Mother-infant interpersonal neural connectivity predicts infants' social learning

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ONE SENTENCE SUMMARY

Mother-infant interpersonal neural connectivity predicts infants' social learning, and is modulated by gaze and speech cues

ABSTRACT

Social learning allows infants to learn vicariously by observing adult behaviour, but how the infant brain accomplishes this feat remains unknown. Here, electroencephalography (EEG) signals were simultaneously measured from forty-seven mothers and infants (10.7 months) during a live social learning task. First, infants observed mothers demonstrate positive or negative emotions toward novel toys. Next, infants' own toy interaction (learning) was measured. Infants' social *learning likelihood* was robustly predicted by mother-infant *interpersonal* neural connectivity in the Alpha (6-9 Hz) band. Stronger dyadic neural connectedness predicted increased learning, and was associated with extended ostensive eye contact and maternal utterances. *Intra-infant* neural connectivity predicted *learning valence* (positive/negative) but was unrelated to learning likelihood. Therefore, interpersonal connectivity is a neural mechanism by which infants learn from their social partners.

(125/125 words)

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49 Social learning is the ability to learn vicariously through observation of others'
50 behaviour and via social interactions (verbal and non-verbal communication), without
51 requiring direct experience (1). Tomasello (2) argues that human mastery of such “second-
52 personal social relations” (3) – in which social partners share and leverage on their joint
53 knowledge, intentionality and goals – is what has distinguished our species and propelled its
54 rise through “cultural intelligence” (4). However, the social transmission of knowledge is
55 challenging – information may be incomplete, irrelevant or misleading, requiring the
56 recipient to weight the reliability and relevance of the source as well as the message.
57 Accordingly, much research has focused on understanding when, how, and why individuals
58 learn from each other (5). In fact, even infants are capable of selecting when and whom to
59 learn from, using cues such as competence, age and confidence as deciding factors (6). For
60 example, after watching an experimenter interact with toys either in an expert or non-expert
61 manner, 12-month old infants are more likely to interact with a new toy when this is
62 presented by the expert (7).

63 One key form of social learning is social referencing – a process of emotional
64 communication in which the partner’s social interpretation of events is used to form one’s
65 own understanding of a situation (8). Social referencing develops over the first year of life,
66 and by 10-12 months of age, infants begin to seek information from others in novel situations
67 and use this information to regulate their own affect and behaviour (9). For example, infants
68 at this age will avoid crossing a short visual cliff (10), show less interaction with toys (11-12)
69 and be less friendly to strangers when their mothers show negative emotion as compared to
70 neutral or happy emotion (13-14). However, Walden & Ogan (15) noted that whereas 10-13
71 month old infants showed predictably less interaction with toys that were associated with a
72 fearful message rather than a positive message from their mothers, older infants (aged 14-22

months) showed the converse pattern, interacting for longer with negatively labelled toys. These findings highlight that infants learn *subjectively* from their adult social partners, showing individual differences in *whether* they learn as well as *what* they learn through social observation.

Despite the importance and early emergence of social learning abilities, little is known about the neural mechanisms that underpin infants' ability to learn through social interaction. Given the complexities of social learning, it is not surprising that the majority of neuroscience research has focussed on how brains learn in isolation through direct experience, rather than from a social partner (16). This applies particularly to understanding the crucial early stages of learning. Here, we address this gap in knowledge. Infants performed a social learning task with their mothers whilst dyadic brain activity was monitored using EEG, (see Figure 1). First, infants observed mothers demonstrate positive or negative emotions toward a pair of novel toys. Next, infants' own interaction with the toys was measured. Two indices of infants' social learning were computed: ***Learning Valence*** (*what* infants learned, positive or negative) and ***Learning Likelihood*** (*when* learning occurred). Here, we report the neural substrates in the infant alpha band (6-9 Hz, see SM S1.1 for explanation) associated with each learning index, as well as maternal behaviours that significantly moderate infants' social learning.

Figure 1. (A) Illustration of the experimental setup and trial structure for the social learning task. During the Observation phase, the parent expressed negatively- or positively-valenced emotions toward a pair of novel objects (order counterbalanced). Infants then interacted with the objects during the Response phase. (B) Intra-personal and inter-personal neural connections assessed during the Observation phase of each trial. Infant and adult heads are shown facing each other, and each dot represents one EEG electrode. Connectivity was measured using the phase-locking value (PLV, see SM S1.8). The relationship between these PLV measures and infants' learning responses was assessed. (C) Illustration of scoring of the two learning indices, Learning Valence and Learning Likelihood, for a single participant (see SM S1.9 for details). In this example, the participant completed 8 trials labelled t_1 to t_8 respectively. As the infant's Valence was >0.5 , her target was the positively-valenced object. Therefore, for each trial, if the infant interacted with the positively-valenced object, this response was scored as 1 in the Learning Likelihood assessment (indicated in blue, upward spokes). If she interacted with the negatively-valenced object, this response was scored as 0 (indicated in red, downward spokes). Here, the infant's Learning Valence was 0.97 and her Learning Likelihood sequence was [1 0 1 1 1 0 1 1]. (D) EEG electrode montage for parent

and infant during data acquisition ($N=32$). Electrodes excluded from data analysis due to motion artifact contamination are marked in red ($N=17$), and electrodes retained for data analysis are marked in green ($N=15$), see SM S1.7 for details. Written informed consent was obtained for the publication of this image.

2 RESULTS

2.1 Neural correlates of infants' Learning Valence and Learning Likelihood

Figure 2A (left column) indicates that infants' Learning Valence (see SM S1.9 for description) was most strongly and extensively correlated to their *own* (i.e. intra-infant) alpha (6-9 Hz) neural connectivity during the Observation phase. A total of 28 neural connections over infants' central and parietal regions were significantly correlated with Learning Valence, after surviving analyses controlling for random correlations and spectral power effects (described in SM S1.10). There were also more limited correlations observed for interpersonal dyadic connectivity (3 significant connections) and for intra-adult left frontal connectivity (1 significant connection). When infants' Learning Likelihood was considered (Figure 2A right column), the opposite pattern of effects was revealed. No intra-infant or intra-adult connections were significantly correlated with infants' likelihood of learning. Only dyadic interpersonal neural connections were significantly correlated with the infants' Learning Likelihood (17 connections survived stringent control analyses). In a subsample replication analysis (see SM S2.2), we confirmed that the reported correlation pattern of alpha (6-9 Hz) intra- and interpersonal connections to Learning Valence and Learning Likelihood respectively was highly internally replicable (82%-90% replicability on average).

Figure 2. (A) PLV-Learning correlations for Valence (left column) and Likelihood (right column), for each intra-infant (top row), intra-adult (middle row) and interpersonal (bottom row) neural connection in the alpha (6-9 Hz) range. Only significant connections (>99.8% of surrogate distribution) whose spectral power was not correlated with each index are shown (see SM S1.10), with hotter colours indicating a stronger positive correlation. The x- and y-axes for all subplots are labelled with electrode names. Correlation values shown are after subtraction of the respective 99.8% threshold value for each connection.

(B) (left panel) Scalp topography of neural connections that were significantly correlated with Learning Valence (top) and Learning Likelihood (bottom). Intra-infant and intra-adult connections are plotted in purple, interpersonal connections are plotted in green. The infant (left) and adult (right) heads are shown facing each other. (right panel) Functional dissociation of Learning Valence (top) and Learning Likelihood (both) with intra-infant and interpersonal neural alpha (6-9 Hz) PLV indices. In each plot, Learning Valence (top) or Likelihood (bottom) is shown as a function of infants' interpersonal (red) and intra-infant (blue) binned PLV index values (see SM S1.10 and S1.11 for details). For both plots, dots show the raw values for each bin, lines indicate the linear line of best fit.

Next, we conducted a confirmatory analysis, described in SM S1.11, to ensure that the identified PLV connections *differentially* predicted variability in infants' Learning Valence and Learning Likelihood respectively (i.e. a functional dissociation). As shown in Figure 2B (right top, blue line), infants with higher intra-infant PLV indices showed more positive Valence whereas there was no relationship between interpersonal PLV indices and Valence (Figure 2B right top, red line). To confirm this dissociation, both intra-infant and interpersonal PLV indices were concurrently entered into a multiple linear regression analysis taking Valence as the dependent variable. The intra-infant PLV index emerged as the sole significant predictor of Learning Valence ($B=.54$ [$SE=.15$], $t=3.64$, $p<.001$ two-tailed). The interpersonal PLV index did not predict Learning Valence ($B=-.26$ [$SE=.15$], $t=-1.76$, $p=.08$ two-tailed).

Conversely, the interpersonal PLV index positively co-varied with infants' likelihood of learning on individual trials (Figure 2B, right bottom, red line) whereas there was no systematic relationship between the intra-infant PLV index and infants' likelihood of learning (Figure 2B, right bottom, blue line). To confirm this dissociation, both interpersonal and intra-infant PLV indices were entered as concurrent predictors into a logistic regression analysis, taking infants' response on each trial as the dependent variable. Only the interpersonal PLV index emerged as a significant predictor of infants' responses ($B=6.00$ [$SE=2.51$], $p<.05$ two-tailed, Wald $\chi^2=5.71$). The intra-infant PLV index was not a significant predictor ($B=.73$ [$SE=3.25$], $p=.82$ two-tailed, Wald $\chi^2=.05$).

Finally, since infants' Learning Likelihood was originally assessed with respect to individual trials, we also conducted a supplementary trend analysis over multiple trials to ascertain that the identified interpersonal PLV connections were significantly correlated to infants' likelihood of learning over time ($r=+.09$, $p<.005$; see SM S2.3).

2.2 Causal relationships between interpersonal connectivity and social learning

Next, we sought to establish whether there was a (temporal) causal relationship between interpersonal neural connectivity and infants' social learning (see SM S1.12 for details). As illustrated in Figure 3, we examined both possible *directions* of temporal causality: do infants' responses affect subsequent dyadic connectivity (Figure 3a blue arrow 1), and do changes in dyadic connectivity affect subsequent responses by infants (Figure 3a blue arrow 2)? To assess the statistical significance of each causal effect, we conducted two McNemar's tests on the relevant contingency data (see Figure 3b(1) and (2) respectively). With regard to the first causal effect (blue arrow 1), McNemar's test revealed that infants' responses were indeed associated with changes in subsequent levels of dyadic neural connectivity (see Figure 3b(1), McNemar Chi-squared = 5.79, $p < .05$, Odds Ratio = 0.69). Nontarget responses were 1.6 times more likely to trigger a subsequent increase than decrease in dyadic connectivity (72↑, 44↓) whereas target responses were 1.2 times more likely to trigger subsequent decrease than increase in dyadic connectivity (85↑, 105↓).

With regard to the second causal effect (blue arrow 2), changes in dyadic neural connectivity indeed had a significant effect on the subsequent distribution of infants' responses (see 3b(2), McNemar Chi-squared = 6.27, $p < .05$, Odds Ratio = 1.60). Increases in dyadic connectivity were 2.3 times more likely to generate a target than nontarget response (109 target: 48 nontarget) whereas decreases in connectivity produced similar proportions of target and nontarget responses (77 target, 72 nontarget). In fact, when *changes* in infants' responses from one trial to the next were considered, instances where infants *corrected* their response from nontarget to target (orange arrow, Figure 3a) were more frequently associated with *increased* dyadic connectivity (57↑, 30↓) than instances where infants did not correct their response (15↑, 14↓) (McNemar Chi-squared = 4.36, $p < .05$, Odds Ratio = 2.00).

(b)		① Infants' <u>current</u> response (trial t)		② Infants' <u>resulting</u> response (trial $t+1$)	
		Target	Nontarget	Target	Nontarget
Change in dyadic connectivity (trial $t+1$)	↑	85 (27.8%)	72 (23.5%)	109 (35.6%)	48 (15.7%)
	↓	105 (34.3%)	44 (14.4%)	77 (25.2%)	72 (23.5%)

Figure 3. (a) Illustration of the chain of temporal causality whereby infants' response during trial t (target or nontarget, defined in SM S1.9) associates with changes in subsequent interpersonal alpha (6-9 Hz) connectivity levels during the Observation phase of trial $t+1$ [numbers indicate the number of trials showing a decrease (red) or increase (green) in dyadic connectivity]; which, in turn, associate with changes in infants' subsequent behaviour during the response phase. Arrow thickness indicates the relative likelihood of occurrence for each type of transition. The orange arrow highlights successful pedagogical events where

infants corrected their responses on the subsequent trial (i.e. nontarget -> target). For each category of responses, the number (percentage) of observed trials is shown. (bottom) Based on these contingencies, two (temporally)-causal hypotheses may be evaluated: (blue arrow 1) do infants' responses affect subsequent dyadic connectivity? (blue arrow 2) do changes in dyadic connectivity drive subsequent choices?

(b) Contingency tables showing (1) the relationship between infants' response on trial t , and subsequent changes in dyadic neural alpha (6-9 Hz) connectivity on trial $t+1$ (Observation phase) and; (2) the relationship between changes in dyadic alpha (6-9 Hz) connectivity from trial t to trial $t+1$ (Observation phase) and infants' resulting response on trial $t+1$. Cells show the number of trials (percentage) in which each possible outcome was observed.

2.3 Modulators of dyadic interpersonal connectivity

Since dynamic changes in dyadic neural connectivity were (temporally) causally associated with infants' learning outcome from trial to trial, we were interested in identifying behaviours that modulated levels of interpersonal connectivity. We focused on trials where infants gave a non-target response as these were more likely to elicit maternal correctional behaviour and consequently, changes in interpersonal connectivity levels. Here, we assessed whether changes in (1) infants' proportional looking to their mother (as compared to the object) and (2) maternal speech characteristics were associated with changes in dyadic connectivity levels and learning outcomes on the subsequent trial (see SM S1.13 for details).

2.3.1 Infants' gaze to mother as compared to object

As shown in Figure 4, increases in infants' proportionate looking to their mother (as compared to the object) were associated with a higher likelihood of successful than unsuccessful corrections in infants' responses on the subsequent trial (78.2% versus 21.8%) as compared to decreases in maternal fixation (McNemar Chi-Square = 11.02, $p < .001$, Odds

Ratio = 3.00). Further, for successful pedagogical events, infants' increased eye contact with their mothers was more frequently associated with increased dyadic neural 6-9 Hz connectivity (31↑:12↓) than decreased eye contact (20↑:16↓; McNemar Chi-Square = 4.17, $p < .05$, Odds Ratio = 1.94). Conversely, for unsuccessful pedagogical events, there was no significant association between infants' proportion of eye contact with their mother and changes in dyadic connectivity (McNemar Chi-Square = 0.10, $p = .75$, Odds Ratio = 0.67).

Figure 4. Diagram illustrating the frequency with which increases (top) and decreases (bottom) in infants' proportional looking to their mothers led to corrections in infants' learning responses. For each category of events, the number (percentage) of trials is shown, along with the number of trials on which decreases (red) or increases (green) in dyadic neural alpha (6-9 Hz) connectivity were observed. Successful corrections are shown in white circles and unsuccessful corrections are shown in grey circles. Arrow thickness and circle size illustrate the relative frequency of events.

2.3.2 Maternal speech properties

Figure 5 shows that increases in maternal speech duration produced a higher proportion of successful corrections in infants' behaviour than unsuccessful corrections (76.3% versus 23.7%) as compared to decreases in speech duration (McNemar Chi-Square = 13.02, $p < .001$, Odds Ratio = 3.00). Further, for successful pedagogical events, longer maternal utterances were more frequently associated with increased dyadic alpha (6-9 Hz) connectivity than shortened utterances (31↑:14↓ versus 26↑:16↓; McNemar Chi-Square = 4.17, $p < .05$, Odds Ratio = 1.94). Conversely, for unsuccessful pedagogical events, there was no significant association between maternal utterance duration and changes in dyadic connectivity (McNemar Chi-Square = 0.06, $p = .80$, Odds Ratio = 1.00).

Figure 5. Diagram illustrating the frequency with which increases (top) and decreases (bottom) in maternal speech duration led to successful or unsuccessful corrections in infants' learning responses. For each category of events, the number (percentage) of trials is shown, along with the number of trials on which decreases (red) or increases (green) in dyadic neural alpha (6-9 Hz) connectivity were observed. Successful corrections are shown in white circles and unsuccessful corrections are shown in grey circles. Arrow thickness and circle size illustrate the relative frequency of events.

Maternal modulations of mean pitch and intensity during successful pedagogical events was not significantly associated with changes in dyadic neural connectivity (Pitch: McNemar Chi-Square = 2.25, $p=.13$; Intensity: McNemar Chi-Square = 1.73, $p=.19$). Further analyses in SM S2.4 and S2.5 provide details of the temporal dynamics of maternal speech acoustics and of infant looking patterns respectively.

4 DISCUSSION

Social observational learning is a powerful but subjective form of early learning, yet little is understood about its neural mechanisms. In this study, we sought to identify the intra- and interpersonal neural alpha (6-9 Hz) correlates of social learning between infants and their parents. We report three main findings. First, a functional dissociation was observed between the neural substrates supporting infants' Learning Valence and Learning Likelihood respectively. Stronger alpha interpersonal connectivity predicted a higher likelihood of social learning on a given trial (irrespective of Learning Valence) whereas intra-infant alpha connectivity predicted Learning Valence only (infants' overall tendency to select the object that was positively- or negatively-modelled). These results suggest that interpersonal alpha connectivity influences when learning will occur during social interaction; whereas intra-personal alpha connectivity influences what information infants learn from their social partners.

Subsequent analyses revealed temporal contingencies between dynamic changes in dyadic neural alpha connectivity and fluctuations in infants' learning from trial-to-trial. Increased dyadic connectivity typically preceded successful learning by infants. Further, unsuccessful learning was frequently followed by increased dyadic connectivity levels on the subsequent trial, perhaps indicating a correctional pedagogical response by parents. Taken together, these results suggest that dynamic changes in the strength of neural connectedness between parent and child are intimately associated with changes in infants' social learning. Third, when we assessed maternal and infant behaviours that associate with changes in dyadic connectivity, we found that longer mutual eye contact and increased maternal speech duration were both associated with up-regulations in interpersonal alpha connectivity during successful pedagogical events.

In the wider developmental context, social co-ordination between parents and their offspring is known to play a vital role in supporting early learning and development (17-18). Parent-child biobehavioural synchronicity (e.g. in gaze, vocalisation and affect) emerges early (during infancy) and influences the development of socioemotional skills like emotional regulation and empathy (19-20). During maternal depression, reduced mother-infant synchronicity may contribute toward long-term deficits in children's learning and development (21-23). Recent studies have indicated that neural synchronisation may support social co-ordination between parents and children. In a study using dual-EEG, Leong et al (24) demonstrated that direct eye contact between adults and 8-month old infants increased their theta and alpha neural synchronisation, concomitantly supporting infants' communicative efforts. An fNIRS study conducted by Reindl et al (25) with parent-child dyads (children aged 7.5 years) found that their prefrontal regions showed significant synchronisation during conditions of social co-operation (but not during competition), with links to children's emotional regulation abilities. However, much remains to be understood about how neural synchronicity between parent and child arises, and its specific role in children's learning and development.

4.1 Mechanistic effect of interpersonal dyadic connectivity on social learning

It is of strong theoretical significance that only interpersonal alpha connectivity (and not intra-infant or intra-adult neural activity) predicted infants' likelihood of learning from a social partner. There are two possible mechanisms for this effect. First, the interpersonal alpha neural signal may act an *attentional filter* (i.e. *gate*) on the sensory and perceptual information that is passed between parent and child during the teaching process. According to this view, only information that is communicated during *mutually* attentive periods (i.e. when parents' and infants' alpha oscillatory cycles are well synchronised) is successfully encoded

by infants for learning. Information that parents communicate during oscillatory misalignment with their infant is attenuated. In this case, interpersonal neural connectivity levels therefore broadly indicate the degree of joint attention between parent and child, which is consistent with previous work (24,26). This view is a logical extension of current models of (individual) attention, which propose that neuronal oscillations act as a mechanism for attentional selection (27).

Alternatively, interpersonal synchrony may act as a *social value signal* which does not directly filter in-coming sensory information, but weights or informs the mental decision to act (i.e. learn) at a later processing stage. This interpretation is consistent with theories of embodied social cognition (28) which suggest that the synchronisation of neurophysiological processes (e.g. mirroring, mimicry or entrainment) allows us to understand and even feel the effects of others' thoughts and emotions (28-29). Indeed, successful social interaction and communication between partners is predicted by widespread neural synchronisation involving brain regions for language processing and prediction (30) as well as higher regions for mentalizing, social cognition and value computation (31). This may explain why biobehavioural synchronisation has long been associated with social affiliation (32), bonding (33) and prosocial behaviour (34). To adjudicate between these two explanations, one would need to perform targeted experimental manipulations that selectively up-regulate joint attention or social value (without affecting the other) to assess downstream effects on interpersonal connectivity and social learning.

4.2 Valence of social learning

Although two-thirds of infants showed a positive learning valence (i.e. they targeted the positively modelled object), approximately one third of infants (16/47=34%) consistently targeted the negatively modelled object instead. This result is consistent with findings by

Walden & Ogan (15) who also observed individual differences in the valence of infants' responses during their social referencing task. Interestingly, Mumme et al (35) also noted that when mothers modelled a fearful expression toward a toy, 12 month old female infants (but not males) tended to look longer at their mother and also approached the toy more, as compared to a neutral baseline. One possible reason for these valence effects could be that infants who favoured the negatively labelled toy may have doubted the veracity of maternal warnings, as these may have been unconvincing in urgency or expressivity. We therefore sought to assess whether maternal speech characteristics, as well as other factors like temperament and maternal sensitivity might contribute toward infants' learning valence. As detailed in SM S2.6, we found that higher temperament similarity between mothers and infants, more succinct (shorter) maternal utterances, and greater sensitivity in maternal behaviour predicted positive learning Valence by infants. However, maternal speech quality (pitch and loudness) did *not* predict Valence, suggesting that variations in maternal performance could not explain infants' tendency to select the positive or negative object. Rather, our results support the view that infants' decision about *what* to learn is more closely related to their prior experience with the social partner. For example, infants whose temperaments were highly dissimilar to their mothers could have previously learned that their social experiences differed from their parents' (e.g. their mother enjoyed a highly-arousing stimulus which they found frightening). This prior knowledge would lead infants to (logically) select the object that their mothers showed dislike rather than liking for, thereby explaining their inverted learning valence.

These results also have strong potential implications for natural pedagogy. If the pedagogical goal of parents is to foster socially appropriate behaviour by infants (i.e, to encourage positively valenced learning), then teaching strategies must first take into account the infants' learning stance (valence). If the infant learner is negatively predisposed (perhaps

due to temperament dissimilarity or other contextual factors), then the use of ostensive signals like eye contact and emphasised speech which increase dyadic connectivity could in fact increase the likelihood of learning socially *disapproved* behaviour. In everyday life, this could create situations of conflict in which parents' efforts to correct infants' behaviour only lead to perpetuation of undesirable behaviour. Rather, our results suggest that increased parental sensitivity during social interaction may be efficacious in changing infants' social learning stance, so that parent and child are attuned in their learning goals. However, an intervention experiment would be needed to confirm these effects.

4.3 Limitations

One limitation of the study is that we relied on natural variations in maternal and infant behaviour (i.e. gaze and speaking) to assess modulatory effects on interpersonal connectivity. Although this approach has the benefit of ecological validity, since these behavioural parameters were not varied in a parametric manner, not all dyads produced large variations in the behaviours of interest, thereby potentially restricting the range of effects observed.

4.4 Conclusion

In conclusion, here we report that dissociable inter- and intra-personal alpha (6-9 Hz) neural circuits underpin infants' social Learning Likelihood and Learning Valence. Whereas interpersonal connectivity predicts the *likelihood* of social learning by infants, intra-infant connectivity predicts their *object* of learning (positive or negative). Temporal contingencies exist between dynamic changes in dyadic neural connectivity and infants' learning from trial-to-trial. Increased dyadic connectivity typically precedes successful learning by infants. Finally, increases in dyadic connectivity associate with greater use of ostensive signals like

389 eye contact and prolonged maternal speech. Our data thus offer insights into the neural
390 mechanisms that underpin the learning of social information during early life.

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ETHICS STATEMENT

This study was carried out in accordance with the recommendations of Cambridge Psychology Research Ethics Committee with written informed consent from all subjects. Parents gave written informed consent on behalf of their children in accordance with the Declaration of Helsinki. The protocol was approved by the Cambridge Psychology Research Ethics Committee (PRE.2016.029).

SUPPLEMENTARY MATERIALS (SM)

Parent-infant interpersonal neural connectivity predicts infants' social learning

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S1 MATERIALS & METHODS

S1.1 Overview of experimental design, analyses and predictions

Infants performed a social learning task with their mothers whilst dyadic brain activity was concurrently monitored using electroencephalography (EEG). The social learning task comprised an *Observation* phase and a *Response* phase (see Figure 1). In the Observation phase, infants watched their mothers express positively- or negatively-valenced emotions towards a pair of novel objects. In the Response phase, both objects were positioned in front of the infant and the object that the infant touched first was recorded. We were interested in the neural substrates that support two distinct aspects of social learning. First, *what* infants learn from their parent (*Learning Valence*), here instantiated as the overall tendency for the infant to choose the object to which their parent had demonstrated positively- or negatively-valenced emotion. Second, *when* learning occurs (*Learning Likelihood*), that is, the likelihood of learning occurring during a given trial. The latter variable reflects the concept that certain neural oscillatory states are optimal for information capture, but neural circuits fluctuate dynamically between more and less optimal states over time (27), accordingly influencing learning probability from trial to trial. In addition, we wished to examine specific teaching behaviours that potentially influence the temporal dynamics of these circuits. Here, we focused on ostensive cues such as eye gaze and vocalisations that extensive previous research has suggested play a role in fostering social connectedness during early learning (17,24).

Three analyses were conducted: First, we examined how the topography of intra-infant, intra-adult and interpersonal neural connections during the *Observation* phase co-varied with infants' *Learning Valence* and *Learning Likelihood* during the *Response* phase that immediately followed. Second, we assessed how infants' social learning changed over the course of the experiment. We specifically sought to establish whether there was a causal relationship between changes in neural connectivity and changes in infants' social learning. We predicted that changes in interpersonal neural connectivity would (temporally) cause changes in infants' learning from one trial to the next. Finally, we assessed whether ostensive signals such as eye contact and infant-directed speech significantly modulated the neural dynamics of social learning by infants. Based on previous work (24), we predicted that these signals would indeed be effective in modulating levels of synchronicity, and therefore learning, within the dyad.

We focused our EEG analysis on the infant Alpha frequency band (6-9 Hz) for a number of reasons. First, because we had successfully observed adult-infant neural synchronicity in this frequency range in our previous research (24). Second, because previous research has suggested that activity in this frequency band plays a crucial role in internally controlled attention (36). Third, because this frequency range is least affected by facial myogenic artifacts which would be generated during this naturalistic task (37-40), see SM S1.7 for processing details).

S1.2 Participants

One hundred and forty participants (70 mothers, 70 infants) took part in the study. The infants showed a 32M/38F gender split. All mothers were native English speakers and infants were aged 11.6 months on average (SEM=0.2). Due to technical issues and excessive EEG artifacts (on account of the naturalistic nature of the task), only neural data from 94 participants (47 mothers, 47 infants) were usable for the final analysis (see Section S1.7 for details on data

exclusion). In this analysed sample, infants were aged 10.9 months on average (SEM=0.32), and showed a 21M/26F gender split. Boys (M=331 days, SEM=9.6) and girls (M=332.7 days, SEM=9.8) did not differ by age ($t_{(45)}=0.12$, $p=0.9$). All infants had no neurological problems and had normal hearing and vision, as assessed by their mother's report.

S1.3 Materials

Four pairs of ambiguous novel objects were used. Within each pair, objects were matched to be globally similar in size and texture, but different in shape and colour. Ambiguous novel objects were chosen to ensure that infants would have no previous experience with these objects.

S1.4 Task protocol

Infants were seated in a high chair, and a table was positioned immediately in front of them. Parents were seated on the opposite side of the table, directly facing the infant. The width of the table was 65cm. Each experimental trial comprised a maternal *Observation* phase involving one pair of novel objects, and a *Response* phase. Each of the four pairs of objects was presented four times to each infant (including both Observation and Response phases for each trial, with trials presented in consecutive sequence and with left and right object locations pseudorandomised), yielding 4 sets of 4 trials, and a maximum of 16 possible trials in total. The task was discontinued if infants showed prolonged fussiness or inattention. On average, infants contributed 10.7 trials (SEM=0.54) to the final analysis. Prior to the start of the experiment, an experimenter trained the parents to deliver the task in a standardised manner. She was also present throughout the session to ensure that participants were interacting as instructed and to code infants' responses. The experimenter provided new pairs of objects as required, but explicitly avoided making prolonged social contact with either participant.

Observation phase. The Observation phase began when the mother attracted her infant's attention by saying "Look", or by holding one of the objects up. Mothers then modelled *positive* emotion toward one object and *negative* emotion toward the other object. Mothers were instructed to limit their speech to simple formulaic verbal descriptions per object (which they repeated for each object), indicating either positive affect ("This is great, we really like this one!") or negative affect ("This is bad, we don't like this one"), and to model positive or negative emotions in a prescribed manner (e.g. smiling versus frowning) (see Figure 1). The order of object presentation (positive or negative) was counterbalanced across trials, and the order of objects was counterbalanced across participants. The Observation phase was completed when maternal utterances ended, which was determined by manual coding of the experimental video (see Sections S1.5, S1.13 and S2.4 for details). Detailed acoustic measurements of maternal utterances and analyses of infants' looking patterns during the Observation phase are provided in the Sections S2.4 and S2.5.

Response phase. After observing their mothers' behaviour toward both objects, mothers placed both objects in front of the infant within the infant's reach (one on the left and the other on the right, consistent with the objects' positioning during the Observation phase). The Response phase started when both objects were stationary and within the infant's reach. The object that the infant touched first was taken as his/her response. After allowing the infant to explore the objects for approximately one minute, they were retrieved from the infant and the next trial commenced. On some trials, infants touched both objects simultaneously. As this response was ambiguous, these trials were excluded from the analysis (a total of 52 trials were excluded).

S1.5 Video recordings and coding of infants' behavioural responses

Throughout the experiment, three Logitech High Definition Professional Web-cameras (30 frames per second) were used to record the actions and utterances of participants. Two cameras recorded a frontal view of the adult and infant respectively, whilst a third camera recorded a side view of their interaction. Afterwards, the video recordings were manually coded for the behaviours of interest. Coding of infants' object selection was performed twice – first live by the experimenter, and later independently verified by a second coder who reviewed the video footage. Agreement between live and video coders was >95%. Coding of infants' gaze patterns and maternal speech acoustics (including utterance durations during the Observation phase) is described further in Section S1.13.

S1.6 EEG acquisition

EEG was recorded simultaneously from the infant and mother using a 2 x 32-channel dual wireless EEG system Mobita (TMSi). Electrode placement corresponded to the international 10-20 system. A high viscosity electrolyte gel SuperVisc (EasyCap, GmbH, Germany) was used to ensure good contact between electrodes and scalp, and electrode impedance was typically kept below 10 k Ω for infants and 20 k Ω for mothers. Data were sampled at 500 Hz, and the ground electrode was placed on the nape. Continuous data were acquired using AcqKnowledge 5.0 software (Biopac Systems, UK).

EEG-video synchronisation. Video recordings of behavioural events were synchronised to the EEG signal by sending triggers via a radio frequency transmitter which marked the EEG trace and also produced a light signal that was visible on the video recording. Synchronisation was performed by manual video coding to identify the exact frame at which the onset of the synchronisation light signal occurred. Thus, the synchronisation accuracy was limited to the temporal resolution of the video frame rate, which was 30 fps or 33 ms.

S1.7 EEG artifact rejection and pre-processing

Overview: We have previously noted that naturalistic experimental EEG data is typically contaminated by a range of movement-related artifacts caused by blinks, saccades, gestures, head movements, speech production and other facial expressions, along with other artifacts specific to infant data including mastication, sucking and other behaviour. Here, we addressed this issue through a combination of approaches: (1) band-pass filtering to remove the most prominent electromyogenic artifacts that occur at frequencies above the Alpha rate; (2) independent component analysis (ICA) to remove stereotypical oculomotor, heartbeat and non-biological artifacts; and (3) exclusion of peripheral channels, which typically show the highest contamination from motion-related artifacts (38). These steps are described in more detail below. Further, to ensure that movement-related artifacts were not systematically biasing our results, in Section S2.1 we present supplementary analyses showing that there was no systematic relationship between the ICA components removed (as an index of the degree of artefactual contamination in the data) and the main neural indices of interest.

Given a relatively brief duration of experimental trials in the present study, and the relatively large data quantities required for successful ICA signal decomposition, inter-trial periods were included during EEG pre-processing. Accordingly, we pre-processed continuous EEG recordings starting 10 sec before the beginning of the first trial and ending 10 sec after the end of the last trial, which were on average 759.6 sec long (Min=220.2, Max=1523.4, SEM=46.1 sec).

Filtering: With the aim to focus the EEG analysis on low Alpha (6-9 Hz) frequencies, continuous recordings were high-pass (1 Hz) and low-pass (16 Hz) filtered using Hamming window FIR filter (cutoff frequencies (-6 dB): [0.5 16.5] Hz; transition band width: 1 Hz). Filtering removed a substantial proportion of electromyogenic artifacts that occur at beta and gamma frequencies (38,40). Recordings were then re-referenced to the average of all channels.

Identification of noisy channels: In order to facilitate the subsequent ICA-based identification and removal of specific artifacts, channels that showed a high general level of noise were first identified and removed using a semi-automatic procedure. First, the power spectrum (1-16 Hz) of individual channels was inspected to identify outliers (more than 3 SD above the mean power spectrum of all channels) which were manually confirmed and then removed. In some cases, data from noisy channels Fp1 and Fp2 were retained when these facilitated the later ICA-based removal of eye-blink artifacts. Additionally, all EEG data were visually inspected and channels which showed excessive noise during critical periods of experimental testing were removed even if their power spectra lay within tolerance limits. Also at this stage, EEG periods that were excessively noisy, were padded to zero (to preserve the temporal structure of the data) but excluded from the subsequent analysis. On average, 61.7 sec (SEM=13) were padded in mothers' datasets, and 146.1 sec (SEM=25.9) in infants' datasets.

Artifact removal using ICA: ICA was conducted using EEGLab (41). In order to facilitate the identification of artifacts, ICA analyses were conducted on all available channels, and then the periphery (outer ring) of the montage were excluded at the final stage of analyses (see below). Only ICA components with clearly identifiable patterns that corresponded to eye blinks and saccades, heartbeat, ground, and muscle noise, were deleted. On average, 9.6 components (SEM=0.39) were removed from mothers' datasets, and 8.9 components (SEM=0.5) from infants' datasets.

Channel interpolation: After the ICA components corresponding to artifacts were deleted, the previously removed noisy EEG channels were then recalculated by spherical spline interpolation of the surrounding channels. On average, 2.9 EEG channels (SEM=0.31) were interpolated in mothers' datasets, and 3.4 channels (SEM=0.28) in infants' datasets.

Exclusion of channels: In a final step, frontal and temporal channels within the periphery (outer ring) of the montage (Fp1, Fp2, AFz, F7, F8, FT9, FC5, FC6, FT10, T7, T8, TP9, P7, POz, P8, TP10 and Oz) were deleted, in order to minimise the influence of residual eye- and muscle-related contamination, retaining 15 channels for the subsequent EEG analysis (see Figure 1 of the Introduction).

Data exclusion. Only 54 out of 70 dyads contributed usable EEG data. Three datasets could not be processed due to a loose ground electrode, two datasets were excluded due to excessive noise across all channels and trials. The remaining excluded datasets could not be reliably time-synchronised due to a hardware error that was rectified for later participants. Individual trials were excluded from the neural analysis if they still contained visually identifiable EEG noise or if either the positive or negative portions of the Observation phase lasted for less than 1 second. Finally, to ensure that the EEG data were representative of participants' performance, only participants who contributed 3 or more usable trials were included in the final analysis. After data exclusion, 47 dyads remained who contributed a total of 463 trials to the final analysis.

S1.8 EEG connectivity analysis: Phase Locking Value (PLV)

We measured intra- and interpersonal neural connectivity over the entire *Observation* phase of each trial by calculating the Phase Locking Value (PLV) between all electrode pairs.

The PLV is a metric that is able to detect frequency-specific transients of phase locking independent of amplitude (42). To examine activity in the Alpha frequency band we selected the frequency range 6-9 Hz, based on previous research into Alpha activity in infants (36,43). To ensure equivalency, the same adjusted Alpha banding (6-9 Hz) was used for both infant and adult computations.

PLV calculation. First, we concatenated the EEG datasets of each member of the dyad, obtaining a single 2D matrix of 30 electrodes (15 infant electrodes and 15 mother electrodes) x time samples of the Observation phase for each individual trial. Second, we performed a band pass filter for the frequency of interest (Alpha 6-9 Hz). Third, we epoched the continuous data from the Observation phase into 1 sec segments using sliding windows with a 50% overlap. Fourth, we standardized the amplitude of each 1 sec EEG segment by calculating its z-score on an electrode by electrode basis, which reduced amplitude variance across participants. Afterwards, we calculated the instantaneous phase of the signal using the Hilbert transform.

Phase synchronisation was calculated between each possible electrode pair, examining both intra-personal connections (between the 15 infant electrodes and the 15 parent electrodes considered independently) and inter-personal connections (between the infant and parent electrodes). Two signals $x(t)$ and $y(t)$ with instantaneous phases $\varphi_x(t)$ and $\varphi_y(t)$ are considered to be phase synchronised if their instantaneous phase difference is constant:

$$\theta(t) = \varphi_x(t) - \varphi_y(t) = \text{constant}. \quad (1)$$

To calculate phase synchronisation, we used the PLV which was defined as:

$$PLV = \left| \frac{1}{T} \sum_{j=1}^T e^{i\theta(t)} \right|, \quad (2)$$

where T is the number of time samples in the segment. PLV is a value within the range [0, 1], where values close to 0 indicate random signals with unsynchronised phases and higher values indicate stronger synchronization between the two signals. Finally, PLV estimates for all 1 sec segments across the entire Observation phase were averaged to obtain a single PLV value for each electrode pair (for all intra- and interpersonal connections).

PLV normalization. To standardise the resulting intra-personal and interpersonal PLV values, we normalised the raw PLV values using a surrogate dataset that represented the distribution of random PLV values between pairs of signals that had identical spectral properties to the real data but were temporally unconnected (i.e. a randomised phase relationship). To create this surrogate dataset, a Fourier transform was applied to each 1 sec EEG segment and a random permutation of its phase values was performed in the frequency domain. Next, an inverse Fourier transform was used to recreate the surrogate data in the time domain. This process retained the original spectral profile of the data whilst selectively disrupting any phase relationships, thereby removing genuine phase-based connectivity patterns. Surrogate PLV values were computed using an identical procedure to the real data, and the phase permutation procedure was repeated 2000 times. Next, the 2000 surrogate PLV values were averaged for each 1 sec data segment, and across segments for each trial.

In order to allow for a comparison between intra- and interpersonal PLV values, we then normalised our original PLV values against the PLV values obtained in this surrogate dataset. To do this, we subtracted the mean surrogate PLV value from the real PLV value for each trial, for each electrode pair (including intra- and interpersonal connections). These normalised trial-level PLV values were used for all later computations. Thresholding for statistical significance was performed separately for individual *connections* via bootstrapping

as part of subsequent analyses, along with controls for spurious effects of spectral power (see SM S1.10 for details). All computations were performed using in-house adaptations of Matlab functions.

S1.9 Social learning indices: Learning Valence and Learning Likelihood

Two independent and separable aspects of infants' social learning behaviour were examined: **Learning Valence** (infants' overall propensity to select objects that were either positively or negatively labelled by the parent) and **Learning Likelihood** (whether learning about a target object took place in a given trial).

Learning Valence. To ascertain this, infants' cumulative behaviour over all trials was considered, taking into account their proportion of positive and negative responses as well as the number of trials completed (i.e. behaviour that was consistently maintained over more trials was rewarded). Valence was computed as the (cumulative) binomial probability of obtaining at least k positive responses out of n total responses, given that the probability, p , of a positive response on any given trial is 0.5, as follows:

$$\sum_{i=0}^{\lfloor k \rfloor} \binom{n}{i} p^i (1-p)^{n-i} \quad (3)$$

Thus, infants' Valence ranged between 0 (all negative responses) and 1 (all positive responses). Further, the number of trials that infants completed was also reflected such that for the same *ratio* of responses, infants who completed more trials received more extreme scores than infants who completed fewer trials, reflecting the lower likelihood that the observed response pattern was obtained by chance. For example, infants who gave 6 positive and 3 negative responses (ratio 2:1, 9 trials) scored 0.91 whereas infants who gave 4 positive and 2 negative responses (ratio 2:1, 6 trials) scored 0.89, thereby "rewarding" infants who maintained a consistent performance over more trials. Conversely, infants who gave more negative than positive responses overall (e.g. 3 positive and 6 negative) achieved lower scores if they completed more trials. Overall, infants selected a positively labelled object on 54.9% (254/463) of all trials completed, and a negatively labelled object on 45.1% (209/463) of trials. The mean learning Valence across all infants was 0.64 (range = 0.09 to 0.99, SE=0.04), reflecting the fact that fewer strongly negatively valenced datasets were present in the cohort. Approximately one third of infants (16/47 = 34%) had a learning Valence of 0.5 or below. These infants consistently chose the object that was negatively modelled by the parent. The remainder of infants (31/47 = 66%) consistently chose the object that was positively modelled by the parent.

Learning Likelihood. The aim of this analysis was to understand **when** infants' learning occurred across different individual trials (independent of their Learning Valence). Therefore, as illustrated in Figure 1 of the Introduction, the Learning Likelihood measure corresponded to infants' trial-to-trial sequence of responding (e.g. [1 0 1 1 1 0]), adjusted for individual infants' Valence. For infants whose Valence was >0.5 (i.e. positively-inclined, 31/47 infants or 66% of cohort), the positively-valenced object was taken as the target object. Therefore each positive response was scored as [1] and each negative response was scored as [0]. For infants whose Valence was ≤0.5 (i.e. negatively-inclined, 16/47 or 34% of cohort), the negatively-valenced object was taken as the target object. Therefore negative responses were scored as [1] whilst positive responses were scored as [0].

S1.10 Neural correlates of Learning Valence and Learning Likelihood (*cf. Results Section 2.1*)

The aim of this analysis was to identify connections in the intra-infant, intra-adult and interpersonal neural network whose strength co-varied with infants' Learning Valence or Learning Likelihood. As the Learning Likelihood measure tracked infants' responses from trial-to-trial, for consistency we conducted both correlation analyses at the *trial level* rather than at the participant level. For Learning Valence, infant's final learning Valence score was correlated to their PLV scores obtained across different individual trials using a Pearson correlation, for each connection. For the Learning Likelihood analysis, infants' response sequences were correlated to their per-trial PLV values using a point-biserial correlation (between a binary and a continuous variable). These analyses were performed separately for the PLV values obtained for each electrode pair.

To ascertain whether the resulting correlation values for each neural connection differed significantly from chance, a permutation analysis was performed to generate the respective surrogate distributions under each null hypothesis (i.e. no relationship between the neural connection and Learning Valence or Learning Likelihood). First, for each neural connection, the PLV values for each trial were randomly permuted across concatenated trials and then correlated to the original Learning Valence scores or Learning Likelihood sequences. This permutation process was repeated 100,000 times in order to generate a surrogate distribution of random correlation values. Only connections whose real PLV correlations exceeded the 99.8% (1/500) highest value of their respective surrogate distributions were identified as being potentially significant (pending also passing the second control analysis).

Next, to remove spurious correlations that were driven by fluctuations in signal power, an identical analysis was performed taking the spectral power of each infant and adult electrode and correlating this power value (for each trial) to the Learning Valence scores and Learning Likelihood sequences. To estimate spectral power, a Fourier transform was performed for each trial using the same segmentation protocol as for the PLV computations (see Section S1.8). Spectral power was calculated for individual frequency bins from 6 Hz to 9 Hz, and the average power across all bins was used. As before, a surrogate distribution of correlation values was created by randomly permutating the power values across concatenated trials and then correlating these permuted values to the original Valence scores and Learning Likelihood sequences. The permutation process was repeated 100,000 times. Connections whose power-Learning Valence and power-Learning Likelihood correlations exceeded the 99.8% (1/500) highest value of their respective surrogate distributions were identified as significant.

Combining these two control analyses, we identified sets of intra- and interpersonal connections whose PLV-Learning Valence or PLV-Learning Likelihood correlations *were* significant (i.e. >99.8% of their surrogate distribution), but whose power-Learning Valence and power-Learning Likelihood correlations were *not* significant. The resulting connections are shown in Figure 2A of the Results section.

For all subsequent analyses in the main text, an *interpersonal PLV index* was computed for each trial as the z-scored mean PLV across all 17 interpersonal connections that were significantly correlated to Learning Likelihood (recall that there were no significant intra-adult or intra-infant connections). An *intra-infant PLV index* was also computed as the z-scored mean PLV across all 28 intra-infant connections that were significantly correlated to Learning Valence. Intra-adult and interpersonal connections were not included in computing the intra-infant PLV index as these were much fewer in number (1 intra-adult and 3 interpersonal connections only) and therefore less representative of the main effect.

S1.11 Confirmation of functional dissociation between identified intra-infant and interpersonal circuits

Here, we sought to confirm a functional dissociation whereby the identified *intra-infant* connections would predict infants' Learning Valence (i.e. their tendency to choose the positively or negatively modelled objects) but not their Learning Likelihood (i.e. whether learning occurs on a given trial). Conversely, we expected the identified *interpersonal* connections to predict Learning Likelihood but not Learning Valence. We used response *probability* as the dependent measure when assessing Learning Likelihood because this is a continuous parameter, whereas the original measure comprised binary responses. For this analysis, we compared the relationship between intra-infant and interpersonal PLV indices (see SM S1.10) with Learning Valence and Learning Likelihood respectively. We performed the same two analyses on each PLV neural index to assess whether each was functionally related to infants' Learning Valence and Learning Likelihood.

Learning Valence. As Valence was computed at the participant level, here the mean PLV index for each participant was accordingly used. Individual infants were rank ordered from lowest to highest by either their interpersonal or intra-infant PLV index. Infants were then divided into 7 equipopulous bins where each bin contained 7 infants, except for the lowest and highest bins, which contained 6 infants each. The mean learning Valence for the infants in each bin was then computed, when rank ordered by either interpersonal or intra-infant PLV values.

Learning Likelihood. As learning likelihood pertains to learning during individual trials, here, the PLV indices for each trial were used. We assessed whether infants' likelihood of learning their target or nontarget object varied systematically as a function of interpersonal and intra-infant PLV indices observed during individual trials. The trial-level intra-infant and interpersonal PLV indices were binned into 7 equidistant bins spanning their respective lowest and highest values across all trials (i.e. the bins corresponded to ± 2.5 , ± 1.7 and ± 0.8 standard deviations from the mean value of each index). For each bin, the mean probability of target responses was computed. Bins where less than 10 trials were observed (representing the extreme values) were excluded. This excluded a total of 16 trials (3.5% of the data) for the intra-infant PLV index and 17 trials (3.7% of the data) for the interpersonal PLV index.

S1.12 Analysis of temporal causal relationships between interpersonal connectivity and social learning (cf. Results Section 2.2)

Here, we sought to establish the causal directionality of the relationship between the *interpersonal PLV index* (see S1.10) and social learning. For this analysis, only trials 1 to 12 were used. The last 4 trials were excluded due to infants dropping out over the course of the experiment (only 47%/38%/23%/11% of infants contributed data respectively for trials 13 to 16). We examined whether infants' learning responses were associated with altered dyadic connectivity levels (i.e. interpersonal PLV index) on the subsequent trial, and whether altered dyadic connectivity from one trial to the next was associated with infants' subsequent response. As these analyses assessed transitional changes, only trials with a subsequent trial could be used. Further, we discarded participants who contributed fewer than 5 usable trials in total (i.e. to ensure that each participant would contribute a minimum of 4 contiguous pairs of trials), leaving a total of 306 trials (66.1% of the dataset) that were analysed.

For each causal effect of interest, a 2x2 contingency matrix was constructed. For the first causal analysis, we assessed the relationship between infants' responses on trial t , and subsequent changes in dyadic neural connectivity during the Observation phase of trial $t+1$. We computed the number of trials in which target or nontarget responses were followed by increases or decreases in the interpersonal PLV index respectively (see Figure 3b(1) of the Results section). For the second causal analysis, we assessed the relationship between changes in dyadic connectivity on trial $t+1$ (Observation phase) relative to trial t , and infants' subsequent response on trial $t+1$. We computed the number of trials on which increases or decreases in the interpersonal PLV index were followed by target or nontarget responses respectively (see Figure 3b(2) of the Results section). All statistical tests used were two-sided.

S1.13 Analysis of modulators of dyadic interpersonal connectivity (*cf. Results Section 2.3*)

For this analysis, we examined the subset of trials where infants gave a nontarget response (116 trials). These were chosen as instances of naturally occurring pedagogical events where mothers sought to change their infants' behaviour. Since mothers enjoyed a high degree of pedagogical success on these trials (i.e. 75% of infants' subsequent choices were for the target object), these trials allowed us to examine the maternal and infant behaviours that were associated with successful pedagogy. Accordingly we calculated the changes in 1) the interpersonal PLV index and 2) in infants' responses between consecutive trials, and we assessed whether these change indices associated with 1) infants' proportional looking to their mother (as compared to the object) and 2) maternal speech characteristics. For each analysis, we constructed 2x2 contingency tables assessing the relative frequency with which increases (or decreases) in the given behaviour were associated with increases or decreases in dyadic connectivity, for successful (nontarget $\rightarrow$ target) and unsuccessful (nontarget $\rightarrow$ nontarget) corrections in infants' responses on the subsequent trial respectively. All statistical tests used were two-sided.

Coding of infants' gaze to mother or object. Video recordings of the Observation phase were manually coded by trained coders who were blind to the experimental hypothesis. For each participant, recordings from two video cameras (showing a frontal and side view) were corroborated. The side view camera was used to assess the mother's positioning of the object whilst the front view camera was used to assess the infant's gaze direction. For each video timeframe (33ms duration), the coder assessed whether the infant was looking at the mother, at the object, or at a different location. The proportion of time that the infant looked at their mother or at the object (out of the total duration of the entire Observation phase) was then computed. Finally, the difference between infants' proportional looking time toward their mother and toward the object was taken for the analysis. When the infants' gaze direction was ambiguous, for example, when the mother was holding the object directly in front of her face, the entire whole trial was excluded from the analysis. By this criteria, 19 trials out of 463 (4% of the data) were excluded.

Coding of maternal speech characteristics. Audio recordings were extracted from the video files and analysed using Praat 5.4 software (www.praat.org). For each trial, the total duration, mean pitch, and mean intensity of maternal utterances during the Observation phase was measured. For accuracy, measurements were only taken between the onset and the offset of maternal speech (excluding initial periods of silence and interruptions by the infant or experimenter). These onsets and offsets were manually checked by a trained rater.

Results of the measurements of infants' gaze and maternal speech characteristics are provided in the Sections S2.4 and S2.5.

S2 SUPPLEMENTARY TEXT

S2.1 Motion artifacts

In addition to our artifact rejection procedures described above, we also performed an additional supplementary analysis. We wished to assess whether infants' and mothers' movement patterns (and the associated EEG distortion) could account for the neural indices of learning that were detected. Accordingly, we conducted a correlation analysis between the severity of motion contamination in the signal (as indexed by number of motion-related independent components [ICs] removed from the EEG signal) and the mean neural phase-locking values obtained within the significant ROIs for learning Valence (intra-infant) and learning Likelihood (interpersonal). Motion-related ICs were defined as having a localised high-frequency peak in scalp areas closest to jaw and neck musculature (i.e. temporal and occipital regions) which would commonly generate speech and head movement EMG artifacts.

Frequency analysis. Tables S1 and S2 show a breakdown of the number of movement-related independent components removed from mothers' and infants' EEG respectively, and the number of mothers and infants showing each number of ICs. For mothers, the mean number of ICs removed was 3.26 (SEM=0.33), ranging from 0 to 8 ICs with the most frequent number of ICs removed being 4 (21% of mothers). Only 7 mothers (15%) had more than 5 ICs removed from their EEG. For infants, the mean number of ICs removed was 3.36 (SEM=0.43), ranging from 0 to 13 ICs with the most frequent number of ICs removed being 3 (21% of infants). Only 7 infants (15%) had more than 5 ICs removed from their EEG. There was no significant difference in the number of ICs removed between infants' and mothers' datasets (Wilcoxon signed rank test: $Z=0.23$, $p=0.82$).

Number of movement-related independent components (ICs)	Frequency (N mothers)	Percentage (%)
0	9	19.1
1	2	4.3
2	7	14.9
3	5	10.6
4	10	21.3
5	7	14.9
6	3	6.4
7	3	6.4
8	1	2.1

Table S1. Movement-related independent components in mothers' EEG datasets.

Number of movement-related independent components (ICs)	Frequency (N infants)	Percentage (%)
0	8	17.0
1	7	14.9
2	3	6.4
3	10	21.3
4	8	17.0
5	4	8.5
6	1	2.1
7	1	2.1
8	1	2.1
9	1	2.1
10	2	4.3
13	1	2.1

Table S2. Movement-related independent components in infants' EEG datasets.

Correlation analysis. There was no significant association between the number of movement ICs detected in infants' EEG (a proxy for amount of movement) and their neural Learning Valence PLV (Spearman's rank order correlation: $\rho=0.11$, $p=0.47$). Similarly, there was no significant association between the number of movement ICs detected in mothers' EEG and their neural Learning Valence PLV (Spearman's $\rho=0.14$, $p=0.33$). Likewise, there was no significant association between the inter-brain neural Learning Likelihood PLV and number of movement ICs detected either in infants' EEG (Spearman's $\rho=-0.09$, $p=0.54$) or mothers' EEG (Spearman's $\rho=-0.08$, $p=0.60$). Therefore, differences in infants' and mothers' movement cannot account for the neural effects observed.

S2.2 Subsample replication analyses

In this analysis, we aimed to assess the internal replicability of our main finding that Learning Valence is primarily associated with intra-infant neural activity whereas Learning Likelihood is only associated with interpersonal neural activity. To conduct this analysis, we randomly subsampled 75% of the dataset a total of 10,000 times. For each random subsample, we computed the (a) original correlation matrix between infants' learning and neural activity; and (b) a surrogate correlation matrix created by randomly permutating the sub-sampled data values prior to performing the correlation (see Section S1.10). Real data correlations that were above 99.8% of the surrogate correlation data values were deemed to be significant.

Figure S1 summarises the internal replicability of the correlations for each intra-infant, intra-adult, and interpersonal neural connection with infants' learning Valence (top) and Likelihood (bottom). The colour of each cell indicates the proportion of subsamples (total 10,000) in which that connection emerged as being significantly correlated to infants' learning behaviour. For example, the Cz-FC2 intra-infant connection was significantly correlated in 98.58% (or 9,858/10,000) of all subsamples. When compared to Figure 2A in the main text, it is clear that *all* the intra-infant (for learning Valence) and *all* the interpersonal (for Learning Likelihood) correlations reported as our main finding fell within the most highly reliable and internally replicable connections. In fact, for Learning Valence, the mean replicability for the reported 28 intra-infant connections was 89.7% (range 77.1% - 99.7%). For Learning Likelihood, the mean replicability for the reported 17 interpersonal connections was 82.3% (range 72.2% - 91.7%). Therefore, the correlations reported in the main text did not arise from only a few outlier data points, but were consistently observed across the cohort.

Figure S1. Internal replicability of (top row, (a)) PLV-Valence correlations for all dyads for each intra-infant (left column), intra-adult (middle column) and interpersonal (right column) neural connection. (left bottom row, (b)) PLV-Likelihood correlations for all dyads for each intra-infant (left column), intra-adult (middle column) and interpersonal (right column) neural connection. The x- and y-axes for all subplots are labelled with electrode names. The colour of each cell indicates the percentage of permuted subsamples (out of a total of 10,000 permutations) where a significant correlation (relative to a surrogate distribution) was observed.

S2.3 Relationship between interpersonal connectivity and learning over time

In this analysis, we sought to confirm whether the relationship between infants' learning likelihood and mother-infant interpersonal connectivity was maintained over the course of the experiment (i.e. both variables were correlated over time). For this analysis, the interpersonal PLV index (see Section S1.10) was re-normalised (z-scored) by participant in order to reduce the effects of inter-individual variability when assessing the learning patterns of individual infants. For each trial position (1 to 12), the mean percentage of target responses was computed across all participants to yield infants' learning pattern over time (see Figure S2). To assess whether there was a statistically significant relationship between infants' learning pattern over time and changes in their corresponding interpersonal neural connectivity with their mothers, a correlation analysis was conducted at the level of individual dyads. For each dyad, the infant's sequence of responses (e.g. [1 0 1 1...]) was correlated to their respective z-scored interpersonal PLV index across those trials (e.g. [0.31 0.15 0.22 0.21...]), yielding one correlation value per dyad. The mean correlation value across all 47 dyads was then computed. Statistical significance was determined via a permutation analysis. For each dyad, their z-scored interpersonal PLV indices were randomly permuted across trials (e.g. [0.15 0.22 0.21 0.31...]) and this permuted sequence of values was correlated with the infant's original response pattern. The mean correlation across all 47 permuted dyad values was then computed. This process was repeated 100,000 times to obtain a surrogate distribution of group mean correlation values. This permutation exercise determined that the real correlation between dyadic neural connectivity and infants' social learning over time was above 99.5% of the values in the surrogate distribution (i.e. $p < .005$), and therefore highly significant.

Figure S2. Infants' pattern of social learning over time. Relationship between infants' average responses toward their target object (left y-axis, solid line) and their interpersonal alpha (6-9 Hz) PLV index (z-scored by participant, right y-axis, dotted line). The x-axis indicates the trial number from 1 to 12.

S2.4 Maternal speech acoustics (across entire observation phase)

Figure S3 (top) shows the distribution of maternal acoustic characteristics that were measured for the observation phase across all trials (463 trials in total). The mean total

duration across all trials was 10.66 seconds (SD = 6.75 seconds). The mean pitch across all trials was 246.0 Hz (SD = 38.82 Hz) and the mean intensity was 69.89 Hz (SD = 5.09 Hz).

Figure S3 (bottom) shows the average acoustic characteristics of trials over time (trials 1 to 12, consistent with the main analysis). Maternal speech duration (left subplot) was longest for the first trial, and then decreased with subsequent trials. There was no consistent pattern of change over time for maternal pitch (middle subplot) or intensity (right subplot).

Figure S3. (top) Histogram plots showing the distribution of maternal acoustic characteristics across all trials for duration (left), pitch (middle) and intensity (right). (bottom) Maternal speech acoustics across trials 1 to 12 for duration (left), pitch (middle) and intensity (right).

S2.5 Infant looking patterns (across entire observation phase)

Figure S4 (top) shows the distribution of the proportion of infant looking time to the object (left), their mother (middle) and either object or mother (right) during the observation phase across all trials (463 trials in total). These proportions were calculated out of the total

duration of the observation phase, thus normalising for differences in the timing of the observation phase. The mean proportion of time that infants looked to the object was 52.75% (SD = 32.60%). The mean proportion of time that infants looked to their mother was 29.19% (SD = 26.55%). On average, infants' total visual attentiveness to either the object or their mother was 81.94% (SD = 27.44%). Therefore, infants were generally highly attentive during the observation phase of the experimental task, and they typically tended to look proportionately for longer at the object than at their mother.

Figure S4 (bottom) shows the average infant looking behaviour over time (trials 1 to 12, consistent with the main analysis). For the first trial, infants' proportion of looking to the object (left subplot) tended to be longer than for the other trials, whilst their looking to their mother (middle subplot) tended to be shorter. There was also a slight trend toward overall decreasing visual attention (i.e. lower looking to either mother object or mother) for later trials (right subplot).

Figure S4. (top) Histogram plots showing the distribution of infants' proportion of looking to the object (left), their mother (middle) or either object or mother (right). (bottom) Infant looking across trials 1 to 12 for the object (left), their mother (middle) and either object or mother (right).

S2.6 Maternal modulators of intra-infant connectivity (learning Valence)

For this analysis, we wished to assess whether aspects of maternal behaviour could explain individual differences in intra-infant PLV index that predicted infants' learning Valence. We computed four maternal predictor variables. To assess *mother-infant compatibility and attunement*, we computed the temperament dissimilarity between infant and mother, as well as maternal sensitivity toward her infant. To assess *maternal speech quantity and quality* we computed her instruction duration as well as the “infant-directedness” of her utterances (an aggregate score of pitch and loudness). All statistical tests used were two-sided.

i. Temperament dissimilarity

Temperament dissimilarity between infants and their mothers was computed by taking the sum of the squared distances between their respective (IBQ for infant, ATQ for adult) dimensional scores on Extraversion, Negative Affect and Orienting Sensitivity. Mother-infant temperament dissimilarity was marginally correlated to their learning Valence, with the most similar dyads achieving the highest (most positive) learning Valence ($r=-0.29$, $p=.058$).

ii. Maternal attunement/sensitivity

Maternal attunement/sensitivity was scored on a scale of 1-5 by video coding of maternal behaviour toward her infant during the observation phase. This scoring took into account maternal responsiveness (e.g. whether she acknowledged if her baby made a gesture or noise, whether she imitated her infant such as smiling when the baby smiled, and whether she adjusted her behaviour in response to her baby's mood), praising her infant, and seeking/maintaining eye contact. Scoring was conducted independently by two coders (inter-rater reliability = 0.7), and the average of their scores was used. Maternal attunement was not significantly correlated to learning Valence ($r=-.02$, $p=.90$).

iii. Speech quantity

Maternal speech quantity was assessed by taking the mean duration of maternal utterances over positive and negative observation phases. There was a significant correlation between speech duration and learning Valence ($r=-.36$, $p<.05$) whereby shorter utterances were associated with higher (more positive) learning Valence.

iv. Speech quality (“IDS-ness”)

Maternal speech quality (or “IDS-ness”) was assessed by taking an index of the mean intensity and pitch over the entire observation phase for each trial. There was no significant correlation between speech quality and learning Valence ($r=.24$, $p=.11$).

When these four maternal variables were entered as predictors into a multiple regression model (best subsets method) taking the intra-infant PLV index (which predicts learning Valence) as the dependent variable, they collectively explained 43.3% (adjusted R^2 ; Model $F(4,38) = 9.04$, $p<.0001$) of variation in intra-infant neural connectivity across participants. As summarised in Table S3, intra-infant neural connectivity was significantly *negatively* predicted by maternal speech duration ($\beta=-.50$, $p<.001$) and temperament dissimilarity ($\beta=-.29$, $p<.05$) as well as marginally *positively* predicted by maternal sensitivity ($\beta=.22$, $p=.07$). More succinct (shorter) maternal utterances, higher temperament similarity

between mothers and infants, and greater sensitivity in maternal behaviour predicted positive learning Valence by infants. However, maternal speech quality was not a significant predictor of Valence ($\beta=.06$, $p=.64$).

<i>Predictors</i>	<i>t</i>	<i>p</i>	<i>Beta (β)</i>	<i>St.Err.β</i>
Maternal speech quantity	-4.06	0.000	-0.50	0.12
Maternal speech quality	0.47	.64	0.06	0.12
Temperament dissimilarity	-2.38	.023	-0.29	0.12
Maternal sensitivity	1.85	0.07	0.22	0.12

Table S3. Results of multiple regression analyses showing the effect of maternal variables on intra-infant neural connectivity