Clinical Child and Family Psychology Review (2022) 25:110–129
https://doi.org/10.1007/s10567-022-00391-7

Parent-to-Child Anxiety Transmission Through Dyadic Social Dynamics: A Dynamic Developmental Model

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Accepted: 14 February 2022 / Published online: 23 February 2022

Abstract

The intergenerational transmission of psychopathology is one of the strongest known risk factors for childhood disorder and may be a malleable target for prevention and intervention. Anxious parents have distinct parenting profiles that impact socioemotional development, and these parenting effects may result in broad alterations to the biological and cognitive functioning of their children. Better understanding the *functional mechanisms* by which parental risk is passed on to children can provide (1) novel markers of risk for socioemotional difficulties, (2) specific targets for intervention, and (3) behavioral and biological indices of treatment response. We propose a developmental model in which *dyadic social dynamics* serve as a key conduit in parent-to-child transmission of anxiety. Dyadic social dynamics capture the moment-to-moment interactions between parent and child that occur on a daily basis. In shaping the developmental trajectory from familial risk to actual symptoms, dyadic processes act on mechanisms of risk that are evident prior to, and in the absence of, any eventual disorder onset. First, we discuss *dyadic synchrony* or the moment-to-moment coordination between parent and child within different levels of analysis, including neural, autonomic, behavioral, and emotional processes. Second, we discuss how overt *emotion modeling* of distress is observed and internalized by children and later reflected in their own behavior. Thus, unlike synchrony, this is a more sequential process that cuts across levels of analysis. We also discuss maladaptive cognitive and affective processing that is often evident with increases in child anxiety symptoms. Finally, we discuss additional moderators (e.g., parent sex, child fearful temperament) that may impact dyadic processes. Our model is proposed as a conceptual framework for testing hypotheses regarding dynamic processes that may ultimately guide novel treatment approaches aimed at intervening on dyadically linked biobehavioral mechanisms *before* symptom onset.

Keywords Anxiety · Development · Synchrony · Emotion modeling · Dyadic social dynamics · Temperament

Introduction

Intergenerational transmission is one of the strongest known risk factors for childhood psychopathology. There are three main pathways for intergenerational transmission: (1) genetic inheritance (Eley et al., 2015; Gregory & Eley, 2007); (2) fetal programming through maternal experiences during pregnancy (Lin et al., 2019; Ostlund et al., 2019); and (3) the active and passive socialization of emotion and stress response through parent–child interactions (Askew & Field, 2008; Dunne & Askew, 2013; Hane et al., 2008; Hastings, Rubin, Smith, & Wagner, 2019). Here, we focus on intergenerational pathways in the emergence of anxiety, one of the most common disorders in children affecting 5–10% of children by age 5 (Essau et al., 1999; Fergusson et al., 1993; McGee et al., 1990), with a lifetime prevalence of 32% by age 18 (Beesdo-Baum & Knappe, 2012). Observational studies have reinforced the link between parental anxiety and the emergence of child anxiety, noting high levels of comorbidity (Biederman et al., 2001; Rosenbaum et al., 1988, 1992). The shared presence of anxiety in the parent–child dyad suggests that we can find unique processes in the emergence of psychopathology that reside within the dyad, rather than the individual, and potentially identify tractable dyadic-level interventions.

In this article, we propose a developmental model in which *dyadic social dynamics* serve as a key conduit in parent-to-child transmission of anxiety based on prior work

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illustrating that dyadic parent–child processes contribute something unique to the transmission of risk from parent to child. That is, these dyadic interactions provide explanatory and predictive power even when taking into account individual parent- and child-level variables (Lunkenheimer, Brown, & Fuchs, 2021; Lunkenheimer et al., 2017, 2020; Moore et al., 2013).

The role of biological intermediaries in parent-to-child anxiety transmission is evident in non-human primate (macaque) models of fearful temperament, indicating high heritability (Williamson et al., 2003), which can then be traced to parallel parent–offspring brain function, particularly in the amygdala (Fox et al., 2018; Fox et al., 2015; Fox et al., 2015). Importantly, genetically informed studies in humans note that while genetic factors account for approximately 30–40% of variance in anxiety symptoms (Eley et al., 2015; Hettema et al., 2001), there is a clear additional environmental pathway that helps explain the association between anxiety in the child and parent (Eley et al., 2015). While studies often cannot disentangle genetic and environmental mechanisms of risk, there is clear evidence that targeting parent–child relations can nonetheless decrease anxiety risk in children (Chronis-Tuscano et al., 2018; Danko et al., 2018; Rapee et al., 2010).

Indeed, both clinical and developmental models of psychopathology have long-centered bidirectional or transactional processes that toggle back and forth from parents to children (Bell, 1968; Hudson & Rapee, 2001; Johnco et al., 2021; Sameroff, 1975). Even though most studies focus on parent-to-child relations, child psychopathology can prospectively predict parent psychopathology. For example, child depression in middle childhood predicts parent depression two years later (Wesseldijk et al., 2018). Silverman and colleagues (Silverman et al., 2021) found that cognitive behavioral therapy (CBT) targeted at child anxiety was associated with post-treatment parental anxiety even though parental anxiety was not the target of any of the study conditions. These bidirectional relations are evident even when one can account for shared risk via genetically informed models (Cioffi et al., 2021). Most often, associations between parent and child are most robust when assessed as a chronic exposure (Borelli et al., 2015; Grabow et al., 2017; Pemberton et al., 2010) versus focusing on a single exposure (Hails et al., 2018). The importance of chronic exposure suggests that smaller time intervals within daily interactions build on each other to generate larger-scale impacts on development and psychopathology risk.

Although our review is not focused on global constructs of parenting as traditionally defined by the developmental psychology literature [e.g., parenting styles, attachment (Baumrind, 1971; Belsky, 1984)], previous research has found associations between parent-linked behaviors and child well-being. Indeed, anxious parents have distinct parenting profiles that impact socioemotional development (Kiel & Buss, 2011, 2014; Kiel & Hummel, 2017; Kiel et al., 2016). For example, anxious parents can be over-protective (Degnan et al., 2008; Hastings et al., 2019; Rubin et al., 1999) or over-solicitous (Buss & Kiel, 2011; Kiel & Buss, 2012; Kiel et al., 2016) in the face of actual or potential child distress.

While these behaviors are motivated by a desire to minimize negative experiences for their child, they can inadvertently prevent children from working to overcome initial anxious tendencies. Indeed, the everyday activities of school and play are often sufficient to ameliorate anxious tendencies in young children (Almas et al., 2011; Phillips et al., 2011; Rudasill & Rimm-Kaufman, 2009). Over-protective parenting behaviors may result in broad alterations to the biological and cognitive functioning of their children (Brooker et al., 2011, 2015; Torpey et al., 2013). Recent work suggests that children show preferential fear-learning when observing a parent versus an unfamiliar adult and this effect is potentiated if the parent is anxious (Silvers et al., 2020). Thus, children at familial risk for anxiety may be exposed to more anxiety-linked behaviors that they, in turn, are more likely to internalize then express.

Extant studies largely examine intergenerational transmission by capturing aggregate measures of parent and child traits or behaviors averaged over set time windows (Anaya & Pérez-Edgar, 2019; Belsky et al., 2012; Brooker & Buss, 2014; Guyer et al., 2015; Hastings et al., 2019; Kiel & Buss, 2011, 2014; Kiff et al., 2011). Although this approach has created a robust knowledge base, parent-to-child transmission is not fully captured by examining child or parental traits and behaviors in isolation. Rather than focus on broader parenting practices or behaviors, many of which have been found to only loosely correlate with child anxiety (McLeod et al., 2007), our model is intended to probe the underlying dynamic, granular, moment-to-moment interactions between parent and child. Repeated daily interactions with caregivers attune the child to parental expressions and regulation of fear and distress, which influence the child's own responses to events (Leerkes et al., 2020). We have a relatively limited understanding of the relations between global risk and moment-to-moment interactions between parent and child.

Thus, the current review argues that we should focus not only on the bidirectional and dyadic relation between parent and child but also explicitly consider the time window of observation. That is, by examining moment-to-moment interactions, occurring within minutes or even seconds or milliseconds, we may be able to generate new insights into long-term developmental and clinical patterns. An emphasis on dynamic relations also reflects larger calls in the field to focus on active mechanisms that influence the full range of psychological and biobehavioral profiles. For example, the

National Institute of Mental Health has called on researchers to examine development and the environment as "bidirectional influences" on the transdiagnostic processes of psychopathology through its Research Domain Criteria (RDoC) (Casey et al., 2014; Sanislow et al., 2010).

Better understanding the *functional mechanisms* by which parental traits are passed on to children can provide (1) novel markers of risk for socioemotional difficulties; (2) specific targets for intervention; and (3) both behavioral and biological indices of treatment response. Specifically, by functional mechanism, we mean one of the critical mechanistic pathways by which parent psychopathology can lead to child psychopathology. Ideally, this mechanism would be capable of modification through targeted intervention or prevention. The current review highlights the importance of incorporating novel methods and analytics for capturing dynamic relations. Here, we present a conceptual developmental model (Fig. 1) focusing on dyadic social dynamics as a key conduit in parent-to-child transmission of anxiety. Dyadic social dynamics capture the moment-to-moment interactions between parent and child that occur on a daily basis, as well as the parental behavioral and socioemotional patterns that children observe.

We note that, although dyadic social dynamics are discussed here in the context of the parent–child relationship, they are present between any two interacting individuals, which distinguishes these fine-grained, dyadic behaviors from general parenting practices. The impact of these dynamics may of course be potentiated in the parent–child relationship in that it uniquely combines long term and repeated exposure and the pivotal role parents play in shaping a child's environment and daily activities. In shaping the developmental trajectory from familial risk to actual symptoms, dyadic processes act on markers of risk that are evident prior to, and in the absence of, any eventual disorder onset. We will delineate two cases of dyadic social dynamics.

First, we will review *dyadic synchrony* or the moment-to-moment coordination of behavioral, neural, and physiological processes between parent and child. We will discuss how novel dynamic measures can be leveraged to capture patterns of concordance. Second, we will discuss how overt *emotion modeling* of distress is observed and internalized by children and later reflected in their own behavior. In doing so we discuss the task and analytic considerations that should be addressed in order to apply the model to research. In conclusion, we highlight future directions, which include a focus on contextual or individual factors that can impact dyadic processes as well as the promise of leveraging dyadic processes for treatment and prevention.

Dyadic Social Dynamics as a Mechanism for Anxiety Transmission

Parental anxiety markedly increases anxiety risk in children. Indeed, up to 33% of children with anxious parents will present with a corresponding psychiatric diagnosis (OR = 1.92) (Johnson et al., 2001). Our ability to target underlying biological transmission mechanisms, such as genetic predisposition or fetal programming of the stress response, is limited and generally untested. Consequently, there exists a robust literature examining how parental behavior can exacerbate or ameliorate genetic and prenatal risk. In particular, over-protective behavior and psychological control tend to restrict children's behavior and may actively encourage dependency (Hastings et al., 2019), which has been associated with social withdrawal and anxiety both concurrently (Chronis-Tuscano et al., 2018; Rubin et al., 2018) and prospectively (Coplan et al., 2008; Degnan et al., 2008) from toddlerhood through early elementary school. In addition, children at temperamental risk for psychopathology appear particularly sensitive to parenting behaviors, as noted below (Hastings et al., 2019).

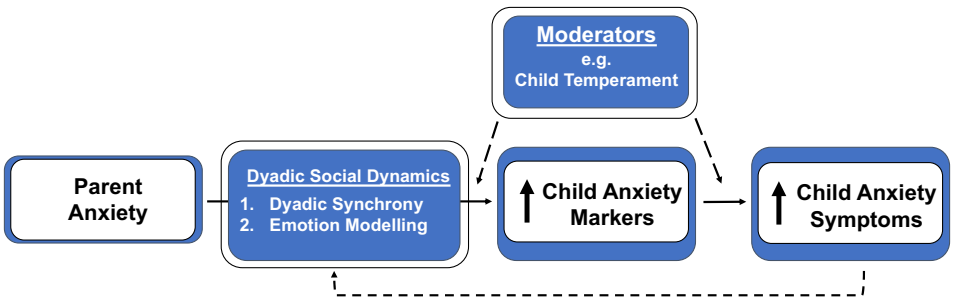

Fig. 1 Conceptual model of trajectory of parent-to-child anxiety transmission. Across development parent anxiety influences the development of child anxiety symptoms through dyadic social dynamics. Over time, emerging child anxiety symptoms can also influence these dyadic interactions. This process can be moderated by a number of individual or contextual factors, illustrated here with child fearful temperament

Parental behavior, in turn, is a core component of adaptive calibration models (ACM) which examine how the environment "gets under the skin" to shape psychobiological development (Del Giudice et al., 2011; Ellis & Boyce, 2008; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). Prior work has focused on broad measures (e.g., over-protection) averaged over set time windows. These measures can provide a stable and robust metric for analysis, which can then be supplemented by measures that capture dynamic interactions in the moment (Shih, Quiñones-Camacho, Karan, & Davis, 2019). It is in these fine-grained interactions that parents may model distinct emotional and stress responses linked to anxiety. Focusing on the temporal context of parenting (Cole, Lougheed, & Ram, 2018; Davis, Brooker, & Kahle, 2020) provides novel ways of assessing moment-to-moment synchrony during interactions, the spill-over effects of observed emotionally salient events, and the long-term arc of change in anxiety-relevant mechanisms and outcomes. Thus, by moving across different time windows researchers can ask interconnected questions, such as (1) Concurrently, how do patterns of dyadic social dynamics vary across parent–child pairs? (2) Across contexts, to what extent does variation in dyadic patterns help predict or explain anxiety risk? (3) Over time, can we predict socioemotional profiles and anxiety risk from earlier patterns captured during dynamic dyadic interactions?

Dyadic Synchrony

Definition of Dyadic Synchrony

The link between partners engaged in a dyadic interaction reflects a contingent relation between the two members of the dyad (Davis et al., 2020; Davis et al., 2018). Synchrony, operationalized behaviorally as the 'temporal co-ordination of discrete micro-level signals between dyadic partners' (Azhari et al., 2019; Bornstein, 2013), is the most basic unit of analysis via which partners are linked. The specific time scale of the interaction of interest often varies with the measure of interest. For example, studies of cardiac synchrony can have thousands of data points embedded within an interaction only a few minutes long. Studies coding for facial expressions, in contrast, will often have fewer discrete data points as they oscillate in slower frequencies (e.g., Fig. 2). However, synchrony measures are computationally intense, so most studies rely on relatively short epochs on the order of 5 to 10 s embedded within interactions that last minutes. One counter example can be found in studies of diurnal cortisol synchrony (e.g., Laurent, Sbrilli, Dawson, Finnegan, & Ramdas-Neal, 2021) or parent–child autonomic synchrony over the course of a day (Smith et al., 2019).

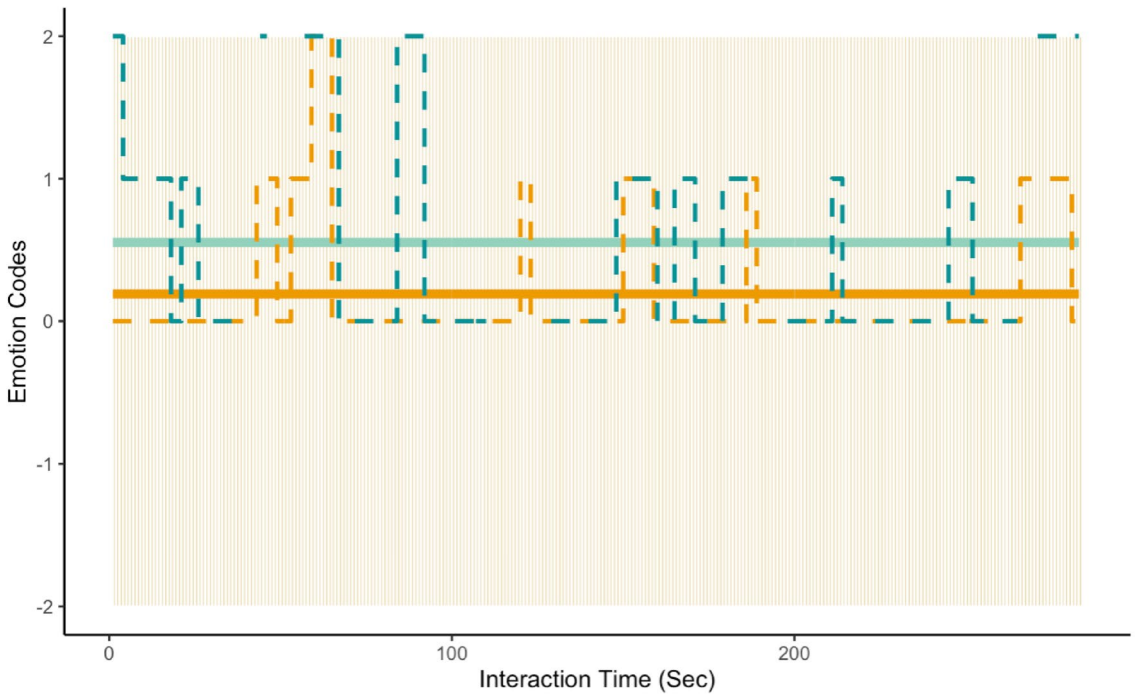

Fig. 2 Example of emotion synchrony in a mother–infant dyad engaged in free play. The solid lines reflect the average emotion level for the mother (green) and infant (orange). The dashed lines capture fluctuations in emotion for each dyadic partner. The temporal linkages between fluctuations are captured in analytic synchrony scores (Color figure online)

Within these epochs, synchronized dyads match, reciprocate, and jointly expand upon one another's actions (e.g., vocalizations, motor activity, and emotions). Synchronized dyads may also exhibit parallel and coordinated autonomic activity, likely reflecting co-created arousal states (Davis et al., 2017; Davis et al., 2018). Synchrony is an emergent property of the dyad's shared experience, not simply reflecting shared space, proximity, or exposure to an event or stimulus (Fishburn et al., 2018). Higher-order social processes, such as engagement and mutual attunement, further potentiate this synchronization (Wass et al., 2020). This basic synchrony, in turn, can serve as the foundation for other attachment-related dyadic constructs, such as co-regulation (Cole & Hollenstein, 2018; Hollenstein, 2013; Lunkenheimer et al., 2011). In following, synchronized signals may provide the latent interpersonal scaffolding onto which more deliberate socialization and regulatory processes are built.

Thus, fine-level measures (e.g., heart beats) are embedded within epochs that can then be used to understand developmental change over longer-time windows, including on the order of years. Dyadic synchrony may also enable the transmission of adaptive or maladaptive, behavioral strategies for addressing uncertainty in the environment by attuning the child to the caregiving context. Thus, we argue that high and low synchrony are not inherently positive or negative indicators of adaptive development, as specific patterns of covariation in biobehavioral systems between parents and children may differentially associate with familial risk for specific disorders (Lunkenheimer, Tiberio, Skoranski, Buss, & Cole, 2018; Smith et al., 2019; Wass, Clackson, & Leong, 2018).

Measurement of Dyadic Synchrony

Traditionally, dyadic synchrony, as defined in the developmental psychology literature, is measured *behaviorally* using second-by-second or frame-by-frame video coding schemes (Feldman, 2007; Feldman & Greenbaum, 1997; Feldman et al., 1996; Harrist & Waugh, 2002; Kellerman et al., 2020; Tronick, 1989; Tronick & Cohn, 1989). These involve careful hand coding by trained research assistants, often noting the vocalizations, eye contact, actions, or emotional expression of each member of the dyad independently and then examining timepoints in which these behaviors coordinate (Beebe et al., 2011; Granat et al., 2017; Moore & Calkins, 2004). This can include lagged time courses in which one member of the dyad might lead and another might follow.

Additionally, recent research has drawn attention to underlying biological components of dyadic synchrony. Dyadic synchrony of the autonomic nervous system has been measured via *heart rate* (Creaven et al., 2014; Suveg et al., 2016), *respiratory sinus arrhythmia* (Lunkenheimer et al., 2015; Ostlund et al., 2017), *pre-ejection period* (Helm et al., 2018), and *salivary alpha amylase* (Laurent et al., 2012), thought to reflect stress, emotional arousal, and engagement in interpersonal interactions. This method has made advances in the mother–child attachment literature from infancy through childhood (Field et al., 1989; Giuliano et al., 2015; Lunkenheimer et al., 2021), but also in intra-fetal mother–offspring dyads (Van Leeuwen et al., 2009). Dyadic synchrony in the *hypothalamic–pituitary–adrenal (HPA) axis* has been assessed via attunement in both cortisol reactivity to a stressor and diurnal cortisol rhythms (Laurent et al., 2021; Pratt et al., 2017), also reflecting dyadic indices of stress regulation.

Neural methods for the measurement of dyadic synchrony include both *electroencephalography (EEG)* and *functional near-infrared spectroscopy (fNIRS)*. Using these methods, the time course of the neural signal is measured in each individual and then compared for moments in which the rise or fall of the neural signal of one individual predicts that of the other (Cui et al., 2012; Fishburn et al., 2018; Kinreich et al., 2017; Liu, Duan, Dai, Pelowski, & Zhu, 2021). EEG methods for measuring neural synchrony capitalize on the high temporal resolution of the method and the mobility of multiple rather than single devices between subjects. The latter has been particularly useful in ecologically valid contexts, such as schools (Bevilacqua et al., 2019; Dikker et al., 2017), musical collaboration (Lindenberger et al., 2009; Müller, Sänger, & Lindenberger, 2013) (i.e., a jazz band), or museums (Dikker et al., 2021). fNIRS, a method for measuring the hemodynamic response of blood oxygen in the brain (Ferrari & Quaresima, 2012) with higher spatial resolution relative to EEG, has been particularly useful for identifying specific neural regions that synchronize between parents and children in such cases as stressful interaction or competition (Quiñones-Camacho et al., 2019; Reindl et al., 2018), language acquisition (Piazza, Cohen, Trach, & Lew-Williams, 2021), or developmental disorders (Kruppa et al., 2020; Quiñones-Camacho et al., 2021; Quiñones-Camacho et al., 2021; Su et al., 2020).

It is important to note the unique time scale of each biobehavioral system and consider theory regarding whether synchrony of that system is meaningful on the specified time scale. For example, neural activation may manifest on the order of milliseconds, whereas behavioral responses may play out across seconds or minutes. Therefore, methodological designs for the study of synchrony may differ depending on the biobehavioral system in question. More consideration of methodological differences and similarities across biobehavioral systems is needed to refine our understanding of the function of dyadic synchrony between parent and child (DePasquale, 2020; Helm et al., 2018).

Dyadic Synchrony in the Parent–Child Psychopathology Context

Parent–child dyadic synchrony is arguably most meaningful in the context of challenging or goal-oriented interactions and thus is typically studied in tasks involving challenging prompts, stimuli, or changing conditions designed to trigger behavioral changes of interest in the dyad. For example, in early parent–child interaction tasks, stimuli may involve experimental alterations to expected parent behavior (e.g., the Still Face Paradigm) (Moore & Calkins, 2004) or the goal of completing a difficult puzzle that requires parental scaffolding of children's efforts (Hoyniak et al., 2021; Lunkenheimer et al., 2018; MacNeill, Fu, Buss, & Pérez-Edgar, in press). Using these paradigms, there is an emerging literature examining how a parent and child synchronize both behavioral and neural and physiological activities (Hasson et al., 2012). While research on parent–child neural synchrony is still scarce, the few published studies suggest that increased parent–child neural synchrony is linked with better emotion regulation (Reindl et al., 2018), less parenting stress (Azhari et al., 2019), and greater behavioral reciprocity (Nguyen et al., 2020), lending additional support for biological synchrony as a mechanism facilitating the transmission of behavioral strategies.

Dyadic synchrony is also modulated based on characteristics of the parent and child, such as parental harshness and psychopathology (Giuliano et al., 2015; Ham & Tronick, 2009; Laurent et al., 2011; Moore et al., 2009; Woody et al., 2016). For example, more maternal psychological aggression (e.g., intent to impose fear) (Lunkenheimer et al., 2018) and greater severity of child maltreatment (Lunkenheimer et al., 2018; Lunkenheimer et al., 2018) have been related to lower physiological synchrony. Synchrony has also been related to temperamental negativity and behavior problems in childhood (Lunkenheimer et al., 2018; Lunkenheimer et al., 2018; Lunkenheimer et al., 2018). Quiñones-Camacho and colleagues (Quiñones-Camacho, Hoyniak, Wakschlag, & Perlman, 2021) found that parent–child dyadic synchrony, measured neurally using fNIRS, positively predicted decreases in internalizing, but not externalizing, symptoms from 4 to 6 years of age. Thus, the mounting, multi-system evidence available underscores the association between synchrony and outcomes and supports future work testing the potential that synchrony acts as a key mechanism in the transmission and/or development of psychopathology in relation to underlying regulatory difficulties.

There is emerging evidence that synchrony may potentiate risk for poor outcomes, particularly in the context of anxiety (Beebe et al., 2011; Granat et al., 2017; Smith et al., 2019; Wass et al., 2020). Children in parent–child dyads that are highly synchronized behaviorally or physiologically exhibit better regulatory abilities and communication skills (Criss et al., 2003; Feldman et al., 1999; Harrist & Waugh, 2002; Im-Bolter et al., 2015; Kochanska et al., 2008; Suveg et al., 2016). However, low prefrontal synchrony is related to psychopathology risk (Quiñones-Camacho et al., 2021; Quiñones-Camacho et al., 2019). One recent study (Smith et al., 2019) captured autonomic synchrony between caregivers and one-year-old throughout a typical day within the home. Anxious parents showed greater autonomic synchrony with their infants throughout the day than their less anxious counterparts (Smith et al., 2019). In particular, non-anxious parents showed elevated attunement with their infant only at moments of high arousal or need and then down-regulated in response to infant arousal. Anxious parents, in contrast, are reactive more often and at lower thresholds of infant arousal (Wass et al., 2020). Wass and colleagues suggest that anxious parents are 'always on,' while non-anxious parents take an approach of being there 'when you need me.'

Additionally, studies with older children find greater behavioral synchrony among dyads with an anxious parent during table top play (Beebe et al., 2011; Granat et al., 2017), in contrast to lower synchrony with depressed (Granat et al., 2017) and maltreating (Lunkenheimer et al., 2018; Lunkenheimer et al., 2018) parents. Thus, while much of the literature has examined the positive effects of dyadic synchrony on child development (Feldman et al., 1999; Feldman et al., 2011; Lunkenheimer et al., 2018; Lunkenheimer et al., 2018; Lunkenheimer et al., 2011; Lunkenheimer et al., 2015; Lunkenheimer et al., 2018; Wass et al., 2020), there is growing evidence that dyadic synchrony may also act as a maladaptive influence (Beebe et al., 2011; Granat et al., 2017; Smith et al., 2019; Wass et al., 2020), particularly within the context of anxiety.

Emotion Modeling

Definition of Emotion Modeling

In the current review we are focused on two distinct forms of dyadic social dynamics: dyadic synchrony and emotion modeling. In the first, the dyad is linked by coupled moment-to-moment fluctuations that can be captured within multiple levels of analysis, including neural, physiological, and behavioral. In the second, we highlight a *cascading* dyadic relation that typically occurs *across* levels of analysis. That is, emotion modeling relies on the observation, and then internalization, of behavior, which is in turn reflected in the child's cognitions, emotions, and behaviors (Denham, Bassett, & Wyatt, 2007). We suggest that emotion modeling is one functional mechanism underlying the relation between parental behavior and child socioemotional outcomes. It is possible that individual differences in dyadic synchrony may influence the strength and impact of emotion modeling

(Feldman et al., 1999; Johnco et al., 2021; Pérez-Edgar, 2019). However, as we note below, this is one of many open questions in this literature.

The transgenerational continuity of stress reactivity (Hipwell et al., 2019) is partially driven by the inheritance of biological tendencies in reactivity profiles. The tendency to have an elevated stress response in both parent and child may be traced to an underlying shared biological predisposition (Bartels, Van den Berg, Sluyter, Boomsma, & de Geus, 2003). However, these tendencies can then be potentiated and triggered by the child directly observing the caregiver's emotional and behavioral response to stress (Laurent, 2014; Silvers et al., 2020). Most learning takes place in social settings, as the brain dynamically adapts to the behavior of others (Wass et al., 2020). Modeling is evident from the first months of life as even infants will mimic emotional displays evident around them. For example, increased autonomic reactivity in a parent during an emotion exposure task is then reflected in increased negative affect in infants (Waters et al., 2014, 2017). This process of spreading or shared responses to the environment is often labeled an 'emotional contagion' effect that is evident even in early infancy (Geangu et al., 2010). Of note, this label may overstate the connection by implying the direct transmission of a shared emotional response in the moment (Ruffman et al., 2017).

However, the clear link between emotion and behavior in one individual and the subsequent shift in the emotion and behavior of another do point to the fact that we are attuned to perceptual and behavioral signals from others even before we have the ability to reflect more complex cognitive or affective responses. Indeed, this attunement appears to be an evolutionarily conserved trait as non-human primates exhibit referential gaze to caregivers and respond differentially to emotional messages based on both gaze behavior and avoidance of the novel objects (Russell et al., 1997). These summative effects then lead to observed variation in sociobehavioral profiles. In particular, observations create 'ostensive cues' that trigger a neural response, which, with repetition, may lead to dyadic attunement (Albert, Schwade, & Goldstein, 2018; Grossmann et al., 2008; Urakawa et al., 2015; Young et al., 2017), which is evident in the child's reflection of modeled behavior. Building on associative and statistical learning, children come to create stable schematic expectations of their environment (e.g., threatening vs. safe) based on how the people around them react to daily life experiences. The child's idiosyncratic socioemotional curriculum can facilitate fear or anxiety learning (Muris et al., 1996) and model how the child should respond.

Measurement of Emotion Modeling

Children likely take in instances of modeling through a number of channels. That is, caregivers and adults can convey information through verbalization, emotion expression, or direct action. The initial steps in studying this process is to capture both the presented signal and the subsequent capture by the child. On the first point, a number of emotion modeling studies have focused on *behavioral observation* of parental emotion expression (Camras, 2019). Behavioral coding schemes have focused on micro-analytic coding of facial affect (Chaplin et al., 2005) as well as broader more gestalt affective expressions, including body language (Lunkenheimer et al., 2020). Parental expressions often emerge spontaneously and are not necessarily directly aimed at the child. As such, their value as a modeling signal comes in the child's equally spontaneous attention to the form and function of these expressions, linking the trigger, the expression, and the subsequent outcomes (Bayet & Nelson, 2019).

Often, the emotion modeling literature then looks to see if the captured parental signal of emotion is correlated with or predicts child emotional behavior (Burstein & Ginsburg, 2010; Eisenberg et al., 1998; Muris et al., 1996). This is a fundamental relation for establishing the impact of modeling. However, as in our discussion of dyadic synchrony, it skips over the functional mechanisms that could be linking parental modeling with child expression (Zhou et al., 2002). Here, we focus on two potential processes that might generate this link: selective attention to parental expression and the child's biological response.

Interestingly, there are relatively less data specifically focused on selective attention to parental modeling (Aktar, Nimphy, Van Bockstaele, & Pérez-Edgar, in press). However, it may be that parents who frequently model negative emotion expression are providing the salient stimuli that feed into, and entrench, attention biases to threat (Aktar & Bögels, 2017). Indeed, there are emerging data suggesting a link between parental anxiety and attention biases in both infancy (Morales et al., 2017) and early childhood (Aktar et al., 2013; Aktar, Van Bockstaele, Pérez-Edgar, Wiers, & Bögels, 2019).

The broader evidence base suggests the likely mechanistic pathways between parental modeling and child outcomes via attention. For example, cognitive theories of anxiety collectively suggest that anxiety is associated with attention bias toward threat (Van Bockstaele et al., 2014). Information processing perspectives (Crick & Dodge, 1994; Morales et al., 2016; Pérez-Edgar, Taber-Thomas, Auday, & Morales, 2014) further argue that attention bias to threat elicits a cascade of effects on downstream information processing and behavioral responses. These processes influence each other through a feedback loop, strengthening a biased mental database that perpetuates and reinforces anxious behaviors over time.

Involuntary orienting toward salient stimuli gradually tunes the visual system, resulting in biased attention, memory encoding, and behavioral enactment in response to previously perceived threats. Thus, attention bias to threat may be

an automatic process produced from repeated pre-tuning of top-down attention selection (Todd et al., 2012). The cyclical relation between attention and socioemotional experiences, mediated through caregiving, may shape long-term anxiety trajectories. For example, temperamentally fearful children are most likely to exhibit maladaptive social withdrawal (Cole et al., 2016; Morales, et al., 2015; Morales et al., 2015; Pérez-Edgar et al., 2011; Pérez-Edgar et al., 2010; Pérez-Edgar et al., 2010) and anxiety (White et al., 2017) if they also exhibit an attention bias to threat.

As with dyadic synchrony, new technology has allowed for more 'child-centered' observation of the instances of modeling they experience. For example, recent work has used *mobile eye-tracking* (Pérez-Edgar et al., 2020) to move beyond the use of static, non-dynamic stimuli that do not fully reflect the rich information provided by one-on-one contingent social interactions (Fu & Pérez-Edgar, 2019). Mobile eye-tracking can provide the child's point of view and supplement observed behavior captured by room-view cameras that may capture the major dynamics of an encounter but not the idiosyncratic input available to the child. One recent study used mobile eye-tracking to capture underlying tendencies in attention as adolescent girls present a speech in front of two judges, one positive and one critical (Woody et al., 2019). They found that the likelihood to visually focus on the critical judge correlated positively with depression symptoms. Patterns of visual attention were also associated with neural connectivity among regions associated with emotion regulation (Sequeira et al., 2021). Importantly, mobile eye-tracking can provide insight into the child's processing of observed behavior even in the absence of any overt behavior on the part of the child. For example, in one laboratory task, six-year-olds rarely directly interacted with an unknown adult. However, mobile eye-tracking measures found that children who visually avoided the stranger, and had visual patterns less coupled with the stranger's actions, demonstrated higher levels of internalizing problems (Gunther et al., in press).

Once attended to, the child's initial response to parental emotion expressions may be evident in *neural or physiological signals*. One classic example can be seen in the visual cliff experiment (Sorce et al., 1985). In one variation, an infant is coaxed to cross the glass cliff by the mother, who is asked to present with either neutral, fearful, or encouraging affect. The infant is less likely to do so if the parent presents with a hesitant or fearful expression, and this hesitation is accompanied by changes in the infant's cardiac response to the cliff (Campos et al., 1992; Klinnert, Campos, Sorce, Emde, & Svejda, 1983). It is important to point out that this initial physiological response need not mean that the infant is also experiencing an emotional response. That is, the infant can attend to the parent's signal, internalize its meaning (e.g., the cliff may be dangerous), and act accordingly (LoBue & Adolph, 2019) without experiencing an emotional response. From this perspective, the physiological or behavioral response is not in and of itself evidence for emotion modeling. Rather, we would need downstream evidence of a fear response, potentially mediated by the cardiac response, to reflect our current conceptualization of emotion modeling.

Observational learning and modeling may also be reflected in a neural response. At a basic level, the motor cortex responds to an action performed by another person (Kerr et al., 2019). This response is part of a larger network, often incorporating the anterior cingulate cortex (ACC), anterior insula, and amygdala (Tramacere & Ferrari, 2016). Of relevance here, higher levels of maternal negative emotion have been linked to greater neural activation in brain regions implicated in detecting (e.g., amygdala, anterior insula) and regulating (e.g., ACC, vlPFC) emotions (Chaplin et al., 2019; Turpyn et al., 2018).

One drawback to imaging studies is that the emotion modeling stimuli are often static pictures or pre-recorded videos in order to account for testing requirements. However, recent work with fNIRS technology suggests that we can couple neural responses in active interactions, much as we have traditionally done with cardiac measures. Future work can use these new technologies to capture the three-needed components of our emotion modeling chain: (1) parental emotional expression, (2) a shift in the child's attention and/or biological response, and (3) a subsequent shift in the child's own emotional expression or behavior. This chain allows researchers to extend the general argument that parental expressions of emotion have a direct impact on a child's emotional arousal and learning about emotions (Eisenberg et al., 1998) by examining parents' active emotional signaling and the dynamic cascade of responses that may follow (Hajal & Paley, 2020).

Emotion Modeling in the Parent–Child Psychopathology Context

Within the emotion development literature, emotion modeling is often approached as a core component of the parental socialization of emotion, along with overt reactions to the child's expressive behavior, explicit discussions of emotion, and managing children's exposure to emotion-inducing situations (Camras, 2019; Camras, Shuster, & Fraumeni, 2014). Parental socialization encompasses a mix of behaviors that are at times overt or covert and can be deliberate or unconscious. Over time, they help shape individual patterns of expression, familial patterns of expressiveness, and contribute to social or cultural profiles of emotional experience and expression (Camras et al., 2014; Leerkes et al., 2020). For example, Holodynski and Friedlmeier (2006) suggest that caregivers shape an infants' undifferentiated negative expressions (e.g., crying) to reflect the response deemed

appropriate to the specific cue or trigger. That is, parents themselves will display the more specific discrete expression (e.g., anger or sadness) to model the expected familial or cultural response. One meta-analytic study (Halberstadt & Eaton, 2002) found that expressivity by family members was significantly related to children's own emotional expressiveness.

Emotion modeling by parents spans a wide range of emotions, including anger (Dollar & Calkins, 2019), sadness (Denham & Kochanoff, 2002), fear (LoBue, Kim, & Delgado, 2019), disgust (Gerull & Rapee, 2002; Shutts et al., 2013), and happiness (Messinger et al., 2019). Through these behaviors, parents can communicate common behavioral tendencies associated with daily events, as well as the personal significance of an eliciting event (Denham, 2019). Importantly, this process influences and shapes the regulation of emotion and socioemotional competence (Eisenberg et al., 2001; Valiente et al., 2004). Intense and frequent expression of anger within parent–child interactions is associated with lowered abilities to appropriately regulate anger and aggressive behaviors (Rubin et al., 2003; Smeekens et al., 2007). However, exposure to well-regulated negative emotion is positively related to components of emotional competence (Garner et al., 1994).

In the specific case of anxiety, anxious parents tend to express higher levels of behavioral and linguistic stress markers in social situations or unfamiliar contexts (Aktar et al., 2013; Murray et al., 2008). In addition, anxious parents are more likely to exhibit interpretation biases that lead them to negatively frame daily events (Creswell, Cooper, & Murray, 2010; Creswell et al., 2011). Parental-negative interpretations are often not confined to their own environment, but also extended to their child's environment and daily activities (Lester et al., 2009). As a result, they are also more likely to spontaneously communicate threat information to their children regarding potentially stressful situations (Murray et al., 2014; Percy et al., 2016). Children receiving threat information are, in turn, more likely to endorse that the targeted person or context is threatening when directly asked (Aktar et al., in press). This process has been observed naturalistically, but also through laboratory induction. One study (De Rosnay et al., 2006) trained mothers to interact with a stranger in either a normative or socially anxious manner. Infants displayed more hesitancy to interact with the stranger after observing the socially anxious interaction of their mother. The potency of this information can increase when combined with individual difference factors, like fearful temperament (LoBue et al., 2019).

Child observation of anxiety-tinged parenting behavior can lead to the subsequent experience of anxiety on the part of the child (Reynolds, Askew, & Field, 2018). For example, anxious child behavior in a modified speech task has been associated with the later emergence of social withdrawal in middle childhood and adolescence (Degnan et al., 2011). It may be that this anxious behavior reflects not only the child's individual traits (e.g., temperament) but also their internalization of observed distress. Indeed, children who observed their caregiver display anxiety during an oral spelling test endorsed higher anxiety levels, anxious cognitions, and desired avoidance of the spelling test versus children who observed low levels of anxious behavior (Burstein & Ginsburg, 2010).

By the same token, repeated observations of non-anxious responses to mild stressors are linked to decreased anxiety in at-risk children. For example, behaviorally inhibited toddlers who experience non-parental child care are less likely to display shyness and social reticence as preschoolers, relative to children in familial care (N. A. Fox et al., 2001). Daily close interactions with an anxious individual, particularly in the absence of countervailing evidence may inadvertently convey a general expectation of negative social evaluations (Bögels et al., 2011; Ollendick & Hirshfeld-Becker, 2002). Compounding this effect, anxious parents may provide fewer social outlets for their children, reflecting their own concerns with social interactions (Ollendick & Hirshfeld-Becker, 2002; Spence & Rapee, 2016). The relatively strong countervailing effect of out-of-home care (Degnan et al., 2008; Phillips et al., 2011) also reflects clinical insights into the use of gradual, but repeated exposure, in cases of social anxiety (Kendall et al., 1997; Radtke, Strege, & Ollendick, 2020).

Outcome Measurement: Early Markers Preceding Symptom Onset

Our model (Fig. 1) proposes that researcher can capture the developmental progression to anxiety by examining intermediate markers that can precede an increase in overt or tractable symptoms. The developmental literature has carefully detailed endogenous factors that are associated with individual patterns of anxiety development (Fox et al., 2006; Hane et al., 2008), including EEG-based markers of regulation, ERP-based metrics of cognitive control, and patterns of attention bias to threat evident in eye-tracking measures (Briggs-Gowan et al., 2015; Brooker et al., 2014; LoBue et al., 2014; Morales et al., 2015; Morales et al., 2015; Pérez-Edgar et al., 2013; Pérez-Edgar, et al., 2010; Pérez-Edgar et al., 2010). To date, however, we do not know how these factors intersect with emerging patterns of anxiety, as influenced by dyadic social dynamics. These biological and cognitive markers of risk in early childhood can be examined across RDoC units of analysis (Casey et al., 2014). Further, they can serve as leading indicators of anxiety progression, given that young children may not yet show elevated symptom levels and provide insight into associated functional mechanisms of risk.

First, the field has often employed neural measures, specifically using EEG, to mark anxiety risk. For example, delta–beta coupling is the moment-to-moment correlation between EEG power in the delta (1 to 3 Hz) and beta (13 to 20 Hz) bands, reflecting the cross-talk between subcortical and higher-order cortical brain networks (Knyazev et al., 2006). Top-down processes (marked by prefrontal cortex activation and beta power) are thought to dampen bottom-up stimuli (marked by limbic activity and delta power) (Knyazev et al., 2006; van Peer et al., 2008). Decoupling between delta and beta power may indicate regulation difficulties, while positive coupling reflects active regulatory processes. However, some studies have reported strong, positive delta–beta coupling in high-anxiety groups (Knyazev, 2011), suggesting that enhanced synchrony can be indicative of "overcontrol" processes induced by anxious states. Stronger delta–beta coupling has also been observed in children with dysregulated fearful temperament (Phelps et al., 2016).

Additionally, EEG signals time-locked to individual task trials can be aggregared to creat event-related potentials (ERPs), which can both capture current processing on the order of milliseconds and act as a predictive indicator of later socioemotional functioning (Hajcak et al., 2019). The N2 component, in particular, is a negative deflection that peaks between 200 and 350 ms after stimulus onset and reflects response inhibition, attention shifting and focusing, and conflict detection (Luck et al., 2000). Thus, the N2 may reflect the extent to which children monitor their environment, take in information, and shift subsequent processing. One study found that six-year-olds high in fearful temperament showed elevated levels of anxiety symptoms only if they also exhibited a small N2 during a flanker task (Morales, 2017).

Analytic Considerations

Our approach to examining moment-to-moment dyadic interaction, often in a longitudinal context, requires careful application of complex statistical methodology. Calculation of synchrony as a dyadic index should employ measures of similarity, such as Pearson product-moment correlations between two sets of scores, covariances, or intraclass correlations where larger values imply greater similarity (Kenny, Kashy, & Cook, 2006). Synchrony indexes are helpful when measuring similarity (i.e., high and low synchrony) across a set of behavioral, physiological, or neural measures to understand contingent relations between members of a dyad. Modeling moment-to-moment interactions that emerge into developmental patterns of risk require longitudinal data that are classified as stable processes or developmental processes (Jongerling et al., 2015; McNeish & Hamaker, 2019).

Emergent Patterns in Dyadic Synchrony

Stable processes allow for the capture of dyadic synchrony within moment-to-moment interactions. These intensive longitudinal data (ILD) structures typically include ten or more measurement occasions that are spaced closer together (e.g., seconds, hours, days) with a focus on instantaneous change (McNeish & Hamaker, 2019). Mean-level changes are typically ignored given that such processes are mean-reverting and stable within an observation window (e.g., Fig. 2). Rather, variability of the outcome (i.e., dyadic synchrony) becomes the focus of the analysis, which includes understanding the relative influence of covariates in explaining within-person variability. A candidate model to analyze stable processes is the two-level dynamic structural equation model (DSEM) appropriate for $N > 1$ analysis of ILD structures (Asparouhov & Muthén, 2020; Asparouhov et al., 2018; McNeish & Hamaker, 2019). Two-level DSEM integrates different modeling approaches (i.e., time-series analysis, multilevel modeling, and structural equation modeling) that specifies a within-dyad (fixed effects) level that meets the stationarity assumption (i.e., mean, variance, and autocorrelations do not systematically change over time) and a between-dyad (random effects) level.

Here, dyadic synchrony can be used as the repeated measures outcome assumed to be mean-reverting, where covariates such as child temperament can be included to examine how or why synchrony patterns might vary. Given that synchrony reflects an emergent property of the parent–child dyad's shared experience, one way to model heterogeneity in these patterns is to employ finite mixture modeling. For example, DSEM mixture models allow estimation of a categorical latent class or continuous latent profile where dyadic synchrony patterns (within-dyad) and the relative influence of covariates (between-dyad) are now dependent on this latent classification or profile. These emergent classes or profiles can then be employed in subsequent examinations of developmental processes.

Long-Term Anxiety Trajectories of Dyadic Synchrony Patterns

Developmental processes, unlike stable processes that focus on variability around a mean, focus on the amount of change in the means of the outcome variable over time. Although still longitudinal in nature, these outcomes are measured in fewer occasions (e.g., 3–10) that occur across months or years. Whereas covariates in stable processes predict fluctuations in each moment-to-moment measurement, covariates in developmental processes are considered in how they explain between-dyad variability or change in growth curves. Therefore, emergent patterns (e.g., latent profiles) derived from the analysis of ILD structures can further be

employed in the examination of developmental trajectories that influence child anxiety symptoms as a result of familial risk factors. For example, estimation of multiple DSEM mixture models from annual parent–child lab visits across ages 3, 4, 5, and 6 years can be examined using latent growth curve modeling. The shape (e.g., linear, quadratic, cubic) of such developmental processes can also be hypothesized based on existing literature and used to inform macro-level data collection occasions. Inclusion of auxiliary variables such as distal outcomes (e.g., child anxiety symptoms) can also be included and precited based on the resultant growth patterns.

Capturing Emotion Modeling in Parent–Child Dyads

Given the availability of child-centered data capture technology (e.g., mobile eye-tracking, heart rate) to measure intensive longitudinal data, differential equation modeling (DEM) could serve as a viable analytic tool. According to Ferrer and Steele (2012), DEMs are useful when considering members of a dyad as an interdependent system where changes result from individual level and/or system-level factors. Each interdependent system is modeled as two differential equations (see Ferrer & Steele, 2012, p. 114) that represent the rate of change in behaviors/emotion for members of a dyad (e.g., parent and child). Framed within the hypothesis that children's phenotypic expression of anxiety results from direct modeling of caregiver response to stress, parameter estimates for DEMs can be modeled to show that changes in a child's behavior/emotion over time are a function of individual factors (e.g., child-related goals) and system-level factors (e.g., caregiver stress response). Variations in the basic model can be employed, resulting in four different predicted patterns of dyadic functioning: approach (child behavior/emotion mirrors that of the caregiver), independent (parent and child behavior/emotion are separate from each other), contrarian (parent and child behavior/emotion repel each other), and unregulated (cyclical oscillations in child and parent behavior/emotion that reach equilibrium or instability).

Conclusions, Future Directions, and Treatment Implications

Our conceptual model of parent-to-child anxiety transmission (Fig. 1) is designed to develop innovative research questions and test tractable hypotheses underlying the mechanisms by which parent-to-child anxiety transmission occurs within early childhood. The current approach builds on decades of work characterizing the transactional nature of the parent–child relationship with respect to emergence of psychopathology over time (Sameroff, 1975; Yirmiya, Motsan, Kanat-Maymon, & Feldman, 2021). As described above, we suggest that parent anxiety is behaviorally transmitted to the child by a process of dyadic social dynamics, leading to maladaptive cognitive and emotion processing. The approach of examining variation across dyadic social dynamics, using multi-modal assessments, especially when applied to a longitudinal context, will allow us to capture trajectories of behavior change over time linked to in-the-moment dynamics (Casey et al., 2014). Importantly, our model presents a flexible empirical strategy that can be applied to developmental and clinically relevant questions across multiple domains of interest. Understanding transmission through dyadic processes may extend to other disorders (e.g., depression, disruptive behavior) or other life stressors (e.g., poverty, family interpersonal stress), to help explain both how emotional dysfunction and adaptive emotional responses occur in familial relationships.

As is evident in the review, there are a number of open questions regarding the form and impact of dyadic social dynamics in the transmission of anxiety. There are, among others, two broad areas of work needed in order to advance this research. First, when and for whom does variation in the context and content of dyadic social dynamics impact anxiety trajectories? Second, once these relations are isolated, do direct perturbations to the systems shift patterns of anxiety? If yes, this would support the premise that dyadic social dynamics can serve as an active mechanism in intergenerational transmission. On the first question, there are a number of strong initial candidates for potential moderators (or mediators) of dyadic effects. Based on the broader anxiety literature, this includes child or parent sex (Bögels & Phares, 2008), match in parent and child sex, co-parent behavior, the specific form of parental psychopathology, patterns of co-morbidity, and individual differences in stress reactivity.

For example, it is unlikely that all children show the same levels of dyadic synchrony nor are as receptive to instances of emotion modeling. In particular, children sensitive to environmental input are more likely to carry the influence of dyadic process, for better and for worse. From the early literature, there is growing evidence that temperamental differences in socioemotional reactivity and regulation are associated with variation in synchrony and modeling. Fearful temperament is the strongest individual difference predictor of anxiety (Chronis-Tuscano et al., 2009; Clauss & Blackford, 2012; Fox et al., 2015; Fox, Snidman, et al., 2015; Hayward et al., 1998; Prior et al., 2000; Schwartz et al., 1999). Temperamentally at-risk children have a three- to fourfold increased likelihood for developing social anxiety (Clauss & Blackford, 2012). The majority of children with extreme temperament are not clinically anxious. Instead, they are more likely to display elevated levels of shyness and social withdrawal and may have difficulty establishing

and maintaining social relationships with peers (Buss et al., 2013; Degnan & Fox, 2007; Fox et al., 2005, 2008).

Over-controlling and over-solicitous parenting behavior potentiate temperamental risk (Kiel & Kalomiris, 2015; Kiel et al., 2016), reflecting a complex relationship between parental behaviors and child anxiety risk (Kiel et al., 2021; Maag et al., 2021). These broad patterns of parental behavior are also evident in moment-to-moment interactions. For example, during a parent–child puzzle task, parents who had higher levels of anxiety spent more time in parent-focused/controlling behaviors with their children, which increased with levels of child fearful temperament (MacNeill et al., in press). Another study, in turn, found that parental anxiety is associated with higher levels of synchrony between parent and child in the same task, providing the underlying conduit for the observed relations (Quiñones-Camacho et al., 2021). Coupled with the fact that temperamentally fearful children display greater biological sensitivity to context (Hastings et al., 2019), attunement with parents may play an outsized role in shaping anxiety trajectories (Muris et al., 1996; Reynolds et al., 2018).

There is, of course, also the open question of whether variation in dyadic synchrony is associated with the impact of emotion modeling within the dyad. To date, we know of no empirical studies that have directly addressed this question. However, one could presume that higher levels of synchrony in parent–child dyads (e.g., neural or behavioral) may be associated with the child being particularly attuned to fluctuations in the parent's expressed emotion, which could precipitate instances of direct modeling. This relation is unlikely to be simple or linear. For example, in an illustrative study with 10-year-olds, levels of behavioral synchrony between child–child dyads varied across structured and unstructured tasks, reflecting the specific in-the-moment goals for an interaction (Anaya, Vallorani, & Pérez-Edgar, 2021a). In addition, mismatches (e.g., one child positive and the other child negative) in delta–beta coupling, a neural marker for emotion regulation (Anaya et al., 2021b), impacted patterns of behavioral synchrony. In particular, negative coupling in the temperamentally fearful child was associated with less synchrony, perhaps reflecting under-regulated cortical–subcortical interactions that may interfere with the ability to get in sync with an unfamiliar partner.

We emphasize that our developmental model has the potential to guide novel treatment approaches aimed at intervening on dyadically linked biobehavioral mechanisms *before* symptom onset. For example, interventions often target children after the emergence of functional impairment. The work described in this review highlights the translation of the parent–child dyad as an intervention point that can counteract evident (e.g., genetic, parenting)-risk markers. For example, future research pinpointing a deficit in dyadic social dynamics that develops along the trajectory to anxiety could be intervened upon using Parent–Child Interaction Therapy (PCIT). PCIT is the most widely used treatment to improve child mental health in early childhood (Herschell et al., 2002). Based on both Attachment Theory and Social Learning Theory, this intervention technique was designed to improve child disruptive behavior through increased attunement during parent–child interaction. PCIT has been adapted to treat anxiety (Puliafico et al., 2012) and depression (Lenze et al., 2011), among other disorders. For example, testing of our model could indicate an increase of dyadic synchrony during moments of increased tension with overt anxious behaviors from the parent (e.g., nervous movement, hovering), with or without a child moderator (e.g., fearful temperament), leading to a lagged response of anxious behaviors in the child (i.e., modeling). As a result, we might use PCIT to teach the parent how to limit their own anxious behaviors. This approach, of course, would only be possible through dyadic approaches to both research and treatment. Thus, in adapting our developmental model to potential treatment implications, we propose that PCIT, and other techniques focused on parent–child interaction, could be modified to treat children who are exposed to increased parental anxiety, especially those with highly anxious temperament, *before* the onset of their own symptoms. This would allow for early intervention and potential prevention prior to symptom onset along the psychopathology trajectory.

Acknowledgements This paper was supported by NIH Grants R56 MH126349 and R01 MH130007 to MPIs Pérez-Edgar and Perlman.

Compliance with Ethical Standards

Conflict of interest The authors have no disclosures.

Ethical approval This manuscript is a review/theoretical paper and does not fall under IRB approval. No subjects were tested as part of this manuscript.

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