Getting in synch: Unpacking the role of parent-child synchrony in the development of internalizing and externalizing behaviors

Laura E. Quiñones-Camacho[1], **Caroline P. Hoyniak**[1], **Lauren S. Wakschlag**[2], **Susan B. Perlman**[1]

[1]Washington University in St. Louis School of Medicine, Department of Psychiatry

[2]Northwestern University, Department of Medical Social Sciences, Feinberg School of Medicine and Institute for Innovations in Developmental Sciences

Abstract

While substantial research supports the role of parent-child interactions on the emergence of psychiatric symptoms, few studies have explored biological mechanisms for this association. The current study explored behavioral and neural parent-child synchronization during frustration and play as predictors of internalizing and externalizing behaviors across a year and a half span. Parent-child dyads first came to the laboratory when the child was 4-5-years-old and completed the DB-DOS: BioSync task while functional near-infrared spectroscopy (fNIRS) data were recorded. Parents reported on their child's internalizing and externalizing behaviors using the Child Behavior Checklist (CBCL) four times over a year and a half. Latent Growth Curve modeling was conducted to assess neural and behavioral synchrony as predictors of internalizing and externalizing trajectories. Consistent with previous investigations in this age range, on average, internalizing and externalizing behaviors decreased over the four timepoints. Parent-child neural synchrony during a period of play predicted rate of change in internalizing but not externalizing behaviors such that higher parent-child neural synchrony was associated with a more rapid decrease in internalizing behaviors. Our results suggest that a parent-child dyad's ability to coordinate neural activation during positive interactions might serve as a protective mechanism in the context of internalizing behaviors.

Keywords

Neural Synchrony; Internalizing; PFC; Parent-child synchrony; Externalizing

Internalizing and externalizing behaviors in early childhood can place children on developmental trajectories toward clinically significant levels of symptoms later in life (Beesdo et al., 2007; Coie & Dodge, 1998; Copeland, Shanahan, & Costello, 2009; Goodwin, Fergusson, & Horwood, 2005; Kovacs & Devlin, 1998; Keiley et al., 2003; Luby et al., 2014), suggesting that levels of internalizing and externalizing behaviors early in life are useful predictors of later risk. Internalizing behaviors— distress responses characterized

by fearfulness, withdrawal, anxiety, and somatic complaints (Achenbach, 1966; Achenbach & Edelbrock, 1978) constitute some of the most common forms of psychopathology (Kessler et al., 2005; Luby et al., 2003; Merikangas et al., 2010) and show some stability throughout the lifespan (Carter et al., 2010). On the other hand, externalizing behaviors— overt distress responses characterized by aggression, hyperactivity, defiance, and destructive behaviors (Achenbach, 1991, Campbell et al., 1995) are common early in life, and tend to show strong declines throughout the childhood years (Coie & Dodge, 1998). Studies probing early onset of both types of behaviors have pointed to the parent-child relationship as a key predictor of concurrent levels and longitudinal trajectories (Hollenstein et al., 2004; Leve et al., 2005; McLeod, Wood, & Weisz, 2007; Rapee, 2012; Wu & Lee, 2020). For example, one study with kindergarteners found that more rigid parent-child interactions were associated with more child externalizing and internalizing behaviors concurrently and predicted growth of externalizing behaviors over a one-year period (Hollenstein et al., 2004). Early childhood is a particularly important time for identifying risk trajectories towards psychopathology (Dougherty et al., 2015; Wakschlag et al., 2019) and represents a developmental stage when children still heavily depend upon their parents to serve as primary emotion socialization agents (Denham, Bassett, & Wyatt, 2007). Given the predictive role of preschool-age internalizing and externalizing behaviors on later psychopathology, and the role that the parent-child relationship plays in the emergence and maintenance of symptoms, a more in-depth exploration of parent-child predictors of trajectories of internalizing and externalizing behaviors in this period is warranted. An explicit exploration of parent-child interactions may help to identify targets for the mitigation of risk for psychiatric disorders in early childhood.

Parent-Child Synchrony

There is growing evidence that parent-child interactions from early in life are reciprocally shaped by both parent and child (Feldman, 2007a, 2007b; Lunkenheimer et al., 2020; Lunkenheimer et al., 2011; Murray et al., 2016), and that these bidirectional influences cannot be fully accounted for by examining each member of the dyad independently (Granic, 2000; Moore et al., 2013). Thus, parent-child interaction measures are a promising avenue for identifying dyadic-level predictors of psychopathology trajectories early in life as they offer unique information about the parent-child relationship. Parent-child synchrony refers to a dyadic pattern of interaction by which behavioral and biological states are coordinated within the dyad, often through co-regulated interactions characterized by contingent responding and social reciprocity (Condon & Sander, 1974; Feldman, 2012; Harrist & Waugh, 2002). Parent-child synchrony is evident from infancy (Condon & Sander, 1974; Feldman, Greenbaum, Yirmiya, 1999; Ham & Tronick, 2009), supports attachment and bond formation within the parent-child dyad (Fleming, O'Day, & Kramer, 1999; Leclere et al., 2014), and remains a beneficial index of adaptive social interactions throughout the lifespan (e.g., Helm, Sbarra, & Ferrer, 2013; Yaniv et al., 2020). Research has consistently demonstrated that stronger positive parent-child behavioral synchrony is an important predictor of better self-control, increased communicative competence, higher empathy and moral internalization, and fewer behavioral problems in cross-sectional and longitudinal work (Feldman, Greenbaum, & Yirmiya, 1999; Feldman, 2007a, 2012; Harrist & Waught

2002; Im-Bolter, Anam, & Cohen, 2015; Jaffe, Beebe, Feldstein, Crown, & Jasnow, 2001; Kochanska et al., 2008; Lindsey et al., 2009; Scholtes et al., 2020). For example, Im-Bolter, Anam, and Cohen (2015) found that 6-10-year-old children with clinical levels of behavioral problems (a combined measure of internalizing and externalizing behaviors) showed significantly lower parent-child behavioral synchrony during play compared to a non-clinical group.

While behavioral synchrony measures are the most commonly used measures of parent-child synchrony, there is work to suggest that biological measures of parent-child synchrony offer complementary information about parent-child interactions. Studies examining the coordination of biological states – termed biological synchrony— often find similar links to those reported in behavioral studies (Feldman, 2007c; Kalomiris & Kiel, 2018; Lunkenheimer et al., 2018; Suveg et al., 2019). For example, in a study with preschoolers and their mothers, lower parent-child autonomic synchrony (measured via respiratory sinus arrhythmia [RSA], an index of parasympathetic activation) during periods of mild stress and free play were associated with a higher risk for behavioral problems (both internalizing and externalizing behaviors; Lunkenheimer et al., 2018). Interestingly, this period of mild stress resulted in weaker autonomic synchrony compared to the period of free play. Importantly, these contexts were also differentially associated with internalizing and externalizing behaviors. While children's externalizing behaviors were associated with weaker autonomic synchrony during the stressful context as well as the free play context, children's internalizing behaviors were associated with weaker autonomic synchrony during a period of free play only, suggesting that context might play a role in these associations (Lunkenheimer, et al., 2018). In other studies, weaker positive parent-child synchrony in cortisol concentration, as well as weaker autonomic synchrony (i.e., RSA synchrony) have also been linked with higher levels of internalizing behaviors in childhood (Kalomiris & Kiel, 2018; Suveg et al., 2019). One additional study by Suveg and colleagues (2019) found that preadolescents with high levels of internalizing behaviors showed negative RSA synchrony with their mothers during a modified Trier Social Stress Task (Kirschbaum, Pirke, & Hellhammer, 1993), while preadolescents with low levels of internalizing behavior showed positive RSA synchrony, suggesting that parent-child synchrony might be disrupted in dyads with a child high in internalizing behaviors. These results suggest that there is growing evidence that disrupted patterns of parent-child synchrony are linked with more internalizing and externalizing behaviors across childhood, and that parent-child biological synchrony might be a particularly important predictor of internalizing trajectories. While much more research is needed, results from these and other similar studies (e.g., Smith et al., 2019) suggest that strong parent-child synchrony might not be universally adaptive, and that in some cases, high parent-child synchrony might increase risk for behavioral problems. For example, stronger parent-child synchrony in the context of strongly negative interactions is likely to increase risk for significant behavioral problems in children already showing signs of behavioral problems or at high-risk of developing them. Thus, while positive forms of parent-child behavioral synchrony are likely to be universally positive, it is still unclear at what point in the spectrum from typically-developing to clinically-impaired and under which contexts (e.g., mildly negative vs. highly negative interactions) parent-child physiological synchrony become a risk rather than a protective factor.

Neural Synchrony

While most research on parent-child synchrony has focused on behavioral and physiological measures, recent work has started to explore the synchronization of neural responses (often referred to as hyperscanning) as a way to assess the neural underpinnings of dyadic coordination during in-vivo interactions (Azhari et al., 2019; Hoyniak et al., 2021; Miller et al., 2019; Montague et al., 2002; Nguyen et al., 2020; Quiñones-Camacho et al., 2021; Reindl et al., 2018). This synchronization of neural activation has been theorized to facilitate the formation of bonds and shared mental states (Redcay & Schilbach, 2019; Wheatley et al., 2012) by helping optimize internal models of complex dynamic environments, enhancing memory and attention to the interacting partner, while reducing the cognitive resources that need to be engaged during the social interaction (Macrae et al., 2008; Miles et al., 2009; Miles et al., 2010). Emerging evidence suggests that increased parent-child neural synchrony in typically developing children might play an important role in children's healthy development (Miller et al., 2019; Quiñones-Camacho et al., 2019b; Reindl et al., 2018). To illustrate this, one study of 5-9-year-olds found that stronger positive parent-child neural synchrony in the prefrontal cortex (PFC) during cooperation was linked with better emotion regulation in both the parent and the child and mediated the association between parent and child emotion regulation (Reindl et al., 2018). In another study, with preschoolers and their mothers, communicative reciprocity (i.e., turn-taking) during a conversation was associated with higher neural synchrony (Nguyen et al., 2020). Thus, research to date suggests that neural synchrony of the PFC might be an underlying biological mechanism for dyadic attunement facilitating behavioral synchronization and socialization processes more broadly. While work on parent-child neural synchrony has started to explore synchrony under various contexts such as cooperation conditions (Miller et al., 2019; Nguyen et al., 2020a), passive video watching (Azhari et al., 2019), a conversation (Nguyen et al., 2020b), or has compared cooperation vs. competition conditions (Reindl et al., 2018), little research has explored how emotionally salient interactional contexts might influence parent-child neural synchrony. This is particularly important when thinking about possible links between parent-child neural synchrony and psychopathology, as contexts that require some level of active emotion co-regulation may be more informative for predicting risk for psychopathology. We know from research on child development that parent-child physiological synchrony during contexts with varied regulatory demands differentially relate to child outcomes (e.g., Lunkenheimer, et al., 2018; Quiñones-Camacho et al., 2019b). Thus, it is possible that measures of neural synchrony during varying tasks contexts would also differentially relate to child outcomes. Demonstrating the utility of assessing parent-child neural synchrony during contexts with varying regulatory demands, a recent study with 4-5-year-olds found that lower parent-child neural synchrony during a period of play following a frustration induction was associated with higher child irritability, a transdiagnostic marker of both externalizing and internalizing behaviors (Quiñones-Camacho et al., 2019b). This study offers, to our knowledge, the only evidence to date that parent-child neural synchrony is linked with early markers of risk for psychopathology.

Current Study

The goal of the current study was to examine behavioral and neural parent-child synchrony as predictors of longitudinal changes in internalizing and externalizing behaviors across the preschool period. In particular, we extend findings from previous work by exploring whether neural synchrony during a frustration induction and a period of play predict trajectories of internalizing and externalizing behaviors across a year-and-a-half period. Moreover, we explored whether similar associations between synchrony and symptoms would be present when using measures of behavioral versus neural synchrony. We hypothesized that there would be modest decreases in internalizing and externalizing behaviors throughout this period, consistent with previous studies that have investigated trajectories of internalizing and externalizing behaviors in this age range (Bub et al., 2007; Coie & Dodge, 1998; Fanti & Henrich, 2010; Schappin et al., 2018). Based on previous work showing that internalizing and externalizing behaviors are differential associated with parent-child physiological synchrony based on task context (e.g., mild stress vs. free play; Lunkenheimer et al., 2018), we hypothesized that higher levels of parent-child neural synchrony in our community sample during a frustration period would predict lower initial levels and a greater rate of change (i.e., greater decreases) in externalizing behaviors and that higher levels of parent-child neural synchrony during the period of play following the frustration induction would predict lower initial levels and greater rate of change (i.e., greater decreases) in internalizing behaviors. Lastly, we explored whether behavioral synchrony would show similar associations with internalizing and externalizing symptoms as did neural synchrony.

Method

Participants

One hundred and fifty-one preschoolers (4-5 years-old at Time 1; M = 4.85 years, SD = .6) and a caregiver (144 mothers) took part in a longitudinal study designed to explore the neural underpinnings of emotional development and the emergence of psychopathology in the preschool period. The current study uses data from four timepoints, an initial visit when the children were 4-5-years-old and three other time points that were separated by 6 months (T1 = 0 months; T2 = 6 months from initial visit; Time 3 = 12 months from initial visit; T4 = 18 months from initial visit). Families were excluded from participating in the study if the parents reported having already sought clinical services for their child or if their child had any current or past psychiatric diagnosis at the first time point. Children were also excluded if they had a neurological disorder, a history of loss of consciousness, or sensory impairments, such as epilepsy, cerebral palsy, ASD, or significant intellectual disability. The study was approved by the Institutional Review Board and all families were consented before participation in the study. Parents reported on their child's race. The racial breakdown was 68% White, 23% Black or African-American, 6% Biracial, 2% Asian American, and 1% Native American or Pacific Islander. Children were identified as being 95% Non-Hispanic. Household income ranged from 70 families (47%) reporting an income of less than $60,000, 55 (36%) reporting an income of $61,000-$120,000, and 26 (17%) reporting an income higher than $121,000 a year.

Child Internalizing and Externalizing Behaviors

Parents completed the Child Behavior Checklist (CBCL; Achenbach, 1991) at all four time points. The CBCL is a widely used assessment of problem behavior in children. Given the focus of the study, we used the internalizing and externalizing behaviors scales. We used two versions of the CBCL, one version that has been validated for ages 1.5-5 and another for ages 6-18; parents were asked to complete the version corresponding to their child's age at the time of assessment. In both versions parents rate their child's behavior using a 3-point Likert scale (0 = *not true*; 1 = *somewhat true*; 2 = *very true*). Given our interest in explicitly mapping longitudinal changes in internalizing and externalizing behavior in a community sample, we used the raw scores rather than the age and gender normalized t-scores for analyses. The psychometric properties of the CBCL have been previously demonstrated (Achenbach, 1991, Achenbach & Rescorla, 2001). Reliability for the internalizing subscale was good in our sample (T1 1.5-5 version: a =.82; T2: 1.5-5 version a =.81; 6-18 version a =.86; T3: 1.5-5 version a =.85; 6-18 version a =.83; T4: 1.5-5 version a =.82; 6-18 version a =.81). As was the reliability for the externalizing subscale (T1 1.5-5 version: a =.91; T2: 1.5-5 version a =.92; 6-18 version a =.80; T3: 1.5-5 version a =.91; 6-18 version a =.90; T4: 1.5-5 version a =.90; 6-18 version a =.88). Means, standard deviations, and ranges for each time point can be found in Table 1. Using CBCL standardized scores, 16 children (11%) were in the borderline or clinical range for internalizing at T1, and 10 (7%) for externalizing. For T2, 16 (11%) were in this range for internalizing, and 16 (11%) for externalizing. T3 scores were on a similar range (18 (12%) internalizing; 20 (13%) externalizing), as were T4 scores (16 (11%) internalizing; 19 (13%) externalizing). Nine children were missing CBCL data at Time 2, ten were missing data at Time 3, and eleven were missing data at Time 4, all children had data at Time 1, and 148 children had data for at least two time points. All missing CBCL data occurred due to missing a visit in the longitudinal study.

Parent-child synchrony task

The current study uses data from the Disruptive Behavior Diagnostic Observation Schedule: Biological Synchrony (DB-DOS: BioSync; Quiñones-Camacho et al., 2019b) during the initial in-lab visit. The DB-DOS: BioSync was adapted from the validated DB-DOS (Wakschlag et al., 2008), which was designed to elicit variations in emotional and behavioral regulation and to assess parent-child dyads' ability to co-regulate across contexts with varying demands. The modified version of the DB-DOS used for this study was developed to fit task requirements of fNIRS and other biological measures (e.g., minimization of movement). The DB-DOS: BioSync consists of two contexts, a 'Frustration' induction context always followed by an unstructured play context. The 'Frustration' context consisted of a period of 10 minutes where the dyads were instructed to complete challenging tangram puzzles together as fast as they could while refraining from playing with attractive toys left next to the table. The puzzles consisted of 7 flat geometric shapes that were combined to form a larger shape (e.g., a cat). This Frustration context consisted of 4 blocks of solving up to 5 puzzles within a 2-minute window, followed by a 15-second inter-block interval. To motivate the dyads to work together to complete the puzzles, they were told that they would receive a prize if they completed the task. To further increase the frustrating nature of this context, the puzzles were too difficult for the child's developmental stage, they were given

1:45 minutes instead of the expected 2:00 minutes, and they were shown a countdown clock on a screen indicating how much time they had left. The Frustration context was followed by the Play context, which also consisted of 10 minutes divided into four blocks of 2 minutes, followed by a 15-second inter-block interval. During this period, dyads were told they could play with the attractive toys that had been originally placed next to them. Each block was an opportunity for the dyad to add a new toy.

fNIRS data acquisition and preprocessing

A NIRScout fNIRS system (NIRx Medical Technologies LLC, Glen Head, NY) was used to collect non-invasive optical imaging (i.e., fNIRS) data using a continuous-wave system at Time 1. fNIRS data was also collected at T3, but this data is not included in the current manuscript. Light was emitted at 760 nm, and 850 nm from eight LED light sources and measured by 4 photodiode light detectors, resulting in 10 ten measurement channels per wavelength. Optical signals were collected at 15.625 Hz. Sensors were mounted on a neoprene head cap, with a source-detector distance of 2.9-3.1 cm. The head caps were placed following the international 10-20 coordinate system for both the parent and the child, with the dorsomedial sources over AF3/AF4, and the ventromedial sources over Fp1/Fp2 (Figure 1). This placement resulted in the probe extending over middle frontal gyrus (MFG) and inferior frontal gyrus (IFG) of each hemisphere of the PFC and were registered to the Colin27 Brain Atlas (Holmes et al., 1998). When necessary, hair was manually separated under the optodes to improve signal detection. Out of the original 151 subjects, 117 dyads had usable fNIRS data (for both members of the dyad) at Time 1, data loss was due to computer errors, poor contact of the sensors with the scalp, or too much movement in either the parent or the child.

The preprocessing of the fNIRS data and activation analyses were carried out in MATLAB (MathWorks, Natick MA USA) using the NIRS Brain AnalyzIR toolbox (Santosa et al., 2018). The raw fNIRS intensity signals were first converted to changes in optical density. These optical density signals were then motion corrected using the temporal derivative distribution repair (TDDR) method (Fishburn et al., 2019). This method utilizes a robust regression approach to remove large fluctuations in the optical density signal (attributed to motion artifacts) while retaining smaller fluctuations (attributed to hemodynamic activity). The motion-corrected signals were then resampled to 4 Hz to reduce computational overhead for the synchrony analyses, and a high-pass filter (cutoff of 0.01 Hz and filter order of 4) was used to remove slow drifts in the signal. After this, the optical density signals were converted to oxygenated hemoglobin concentration using the modified Beer-Lambert law.

Quantification of Neural Synchrony

Parent-child neural synchrony was defined as the concurrent lateral PFC activation of the parent and the child during the 'Frustration' and 'Play' contexts separately. First, timings were standardized across participants. After this, signals were whitened by eliminating the temporal autocorrelations using an autoregressive model. This was done as serial correlations are a common source of noise in fNIRS data and could inflate correlation estimates Santosa et al, 2017). There is evidence that serial correlations in time series data

can artificially inflate functional connectivity estimates from wavelet transform coherence or Pearson correlations, and that this increased false discovery rate can be controlled by using a robust correlation approach with temporally whitened signals (Santosa et al., 2017). Thus, we chose this approach for our analyses. The Bayesian Information Criterion was used to choose the order of the autoregressive model from a minimum value of 1 to a maximum of 32 (20 has been shown to be enough; Santosa et al., 2017). A robust regression approach was then used to calculate robust correlation coefficients between participants (Shevlyakov & Smirnov, 2011). Parent-child neural synchrony was then quantified using the Fisher r-to-z transformation of the absolute value of the robust correlation coefficient. This was done for all possible channel-pairs. Reciprocal connections were enforced to reduce the number of unique connections and thus prevent multiple comparisons corrections from being overly-conservative.

Parent-Child Neural Synchrony

Significance of the neural synchrony analyses was estimated via permutation testing with random dyads (e.g., parent of dyad B with child of dyad D). This approach allowed us to confirm that the synchrony was driven by a dyad's active interaction rather than being driven by two people completing similar tasks. Neural synchrony was calculated between all possible subject pairs to determine the appropriate null distribution of neural synchrony values. Due to some data loss, there were neural synchrony values for 117 concurrent parent-child dyads and 27,144 non-concurrent (null) parent-child dyads ($N_{null} = \frac{N_{subject}^2 - N_{subject}}{2} - N_{dyad}$). Permutation testing was then conducted to calculate the p-value associated with each concurrent dyad's neural synchrony value by calculating the proportion of values from null-pairings that were equal to or greater than the observed value, e.g. $\hat{p} = \frac{\sum (Z_{null} \geq Z_{observed}) + 1}{N + 2}$. The constant terms were chosen to guarantee that the resulting p-values would be between 0 and 1. After this, adjusted z-values were calculated from the estimated p-values using an inverse cumulative density function for the standard normal distribution. One dyad had an adjusted Z-value that was over 4 SD and were removed from analyses. One additional dyad had usable data but was excluded due to a child's brain abnormality identified via MRI at a later timepoint. Adjusted Z-values were submitted to a mixed-effects model with task condition modeled as a fixed effect and dyad ID modeled as a random effect. The presence of parent-child neural synchrony was assessed for each condition using the t-contrast corresponding to a 1-sample t-test. Lastly, the corresponding p-values were corrected for multiple comparisons by calculating the Benjamini-Hochberg FDR-corrected p-value (Benjamini & Hochberg, 1995) across all unique channel pairs. Both conditions resulted in significant neural synchrony compared to the null-distribution, and no significant differences in neural synchrony emerged between conditions (for a full description of the neural synchrony findings, which are not described further in this manuscript, please refer to Quiñones-Camacho et al., 2019b). In order to include parent-child neural synchrony as a predictor in the latent growth models, we extracted synchrony values for each context from a channel-pair that showed the strongest effect in the mixed-effect model (i.e., peak channel) to be used for further analyses. This channel generally corresponds to the right DLPFC. This allowed to reduce the number of parameters estimated

from using all significant channel pairs and allowed for a closer comparison of the neural and behavioral synchrony models.

Parent-Child Behavioral Synchrony

Instances of parent-child behavioral synchrony were assessed during both contexts of the DB-DOS: BioSync task. Behavioral synchrony was a global code and was defined as the amount of time the dyad spent engaged in mutually responsive and co-regulated interactions via shared attention, reciprocal communication, eye contact, and coordinated behaviors (Coding Scheme can be found in Supplemental Material 1). Every second of each of the contexts (separately) was coded as being either synchronous or asynchronous. A synchronous code, thus, indicated that the dyad engaged in a mutually responsive and co-regulated interaction during that second of the interaction. Specifically, each second of the interaction was coded as synchronous if the dyad showed reciprocal communication, eye contact, and coordinated behaviors with directed gaze during that period. Before the first synchrony code was given, the dyad had to exchange three verbal or behavioral turns. Since our coding of behavioral synchrony was a single global score, waiting for the dyad to exchange three verbal or behavioral turns before coding the interaction as synchronous ensured that the dyad was in fact engaging in reciprocal responding, which is necessary to establish synchrony and is likely to take more than a second. Behavioral synchrony continued to be coded until there was a break in the reciprocal exchanges (e.g., the dyad did not show any reciprocal responding for more than three seconds). After a period of asynchrony, parent-child dyads could regain synchrony by engaging in coordinated and reciprocal interactions for at least three seconds. These individual second-by-second measures were summed to create a general behavioral synchrony score for each context (i.e., the total time spent in synchrony during Frustration and Play). Videos were coded offline. Of the 151 dyads who participated in the study, only 127 had codable videos (missingness was due to problems with the video camera or audio). Parent-child behavioral synchrony was coded by six trained research assistants who did not interact with the dyad during the visit. Behavioral synchrony training comprised of an initial conceptual grounding, followed by coding for eight master tapes to 0.80 reliability (kappa) of the master codes. After this, coders were assigned new videos to code. Reliability was calculated on 20% of data for all codable videos and was acceptable ($K = .81$). There were no significant differences in behavioral synchrony between conditions.

Data Analysis Plan

To examine the association between parent-child neural synchrony and change in internalizing and externalizing behaviors across early childhood, we used Latent Growth Curve (LGC) modeling. LGC analysis allows for the modeling of change over time in internalizing and externalizing behaviors while also allowing for the investigation of between-person variability in change and predictors of rate of change (McArdle & Epstein, 1987; Willett & Bub, 2005). LGC modeling was done using MPlus version 8.4 (Muthen & Muthen, 1998-2017). Internalizing and externalizing behaviors data from the CBCL across the four time points were used to estimate latent intercept and slope factors. The latent intercept factor was centered at Time 1, making its interpretation equivalent to the level of internalizing (or externalizing) behaviors at Time 1. The latent slope factor represents the

rate of change from Time 1 to Time 4 (1.5 years later). Variances for the latent intercept and slope factors reflect the presence of individual differences in initial levels and rate of change (for latent intercept and slope, respectively). After an initial growth model was established, models were fitted for parent-child neural synchrony during Frustration and Play, separately. Specifically, the single neural synchrony score extracted for each condition (described in pg. 13) was entered as a predictor of the intercept and slope in the growth curve model. All models were estimated using Full Information Maximum Likelihood (FIML), and model fit was determined via examination of the Chi-square, the Comparative Fit Index (CFI), Tucker Lewis Index (TLI), the Root Mean Squared Error of Approximation (RMSEA), and the Standardized Root Mean Square Residual (SRMR). Standard guidelines were used to estimate good model fit, such as non-significant chi-square, CFI and TLI values higher than .95, and RMSEA and SRMR values smaller than .06 (Hu & Bentler, 1998).

Results

Preliminary results

Descriptive statistics and correlations among study variables can be found in Table 1. There were no sex differences in any of our variables of interest ($ts < .983$, $ps > .325$). Age at the initial visit was also not associated with any of the variables of interest ($rs < -.141$, $ps > .095$). As expected, internalizing behaviors were correlated across all four time points ($rs > .519$, $ps < .001$), as were externalizing behaviors $rs > .575$, $ps < .001$.

Trajectory of internalizing and externalizing behaviors

Internalizing behaviors.—A linear growth model showed excellent fit for internalizing behaviors, $\chi^2(5) = 4.839$, $p = .436$, CFI = 1.000, TLI = 1.000, RMSEA = .000, SRMR = .031 and was an improvement over a random intercept only model ($\chi^2(8) = 19.171$, $p = .014$, CFI = .958, TLI = .969, RMSEA = .096, SRMR = .070). Fit indices and parameter estimates can be found in Table 2. The mean for the intercept (corresponding to internalizing behaviors at Time 1) was significant, indicating that initial levels of internalizing behaviors were significantly different from zero. The linear slope was also significant and negative, indicating that, on average, children showed a linear decrease in internalizing behaviors over the four time points. The variances for both the intercept ($p < .001$) and slope ($p = .005$) were significant, indicating significant variability across children in their initial levels and rate of change in internalizing behaviors, supporting the addition of predictors to the model.

Externalizing behaviors.—A linear growth model showed adequate fit for externalizing behaviors, $\chi^2(5) = 16.723$, $p = .005$, CFI = 0.967, TLI = 0.961, RMSEA = .125, SRMR = .059 and was an improvement over a random intercept only model ($\chi^2(8) = 47.255$, $p < .001$, CFI = .890, TLI = .918, RMSEA = .180, SRMR = .115). Fit indices and parameter estimates can be found in Table 3. The mean for the intercept (corresponding to externalizing behaviors at Time 1) was significant, indicating that initial levels of externalizing behaviors were significantly different from zero. The linear slope was also significant and negative, indicating that, on average, children showed a linear decrease in externalizing behaviors over the four time points. The variances for the intercept was significant ($p < .001$), while the variance for the slope was marginal ($p = .066$), indicating

some variability across children in their initial levels and rate of change in externalizing behaviors.

Neural Synchrony as a predictor of internalizing and externalizing trajectories

Internalizing Behaviors.—Two models were fitted by adding each neural synchrony measure (i.e., Frustration and Play) to the linear growth model. The model for neural synchrony during 'Frustration' resulted in a similar fit as the model with no predictors, $\chi^2(7)$ = 2.327, p = .940, CFI = 1.00, TLI = 1.00, RMSEA = .000, SRMR = .016. However, neural synchrony during this context did not predict initial levels or rate of change in internalizing behaviors. The second model with 'Play' as the predictor (Figure 2) also showed a similar fit as the model with no predictors, $\chi^2(7)$ = 2.115, p = .910, CFI = 1.00, TLI = 1.00, RMSEA = .000, SRMR = .019. Neural synchrony during 'Play' was a predictor of the rate of change only (b = .317, p = .027) suggesting that stronger parent-child neural synchrony during a period of play was associated with a greater rate of change in internalizing behaviors, in this case, a more marked decrease over the four time points.

Externalizing behaviors.—Two additional models were fitted by adding each neural synchrony measure (i.e., Frustration and Play) to the linear growth model. The model for neural synchrony during 'Frustration' resulted in a better fit than the model with no predictors, $\chi^2(7)$ = 12.802, p = .077, CFI = 0.980, TLI = 0.971, RMSEA = .085, SRMR = .056. However, neural synchrony during this context did not predict initial levels or rate of change in externalizing behaviors. The second model with 'Play' as the predictor also showed an improved fit from the model with no predictors, $\chi^2(7)$ = 12.303, p = .091, CFI = 0.982, TLI = 0.974, RMSEA = .081, SRMR = .056. Neural synchrony during 'Play' also did not predict rate of change.

Follow-up analyses with behavioral synchrony

Internalizing behaviors.—To assess whether the associations observed between internalizing behaviors and parent-child neural synchrony could be explained by behavioral synchrony, an additional set of models with behavioral synchrony added as a second predictor were fitted. A model including behavioral and neural synchrony for the period of Play was fitted for internalizing behaviors first, this model showed a good fit $\chi^2(9)$ = 9.059, p = .432, CFI = 1.000, TLI = 1.000, RMSEA = .008, SRMR = .032. Behavioral synchrony marginally predicted initial level (b = −.008, p = .071) and neural synchrony still significantly predicted rate of change (b = .364, p = .014). A similar model was then fitted for internalizing behaviors with behavioral and neural synchrony during the Frustration context as predictors. This model showed good fit $\chi^2(9)$ = 6.489, p = .690, CFI = 1.000, TLI = 1.000, RMSEA = .000, SRMR = .031. However, neither synchrony measure predicted initial levels or rate of change (p's > .350).

Externalizing behaviors.—Two final models were then fitted for externalizing behaviors. The model including behavioral and neural synchrony for the 'Play' context showed adequate fit $\chi^2(9)$ = 16.589, p = .056, CFI = 0.972, TLI = 0.956, RMSEA = .093, SRMR = .066. Behavioral synchrony predicted initial levels of externalizing behaviors (b = −.020, p = .002), neural synchrony did not predict initial levels of rate of change (p's > .319).

The last model using behavioral and neural synchrony during the 'Frustration' condition as predictors of externalizing behaviors also showed adequate fit $\chi^2(9) = 19.018$, $p = .107$, CFI = 0.963, TLI = 0.943, RMSEA = .107, SRMR = .069. As with the previous model, behavioral synchrony during the 'Frustration' condition predicted initial levels of externalizing behaviors ($b = -.018$, $p < .001$), but neural synchrony did not predict initial levels of rate of change ($p's > .265$).[1]

Discussion

The current study examined behavioral and neural forms of parent-child synchrony as predictors of trajectories of internalizing and externalizing behaviors across the preschool period. As expected, and consistent with previous studies (e.g., Bub et al., 2007; Fanti & Henrich, 2010; Schappin et al., 2018), both internalizing and externalizing behaviors decreased over the one- and-a-half-year period, with more sharp decreases in externalizing behaviors, indicative of a developmentally normative transition towards greater self-regulation (Thompson & Meyer, 2007). As we hypothesized, parent-child synchrony during the Play context predicted modest decreases in internalizing behaviors, but this was only true for our measure of neural synchrony. For externalizing behaviors, we failed to find associations with neural synchrony. However, behavioral synchrony emerged as a predictor of initial levels of externalizing symptoms even when considering a neural measure of parent-child synchrony for the same context. Our findings serve as evidence of the role of parent-child neural synchrony, in particular neural synchrony of the PFC, as an important buffer against internalizing psychopathology. The fact that these findings were specific to a period of play following a frustration induction that served as a period of recovery from the frustrating context (rather than during the frustration-induction per se) supports previous work on the importance of recovery periods for understanding the regulation of distress in children (Kahle et al., 2016; Quiñones-Camacho et al., 2019b; Santucci et al., 2008). This is particularly important as both internalizing and externalizing disorders are linked with emotion dysregulation. Our findings offer some neural support for the role that parent-child co-regulation following a period of distress plays in the emergence and maintenance of internalizing behaviors in early childhood. This is particularly meaningful given that we focused on parent-child neural synchrony of the PFC, and area not only associated with emotion regulation (e.g., Levesque et al., 2004; Ochsner et al., 2002), but a region that undergoes substantial development during the preschool years (Diamond, 2002), and a region that has been found in previous studies on parent-child neural synchrony to partially mediate links between parent and child emotion regulation (Reindl et al., 2018).

Our finding that neural synchrony during a period of play *following* a frustration induction was related to internalizing behaviors, but not neural synchrony *during* the frustration period, is not completely surprising, as previous work has shown that positive interactions

---

[1]Because the two CBCL versions differed in the number of items that went into the internalizing and externalizing subscales, we re-ran the models with the mean rather than the sum of the items. The results remained largely the same. Neural synchrony during the play context remained a significant predictor of the internalizing slope only (B = .009, β = .260, p = .034). For a more direct comparison, the standardized coefficients of the effects of neural synchrony on the slope of internalizing were: β = .244 for the sum model and β = .260 for the mean model. Behavioral but not neural synchrony still predicted the externalizing intercept (B = −.001, β = −.343, p = .001) but not the slope. The standardized coefficients of the effects of behavioral synchrony on the intercept of externalizing were: β = −.328 for the sum model and β = −.343 for the mean model."

following challenging dyadic interactions are particularly critical predictors of positive child outcomes (Ham & Tronick, 2009; Quiñones-Camacho et al., 2019b; Tronick, 2007). Previous work has found that returning to synchrony after a distressing event is a particularly important indicator of adaptive parent-child interactions (Ham & Tronick, 2009; Scholtes et al., 2020). It is possible that this is the case because a period of play immediately following a challenging and mildly frustrating task allowed the dyad to return to a more positive interactive state, thus, serving as a period of repair from the dyadic stress generated during the frustration context. This is likely to be driven by the fact that the play context offered the opportunity for parents to socialize emotion regulation and for children to practice regulating their negative affect in the absence of a distressing stimulus or event. For children with higher internalizing behaviors, periods of distress might be particularly hard to navigate, and the negative emotions elicited by distressing events might be sustained for longer periods of time in comparison to peers with lower levels of internalizing behaviors. As such, positive interactions following distressing events may be particularly critical opportunities for parent-child co-regulation. Higher synchrony in this context would then allow children to stay attuned to the parent, facilitating parent-child co-regulation, allowing the child to navigate the sustained negative emotions more easily and thus increasing the child's confidence in their ability to cope with negative emotions. Over time, this could support an increase in self-regulatory competence, decreasing the risk for later psychopathology. There is behavioral (e.g., Feldman, Greenbaum, & Yirmiya, 1999; Kochanska et al., 2008), physiological (e.g., Feldman, 2007b), and, to a lesser extent, neural (Reindl et al., 2018) work demonstrating that parent-child synchrony supports the development of adaptive self-regulation. Our work extends those findings by demonstrating the role parent-child neural synchrony plays in internalizing trajectories.

While neural synchrony of the PFC emerged as an important predictor of internalizing trajectories, the same was not true for externalizing trajectories. However, given that the growth models showed better fit for internalizing behaviors and that the variance in the slope of externalizing was marginal, this result should be interpreted with caution. It is possible that we did not find a link between neural synchrony and externalizing behaviors because there was not sufficient variability in the trajectories of externalizing behaviors to find this effect. It is also possible that higher parent-child neural synchrony during a period of play following a frustrating episode is particularly important in the context of internalizing behaviors, as it might be indicative of factors, such as a lack of behavioral withdrawal on the part of the child, that might be protective. Given that a tendency to withdraw is a common characteristic of children high in internalizing behaviors (Achenbach, 1991), being able to continue to interact with their parent after a frustrating episode might allow children to continue to engage in co-regulation with their parents, reducing their distress, and serving as a protective factor against increasing internalizing trajectories. However, given that withdrawing from interactions is not common in externalizing disorders, children showing high levels of externalizing behaviors might not benefit to the same extent from higher parent-child neural synchrony during periods of repair. Lastly, given that neural synchrony facilitates social interactions by helping reduce the cognitive burden necessary for enhanced memory and attention to an interacting partner (Macrae et al., 2008), it is possible that increased parent-child neural synchrony might be particularly useful in the context of

internalizing behaviors as it might facilitate the modification of children's internal models of difficult social interactions, resulting in a decreased need to withdraw during distressing events, and allowing for greater parent-child co-regulation. This, in turn, would result in greater changes in self-regulation and increased sense of control over time, potentially explaining why neural synchrony was associated with the slope (i.e., change) and not the initial levels of internalizing behaviors.

It is noteworthy that the patterns for behavioral and neural synchrony were different, with behavioral synchrony associated with the intercept of internalizing (marginally) and externalizing behaviors, whereas neural synchrony was associated with the slope for internalizing only. There are various reasons for why this might be the case. For example, while our measure of neural synchrony represents the level of coordination within the DLPFC of parent and child neural activity throughout the task, our behavioral measure considers parent and child behaviors as part of a single measure, and thus, represents a dyadic level global approach to parent-child behaviors. While we cannot conclude from the results of this study exactly why behavioral and neural synchrony showed different patterns of associations with internalizing and externalizing behaviors, our finding that behavioral and neural synchrony were differentially linked with child outcomes is not entirely unexpected as other studies have found similar differences between biological and behavioral measures of synchrony (Suveg, Shaffer, & Davis, 2016; Woltering et al., 2015). It is possible that our measure of behavioral synchrony was not able to fully capture the type of parent-child dynamics that were driving neural synchrony during the play context. More research is needed, however, to fully understand how behavioral and neural synchrony relate to each other across interactional contexts. This is particularly important as the associations between behavioral and neural synchrony with problem behaviors differed between internalizing and externalizing disorders. A more systematic approach to assessing behavioral and neural forms of parent-child synchrony is needed to clarify whether neural synchrony is only linked to internalizing behaviors, or if the associations between neural synchrony and externalizing and behaviors are context dependent. Nonetheless, our study demonstrates the utility of using measures of neural synchrony as a viable approach to assessing biological underpinnings of the parent-child interaction, offering information beyond what can be captured from behavioral synchrony measures alone. It is possible that parent-child neural synchrony is, in fact, only associated with internalizing behaviors, even when other interactional contexts are considered. If this is the case, this provides further evidence of the ways in which internal models in internalizing disorders are developed and modified early in life though parent-child interactions. This has substantial implications for our understanding of early psychopathology and the parent-child relationship more broadly.

While our study has several notable strengths, such as the multi-wave assessment of internalizing and externalizing behaviors during the preschool years and our use of both behavioral and neural measures of parent-child synchrony, some limitations should be noted. First, while it is important to take dimensional approaches to early psychopathology, as this allows for a greater understanding of trajectories towards clinical disorders, internalizing behaviors in our sample were generally at or below the subclinical threshold. The CBCL also has floor effects at the lower end of the normal: abnormal dimensional spectrum (Kaat et al., 2018). However, there was still significant variability in internalizing behaviors,

as suggested by the significant variances for both intercept and slope. Relatedly, we use two different versions of the CBCL throughout the study, this means that some of the questions included in the internalizing and externalizing subscales changed from the initial visit to later visits. However, due to the heterotypic continuity of psychopathology across childhood, it is critical to use measures appropriate to capture the different developmental manifestations of psychopathology. Changing versions of the CBCL as children became older ensured we were able to capture these changing developmental manifestations. Additionally, while our use of a neural measure of parent-child synchrony is a notable strength and provides important information about the neural underpinnings of co-regulation, we do not have detailed coding of the specific co-regulatory behaviors used during the task or detailed coding of the dyads affective responding. There is evidence that adaptive parent-child interactions are not monotonously synchronous (Tronick, 2007), and that periods of asynchrony during negative interactions and periods of repair are adaptive (Scholtes, Lyon, & Skowron, 2020). Thus, exploration of parent-child behavioral synchrony using a more thorough coding approach is necessary to fully disentangle the patterns of behavioral and affective synchrony that are most adaptive. Given that the current manuscript only included a neural synchrony measure at time 1, we were unable to test whether trajectories of internalizing (and externalizing) behaviors influence parent-child neural synchrony at later timepoints, it is likely that the association between parent-child neural synchrony and child psychopathology is bidirectional and that higher child symptoms influence patterns of dyadic synchrony at later timepoints. Lastly, fNIRS is a neuroimaging technique that is restricted to the measurement of cortical regions, and our analyses focused on neural synchrony within the PFC. Thus, we were limited in the emotion-related regions and networks that we could probe, and in our ability to address whether neural synchrony across regions or within regions of the PFC would show similar associations. Moreover, because we did not have anatomical MR data for each child at T1 and did not have 3-D digitizer data available to confirm optode placement, it is possible that the peak channel measured activity of slightly different regions within the larger right PFC area. However, given the limitations of other neuroimaging techniques for measuring neural activity during in-vivo social interactions, our study still represents a notable advancement over previous neuroimaging studies on parent-child interactions and child psychopathology. Moreover, our analyses showing that neural synchrony within the PFC was linked with trajectories of preschool psychopathology extends work on the role of the PFC for social processes and its implications for child outcomes.

While research on parent-child neural synchrony is still emerging, findings from this line of work seem promising for identifying biological mechanisms for the parent-child co-regulation of distress, which in turn may help clarify dyadic-level biological correlates of risk for psychopathology. However, before truly clarifying the utility of these measures, a more careful exploration of interactional context will be necessary. This, with a more careful exploration of the role of age (using both concurrent and longitudinal approaches), an explicit consideration of parental psychopathology, and direct comparison of neuroimaging modalities (e.g., EEG vs. fNIRS), as well as comparisons of neural and physiological measures of synchrony will be needed. Additionally, while work on parent-child neural synchrony and early psychopathology in community samples is necessary for understanding

transitions from normative to clinical behaviors, the results might change for children in the more clinical/severe range, in fact, there is reason to believe that these associations would look different in samples of children and/or parents with high symptomatology. For example, in a study by Suveg and colleagues (2019), preadolescents high in internalizing symptoms showed negative RSA synchrony with their mothers while preadolescents with low internalizing symptoms showed positive RSA synchrony. Although we are not aware of any data, to date, that has focused on parent-child neural synchrony in high-risk groups, we have reason to believe based on work on physiological measures that the most adaptive patterns of parent-child neural synchrony might be different in dyads with higher levels of symptoms.

Though substantial work is still needed, the findings from this study advance our understanding of the role of the neurobiological underpinnings of parent-child co-regulation of emotion on trajectories of internalizing and externalizing behaviors during the preschool years. As such, our study offers initial evidence of the longitudinal implications of the parent-child synchronization of neural responses on psychopathology trajectories, and underscore the importance of considering the biological mechanisms for parent-child co-regulation as a potential target for the mitigation of risk for internalizing disorders in early childhood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements.

References

Achenbach TM (1966). The classification of children's psychiatric symptoms: A factor-analytic study. Psychological Monographs: General and Applied, 80(7), 1–37. 10.1037/h0093906.

Achenbach TM (1991). Manual for the Child Behavior Checklist/4–18 and 1991 Profile. Burlington, VT: Department of Psychiatry, University of Vermont.

Achenbach TM, Dumenci L, & Rescorla LA (2001). Ratings of relations between DSM-IV diagnostic categories and items of the CBCL/6-18, TRF, and YSR. Burlington, VT: University of Vermont, 1–9.

Achenbach TM, & Edelbrock CS (1978). The classification of child psychopathology: A review and analysis of empirical efforts. Psychological Bulletin, 85(6), 1275–1301. 10.1037/0033-2909.85.6.1275. [PubMed: 366649]

Azhari A, Leck WQ, Gabrieli G, Bizzego A, Rigo P, Setoh P, … & Esposito G (2019). Parenting stress undermines mother-child brain-to-brain synchrony: A hyperscanning study. Scientific Reports, 9(1), 1–9. 10.1038/s41598-019-47810-4. [PubMed: 30626917]

Beesdo K, Bittner A, Pine DS, Stein MB, Höfler M, Lieb R, et al. (2007). Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. Archives of General Psychiatry, 64(8), 903–912. https://psycnet.apa.org/doi/10.1001/archpsyc.64.8.903. [PubMed: 17679635]

Benjamini Y, & Hochberg Y (1995). Controlling the False Discovery Rate: A Practical and Powerful Approach to Multiple Testing. Journal of the Royal Statistical Society. Series B (Methodological), 57(1), 289–300.

Bub KL, McCartney K, & Willett JB (2007). Behavior problem trajectories and first-grade cognitive ability and achievement skills: A latent growth curve analysis. Journal of Educational Psychology, 99(3), 653–670. 10.1037/0022-0663.99.3.653

Campbell SB (1995). Behavior problems in preschool children: A review of recent research. Journal of Child Psychology and Allied Disciplines, 36, 113–149. 10.1111/j.1469-7610.1995.tb01657.x.

Carter AS, Wagmiller RJ, Gray SA, McCarthy KJ, Horwitz SM, & Briggs-Gowan MJ (2010). Prevalence of DSM-IV disorder in a representative, healthy birth cohort at school entry: Sociodemographic risks and social adaptation. Journal of the American Academy of Child and Adolescent Psychiatry, 49(7), 686–698. 10.1016/j.jaac.2010.03.018.. [PubMed: 20610138]

Coie JD, & Dodge KA (1998). Aggression and antisocial behavior. In Damon W & Eisenberg N (Eds.), Handbook of child psychology: Vol.3. Social, emotional, and personality development (pp. 779–862). New York, NY: Wiley.

Condon WS, & Sander LW (1974). Synchrony demonstrated between movements of the neonate and adult speech. Child Development, 45(2), 456–462. 10.2307/1127968. [PubMed: 4837718]

Copeland WE, Shanahan L, Costello EJ, & Angold A (2009). Childhood and adolescent psychiatric disorders as predictors of young adult disorders. Archives of General Psychiatry, 66(7), 764–772. https://psycnet.apa.org/doi/10.1001/archgenpsychiatry.2009.85. [PubMed: 19581568]

Denham SA, Bassett HH, & Wyatt T (2007). The socialization of emotional competence. Handbook of socialization: Theory and research, 614–637.

Diamond A, 2002. Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy, and biochemistry. In: Stuss D, Knight R (Eds.), Principles of Frontal Lobe Function. Oxford University Press, New York, NY, pp. 466–503.

Dougherty LR, Leppert KA, Merwin SM, Smith VC, Bufferd SJ, & Kushner MR (2015). Advances and directions in preschool mental health research. Child Development Perspectives, 9(1), 14–19. 10.1111/cdep.12099.

Fanti KA, & Henrich CC (2010). Trajectories of pure and co-occurring internalizing and externalizing problems from age 2 to age 12: Findings from the National Institute of Child Health and Human Development study of early child care. Developmental Psychology, 46, 1159–1175. https://psycnet.apa.org/doi/10.1037/a0020659. [PubMed: 20822230]

Feldman R (2007a). Mother-infant synchrony and the development of moral orientation in childhood and adolescence: Direct and indirect mechanisms of developmental continuity. American Journal of Orthopsychiatry, 77(4), 582–597. 10.1037/0002-9432.77.4.582. [PubMed: 18194038]

Feldman R (2007b). Parent–infant synchrony and the construction of shared timing; physiological precursors, developmental outcomes, and risk conditions. Journal of Child Psychology and Psychiatry, 48(3/4), 329–354. 10.1111/j.1469-7610.2006.01701.x.. [PubMed: 17355401]

Feldman R (2007c). Parent–infant synchrony: Biological foundations and developmental outcomes. Current Directions in Psychological Science, 16(6), 340–345. 10.1111/j.1467-8721.2007.00532.x..

Feldman R (2012). Parent-infant synchrony: a biobehavioral model of mutual influences in the formation of affiliative bonds. Monographs of the Society for Research in Child Development, 77, 42–51. 10.1111/j.1540-5834.2011.00660.x

Feldman R, Greenbaum CW, & Yirmiya N (1999). Mother–infant affect synchrony as an antecedent of the emergence of self-control. Developmental Psychology, 35(1), 223–231. https://psycnet.apa.org/doi/10.1037/0012-1649.35.1.223. [PubMed: 9923477]

Fishburn FA, Ludlum RS, Vaidya CJ, & Medvedev AV (2019). Temporal Derivative Distribution Repair (TDDR): A motion correction method for fNIRS. Neuroimage, 184, 171–179. 10.1016/j.neuroimage.2018.09.025. [PubMed: 30217544]

Fleming AS, O'Day DH, & Kraemer GW (1999). Neurobiology of mother–infant interactions: experience and central nervous system plasticity across development and generations. In Neuroscience & Biobehavioral Reviews (Vol. 23, Issue 5, pp. 673–685). 10.1016/s0149-7634(99)00011-1. [PubMed: 10392659]

Goodwin RD, Fergusson DM, & Horwood LJ (2004). Early anxious/withdrawn behaviours predict later internalising disorders. Journal of Child Psychology and Psychiatry, 45(4), 874–883. 10.1111/j.1469-7610.2004.00279.x.. [PubMed: 15056317]

Granic I (2000). The self-organization of parent–child relations: Beyond bidirectional models. In Lewis MD & Granic I (Eds.), Emotion, development, and self-organization: Dynamic systems approaches to emotional development (pp. 267–297). New York, NY: Cambridge University Press.

Ham J, & Tronick E (2009). Relational psychophysiology: Lessons from mother–infant physiology research on dyadically expanded states of consciousness. Psychotherapy Research, 19(6), 619–632. 10.1080/10503300802609672. [PubMed: 19235090]

Harrist AW, & Waugh RM (2002). Dyadic synchrony: Its structure and function in children's development. Developmental Review, 22(4), 555–592. 10.1016/S0273-2297(02)00500-2.

Helm JL, Sbarra DA, & Ferrer E (2014). Coregulation of respiratory sinus arrhythmia in adult romantic partners. Emotion, 14(3), 522–531. https://psycnet.apa.org/doi/10.1037/a0035960. [PubMed: 24708502]

Holmes CJ, Hoge R, Collins L, Woods R, Toga AW, & Evans AC (1998): Enhancement of MR images using registration for signal averaging. Journal of Computer Assisted Tomography, 22(2), 324–333. 10.1097/00004728-199803000-00032. [PubMed: 9530404]

Hollenstein T, Granic I, Stoolmiller M, & Snyder J (2004). Rigidity in parent—child interactions and the development of externalizing and internalizing behavior in early childhood. Journal of Abnormal Child Psychology, 32(6), 595–607. 10.1023/b:jacp.0000047209.37650.41. [PubMed: 15648527]

Hoyniak CP, Quiñones-Camacho LE, Camacho MC, Chin JH, Williams EM, Wakschlag LS, & Perlman SB (2021). Adversity is Linked with Decreased Parent-Child Behavioral and Neural Synchrony. Developmental Cognitive Neuroscience, 48, 100937. 10.1016/j.dcn.2021.100937. [PubMed: 33639519]

Hu LT, & Bentler PM (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. Psychological Methods, 3(4), 424–453. 10.1037/1082-989X.3.4.424.

Im-Bolter N, Anam M, & Cohen NJ (2015). Mother–child synchrony and child problem behavior. Journal of Child and Family Studies, 24(7), 1876–1885. 10.1007/s10826-014-9989-1.

Jaffe J, Beebe B, Feldstein S, Crown CL, Jasnow MD, Rochat P, & Stern DN (2001). Rhythms of dialogue in infancy: Coordinated timing in development. Monographs of the Society for Research in Child Development, 66(2), vi–131.

Kaat AJ, Blackwell CK, Estabrook R, Burns JL, Petitclerc A, Briggs-Gowan MJ, … Wakschlag LS (2018). Linking the Child Behavior Checklist (CBCL) with the Multidimensional Assessment Profile of Disruptive Behavior (MAP-DB): Advancing a Dimensional Spectrum Approach to Disruptive Behavior. Journal of Child and Family Studies, 1–11. 10.1007/s10826-018-1272-4. [PubMed: 33311964]

Kahle S, Miller JG, Lopez M, & Hastings PD (2016). Sympathetic recovery from anger is associated with emotion regulation. Journal of Experimental Child Psychology, 142, 359–371. 10.1016/j.jecp.2015.10.004. [PubMed: 26597139]

Kalomiris AE, & Kiel EJ (2018). Mother–toddler cortisol synchrony moderates risk of early internalizing symptoms. Infancy, 23(2), 232–251. 10.1111/infa.12216. [PubMed: 29576751]

Keiley MK, Lofthouse N, Bates JE, Dodge KA, & Pettit GS (2003). Differential risks of covarying and pure components in mother and teacher reports of externalizing and internalizing behavior across ages 5 to 14. Journal of Abnormal Child Psychology, 31, 267–283. 10.1023/a:1023277413027. [PubMed: 12774860]

Kessler RC, Chiu WT, Demler O, & Walters EE (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Archives

of General Psychiatry, 62(6), 617–627. https://psycnet.apa.org/doi/10.1001/archpsyc.62.6.617. [PubMed: 15939839]

Kirschbaum C, Pirke K-M, & Hellhammer DH (1993). The 'trier social stress test'—A tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology, 28, 76–81. 10.1159/000119004. [PubMed: 8255414]

Kochanska G, Aksan N, Prisco TR, & Adams EE (2008). Mother-child and father-child mutually responsive orientation in the first 2 years and children's outcomes at preschool age: Mechanisms of influence. Child Development, 79, 30–44. doi: 10.1111/j.1467-8624.2007.01109.x.. [PubMed: 18269507]

Kovacs M, & Devlin B (1998). Internalizing disorders in childhood. The Journal of Child Psychology and Psychiatry and Allied Disciplines, 39(1), 47–63. 10.1017/S0021963097001765. [PubMed: 9534086]

Leclere C, Viaux S, Avril M, Achard C, Chetouani M, Missonnier S, & Cohen D (2014). Why synchrony matters during mother-child interactions: A systematic review. PloS one, 9(12). 10.1371/journal.pone.0113571.

Leve LD, Kim HK, & Pears KC (2005). Childhood temperament and family environment as predictors of internalizing and externalizing trajectories from ages 5 to 17. Journal of Abnormal Child Psychology, 33(5), 505–520. 10.1007/sl0802-005-6734-7. [PubMed: 16195947]

Levesque J, Joanette Y, Mensour B, Beaudoin G, Leroux JM, Bourgouin P, & Beauregard M (2004). Neural basis of emotional self-regulation in childhood. Neuroscience, 129(2), 361–369. 10.1016/j.neuroscience.2004.07.032 [PubMed: 15501593]

Lindsey EW, Cremeens PR, Colwell MJ, & Caldera YM (2009). The structure of parent–child dyadic synchrony in toddlerhood and children's communication competence and self-control. Social Development, 18(2), 375–396. 10.1111/j.1467-9507.2008.00489.x.

Luby JL, Gaffrey MS, Tillman R, April LM, & Belden AC (2014). Trajectories of preschool disorders to full DSM depression at school age and early adolescence: Continuity of preschool depression. The American Journal of Psychiatry, 171(7), 768. 10.1176/appi.ajp.2014.13091198.. [PubMed: 24700355]

Luby J, Heffelfinger A, Mrakotsky C, Brown K, Hessler M, Wallis J, & Spitznagel EL (2003). The clinical picture of depression in preschool children. Journal of American Academy of Child Adolescent Psychiatry, 42(3), 340–348. 10.1097/00004583-200303000-00015.

Lunkenheimer E, Hamby CM, Lobo FM, Cole PM, & Olson SL (2020). The role of dynamic, dyadic parent–child processes in parental socialization of emotion. Developmental Psychology, 56(3), 566–577. https://psycnet.apa.org/doi/10.1037/dev0000808. [PubMed: 32077725]

Lunkenheimer ES, Olson SL, Hollenstein T, Sameroff AJ, & Winter C (2011). Dyadic flexibility and positive affect in parent-child coregulation and the development of child behavior problems. Development and Psychopathology, 23, 577–591. 10.1017/S095457941100006X. [PubMed: 23786697]

Lunkenheimer E, Tiberio SS, Skoranski AM, Buss KA, & Cole PM (2018). Parent-child coregulation of parasympathetic processes varies by social context and risk for psychopathology. Psychophysiology, 55(2), e12985. 10.1111/psyp.12985.

Macrae CN, Duffy OK, Miles LK, & Lawrence J (2008). A case of hand waving: Action synchrony and person perception. Cognition, 109(1), 152–156. 10.1016/j.cognition.2008.07.007. [PubMed: 18755450]

McArdle JJ, & Epstein D (1987). Latent growth curves within developmental structural equation models. Child Development, 58(1), 110–133. 10.2307/1130295. [PubMed: 3816341]

McLeod BD, Wood JJ, & Weisz JR (2007). Examining the association between parenting and childhood anxiety: A meta-analysis. Clinical Psychology Review, 27(2), 155–172. 10.1016/j.cpr.2006.09.002. [PubMed: 17112647]

Merikangas KR, He JP, Burstein M, Swanson SA, Avenevoli S, Cui L, … & Swendsen J (2010). Lifetime prevalence of mental disorders in US adolescents: Results from the National Comorbidity Survey Replication–Adolescent Supplement (NCS-A). Journal of the American Academy of Child & Adolescent Psychiatry, 49(10), 980–989. 10.1016/j.jaac.2010.05.017. [PubMed: 20855043]

Miles LK, Griffiths JL, Richardson MJ, & Macrae CN (2010). Too late to coordinate: Contextual influences on behavioral synchrony. European Journal of Social Psychology, 40(1), 52–60. 10.1002/ejsp.721.

Miles LK, Nind LK, & Macrae CN (2009). The rhythm of rapport: Interpersonal synchrony and social perception. Journal of Experimental Social Psychology, 45(3), 585–589. 10.1016/j.jesp.2009.02.002

Miller JG, Vrti ka P, Cui X, Shrestha S, Hosseini SH, Baker JM, & Reiss AL (2019). Interbrain synchrony in mother-child dyads during cooperation: An fNIRS hyperscanning study. Neuropsychologia, 124, 117–124. 10.1016/j.neuropsychologia.2018.12.021. [PubMed: 30594570]

Montague PR, Berns GS, Cohen JD, McClure SM, Pagnoni G, Dhamala M, Wiest MC, Karpov I, King RD, Apple N, et al. , 2002. Hyperscanning: simultaneous fMRI during linked social interactions. Neuroimage 16, 1159–1164. 10.1006/nimg.2002.1150. [PubMed: 12202103]

Moore GA, Powers CJ, Bass AJ, Cohn JF, Propper CB, Allen NB, & Lewinsohn PM (2013). Dyadic interaction: Greater than the sum of its parts?. Infancy, 75(4), 490–515. 10.1111/j.1532-7078.2012.00136.x.

Murray L, De Pascalis L, Bozicevic L, Hawkins L, Sclafani V, & Ferrari F (2016). The functional architecture of mother-infant communication, and the development of infant social expressiveness in the first two months. Scientific Reports, 6, 39019. 10.1038/srep39019. [PubMed: 27966659]

Muthén LK and Muthén BO (1998–2017). Mplus User's Guide. Eighth Edition. Los Angeles, CA: Muthén & Muthén

Nguyen T, Schleihauf H, Kayhan E, Matthes D, Vrti ka P, & Hoehl S (2020). The effects of interaction quality on neural synchrony during mother-child problem solving. Cortex, 124, 235–249. 10.1016/j.cortex.2019.11.020. [PubMed: 31927470]

Nguyen T, Schleihauf H, Kayhan E, Matthes D, Vrti ka P, & Hoehl S (2020). Neural synchrony in mother-child conversation: Exploring the role of conversation patterns. Social Cognitive and Affective Neuroscience, nsaa079. 10.1093/scan/nsaa079.

Ochsner KN, Bunge SA, Gross JJ, & Gabrieli JD (2002). Rethinking feelings: an FMRI study of the cognitive regulation of emotion. Journal of Cognitive Neuroscience, 14(8), 1215–1229. 10.1162/089892902760807212. [PubMed: 12495527]

Quiñones-Camacho LE, Fishburn FA, Camacho MC, Wakschlag LS, & Perlman SB (2019a). Cognitive Flexibility-Related Prefrontal Activation in Preschoolers: A Biological Approach to Temperamental Effortful Control. Developmental Cognitive Neuroscience, 100651. 10.1016/j.dcn.2019.100651.. [PubMed: 31154272]

Quiñones-Camacho LE, Fishburn FA, Camacho MC, Hlutkowsky CO, Huppert TJ, Wakschlag LS, & Perlman SB (2019b). Parent-child neural synchrony: A novel approach to elucidating dyadic correlates of preschool irritability. Journal of Child Psychology and Psychiatry. 10.1111/jcpp.13165.

Quiñones-Camacho LE, Fishburn FA, Belardi K, Williams DL, Huppert TJ, & Perlman SB (2021). Dysfunction in interpersonal neural synchronization as a mechanism for social impairment in autism spectrum disorder. Autism Research 1–12. 10.1002/aur.2513.

Rapee RM (2012). Family factors in the development and management of anxiety disorders. Clinical Child and Family Psychology Review, 15(1), 69–80. https://psycnet.apa.org/doi/10.1007/s10567-011-0106-3. [PubMed: 22116624]

Redcay E, & Schilbach L (2019). Using second-person neuroscience to elucidate the mechanisms of social interaction. Nature Reviews Neuroscience, 20, 495–505. 10.1038/s41583-019-0179-4. [PubMed: 31138910]

Reindl V, Gerloff C, Scharke W, Konrad K (2018). Brain-to-brain synchrony in parent-child dyads and the relationship with emotion regulation revealed by fNIRS-based hyperscanning. Neuroimage, 178, 493–502. 10.1016/j.neuroimage.2018.05.060. [PubMed: 29807152]

Santosa H, Aarabi A, Perlman SB, & Huppert T (2017). Characterization and correction of the false-discovery rates in resting state connectivity using functional near-infrared spectroscopy. Journal of Biomedical Optics, 22(5), 055002. 10.1117/1.JBO.22.5.055002. [PubMed: 28492852]

Santosa H, Zhai X, Fishburn F, & Huppert T (2018). The NIRS Brain AnalyzIR Toolbox. Algorithms, 11(5), 73. 10.3390/a11050073. [PubMed: 38957522]

Santucci AK, Silk JS, Shaw DS, Gentzler A, Fox NA, & Kovacs M (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. Developmental Psychobiology, 50, 205–216. https://psycnet.apa.org/doi/10.1002/dev.20283. [PubMed: 18335488]

Schappin R, Wijnroks L, Venema MU, & Jongmans M (2018). Exploring predictors of change in behavioral problems over a 1-year period in preterm born preschoolers. Infant Behavior and Development, 50, 98–106. 10.1016/j.infbeh.2017.12.003 [PubMed: 29257995]

Scholtes CM, Lyons ER, & Skowron EA (2020). Dyadic synchrony and repair processes are related to preschool children's risk exposure and self-control. Development and Psychopathology, 1–13. 10.1017/s0954579420000358

Shevlyakov G, & Smirnov P (2011). Robust estimation of the correlation coefficient: An attempt of survey. Austrian Journal of Statistics, 40, 147–156. 10.17713/ajs.v40i1&2.206

Smith CG et al. (2019) Anxious parents show higher physiological synchrony with their infants. PsyArXiv. Published online 20 June 2019. 10.31234/osf.io/m2rsg

Suveg C, Braunstein West K, Davis M, Caughy M, Smith EP, & Oshri A (2019). Symptoms and synchrony: Mother and child internalizing problems moderate respiratory sinus arrhythmia concordance in mother–preadolescent dyads. Developmental Psychology, 55(2), 366–376. 10.1037/dev0000648. [PubMed: 30507218]

Suveg C, Shaffer A, & Davis M (2016). Family stress moderates relations between physiological and behavioral synchrony and child self-regulation in mother–preschooler dyads. Developmental Psychobiology, 58(1), 83–97. 10.1002/dev.21358. [PubMed: 26376933]

Thompson RA, & Meyer S (2007). The socialization of emotion regulation in the family. In Gross J (Ed.), Handbook of emotion regulation (pp. 249–268). New York, NY: The Guilford Press.

Tronick EZ (2007). The neurobehavioral and social-emotional development of infants and children. New York: Norton.

Wakschlag L, Hill C, Carter AS, Danis B, Egger HL, Keenan K, … & Briggs-Gowan MJ (2008). Observational Assessment of Preschool Disruptive Behavior, Part I: Reliability of the Disruptive Behavior Diagnostic Observation Schedule (DB-DOS). Journal of the American Academy of Child & Adolescent Psychiatry 47(6), 622–631. [PubMed: 18434926]

Wakschlag LS, Roberts MY, Flynn RM, Smith JD, Krogh-Jespersen S, Kaat AJ, … & Davis MM (2019). Future directions for early childhood prevention of mental disorders: A road map to mental health, earlier. Journal of Clinical Child & Adolescent Psychology, 48(3), 539–554. 10.1080/15374416.2018.1561296. [PubMed: 30916591]

Wheatley T, Kang O, Parkinson C, & Looser CE (2012). From mind perception to mental connection: Synchrony as a mechanism for social understanding. Social and Personality Psychology Compass, 6(8), 589–606. 10.1111/j.1751-9004.2012.00450.x.

Willett JB, & Bub KL (2005). Latent growth curve analysis. In Rindskopf D (Ed.), Encyclopedia of statistics in behavioral science (2nd ed.). Oxford, England: Wiley.

Woltering S, Lishak V, Elliott B, Ferraro L, & Granic I (2015). Dyadic attunement and physiological synchrony during mother-child interactions: An exploratory study in children with and without externalizing behavior problems. Journal of Psychopathology and Behavioral Assessment, 37, 624–633. 10.1007/s10862-015-9480-3.

Yaniv AU, Salomon R, Waidergoren S, Shimon-Raz O, Djalovski A, & Feldman R (2020). Synchronous Caregiving from Birth to Adulthood Tunes Humans' Social Brain. bioRxiv. 10.1101/2020.03.09.974659..

Wu CY, & Lee TSH (2020). Impact of parent–child relationship and sex on trajectories of children internalizing symptoms. Journal of Affective Disorders, 260, 167–173. 10.1016/j.jad.2019.09.016 [PubMed: 31494367]

Figure 1.
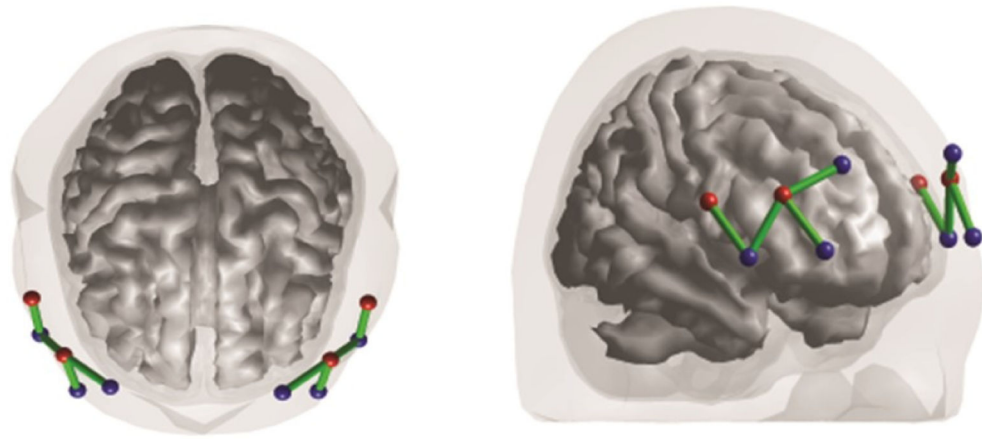Probe configuration visualized on the surface of the scalp after registration to the Colin27 atlas. Green lines represent measurement channels.
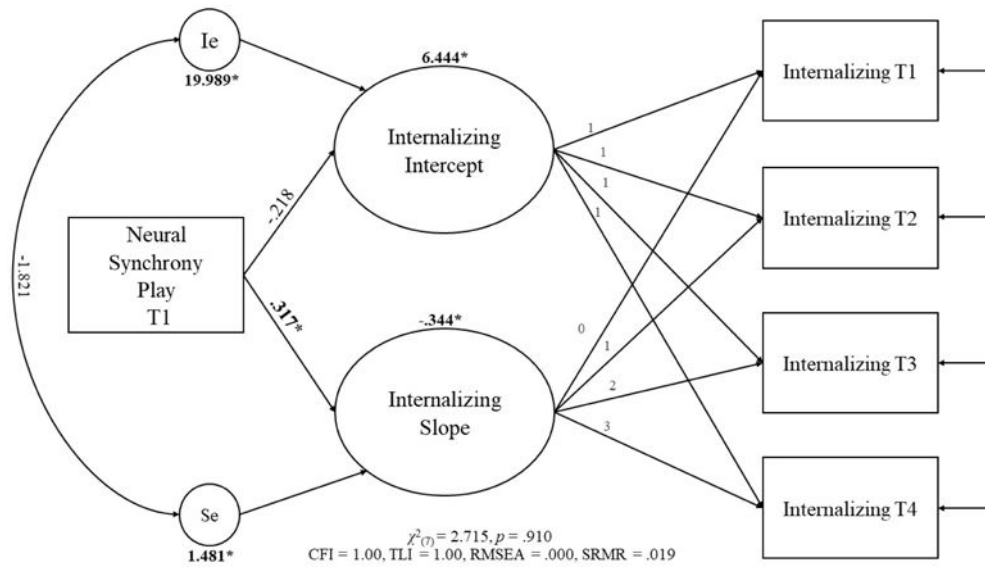
Figure 2.
Latent Growth Curve Model with parent-child neural synchrony during play predicting children's internalizing trajectories. Bolded values with an asterisk represent significant unstandardized parameters. Ie = Error of the intercept; Se = Error of the slope.

Table 1.

Descriptive statistics and correlations among variables of interest

	Mean	SD	Range	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Internalizing T1	6.43	5.26	0-29	-												
2. Internalizing T2	6.28	5.86	0-29	**.647**	-											
3. Internalizing T3	5.99	5.39	0-22	**.663**	**.654**	-										
4. Internalizing T4	5.49	4.96	0-26	**.519**	**.615**	**.678**	-									
5. Externalizing T1	9.76	7.46	0-35	**.592**	**.506**	**.409**	**.362**	-								
6. Externalizing T2	9.44	7.77	0-42	**.450**	**.676**	**.388**	**.434**	**.748**	-							
7. Externalizing T3	8.76	7.35	0-40	**.466**	**.520**	**.579**	**.433**	**.697**	**.752**	-						
8. Externalizing T4	7.09	6.49	0-35	**.324**	**.493**	**.307**	**.553**	**.575**	**.742**	**.676**	-					
9. Neural Synchrony Frustration T1	.504	1.10	−2.40-3.02	.079	.050	.052	−.030	.046	−.034	−.043	−.125	-				
10. Neural Synchrony Play T1	.490	1.04	−1.85-3.67	−.035	.016	.033	**.207**	.042	.033	.019	.093	.053	-			
11. Behavioral Synchrony Frustration T1	292.22	128.39	0-488	−.040	−.188	−.083	−.038	**−.347**	**−.288**	**−.279**	−.161	−.010	.070	-		
12. Behavioral Synchrony Play T1	305.77	106.25	18-517	−.149	**−.265**	**−.209**	−.141	**−.290**	**−.238**	**−.207**	−.134	−.117	−.036	**.476**	-	
13. Age T1 *	57.83	7.12	48-71	.055	−.090	−.050	−.026	−.078	−.141	−.121	−.136	−.047	−.005	.162	.111	-

Note. Bold = $p < .05$.

*
Age in months

Table 2.

Parameter estimates, standard errors, and fit indices for Latent Growth Curve (LGC) Models for children's internalizing behaviors with neural synchrony during Frustration and Play as predictors.

	LGC with no predictors			LGC with Neural Synchrony during Frustration as predictor			LGC with Neural Synchrony during Play as predictor		
	B	*SE*	*p*	*B*	*SE*	*p*	*B*	*SE*	*p*
Means									
Intercept	**6.487**	**.424**	**<.001**	**6.137**	**.510**	**<.001**	**6.444**	**.514**	**<.001**
Slope	**−.280**	**.135**	**.038**	−.123	.164	.455	**−.344**	**.163**	**.035**
Variances									
Intercept	**22.432**	**3.408**	**<.001**	**19.797**	**3.590**	**<.001**	**19.989**	**3.607**	**<.001**
Slope	**1.428**	**.508**	**.005**	**1.543**	**.528**	**.003**	**1.481**	**.517**	**.004**
Covariance	**−2.582**	**1.045**	**.013**	−1.775	1.035	.086	−1.821	1.025	.076
Predictor on									
Intercept				.394	.425	.354	−.218	.449	.628
Slope				−.126	.137	.357	**.317**	**.143**	**.027**
Fit Indices									
χ^2	4.839			2.327			2.715		
df	5			7			7		
CFI	1.00			1.00			1.00		
TLI	1.00			1.00			1.00		
RMSEA	.000			.000			.000		
SRMR	.031			.016			.019		

Note. B = unstandardized coefficient; SE = standard error; **Bold = *p* <.05.**

Table 3.

Parameter estimates, standard errors, and fit indices for Latent Growth Curve (LGC) Models for children's externalizing behaviors with neural synchrony during Frustration and Play as predictors.

	LGC with no predictors			LGC with Neural Synchrony during Frustration as predictor			LGC with Neural Synchrony during Play as predictor		
	B	SE	p	B	SE	p	B	SE	p
Means									
Intercept	**10.074**	**.613**	**<.001**	**9.968**	**.751**	**<.001**	**9.901**	**.749**	**<.001**
Slope	**−.797**	**.168**	**<.001**	**−.766**	.188	**<.001**	**−.849**	**.191**	**<.001**
Variances									
Intercept	**47.181**	**6.616**	**<.001**	**43.614**	**7.152**	**<.001**	**43.427**	**7.143**	**<.001**
Slope	1.508	.822	.066	1.591	.815	.051	1.604	.821	.051
Covariance	**−3.830**	**1.684**	**.023**	−2.278	1.608	.156	−2.177	1.613	.177
Predictor on									
Intercept				.174	.621	.779	.324	.652	.620
Slope				−.204	.154	.186	−.037	.165	.824
Fit Indices									
χ^2	16.723			12.802			12.303		
df	5			7			7		
CFI	.967			.980			.982		
TLI	.961			.971			.974		
RMSEA	.125			.085			.081		
SRMR	.059			.056			.056		

Note. B = unstandardized coefficient; SE = standard error; **Bold** = *p* < **.05.**