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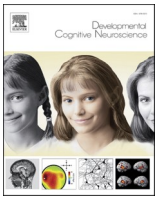

From movement to action: An EEG study into the emerging sense of agency in early infancy

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ABSTRACT

Research into the developing sense of agency has traditionally focused on sensitivity to sensorimotor contingencies, but whether this implies the presence of a causal action-effect model has recently been called into question. Here, we investigated whether 3- to 4.5-month-old infants build causal action-effect models by focusing on behavioral and neural measures of violation of expectation. Infants had time to explore the causal link between their movements and audiovisual effects before the action-effect contingency was discontinued. We tested their ability to predict the consequences of their movements and recorded neural (EEG) and movement measures. If infants built a causal action-effect model, we expected to observe their violation of expectation in the form of a mismatch negativity (MMN) in the EEG and an extinction burst in their movement behavior after discontinuing the action-effect contingency. Our findings show that the group of infants who showed an MMN upon cessation of the contingent effect demonstrated a more pronounced limb-specific behavioral extinction burst, indicating a causal action-effect model, compared to the group of infants who did not show an MMN. These findings reveal that, in contrast to previous claims, the sense of agency is only beginning to emerge at this age.

1. Introduction

As adults, we take our sense of agency — the feeling of controlling one's actions and their consequences (Haggard and Chambon, 2012) — for granted and are readily able to predict the causal effects of our actions. However, it is unknown how infants come to experience their own agency and understand that their movements have consequences. This capacity allows for better causal learning (Lagnado and Sloman, 2002), self-other distinction (Jeannerod, 2004; Tsakiris et al., 2007) and social and moral interactions (Caspar et al., 2016; David, 2012). On an even more basal level, developing a sense of agency and using one's body to achieve goals means that infants can learn to use their movements to perform coordinated, intentional actions.

Previous research suggests that sensitivity to sensorimotor contingencies is present in early infancy (Rochat and Striano, 1999; Watanabe and Taga, 2006) and possibly already in utero (Myowa-Yamakoshi and Takeshita, 2006; Zoia et al., 2007; for review see Fagard et al., 2018). Furthermore, in an fNIRS study, Filippetti et al. (2014) found that infants at 5 months of age show specific cortical processing for body-related contingent versus non-contingent stimuli. Behaviorally, infants have been shown to increase the movement frequency of an action that yields an effect, which has previously been taken as evidence for the early presence of a sense of agency in infants as young as 2 months of age (Gergely and Watson, 1999; Rochat and Striano, 2000; Watanabe and Taga, 2011). Recent computer simulation work, however, has shown that this behavioral pattern does not provide evidence for the presence of an underlying causal model, which is required for the sense of agency (Zaadnoordijk et al., 2018). The increase in movement frequency could be produced by a simulated agent that had a cognitively plausible learning mechanism but, by design, did not have the capacity to learn causal relations. That is, the behavior can be caused both by mechanisms that enable the infant to learn causal relations and by mechanisms that do not. However, we do not know which of these two types of mechanisms is underlying infants' behavior. As such, the

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behavioral pattern cannot be considered sufficient evidence to conclude that infants have learned the causal relation. In order to reach this conclusion, evidence must be presented that cannot be explained by a mechanism unable to learn causal relations. In this study, we set out to investigate whether 3- to 4.5-month-old infants build causal action-effect models. Based on results of the simulation work, we focused on the neural and behavioral response following the *discontinuation* of an audiovisual action effect. We investigated the presence of a violation of expectation, as this indicates that infants made a prediction regarding the consequences of their movements, i.e., built an internal model.

Three- to 4.5-month-old infants were tested in a computerized version of the mobile-paradigm in which movement of one of the infant's limbs causes an audiovisual effect (Rovee and Rovee, 1969). Infants were seated in a car seat in front of a computer screen during three phases of the experiment. During the first phase ('baseline'), they were presented with a static visual display. During the second phase ('connect'), infants' arm movements triggered an audiovisual animation. During the third phase ('disconnect'), the action-effect contingency was discontinued, and infants again saw only the static display. We analyzed movement frequency patterns over the three phases as well as the mismatch negativity (MMN) event-related potential (ERP) in the electroencephalogram (EEG) data. EEG has previously been used to study infants' neural body maps, that is, the development of somatotopic representations in the brain (e.g., Saby et al., 2015; Meltzoff et al., 2019), but has not yet been extended to the context of the sense of agency in early infancy.

In line with previous empirical findings (Heathcock et al., 2004; Rovee and Rovee, 1969; Rovee-Collier et al., 1978; Watanabe and Taga, 2006, 2011), we expected to see an increase in movement frequency during the connect phase as an indication that the infants detected the contingency. In contrast to earlier research, we were especially interested in the neural and behavioral response following the cessation of the audiovisual effect — if infants had built expectations about the causal relation between their movements and the audiovisual effect, we expected to find evidence for a violation of expectation (an MMN) in the EEG data upon movement during the disconnect phase compared to the baseline phase. The input was identical for both these phases (i.e., a static picture) but in contrast to the baseline phase, the disconnect phase was preceded by a learning phase (the connect phase). Furthermore, during the disconnect phase, infants were expected to initially increase their movement frequency to gather information about the new state of the world and update their model, then decrease the movement frequency once more once the infants' internal model updated anew (i.e., an extinction burst).

2. Methods

2.1. Open research practices

The anonymized raw and preprocessed data as well as the experiment can be obtained here: http://hdl.handle.net/11633/aabrg7pr.

2.2. Participants

Sixty-five full-term infants (M_{Age} = 115.06 days, SD_{Age} = 12.47; 29 male) were tested in the Baby EEG Lab at the Donders Centre for Cognitive Neuroimaging (DCCN), Nijmegen. Parents gave written consent. Participation was rewarded with age-appropriate books or monetary compensation. Ethical approval for the project was granted by the regional medical ethical committee, Commissie Mensgebonden Onderzoek (CMO) regio Arnhem-Nijmegen (NL39352.091.12, CMO 2012/012).

2.3. Materials

2.3.1. Apparatus

Movement and EEG data were recorded concurrently. Four accelerometer bracelets were attached to the infants' limbs. The triggering bracelet that controlled the audiovisual effect was fastened around one of the wrists (counterbalanced across participants). EEG was recorded from 32 active Ag/AgCl electrodes referenced online to the left mastoid (TP9), using infant-sized caps (ActiCAP) following the international 10–20 system. Data were sampled with a Brain Amp DC amplifier via Brain Vision Recorder Software (Brain Products GmbH, Germany) with a sampling frequency of 500 Hz.

2.3.2. Stimuli

Infants were presented with a colorful image of a mobile toy against a black background on a computer screen. During the baseline and disconnect phases, the image remained static on the screen. In the connect phase, an animated version of the mobile toy with a simultaneous bell-like auditory stimulus was triggered upon movement of the infant's trigger arm. The animation lasted 650 ms and was assembled from 44 rotated versions of the static image (ranging between -10° and 10°) using the visual processing software Virtual Dub 1.10.4. The stimuli were presented via Presentation software (Neurobehavioral Systems; http://www.neurobs.com/).

2.4. Design and procedure

After briefing the parent(s), infant was capped and electrode impedances were checked in an experimental room designed to minimize external noise and electromagnetic interference. To mitigate parental interference while at the same time maintaining the infants' proximity to their parent, the infant was placed in a baby car seat (Maxi-Cosi) on the parent's lap, approximately 50 cm away from a computer screen. The parent was instructed to remain passive throughout the task. The four accelerometers were secured around the infant's wrists and ankles. Once the infant accommodated to this set-up, the experiment was initiated. The image of a mobile toy was presented across the three phases in a fixed, uninterrupted sequence – baseline, connect, and disconnect. During the baseline and disconnect phases (2 min each), the image was static. In the connect phase (3.5 min), movement of the trigger arm elicited the audiovisual effect (Fig. 1).

The experiment ended after the three phases had elapsed or if the infant repeatedly showed signs of fussiness or discomfort. The parents were then debriefed and compensated. A complete experiment lasted 7.5 min and a full testing session lasted approximately an hour.

2.5. Data acquisition

2.5.1. Movement frequency recordings

Movement was registered for each limb whenever the change in the limb's velocity exceeded a threshold value that was kept constant across infants. The threshold's sensitivity level was based on pilot data and was adjusted as to minimize the measurement noise resulting from head or torso movements, yet allowing infants in this age group to easily set off the audiovisual effect. Above-threshold movements were logged as count data at 20 Hz.

2.5.2. EEG recordings

Data were sampled at 500 Hz, applying 0.016 Hz high-pass and 125 Hz low-pass filters online. We strived to keep the impedances below 50k℧. Because infants were lying in the baby seat, we were often unable to obtain clean signals from the occipital and parietal sites, making an averaged reference inappropriate (Trainor et al., 2003). Relative to the mastoids, the MMN response appears negative at frontal sites (Trainor et al., 2001); furthermore, the identification of adult MMN responses has shown to be more robust in mastoid-referenced data (Walker-Black and

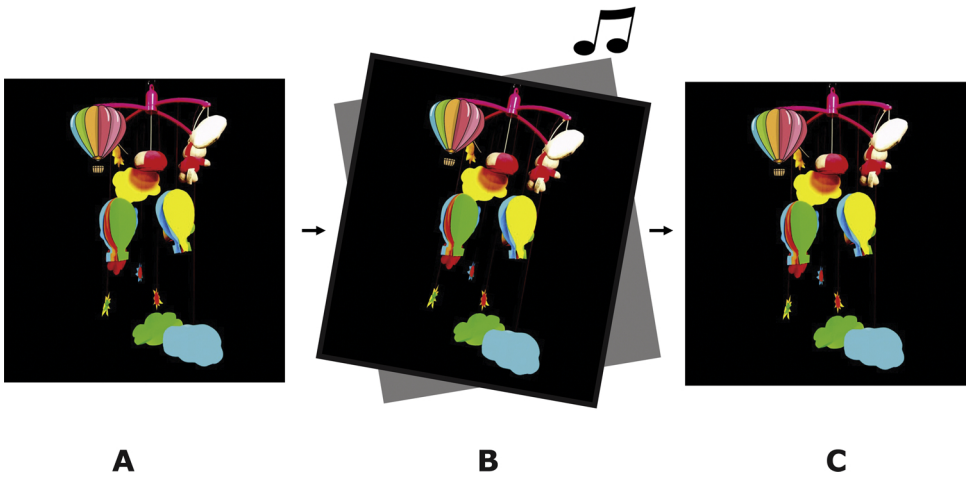

Fig. 1. The visual stimuli used in the three phases. **A**. In the baseline phase, which lasted 2 min, the static image was shown. **B**. In the connect phase, which lasted 3.5 min, the image wiggled, and a sound was played when the infant moved the trigger arm. **C**. In the disconnect phase, which lasted 2 min, the static image was shown again.

Stuart, 2008). Thus, the signal was re-referenced offline to the mastoid average (TP9, TP10).

2.5.3. Video recordings

Experimental sessions were filmed to monitor the experimental process on-site.

2.6. Data preparation and analyses

Behavioral data were pre-processed in Excel (Microsoft Office Professional Plus 2013). Statistical analyses were performed using IBM SPSS Statistics, Version 21.0. EEG data pre-processing and analyses were done using the open-source Matlab toolbox Fieldtrip (Donders Institute for Brain, Cognition, and Behavior; http://www.fieldtriptoolbox.org/, Oostenveld et al., 2011).

2.6.1. Movement frequency data

The experiment was segmented into 45 time bins by computing the movement frequency over 10-second intervals. We opted for this timescale because treating repeated measures as a continuous outcome increases the chances of detecting growth effects (Kwok et al., 2008), such as the linear and quadratic trends we were primarily interested in. Moreover, the finer-grained the scale, the more data points can be preserved per infant. All infants who completed the connect phase were included in the analyses; at this cut-off point a participant's behavioral response pattern could offer a meaningful contribution to the parameter estimation.

The behavioral data were modeled using multilevel time series analyses, which is an especially suitable technique for hierarchical data structures, such as movement series nested within infants (Vossen et al., 2011). Even more crucial for dealing with infant data, multilevel analyses rely on likelihood-based estimations and thus can handle missing data without requiring list-wise deletion, resulting in a considerable power gain (Kwok et al., 2008; Vossen et al., 2011). The choice of multilevel modeling was further motivated by the observed variance-component coefficient (VCC) of 0.41, indicating that almost half of the variability in movement frequency over time was found at the between-individual level.

To distinguish between the within-infant and between-infant differences in outcome trajectories over time, a multilevel model partitions the variance into a fixed and a random component. Fixed effects estimate a single population parameter (e.g., the mean movement frequency at baseline), whereas random effects describe the random probability distribution around that fixed effect for each infant (e.g., the variance of the baseline movement frequency) (Curran et al., 2010). The appropriate modeling of the random part is beneficial to the valid estimation of the fixed model part (Jacqmin-Gadda et al., 2007), as well as quantifying the descriptive quality of the model by estimating the amount of variation at each level that is still unexplained after taking into account the specified model.

In the fixed model parts, we introduced 1) dummy predictors to assess the main effect of each phase, and 2) time by phase interaction terms testing for linear and quadratic trends during each phase. All predictors were centered with respect to the arithmetic mean prior to computing the quadratic terms and were therefore orthogonal.

In all of our models, the random part combined a random intercept to capture the residual between-subject variation in movement frequency with an Autoregressive Moving Average Error (ARMA(1,1)) to model the within-subject residual variation over time. ARMA(1,1) matches the autoregressive structure inherent to most time series data as it assumes that behavior at adjacent time points would correlate more strongly. By allowing individual intercepts to vary randomly, the model accounts for individual differences in movement frequency, i.e., that behavior within the individual tends to be more similar than behavior across individuals. ARMA(1,1) was chosen as it is the most general covariance structure for within-subject variation that can be combined with a random intercept and is still parsimonious in the presence of a large number of repeated measures within individual (Nentjes et al., 2015; Vossen et al., 2011).

The first model looked at phase-specific changes in movement frequency over time, while allowing this change to be relative to each infant's own intercept. The analytic strategy was to 1) saturate the fixed model part, 2) remove the non-significant predictors backwards unless that would lead to a significant deterioration in model fit or parsimony. Predictors were pruned hierarchically (i.e., from second-order polynomials to main effects). As all models shared the same random part, they were estimated using the Maximum Likelihood (ML) method in order to be compared with Likelihood Ratio tests in terms of their model fit to parsimony. This way, we could also evaluate the effect of imposing constraints in the fixed part at each step. The final model was re-estimated with the Restricted Maximum Likelihood (REML) method to obtain the unbiased covariance estimates (Browne and Draper, 2006).

2.6.2. EEG data

Each movement of the trigger arm was considered a trial; thus, the onset of each trial was defined by a marker sent to the EEG system upon trigger arm movement as detected by the accelerometers. The MMN analyses and artifact rejection were done on the frontal sites (F3, F4), where the MMN's morphology has shown to be most pronounced in 3- to

4-month-olds (He et al., 2007; Trainor et al., 2003). A 0.5–20 Hz bandpass filter was applied and the mean signal of each trial was subtracted from the data. The continuous output was segmented into 600-ms movement-locked epochs, including a 100-ms pre-movement baseline (henceforth PMB to disambiguate the pre-movement EEG baseline from the experiment's baseline phase); correction was set at the mean amplitude over the PMB. High-amplitude artifacts were rejected manually; as a general rule of thumb, trials with measured activity exceeding 50μV during the PMB and 150μV during the epoch were rejected.

All individual datasets with at least five artifact-free trials in the baseline and in the disconnect phase entered the analysis. 16 out of 22 infants had more than 20 trials for each phase, and apart from one infant who had eight artifact-free trials for one phase, all participants contributed at least 10 trials per phase. Baseline and disconnect trials were averaged separately across participants. To form difference waves, the averaged disconnect phase waveforms were subtracted from the averaged baseline phase waveforms. The mean amplitudes for the 200-350-ms window, within which we expected the MMN-response (Basirat et al., 2014; Trainor et al., 2003), were derived from the averaged signal over 20-ms data segments. A one-tailed paired *t*-test checked for waveform differences between the two phases.

To rule out the alternative of the group-level result being caused by the averaging of two distinct ERP morphologies as reported by Trainor and colleagues (2003), infants' MMN responses were classified either in the positive waveform group, or in the MMN group. The split was based on the deflection of the averaged measured activity over the window of interest. The groups were then used as a predictor in the neurobehavioral analysis.

2.6.3. Neurobehavioral model

It was of primary interest to assess the extent to which the behavioral patterns were consistent with the neural mismatch responses pattern. Thus, this analysis focused only on infants from whom we had obtained sufficient data for the EEG analysis, as well as movement frequency data. Furthermore, we were interested in testing for movement specificity and whether its presence can be related to the infants' deflection of the ERP waveform. Thus, we extended the behavioral model into a growth model. This allowed us to assess the interaction of ERP waveform group (positive or negative) and phase-specific behavioral patterns (Curran et al., 2010; Kwok et al., 2008). Moreover, we assessed to what extent the behavioral patterns were observed differentially for the trigger arm compared to the other arm since limb specificity indicates that movement frequency increase is not merely caused by arousal.

3. Results

3.1. Behavioral analysis

Thirty-six infants completed the connect phase and were included in the behavioral analysis (M_{Age} = 117.56 days, SD_{Age} = 12.18 days). An additional 29 infants were excluded from the analysis due to excessive crying before the end of the connect phase (21 infants), falling asleep (2 infants) or technical errors (6 infants).

Averaging the movement of all limbs, infants moved more during the disconnect phase relative to baseline and connect phases (B = 15.04, SE = 2.90, t(60.774) = 5.19, $p < 0.001$, 95 % CI [9.24, 20.84]), but the baseline and connect phases did not differ significantly in terms of mean movement frequency (B = -1.15, SE = 2.13, t(154.523) = -0.54, p = 0.592, 95 % CI [-5.35, 3.06]). During the connect phase, infants responded to the contingent effect by linearly increasing their overall movements (B = 0.65, SE = 0.19, t(206.5) = 3.45, p = 0.001, 95 % CI [-0.28, 1.02]). However, the infants' behavioral response to the absence of the audiovisual effect did not follow the predicted quadratic trend in the disconnect phase (B =. −12, SE = 0.09, t(382.82) = −1.29, p = 0.20), suggesting no evidence for a group-level extinction burst. These findings match the results obtained when re-running the analysis with limb specificity (trigger arm versus contralateral arm) as additional factor. Limb specific behavior would indicate that increased movement frequency is not caused by mere arousal. In the limb-specificity analysis, an additional marginally significant difference in mean movement frequency during the connect phase was found for the trigger arm compared to the contralateral arm (B = 0.67, SE = 0.34, t(1936.62) = −1.95, p = 0.051, 95 % CI [-1.35, 0.00]). However, the corresponding random effect was also significant, indicating substantial variation across infants' response patterns. See Fig. 2 for the movement frequency patterns.

3.2. ERP analysis

Twenty-two infants had sufficient data for the ERP analysis (see Methods for criteria; M_{Age} = 116.68 days, SD_{Age} = 14.52). The total number of artifact-free trials was 956 during the baseline phase (M_{Trials} = 43.45, SD = 26.30) and 1193 during the disconnect phase (M_{Trials} = 54.23, SD = 30.99). No significant MMN component was found 200−350 ms after onset (t(21) = −1.05, p = 0.847, one-tailed; Fig. 3), the expected time window for infants of this age (latency based on Basirat et al., 2014; Trainor et al., 2003). Thus, as a group, the 3- to 4.5-month-old infants showed no evidence of differential neural processing during the disconnect and baseline phases.

However, although the exact biological or cognitive causes are not yet known, infant ERPs have been shown to undergo changes during development, such as decreases in latency (de Haan, 2013) and increases in amplitude (e.g., sensory-evoked potentials like N1 and P1; Wunderlich and Cone-Wesson, 2006). Also, previous research suggests that, especially in the first months of life, the direction of ERP deflections can change due to brain maturation (de Haan, 2013; Thierry, 2005). Importantly, Trainor and colleagues showed that infants between 2 and 6 months of age transition from showing a positive waveform to an adult-like negative MMN in a mismatch paradigm (Trainor et al., 2003). Based on these findings, we explored whether our sample consisted of some infants showing a negative and some infants showing a positive deflection, leading to the appearance of no MMN overall. Following Trainor and colleagues (2003), we split our sample into two groups based on the mean amplitude of the difference wave in the time window of interest (see Fig. 4A and Fig. 4B). The mismatch negativity subgroup consisted of ten infants (M_{Age} = 113.40 days, SD = 13.95), yielding 460 artifact-free trials during the baseline phase (M_{Trials} = 46.00, SD = 26.19) versus 494 trials in the disconnect phase (M_{Trials} = 49.40, SD = 28.54). The positive waveform subgroup included twelve infants (M_{Age} = 119.417 days, SD = 15.01), with 496 baseline trials (M_{Trials} = 46, SD = 26.19) and 699 disconnect trials (M_{Trials} = 58.25, SD = 33.58). The average ERP of the mismatch negativity subgroup showed a clear MMN morphology, whereas the positive waveform subgroup exhibited no clear ERP. The two subgroups did not differ in age (t = 0.966, p = 0.346). We did not perform any statistical tests on the resulting waveforms after splitting the groups to avoid circular statistical analysis. Instead, we re-analyzed the behavioral data using the ERP group as a predictor, as described below.

3.3. Neurobehavioral analysis

After finding two mismatch response profiles in the electrophysiological data, we were specifically interested in testing whether these two subgroups showed any differences in their movement patterns. In particular, we investigated differences in the movement patterns between trigger arm and contralateral arm. To estimate potential differences between the subgroups, we tested for cross-level interactions in a model with ERP deflection predicting movement pattern and limb specificity.

The mean movement frequency per arm in the connect (B = 2.46, SE = 0.70, t(1154.72) = 3.52, $p < 0.001$, 95 % CI [1.09, 3.83]) and

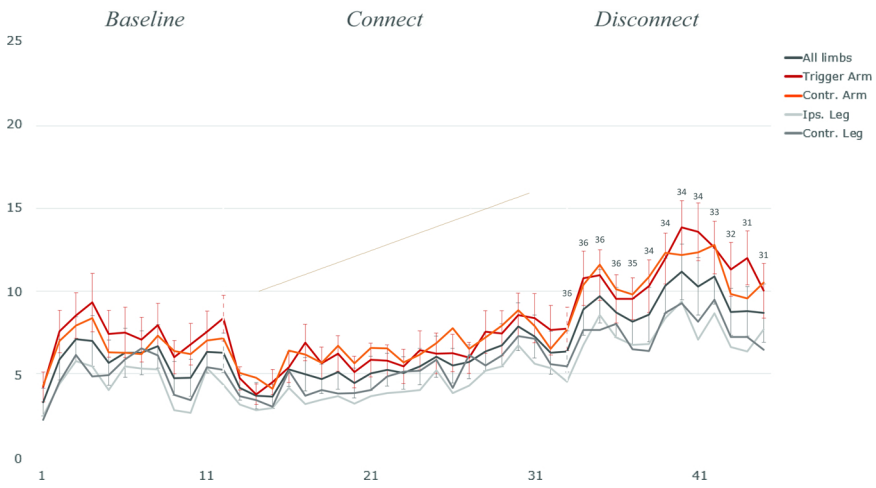

Fig. 2. Movement frequency over time binned in 10-second segments. Until the disconnect phase, 36 infants are included; the number of infants after that point are indicated in the figure. The average movement behavior over all limbs is indicated in black. Additionally, the movement behavior of each individual limb (trigger arm, contralateral arm, and the legs ipsilateral and contralateral to the trigger arm) is shown in the plot. Error bars, shown for the average of all limbs and for the trigger arm, reflect one standard error around the mean.

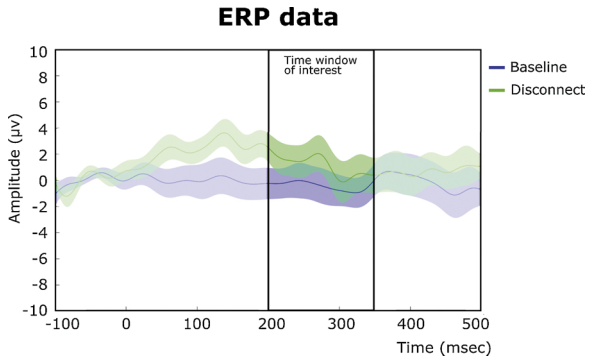

Fig. 3. ERP results of the MMN analysis (shaded area reflects one standard error around the mean). We found no statistical evidence for a difference between the disconnect and baseline phases in the time window of interest (200−350 ms after trigger arm movement).

disconnect ($B = 5.45$, $SE = 0.96$, $t(1157.44) = 5.71$, $p < 0.001$, 95 % CI [3.57, 7.32]) phases was conditional on the deflection of the mismatch response, such that the trigger arm moved more frequently than the contralateral in the mismatch negativity relative to the positive waveform group. The difference per arm in the shape of the extinction burst during the disconnect phase was also conditional on the deflection of the mismatch response ($B = 0.26$, $SE = 0.06$, $t(1248.13) = 4.32$, $p < 0.001$, 95 % CI [0.14, 0.39]); that is, the movement of the trigger arm followed a more pronounced limb-specific extinction burst relative to that of the contralateral arm in the mismatch negativity compared to the positive waveform group (Fig. 4). Crucially, there was no significant difference between the groups regarding the linear increase during the connect phase ($B = -0.24$, $SE = 0.12$, $t(1183.76) = -0.21$, $p = 0.836$, 95 % CI [−0.25, 0.20]).

3.4. Exploratory analyses

The difference between the groups in number of movements in the connect phase could potentially be an explanation for the group differences in the disconnect phase. A Pearson correlation between the number of movements in the connect phase and the mean amplitude of the difference wave between the baseline and disconnect phase (by which the groups were determined) revealed no significant effect ($r = -0.120$, $p = 0.595$), indicating that number of movements by itself could not explain our group differences. Since the positive waveform group still moved on average 152 times in the connect phase, it seems they had ample learning opportunity.

We chose an audiovisual effect to maximize infants' processing of the auditory stimulus (Hyde et al., 2010) while at the same time providing infants with sufficient information to extract the action-effect contingency without the need to look at the screen. Still, to ensure that attention to the screen did not bias our results, we analyzed the videos of each group. Our video analysis (in which we coded for turning away from the screen and closed eyes for longer than a second) showed that there was no significant difference between the two groups ($t(20) = 1.366$, $p = 0.187$, two-tailed).

4. Discussion

In this study, we investigated whether 3- to 4.5-month-old infants build a model of the effects of their own movements, a crucial prerequisite for the sense of agency. We obtained electrophysiological and behavioral measures to inform us about infants' action-effect models and in particular about infants' violation of expectation upon discontinuation of a sensorimotor contingency. We hypothesized that if infants built a causal action-effect model, we would observe this in the data in two ways: a mismatch negativity response in the electrophysiological data, and an extinction burst, a temporary additional increase and then decrease in movement frequency, after the effect was discontinued. We found that only a subset of infants showed a mismatch negativity response to a violation of expectation of the action consequences, and thus establish the causal connection between their actions and the consequences of their actions. Notably, these infants also had a greater extinction burst for the arm that triggered the effect as compared to the contralateral arm, indicating that these infants had built not only built a causal action-effect model, but also had learned which specific limb triggered the effect. The other infants did not show an electrophysiological violation of expectation. Moreover, they did not demonstrate limb specificity during the disconnect phase. The exploratory analyses do not provide an indication that the results are based on a difference in learning opportunities. Rather, our results are better explained by the sense of agency not yet being present in the positive waveform group, causing this group of infants not to build a causal action-effect model regardless of the learning opportunities. Therefore, we suggest that the group differences may reflect a difference in the development of the sense of agency. In sum, these findings suggest that not all infants were able to build an action-effect model, and thus that the sense of agency is still emerging in infants between 3 and 4.5 months of age.

Since infants increase their movement frequency when a movement produces an effect, researchers have previously suggested that a sense of agency is present from 2 months of age (Rochat and Striano, 1999, 2000; Watanabe and Taga, 2006). This behavior was replicated in the current study, as infants showed a linear increase in movement frequency in the

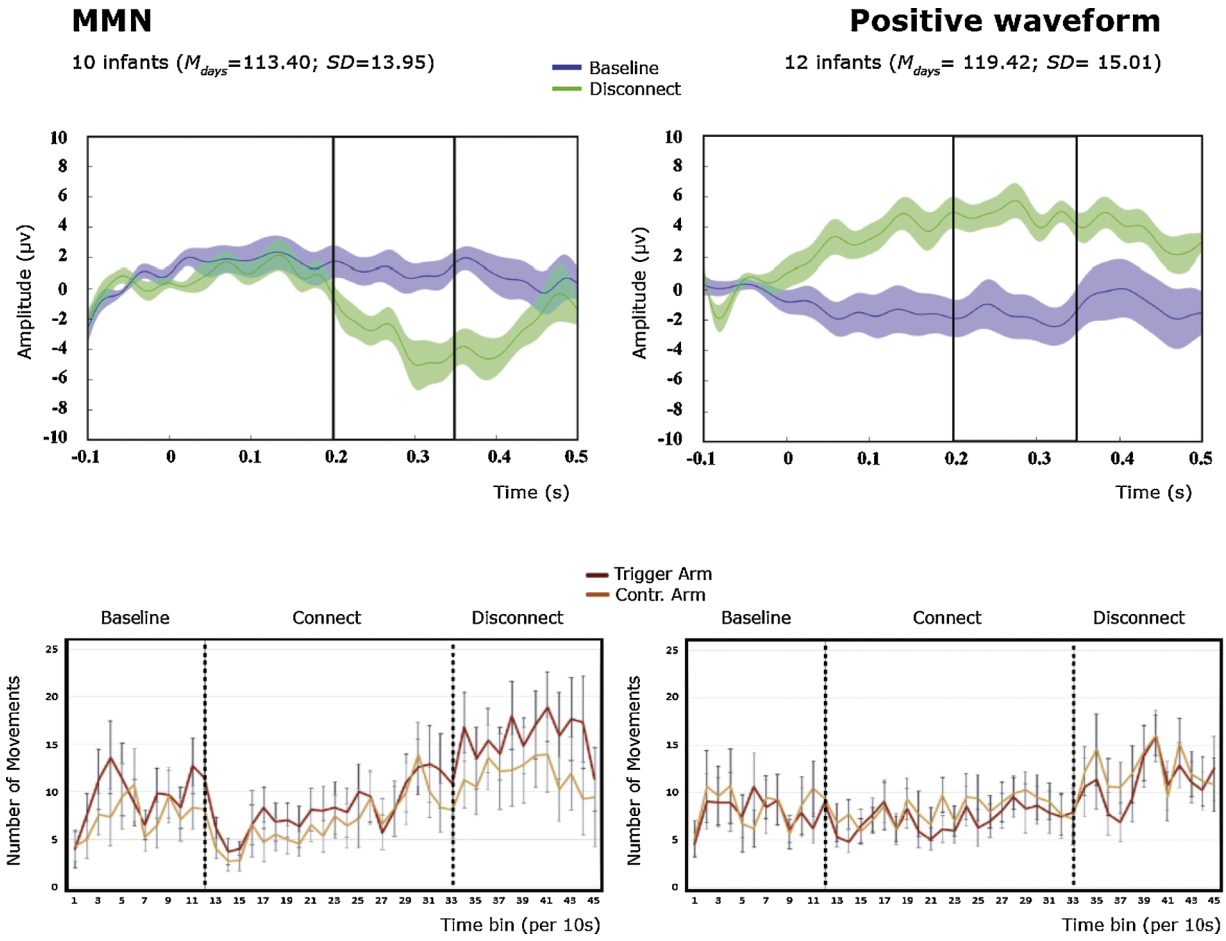

Fig. 4. The ERP waveforms per group and their corresponding behavioral movement frequency patterns (one standard error around the mean indicated by shaded area (ERPs) and bars (behavioral data)). **A**. ERP waveform of the group of infants with a negative mean deflection in the time window of interest (MMN group). **B**. ERP waveform of the group of infants with a positive mean deflection in the time window of interest (positive waveform group). **C**. Behavioral patterns of the three phases for the MMN group. **D**. Behavioral patterns of the three phases for the positive waveform group.

connect phase. Computer simulation research, however, has demonstrated that an underlying causal model cannot be inferred from this behavioral pattern (Zaadnoordijk et al., 2018). This was why, in contrast to previous research, our focus was on the violation of expectation, as this indicates that infants have made a prediction regarding the consequences of their action and thus have built an internal model. In previous work on infants' abilities to predict the consequences of their own actions, 10-month-olds were presented with a visual stimulus upon pressing a button (Kenward, 2010). The infants were able to make anticipatory fixations towards the location on the screen before they pressed the button and the effect appeared. Building on these ideas, our study is the first to obtain both behavior and EEG data to show that the ability to predict the consequences of one's actions emerges between 3 and 4.5 months of age.

Due to the developmental properties of the MMN, and in line with previous research (Trainor et al., 2003), we split our sample based on the mean amplitude of the EEG signal during the time window of interest. This procedure gave us important insights into the emergence of the sense of agency, as it revealed the underlying interactions between the neural signal and infants' behavior. Splitting our sample according to the direction of deflection of the individual's averaged measured activity may have introduced some individual misclassifications. Crucially, however, the resulting waveforms — a global drift in the positive waveform group and an ERP-like waveform in the mismatch negativity subgroup — are a direct consequence of our design and not an artifact of our analysis. The two observed waveforms are in line with the latency and morphology reported by others studying the MMN in this age group, who also find a split across infants in positive and negative amplitudes (e.g., Trainor et al., 2003). Moreover, the observed neural processing differences translated to a specific behavioral difference, evident from the limb specificity found in the mismatch negativity but not in the positive waveform subgroup during the extinction burst in the disconnect phase.

It remains an open question whether the infants in the positive waveform group did not detect that their movement caused the audiovisual effect, or whether they built an incorrect causal model (e.g., a model in which another limb caused the effect). An additional open question is which factors influence the development of a sense of agency. The infants who displayed a violation of expectation and a limb specific extinction burst may be further in their general development or may have specific cognitive advantages. Since the sense of agency emerges in all infants eventually, we expect that the split in neural and behavioral data disappears and becomes similar to the MMN group as infants grow older. Similarly, we expect that a group of much younger infants would react more similar to the positive waveform group. Future research could confirm or disprove these hypotheses as well as address the open questions.

The question regarding which patterns of behavior demonstrate an underlying causal model is not only within the purview of infant development; animal behavior researchers also debate this topic. Comparable to the results of computer simulation work (Zaadnoordijk et al., 2018) showing that behaviors previously taken as evidence for an underlying causal model could be explained with a simpler model-free mechanism, Taylor and colleagues (2014, 2015) have argued that

complex tool-use does not necessarily indicate capacity for causal reasoning. Crows were shown to be unable to produce causal interventions and thus lack certain causal learning capacities, even though the ability for causal interventions had previously been taking to underlie complex tool use (Taylor et al., 2014). Thus, the underlying mechanisms of behavioral data, rather than their complexity or intuitive explanation, provides a measure of causal model building capacities of human and non-human populations.

In the present study, we shed light on infants' ability to learn causal relations between their actions and the subsequent consequences, by complementing behavioral data with neural measures. This combination allowed us to show that behavior alone is not sufficiently nuanced for assessing the developing sense of agency. While we replicate previously found behavior that has been taken as indicative of a sense of agency, the electrophysiological data demonstrate — and thereby confirm theoretical research (Zaadnoordijk et al., 2018) — that this is a flawed interpretation. We do not find an interaction between subgroup and linear increase in the connect phase. Since researchers in previous studies only acquired behavioral data, it was impossible to determine whether infants experienced a violation of expectation when the causal connection was manipulated. The EEG results indicate that only a subset of 3- to 4.5-month-old infants built an action-effect model, and thus that the sense of agency is emerging but not evident for all infants in this age group. Our findings therefore demonstrate limitations of the previous claims about infants' sense of agency and the evidence by which these claims were justified. By going beyond the behavioral data, we were able to demonstrate for the first time how infants' movements turn into actions as sense of agency emerges.

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Declaration of Conflicting Interest

The authors have no conflicts of interest with respect to the authorship or the publication of this article.

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