i.e. their lowest possible arousal state). For each combination, we calculated the average change between all epochs in that category, and the epoch immediately following. This change score is drawn on the vector plot as a red line. Thus, for the point located at [1,1] on the vector plot, the vector extends +0.3 on the x-axis (representing change in infant arousal), and +0.7 on the y-axis (representing change in adult arousal). This indicates that, across all epochs starting from [1,1], the average change to the next epoch was a gain of +0.3 bins in infant arousal, and +0.7 bins in adult arousal (see Figure 4a, 4b).

Across all data, the vectors tend to point towards the centre of the plot. This indicates regression to the mean: in an epoch where infants’ and parents’ arousal starts low, an increase is expected to the next epoch; whereas for an epoch that starts high, a decrease is expected. The centre point of the vectors appears to be around bin 4 (out of 6), consistent with the lightly positively skewed distribution observed across all data (see Wass et al., in press).

In order to examine how associations between parent and infant arousal vary contingent on the arousal level of parent and child we can examine, for example, the bottom segments of each vector plot (Fig 4a-b), which show instances in which the adult’s arousal is low. The left quadrant (shaded
yellow on Figure 4c) shows instances in which both parent and infant arousal is both low; the right quadrant (shaded red) shows instances in which the parent arousal is low but infant arousal is high. The height of the vector shows the change in adult’s arousal between epochs. It appears that, in both groups, the change (increase) in adult arousal is greater where the infant’s arousal is high than when it is low. To estimate this, we calculated the change in adult arousal between the bottom right and bottom left quadrants of the vector plot (Fig. 4c), and comparing the observed results to a chance value of 0 using a t-test. Results from four participants were excluded (three low/one high) were excluded using the +/- 2IQR rule. For both the low (t(30)=2.03, p=.05) and high (t(32)=2.39, p=.02) GAD-7 groups, marked differences from zero were observed. This indicates that parental arousal increases (to time t+1) more at times when infant arousal at time t is high, than when it is low. There was no significant difference between groups on this measure. This suggests that, when parental arousal is low, parents reactively upregulate their own arousal in response to changes in their child. This finding is true for both the high and low parental anxiety groups.

The top segments of the vector plot (Fig 4c-4d) show instances in which the adult’s arousal is high. In the non-anxious group it appears that the negative vertical displacement of the lines is greater in the top right quadrant (shaded green on Figure 4c), comparing the top left quadrant (shaded brown). If true, this would indicate that, when the adult’s arousal starts high, their arousal decreases more in instances where the infant’s arousal is high, than when it is low. To estimate this, we calculated the change between quadrants, and compared the observed results to a chance value of 0 using a t-test. Results from three participants were excluded (one low/two high) were excluded using the +/- 2IQR rule. For the lower anxiety (t(32)=2.16, p=.04) but not the higher anxiety (t(31=0.75, p=.46) groups, a significant difference was observed. An independent samples
t-test also identified a significant difference between groups on this measure $t(63)=2.05$, $p=.045$. This suggests that, when their own arousal is high, parents down-regulate their own arousal in response to increases in the infant – but that this finding is specific to the non-anxious group.

Figure 4: a)-b) Vector plot illustrating transitions between arousal bins, contingent on starting arousal state. a) shows non-anxious (low GAD-7) group; b) shows anxious (high GAD-7) group. Data were averaged into 60-second epochs and binned from 1 (low) to 10 (high), for infant and parent separately. Thus, an epoch classified as [1,1] indicates an epoch in which both infant and parent were in a low arousal state. The red line indicates the average direction of travel between that and the subsequent epoch, averaged across all epochs in that bin. Thus, for the position [1,1]
on plot a, the red line shows a displacement of +0.3 on the x-axis and +0.4 on the y-axis, indicating that the average epoch starting at [1,1] showed an increase of +0.3 in infant arousal and +0.7 in adult arousal to the subsequent epoch. c) schematic illustrating the analysis whose results are shown in d). Each vector plot was divided into four quadrants – 1 Parent low/Infant low (yellow), 2 Parent low/Infant high (red), 3 Parent high/Infant low (brown) and 4 Parent high/Infant high (green). In order to investigate how infant arousal and adult arousal interacted to predict the change in adult arousal, we subtracted the average adult change scores in quadrant 4 from quadrant 3, and quadrant 2 minus quadrant 1. This was performed separately for the two groups.

d) bar chart showing the results of the analysis. * indicates the significance of the analyses comparing the observed values to a chance level of 0. * - p<.05, † - p=.05.

Analysis 5 – Parental reactivity to ‘peak’ moments of infant arousal – differences contingent on high- vs low parental anxiety

Analysis 3 examines the continuous association between parent and infant arousal across all data. In addition, and motivated by previous findings (Wass et al., in press) we also examined adult reactivity to ‘peak’ arousal events from the infant. Figure 5a shows a schematic illustrating this analysis. First, adult’s arousal data were z-scored, participant by participant. Next, instances where the infant’s arousal crossed a centile threshold (e.g. exceeded the 97th centile of samples for that infant in that day) were identified. Then, for each instance, the change in adult arousal from 600 seconds before to 600 seconds after the infant peak arousal moment was excerpted. Individual instances were averaged to index how the adult’s arousal level changed relative to the ‘peak’
arousal moment of the infant. The analysis was repeated using different values for the centile threshold (Figure 5b). Summary results are shown in Figure 5c. We also performed permutation-based temporal clustering analyses to examine whether, after correcting for multiple comparisons, a significant peak in parent arousal was observed relative to the peak arousal moment in the infant (see SM section 1.5). Instances where a significant peak was observed are drawn as coloured datapoints on Fig 5c (blue/red for high/low GAD-7 groups); instances where no significant peak was observed are drawn as black datapoints.

Results suggest that, for the non-anxious group, significant peaks in adult arousal were observed only relative to the 95th and 97th centile thresholds, whereas, anxious parents show peaks in arousal relative to all thresholds except the 80th centile threshold. This suggests that non-anxious parents upregulate their own arousal relative to ‘peak’ arousal moments in their infant, but not relative to more small-scale fluctuations, whereas anxious parents show greater autonomic reactivity to small-scale fluctuations.
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Figure 5: a) schematic illustrating the analysis shown in b)-c). First, adult’s arousal data are z-scored, participant by participant. Next, instances where the infant’s arousal crosses a centile threshold (e.g. exceeded the 95th centile of samples for that infant in that day) were identified. Then, for each instance, the change in adult arousal from 600 seconds before to 600 seconds after the infant peak arousal moment was excerpted. Individual instances were averaged to index how the adult’s arousal level changed relative to the ‘peak’ arousal moment of the infant. The analysis was repeated using different values for the centile threshold. b) change in parent arousal relative to ‘peak’ arousal moments of the infant, defined using variable centile thresholds. c) summary plot showing just the time 0 parent arousal levels from the plots in b. Where the permutation-based temporal clustering analyses indicated that a significant peak in adult arousal was observed.
relative to the infant ‘peak’ arousal event, the datapoint has been drawn in colour (blue/red for anxious/non-anxious group, i.e. high/low GAD-7 groups). Where no significant peak in adult arousal was observed, the datapoint has been drawn black.

Analysis 6 – infant recovery from extremes of vocal affect – relationship to parental behaviours

Analysis 5 examines how adults react to naturally occurring ‘peak’ moments in infant arousal during the day. In addition, we wished to examine how these differences in adult reactivity in turn affect the infant. To examine this, we first identified all infant vocalisations that occurred during the day; for each vocalisation, we examined the rate of change of infant physiological arousal relative to these vocalisations (Figure 6a-6c).

The significance of group differences was calculated by first conducting t-tests separately for each individual time bin, and then correcting for multiple comparisons using a permutation-based clustering analysis (see SM section 1.5). As expected, all vocalisations showed a significant peak in infant autonomic arousal at time 0 – i.e. the time of the infant vocalisation (all permutation-based clustering ps<.001). The anxious group showed significantly higher physiological arousal at the time of the vocalisation, along with significantly high arousal during the period 8-12 minutes after the vocalisation, indicating slower recovery (Fig 6a, p=.023). A similar pattern was evident following positive affect vocalisations (Figure 6b, p<.001), but not following neutral affect vocalisations. These differences were not attributable to differences in the frequency of vocalisations as these did not differ significantly between groups (z=.31/1.50/.97, p=.75/.30/.33 for negative/positive/neutral affect vocalisations respectively).
Figure 6 – a)-c) change in infant autonomic arousal relative to: a) negative affect vocalisations; b) positive affect vocalisations; c) neutral affect vocalisations. For each plot, the blue line shows the anxious group (High GAD-7), and the red line the non-anxious group (Low GAD-7). Shaded area shows the S.E. Areas identified as showing above-chance group differences following correction for multiple comparisons using the permutation-based clustering analysis are highlighted with *.

We also wished to assess how infant recovery related to the differences in parental reactivity described in analysis 4 above. To do this, we measured the degree to which maternal autonomic reactivity is specific to ‘peak’ infant arousal moments using the following method. For each participant, the maternal arousal response to >97th centile infant arousal moments was calculated (see Figure 5b). This was done by averaging the z-scored maternal arousal values from 3 minutes before and after the peak infant arousal moment (corresponding to the peaks visible on Figure 5b; analyses were also repeated using other time windows with similar results). For each participant, the maternal arousal response to >75th centile arousal moments was also calculated (see Figure 5b). The degree to which maternal autonomic reactivity is specific to ‘peak’ infant arousal moments was calculated by subtracting the >97th centile arousal responses from the >75th centile
responses, so that a larger value indicates that maternal autonomic reactivity is more specific to ‘peak’ infant arousal moments.

Infant recovery was assessed by calculating the average infant arousal during the period from 1200 seconds before and after the positive and negative affect vocalisations (corresponding to the time periods shown in Figure 6), and subtracting the average arousal during the period after the vocalisation from the average arousal during the period before. (Again, results of analyses using other time windows were highly similar.) In order to assess how infant recovery related to parental reactivity, we calculated the bivariate correlation between the two measures. Infant recovery following negative affect related to more selective parental reactivity (i.e. a bigger difference between >97th centile and >75th centile arousal responses) $\rho=-.33$ $p=.045$. This finding was observed consistently in the lower ($\rho=-.31$) and higher ($\rho=-.50$) parental anxiety groups. No relationship was observed between the same variable and infant recovery following positive affect ($\rho=-.07$). Overall, these findings suggest that parents whose autonomic reactivity is more specific to ‘peak’ infant arousal moments have infants who recover faster following negative affect.

**Discussion**

The present study aimed to examine how anxious symptoms in parents relate to affect co-regulation in parent-infant dyads. To address this, we used miniaturised microphones and cameras, and wearable physiological monitors, to record vocalisations and day-long physiological fluctuations in 12-month-old infants and their parents. Participating parents completed a self-rating scale of current anxiety symptoms (the GAD-7).
Our results indicated that mean levels of autonomic arousal did not differ between the more or less anxious groups for either parent or infants (Analysis 2). This is informative, because previous research has concentrated on physiological reactivity to anxiogenic situations in children of anxious parents (Nikolić, Aktar, Bögels, Colonnesi, & Vente, 2018), rather than examining mean physiological arousal in home settings. We did, however, find differences in how arousal levels in dyads associated with each other throughout the day (Analysis 3). Overall, dyads in the more anxious group showed higher synchrony in physiological arousal. Conversely, in the less anxious group, mothers’ arousal levels were less tightly coupled with infant levels (Fig 3b and SM section 2.1).

Higher levels of matched dyadic biobehavioral activity have traditionally been associated with adaptive parenting and positive child outcomes (Leclère et al., 2014). However, recent research has suggested that excessive coupling may in fact be characteristic of interactions between anxious mothers and their infants. Analyses investigating behavioural synchrony found that anxious mothers showed greater synchrony in gaze and touch than depressed mothers or control mothers (Granat et al., 2017), and research into highly vigilant parents has also shown high levels of adrenocortical synchrony (Pratt et al., 2017). Theories of psychopathology and synchrony suggest that there may be an ‘optimal midrange’ of synchrony (where too little coordination marks withdrawal, and too high coordination marks vigilance; Beebe et al., 2011).

Also consistent with these theoretical perspectives are our findings examining how parents react to small- vs large-scale arousal fluctuations in their child (Analysis 5). Non-anxious mothers were
‘there when you need me’: they showed reactive autonomic changes relative to peak arousal events from their child, but did not show changes to their child apart from that. Conversely, anxious mothers were ‘always on’: they did not show greater autonomic reactivity to these peak arousal events in their child, but showed more reactivity to small-scale fluctuations of child physiological arousal. Parents whose autonomic reactivity is more specific to ‘peak’ infant arousal moments have infants who recover faster following negative affect (Analysis 6). These findings support evidence for an ‘overloaded, highly stimulating’ behavioural profile in anxious mothers (Feldman, 2007), that leave insufficient time for infants to experience neutral affect, or ‘time off’, thereby losing opportunities to practice self-regulation.

Finally, our results provide evidence on anxious parents’ capacity to change the overall arousal levels of the dyad contingent on the starting arousal level of parent and child, considered separately (Analysis 4). When the overall level of physiological arousal in the dyad was high, the less anxious mothers decreased their own physiological arousal, bringing the total level of physiological arousal in the dyad down - showing behaviours akin to ‘stress buffering’ (Hennessy, Kaiser, & Sachser, 2009). This behaviour was absent among more anxious mothers. Our findings suggest that the mechanism by which affective and arousal states are transmitted from one partner to another does not operate consistently across more anxious and less anxious dyads, and may therefore be a fruitful target for further research.

Our research is limited by several factors. Firstly, our sample was sourced from the community, and group differences were established from a median split of a uni-dimensional measure of parental anxiety (the GAD-7). Though there is genetic evidence that total GAD-7 scores have the
same genetic underpinnings as professionally diagnosed anxiety disorders (Purves et al., 2019), our findings nonetheless only represent moderate levels of anxiety in the ‘more anxious’ group. Further research with a clinical sample would be needed to investigate the effects of moderately severe and severe levels of anxiety in mothers. A second limitation of our study is that we investigated biobehavioural relations between mother-infant dyads, and not father-infant dyads; research has suggested that gender differences in parents are relevant for childhood anxiety disorders, and should be a focus in the future (Majdandžić, Möller, Vente, Bögels, & Boom, 2014; Möller, Majdandžić, & Bögels, 2015). A third limitation is the study’s cross-sectional design; although our results provide information about dyadic interaction in the short term, longitudinal studies could consider longer term relationships between parental anxiety, excessive synchrony and later child outcomes. Finally, our research did not differentiate anxiety subtypes, for example general anxiety disorder versus panic disorder or social anxiety disorder; evidence suggests children respond differentially to parents on these bases, and therefore these subtypes should be incorporated into future research among mother-infant dyads (de Rosnay, Cooper, Tsigaras, & Murray, 2006; Murray, Cooper, Creswell, Schofield, & Sack, 2007).

Our research provides new information on how the regulatory profiles of anxious mother-infant dyads are inter-dependent on one another, and how this informs the individual partners’ capacity to downregulate arousal levels. It also contributes to the evidence-base on the intergenerational transmission of anxiety from parent to infant, building on our understanding of how parent-child interactions differ in anxious parents during the first year of life. The research also provides evidence that even in mothers without a professional diagnosis of anxiety, there are apparent effects of maternal anxiety on physiological processes in both mother and infant. This information
is helpful for developing our knowledge of the environmental mechanisms underlying the development of anxiety disorders. It may also inform future intervention studies focused on reducing overall levels of anxiety in the dyad, whether or not the parent has a clinical diagnosis; for example, targeting interoceptive capacities in the parent.

Acknowledgements. This research was funded by ESRC grant number ES/N017560/1. We wish to thank Joan Eitzenberger, Caitlin Gibb, Leanne Barnes and Louise Stubbs for contributing to data coding used in the manuscript, and all families who participated in the research.

Key points

- Infants of more anxious mothers take longer to calm following extremes of positive or negative affect.
- In home settings, physiological arousal is more strongly synchronised between more anxious mothers and their infants.
- More anxious mothers show greater autonomic reactivity to small-scale fluctuations in their infant.
- Less anxious mothers show physiological responses akin to stress buffering, whereas this is absent in more anxious mothers.
References


ANXIETY AND PHYSIOLOGICAL SYNCHRONY


