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Sense of agency in infancy:
Testing for causal model building

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Abstract

The sense of agency has been described as the experience that is sensed when we performed our own actions, such that we control events in the outside world through them. The development of sense of agency is crucial for children to be able to learn from their experience and interaction with the surrounding world. To date, evidence of a sense of agency in infancy is limited to a behavioral data pattern. However, a simulation with no capability to build internal model was able to replicate a similar data pattern. Therefore, additional investigation on different phenomena is necessary. It is assumed that to experience a sense of agency, an internal model is required. Having an internal model would enable one to make predictions upon it. When there is a mismatch between the predicted events with the actual events, a mismatch response in terms of the brain's event related potential is elicited. In this study, we proposed to investigate the presence of sense of agency in infants based on how the brain would react to a violation of its internal model. In this study, we investigated 3-to-4-month old infants' movement response along with the ERP response to movement in different experimental phases. There were three phases, namely baseline, connect, and disconnect. One of the infants' limb movement would trigger stimulus effect during the connect phase, whereas baseline corresponds to the phase before and disconnect corresponds to the phase after. The omission of the movement's effect was applied in the disconnect phase and aimed to violate infants' expectations. The results indicated infants experience violation of expectation in response to the omission of their movement effect, as shown in the ERP mismatch response. It indicates that infants built an internal model linking their movement with its effect, suggesting the presence of sense of agency.

Keywords: sense of agency, development, mobile paradigm, event related potential, mismatch response

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1. Introduction

We perform actions in our everyday life to accomplish our goals, to fulfill our needs, and to do our obligations. We open the window when the temperature in the room becomes too warm and we turn on the light when it is dark. By opening the window we know that the wind will enter the room and the room will become cooler. By turning on the lights we know that it will be brighter. Such experience that is sensed when we performed our own actions has been described as a sense of agency (Haggard & Chambon, 2012). Amongst various definitions, Haggard proposed sense of agency as "the experience of controlling one's own actions, and through them, events in the outside world" (Haggard, & Chambon 2012, p390).

Undoubtedly, having a sense of agency is important and its development is essential as it enables us to learn from our surrounding world. Understanding the causal structure between actions and their consequences enables children to learn and benefit from their interaction with the physical and social world (Lagnado & Sloman, 2002; Schulz, Gopnik, & Glymour, 2007; Rochat & Striano, 2000). It allows them to optimally adjust their actions to yield the best outcome based on their experience. Thus, developing a sense of agency may be considered crucial for human cognitive development in general (Zaadnoordijk et al., 2016).

Rochat and Striano (2000) propose that a sense of agency in infancy develops on the basis of interaction between sensory modalities, through systematic exploration of own bodies, self-produced actions, and observation of the resulting perceptual consequences. Some developmental psychologists have suggested that it may present at birth (Kalnins & Bruner, 1973; Lewis, Sullivan, & Brooks-Gunn, 1985; Siqueland & Delucia, 1969). In accord with the notion, Rochat and Hespos (1997) did a study on infants' rooting response to self- (cheek being touched by own hand) and external stimulation (touched by experimenter's hand). Despite there was no significant difference found in newborns' response to self- and external stimulation, they reported a significant result in the 4-week old infants' response. The 4-week old infants in their study were able to discriminate between conditions by showing more response to self-relative to external stimulation. They interpreted these findings as evidence that young

infants were able to dissociate intero- (self-) –ceptive from exteroceptive stimulation, indicating the capability to place themselves as a differentiated agent within the environment. (Rochat & Hespos, 1997). This capability to differentiate their own body based on interoceptive information from their hand movement and the tactile information from the cheek being touched is stated as an indication of sense of agency existence in newborns (Geangu, 2008). In line with this, a study conducted by Kalnis and Bruner (1973) reported that by the age 5 to 12 weeks infants significantly increase their sucking response when it is followed by a stimulus movie becoming more clear (Kalnins & Bruner, 1973). In fact, early research proposed that there is a simple form of sense of agency which present at birth and is the result of active discovery by the infants of the contingency between stimuli and proprioception of body movement (Guillaume, 1926).

However, Rochat and Striano (1999) argued that mere contingency learning is not enough to claim that newborns have a sense of agency. They suggested that experiencing a sense of agency presupposes circumstances where someone is able to do self-exploration as an agent and understand the causal link between own actions and the corresponding perceptual consequences (action effect). Based on that notion, they argued that there is a fundamental difference between contingency detection and the detection of a causal link between action and its perceptual consequences, as the latter presupposes systematic voluntary control performed by the infant (Rochat & Striano, 1999).

In line with that, there are several studies investigating sense of agency by probing infants' self-exploration in response to a perceptual consequence (Angulo-Kinzler & Horn, 2001; Watanabe & Taga, 2006, 2009, 2011). They investigated this by utilizing a mobile paradigm that was originally used by Rovee-Collier (1978) to study infants' memory retention. In their study, one of the 3-month-old infants' legs was connected to a hanging mobile toy (thus served role as the 'trigger' leg) in a way so that their movement would trigger the mobile movement (e.g. by tying a ribbon connecting infant's leg and a mobile toy in Rovee-Collier, 1978), providing an action's effect. The findings showed that 3-month-old infants significantly increased the kicking

frequency of the connected (trigger) leg (Watanabe & Taga, 2006, 2009). These findings has been replicated by several other studies (Angulo-Kinzler & Horn, 2001; Heathcock et al., 2005; Watanabe & Taga, 2011). The pattern of increased frequency was further interpreted as evidence that young infants were able to learn and memorize the causality links of their action and environmental change, leading them to adjust their movements accordingly (Watanabe & Taga, 2006, 2009, 2011). Further interpretation on this results has also been proposed as an evidence of early sense of agency (Watanabe & Taga, 2011; Kelso, 2016).

Nevertheless, the proposed evidence of early sense of agency was made purely based on interpretation of behavioral result (Watanabe & Taga, 2006, 2009, 2011), which leaves rooms for various alternative interpretations. One way to address this issue is to conduct further investigation based on the knowledge of how the sense of agency is experienced. In line with Rochat and Striano's (1999) notion that one of the indicators for sense of agency is an observed systematic voluntary control, the phenomena of sense of agency has been explained through an approach based on the theory of motor control. The theory of motor control suggested that motor control involves an internal model, namely forward models and inverse models. Forward models mimic the causal course of a process by making prediction of its next state. Inverse models invert the causal course by estimating the motor command responsible for causing a particular state transition (Wolpert et al., 1995). On a similar note, the assumption derived from cognitive phenomenology also stated experiencing a sense of agency presupposes an internal representation of events as being caused by own action (Bayne, 2008; Bayne & Levy, 2006). Therefore, investigation on the presence of internal model, linking actions with its effect, might provide a new insight into the research on sense of agency in infancy.

To our knowledge, there has been one study which investigate infant sense of agency based on the assumption of the presence of internal model (Zaadnoordijk et al., 2016). Zaadnoordijk and colleagues simulated infants' responses to a mobile movement through a computer program. By assuming that one needs and underlying internal (causal) model to experience sense of agency, they tested whether the behavioral data

patterns (i.e. increased movement frequency) seen in previous studies (Watanabe & Taga, 2006, 2009, 2011) is evidence for the presence of an underlying internal model. Rather than testing it by simulating an infant who built internal model, they did it in a reverse way by simulating a 'model free' infant with no internal model building capability. Their findings revealed that increase movement frequency could also be replicated in the simulation. To relate back to study done by Watanabe & Taga (2009, 2011), it seems that this increase in behavioral response due to stimulus feedback was not necessarily mean the infants built an internal model (Zaadnoordijk et al., 2016). Accordingly, Zaadnoordijk and colleagues (2016) argued that the observed change in infants' behavior is insufficient to claim the presence of a sense of agency. Consequently, it cannot be concluded that sense of agency is present in young infants based on these data patterns.

Nevertheless, the simulation work by Zaadnoordijk and colleagues (2016) opens another avenue to be explored further. They reported that the 'model-free' infant simulation may have been able to replicate the data pattern that has been taken as evidence for an underlying causal model, but did fail to replicate another behavioral effect. In Watanabe and Taga's study (2011, also in Watanabe & Taga, 2006, 2009; Rovee-Collier et al., 1978), it was reported that when the infants' connection to the mobile was severed, thus stopping the effect of their movement upon the mobile movement, infants increased their movement frequency for a period of time. These behavioral patterns was not replicated by the simulation (Zaadnoordijk et al., 2016). Further investigation on the presence of internal (causal) model in infants by focusing more at this part of the experiment (i.e. when infants' movement effect was omitted) might provide more information to address this issue.

Accordingly, this study investigated the presence of sense of agency based on how the brain would react to a violation of its internal model. Friston (2012) proposed that the brain actively making predictions based on the internal models. These predictions represent a state of neural acquired readiness to experience events with particular characteristics (Todd et al., 2012). Todd and colleagues (2012) suggested that prediction can be considered to be synonymous with expectation. If the prediction

outcome does not match with the current internal model, expectations are violated, then a prediction error occurs (Todd et al., 2012). Accordingly, prediction error signal pertaining to self-produced action's sensory consequences might then serves role as an indication of the existence of the internal models and infer the presence of sense of agency. Thus, we would focus current study on the prediction error response to the violation of infant's expectations on their action's effect.

There have been a lot of studies proving that prediction error could be measured in terms of event related potential (ERP) response, particularly a mismatch response (Baldweg, 2007; Trainor, 2012; Bendixen, 2012; Friston, 2012). One of those mismatch responses is the mismatch negativity (MMN; Baldweg, 2007; Bendixen, 2011). Normally, the MMN is elicited when a violation of an established pattern in sensory input is detected by the brain (Näätänen and Alho, 1997), and as such, in auditory perception studies, an MMN is usually elicited by a deviant tone amongst a series of standard tones (Todd et al., 2012; Trainor et al., 2003). Accordingly, Winkler and colleagues (1996) proposed that one of the functional role of MMN is to reflect the incorporation of recent change in the events by the brain to its representational model in order to minimize future prediction errors. In addition, the presence of mismatch response to deviant stimuli in infants has also been proposed as evidence of predictive processing in young infants, such that infants act as predictors, readily building up expectations for the upcoming events based on previous events (Trainor, 2012).

In current study, we investigated 3-to-4-month old infants' response to a mobile paradigm (modified) as used by Watanabe and Taga (2009, 2011, originally used by Rovee-Collier, 1978), combined with EEG measurement. To our knowledge, this is the first study that combined mobile experiment with EEG measurement. In contrast to what have been done by Watanabe and Taga (2009, 2011), present study focused more to infants' response following the omission of their movement's effect. This study will therefore address the following research question: do infants build a model of how their actions cause effects?

As what had been done in the mobile paradigm study (Watanabe & Taga, 2011), we implemented 3 phases in this study, namely baseline, connect, and disconnect. One of the infants' limb movement would trigger an audio-visual effect during the connect phase, whereas baseline corresponds to the phase before and disconnect corresponds to the phase after. The omission of the movement's effect was applied in the disconnect phase and aimed to violate infants' expectations. We measured the movement response in all phases along with the ERP response to movement in baseline and disconnect phase. For the behavioral analysis, in accord with findings from previous studies (Watanabe & Taga, 2006, 2009, 2011; Rovee-Collier, 1978), we hypothesize that there would be an increased movement frequency from baseline to connect phase, and from connect to disconnect phase. In addition, as observed by Watanabe and Taga (2006), we also expected to see more response of the arms (both the attached and opposite) relative to the legs. For the ERP analysis, we assumed that if infants predict their movement's effect based on previous events, once the effect was omitted, we expected to detect a mismatch response to the movement occurred during the 'disconnect' phase. Our hypothesis was that if the infants built an internal model of the causal link between their action and its effect during the connect phase, the mismatch response to events comprised of violation of expectation during the disconnect phase should be detected. Therefore, we expected to find a larger amplitude of mismatch response relative to the baseline phase, when the model was not present yet.

Based on literature, the MMN is obtained by subtracting the event-related potential (ERP) response to standard stimuli from those to deviant stimuli (Wacongne et al., 2012). In adults, the MMN appears as a negative deflection to the deviant sound which peaks over frontocentral regions of the brain around 100-200ms from the deviation point (Todd et al., 2012). However, since the MMN in infancy is still immature, the latency occurred a bit later. Alho and Cheour (1997), for instance, reported that in 3-month-old infant, the frontally distributed MMN started to appear at approximately 200ms after the stimulus onset. Other studies also reported to find frontally distributed MMN in infants at latency around 200ms-300ms (Kutzberg et al., 1995; Trainor et al., 2003; Basirat et al., 2014). Additionally, despite its name, several other studies (Trainor et al., 2003; Dehaene-Lambertz & Baillet, 1998; Dehaene-Lambertz and

Dehaene, 1994; Morr et al., 2002) reported to find mismatch response in infants in the positive direction at the same latency range. It was suggested that this inconsistency in the direction of the difference wave might be due to maturational process of the neuronal system responsible for eliciting such response (Trainor et al., 2003). Despite the inconsistency in the findings, we expected to find the mismatch response as negativity in the difference wave (i.e. response in disconnect being more negative relative to baseline). A mismatch response would serve as evidence of the presence of the internal model.

Additionally, unlike standard MMN studies, present study locked the ERP response to movement rather than a series of passively received standard and deviant stimuli. Movements in baseline phase were analogue to standard stimuli while movements in disconnect phase were analogue to deviant stimuli. By doing so, we aimed to test specifically the presence of internal model underlying sense of agency, i.e. the one that related to infants' expectation of their movement effect.

2. Methods

2.1 Participants

In total, 31 infants (22 females, mean age 117.6 days, SD=8.7) participated in this experiment. Data analysis was based on a final sample of 7 infants (3 females, mean age 123.85 days, SD=6). The remaining infants had to be excluded from the analysis due to technical problems (2), fussiness/excessive crying (13), insufficient amount of movement (1), experimenter error (1), or insufficient number of valid ERP trials (7).
The infants were recruited from a database of the Baby Research Center Nijmegen. This database contains contact information of families living in the surrounding area of Nijmegen who had shown interest to participate in a scientific experiments with their children. A small present or monetary compensation was given for participation. All parents were informed about the experiment and gave written consent before the experiment begun. Ethical approval for this research was granted by the local ethical committee.

2.2 Stimuli

Fig 1. Picture of mobile toy used as stimulus (static and video)

Infants were presented with a static image of a mobile toy during first phase and last phase of the experiment (Figure 1). The mobile toy image was used to make the experiment comparable to previous experiments performed by Rovee-Collier and colleagues (e.g. 1978) as well as Watanabe and Taga (2006, 2011) who used a real mobile toy. A static image of the mobile toy was used rather than a real mobile toy in order to avoid movement persistence effects caused by the physical characteristics of the real mobile toy.

An audiovisual stimulus, an animated version of the static image, was presented during the middle phase of the experiment. This animated version of the static image was a video showing a wiggling version of the image along with a sound. The animation was created using 44 rotated versions of the static image from -10^{o} to 10^{o} rotation. Those 44 pictures were then edited and cut to a length of approximately 650ms using visual processing utility Virtual Dub 1.10.4 to produce a final video stimuli (http://www.virtualdub.org/).

2.3 Materials

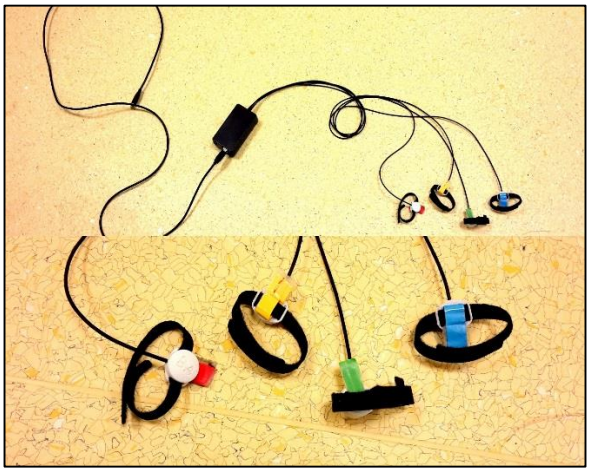

Fig 2. Color-coded accelerometers: target (yellow), opposite wrist (blue), ipsilateral leg (red), contralateral leg (green)

Similar to the experiment with a real mobile toy (Rovee-Collier et al., 1978), this experiment was also designed in a way such that infants' movements could trigger the audio-visual effect. This was done by using accelerometers as movement sensors for infants' limbs, i.e. one 'target' accelerometer and three additional accelerometers (Figure 2). During the experiment, the 'target' accelerometer was attached to one of the infant's wrists (counterbalanced across participants). The arm to which the 'target' accelerometer was attached is defined as 'trigger' arm from here on. Only the 'target' accelerometer served a function as both movement sensor and a trigger for the video

playback during one of the experimental phases. A marker for each above-threshold movement detected by the 'trigger' accelerometer would be sent to the infant's EEG data during the whole experiment. Movement of the opposite arm and both infant's legs were also monitored with 3 additional accelerometers. Input from all accelerometers were sampled at 20Hz sampling frequency and logged for each limb separately for further behavioral analysis.

2.4 Task

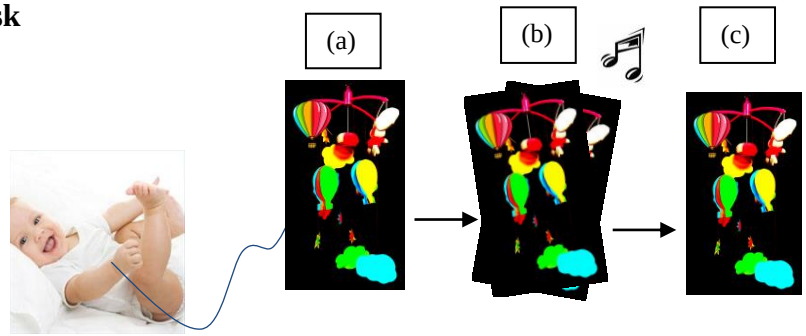

Fig 3. Task of the experiment, consisting of 3 phases: (a) baseline, (b) connect, (c) disconnect

The experiment consisted of three phases, the baseline phase, the connect phase, and the disconnect phase. As illustrated in Figure 3, infants were presented with the static image of mobile toy for 2 minutes during the baseline phase. Following this phase was the connect phase. During the connect phase, infants' movement would trigger the video stimulus playback along with the sound effect. The connect phase lasted for 3.5 minutes. The disconnect phase followed directly after a period of connect phase without any gap. During the disconnect phase, infants were presented with the same stimulus as in the baseline phase for 2 minutes. Their movement did no longer trigger any effect on the stimulus presentation. All stimuli were presented using Presentation Software (Neurobehavioral Systems; https://www.neurobs.com/). The phases were presented in a fixed order.

2.5 Procedure

Parents and infants were invited to the Baby EEG Lab in Donders Centre for Cognitive Neuroimaging for a testing session that lasted approximately one hour including explanation and instruction to parents, preparation, and debriefing. We informed the parents about the procedure of the experiment before we started. The infant's head circumference was measured and an EEG cap was chosen based on the size of the head circumference. Then the infant was brought to a shielded room built to minimize EEG interference by other electrical sources. The EEG capping was done in the shielded room, and after the EEG cap had been fitted, the experimenter controlled the impedances. Infants were seated in a child-seat (Maxi-Cosi) which was positioned on the parent's lap approximately 50cm from a computer screen. This set up was used in order to minimize parental influence and ensure that the infant's sitting position was stable. All of the visual stimuli used in this experiment was presented on the computer screen (see Figure 4 for experimental set-up). When the infant seemed comfortable, the experimenter attached the four accelerometers to the infant's limbs. The experiment began once the infants were comfortable with the overall set up. If the infant seemed to lose interest to the stimuli, became fussy, started to cry, or showed other signs of discomfort, the experimenter could pause the experiment until the infant regained attention to the screen. The experiment ended after the infant finished the three phases or if the infant continued to show signs of discomfort, cry, or fussiness. Infants' EEG and movement frequency were recorded throughout the experiment. A video of the whole experiment was also recorded

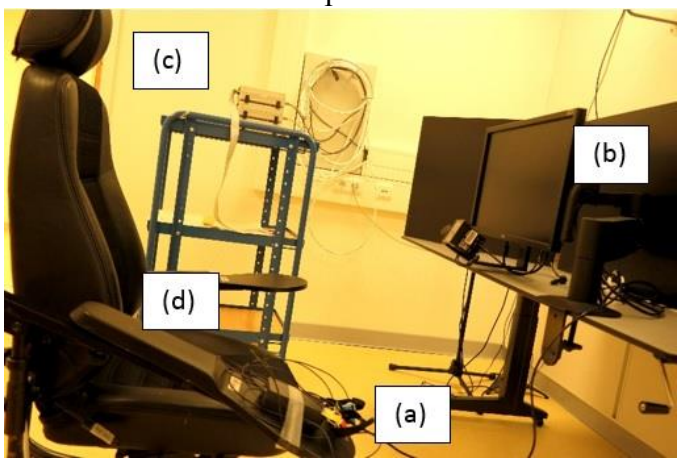

Fig 4. Experimental et-up: (a) accelerometers, (b) stimulus monitor, (c) EEG set-up, (d) maxi-cosi position

2.4 Data Acquisition

EEG was recorded from 32 active Ag/AgCL electrodes using infant-sized caps (ActiCAP), Brain Amp DC, and Brain Vision Recorder software (Brain Products GmbH, Germany). The electrodes were arranged according to the International 10-20 system configuration. The data was sampled at 500 Hz and online filtered with 0.016 Hz high-pass and 125 Hz low-pass filter. Left mastoid was used as online reference and the impedance was kept below 20 kʊ. The EEG data was re-referenced to the averaged of all channels for data analysis.

2.5 Data Analysis

The EEG data was analyzed using Fieldtrip (open source Matlab toolbox, developed at Donders Institute for Brain, Cognition, and Behavior; http://www.fieldtriptoolbox.org/). Statistical analyses was performed using IBM SPSS Statistics, version 22. We validated that the data was normally distributed prior to any parametric statistical analyses.

2.5.1 Analysis of Infants' Movement Frequency

Infants' movement frequency was recorded by the accelerometers. The accelerometers recorded movement acceleration on 3D positions (x,y,z) and obtained the resultant value. Increase in movement frequency was detected when the resultant value difference between two consecutive movements exceeded a threshold. Any differences in number of movement and movement frequency across conditions was tested using 2-way repeated measures ANOVA with factors of phases in which the movement occurred (baseline, connect, and disconnect) and the limbs (trigger arm, opposite arm, ipsilateral leg, and contralateral leg).

2.5.2 Analysis of ERP Mismatch Negativity

2.5.2.1 Segmentation and ERP Epoch Selection

All of the EEG data were preprocessed first before being analyzed. Preprocessing included data segmentation, eye artifacts rejection, other artifacts rejection, baseline

correction, demeaning, and filtering. For the analysis of the ERP Mismatch Negativity response, windows of interest for baseline and disconnect phase were selected from each phase. More specifically, the part where the movement happened, as indicated by the movement marker. The continuous EEG data was segmented into 800ms movement-onset-locked epochs, including a baseline window defined as 200ms prior to the movement onset. Each epoch were filtered by a bandpass 0.5 – 20 Hz filter and baseline corrected with respect to the mean 100-ms pre-stimulus voltage. These segmented epochs are called trials from here on. Valid EEG trials of these windows of interest were chosen to be included in the data analysis. Infants were included at the final analysis if they had at least 5 valid trials for each phase. Trials were defined as valid if it did not contain any EEG artifacts (such as eye blinks, eye movements, noise, or electrode drifts). Eye artifacts were rejected by utilizing independent component analysis (ICA). Other artifact rejection was done manually on each data from every individual EEG trial. After the pre-processing, the criteria of valid trials for each phase were met by 7 infants and only those data will be reported in the result section. On average, infants completed 32 valid trials (SD = 17) for the baseline phase, 76 valid trials (SD = 26) for the connected phase, and 24 valid trials (SD= 20) for the disconnected phase.

2.5.2.2 Determining Difference in Waveform of Mismatch Negativity Response

In MMN studies with standard and deviant trials, the difference waveforms were obtained by subtracting the standard waveforms from deviant waveforms (Trainor et al., 2003). In this experiment, the standard trials were analogous to trials in the baseline phase while the deviant trials were analogous to trials in the disconnected phase. ERPs from both phases were averaged separately. The difference waves was obtained by subtracting the general average of trials from the baseline phase from the general average of trials from the disconnected phase. Grand average was obtained by averaging across participants. Data was recorded from 32 sites, 12 frontal sites, 7 central sites, 7 parietal sites, 3 temporal sites, and 3 occipital sites. Based on the study conducted by Trainor and colleagues (2003) investigating MMN in infants, we focused on the frontal electrodes, particularly F3 and F4. Mean ERP amplitudes were calculated as a mean voltage obtained from consecutive 20ms time points over

particular time window. The mean amplitudes were compared over 200ms-300ms time window across conditions based on infants MMN literature (Trainor et al., 2003). Additional exploratory measurement was done on ERPs amplitude from 0-600ms time window in 100-ms intervals. A two-tailed dependent *t*-test was performed on the mean amplitudes to determine the regions of the waveform that were significantly different across conditions.

3. Results

3.1 Number of movement and movement frequency

Behavioral data from 7 participants who met the EEG inclusion criteria are included in the analysis. Given that one of the criteria of inclusion for EEG was a minimum of five valid trials per condition, we also included participants who did not finish the disconnect phase as long as the minimum number of valid trials in disconnect phase was obtained. Analysis on the behavioral data was done based on the accelerometer log files. Table 1 gives an overview of the number of movements that occurred in each phase for each limb averaged across participants. The number of movements was obtained by averaging the movements for each limbs in 30s time bins. Figure 5 shows how the number of movements of each limb changed across conditions. The average number of movements over time shows a slight increase in all limbs across conditions, from baseline to connect to disconnect phase. To validate the difference across conditions by including all limbs as a factor (trigger arm, opposite arm, contralateral arm, and ipsilateral arm) and examine the effect of conditions and limbs on the observed number of movement, a two-way ANOVA was conducted. The analysis was based on the total number of movement within each phase. There was no significant interaction between the effects of limbs and conditions on the observed movement frequency, $F(6) = .011$, $p = 1.0$. There was no significant effect of either limbs ($F(3) = 1.64$, $p = 0.186$) or conditions ($F(2) = 1.48$, $p = 0.233$) on the number of movement. Therefore, we conclude that there was no difference between the number of movements performed by the trigger arm and the movement performed by other limbs in all experimental conditions.

Table 1 Movement count across conditions (averaged over 7 participants)

Limbs/Condition	Movement/second (averaged over 7 participants)							
	Trigger arm		Opposite arm		Ipsilateral leg		Contralateral leg	
	M	SD	M	SD	M	SD	M	SD
Baseline	27.8	13.7	19.8	16.3	15.8	15.1	16.6	18.7
Connect	34.1	17.2	24.8	20.7	21.1	23.7	22.0	27.8
Disconnect	38.9	19.2	29.2	23.5	26.4	26.2	24.4	26.1

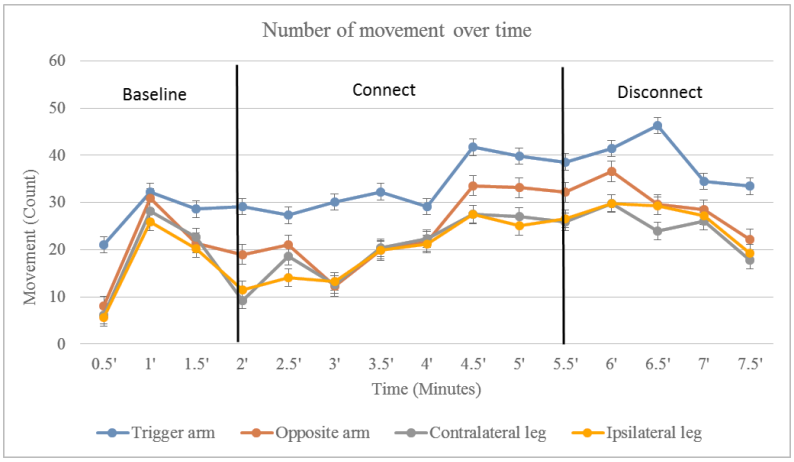

Fig 5 Number of movements across conditions (averaged over 8 participants): baseline, connect, disconnect for trigger arm, opposite arm, contralateral leg, and ipsilateral leg

Additionally, we analyzed how the movement frequency differs across conditions. As our participants did not have to finish the entire disconnect phase, the duration of this phase is on average shorter than the baseline phase. Therefore, finding no significant difference in number of movements between the baseline and disconnect phase does not necessarily mean that there is no difference in the movement frequency. Table 2 gives an overview of movement per second in each phase for all limbs averaged across participants. The frequency was obtained by dividing the total movement for each limb in each condition by the duration of the condition. The average movement per second shows an increase in all limbs across condition, from baseline to connect to disconnect phase. A two-way ANOVA with Bonferroni correction was conducted to

examine the effect of the conditions and limbs on the movement frequency. There was no significant interaction between the effects of limbs and conditions on the observed movement frequency, $F(6) = .143$, $p = .99$. The effect of limbs on movement frequency was also not significant, $F(3) = 2.49$, $p = .06$. Thus, there was no significant difference in movement frequency between trigger arm, opposite arm, contralateral leg, and ipsilateral leg. However, there was a significant effect of condition, $F(2) = 7.27$, $P < 0.005$. In general, there was a significant difference of movement frequency between baseline - disconnect phase ($P < 0.005$) and connect – disconnect phase ($P < 0.05$) but surprisingly not between baseline – connect phase ($p = 1.0$). Mean difference values revealed that movement frequency was highest in the disconnect phase and lowest in the baseline phase. Figure 6 shows how movement frequency of the trigger arm changed across condition for all participants.

Table 2 Movement frequency across conditions for all limbs (averaged over 7 participants)

Limbs/Condition	Movement/second (averaged across 8 participants)							
	Trigger arm		Opposite arm		Contralateral leg		Ipsilateral leg	
	M	SD	M	SD	M	SD	M	SD
Baseline	0.9	0.5	0.7	0.6	0.6	0.5	0.5	0.5
Connect	1.1	0.5	0.8	0.6	0.7	0.8	0.7	0.7
Disconnect	1.9	0.6	1.4	0.8	1.1	1.2	1.1	1.1

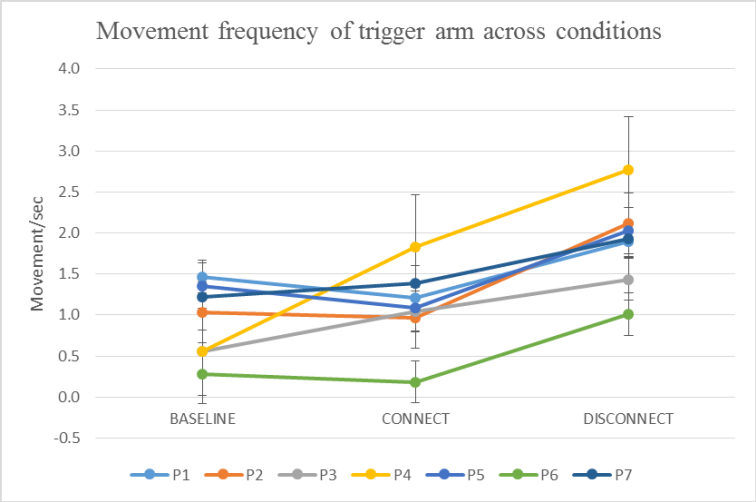

Fig 6. Changes in movement frequency of trigger arm across conditions per participant.

3.2 ERP Response

Based on the inclusion criteria described in the Method section, 7 participants were included in ERP final analysis. The preprocessed EEG data for each participant was obtained for both conditions in consecutive 20ms time windows, ranging from 200ms prior to and 600ms after movement onset. The mean amplitude of ERP for each participant was obtained by averaging the raw data within the range of each time of interest. Grand average data was computed from the average of the mean amplitude over 7 participants. Figure 7 shows the ERP response of all electrodes averaged across participants.

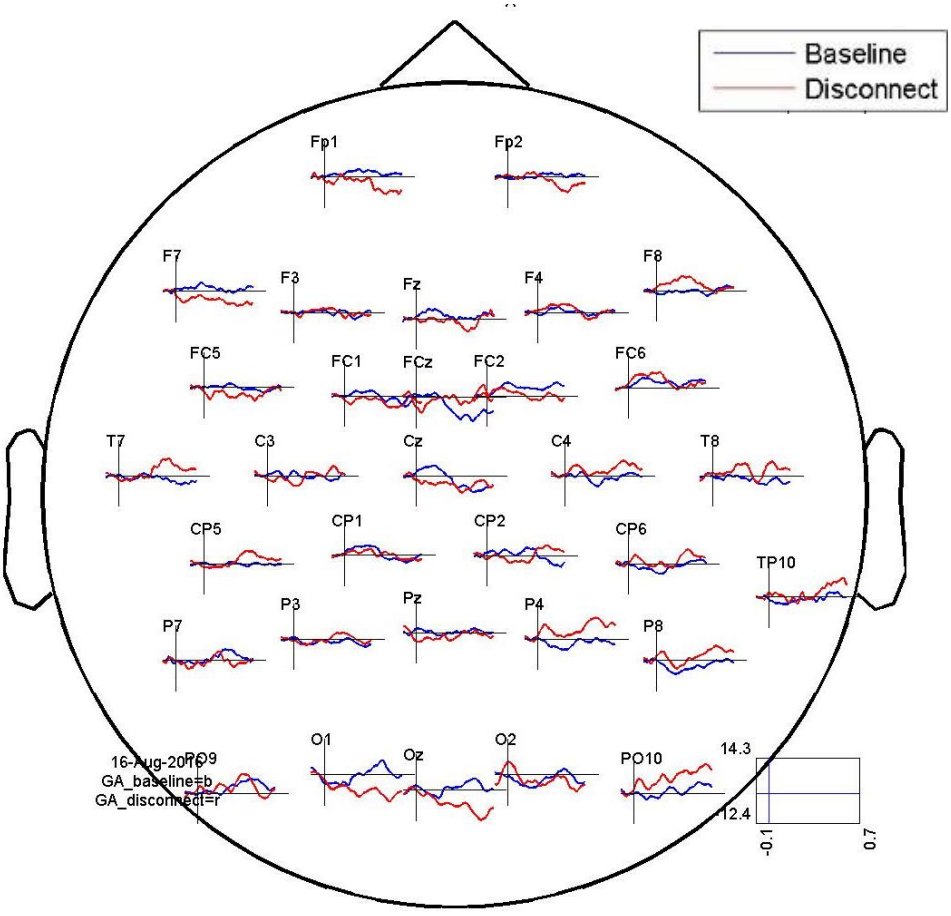

Fig 7 Grand average event-related potentials (ERPs; averaged across 7 infants) locked to movement in baseline (blue lines) and disconnect phase (red lines) in all electrodes (negative plotted downwards)

According to our a priori hypothesis, we expected to see the MMN on F3 and F4 around 300 ms after movement onset. Therefore, focusing on our electrodes of interest (i.e. left frontal/F3 and right frontal/F4, according to international 10/20 system), we

plotted ERPs response to movement in baseline against the response to movement in disconnect, as recorded in F3 and F4, in Figure 8. The difference wave between the two conditions was obtained by subtracting baseline movement-locked ERPs response from disconnect movement-locked ERPs response and was plotted in Figure 9. As shown in Figure 8, movement-locked ERPs in the disconnect phase over our time of interest seems to be more positive relative to movement-locked ERPs in the baseline phase both on F3 and F4. This was confirmed by the difference wave as shown in Figure 9. To validate the visual observation, we compared average of mean amplitude at latency 200-300ms recorded from both electrodes in baseline with disconnect phase. A two-tail dependent t-test was performed to measure any significant difference between conditions on data from each electrodes separately. We found no statistically significant difference between mean amplitudes in baseline (F3, M=0.1389 μV, SD = 2.8; F4, M=1.49 μV, SD = 3.17) and disconnect (F3, M=0.17 μV, SD = 2.81; F4, M=2.67 μV, SD = 4.13) for both electrodes (F3, p=0.27; F4, p=0.08). Post hoc comparisons using FDR correction indicated the similar result (F3, p=0.27; F4, p=0.16).

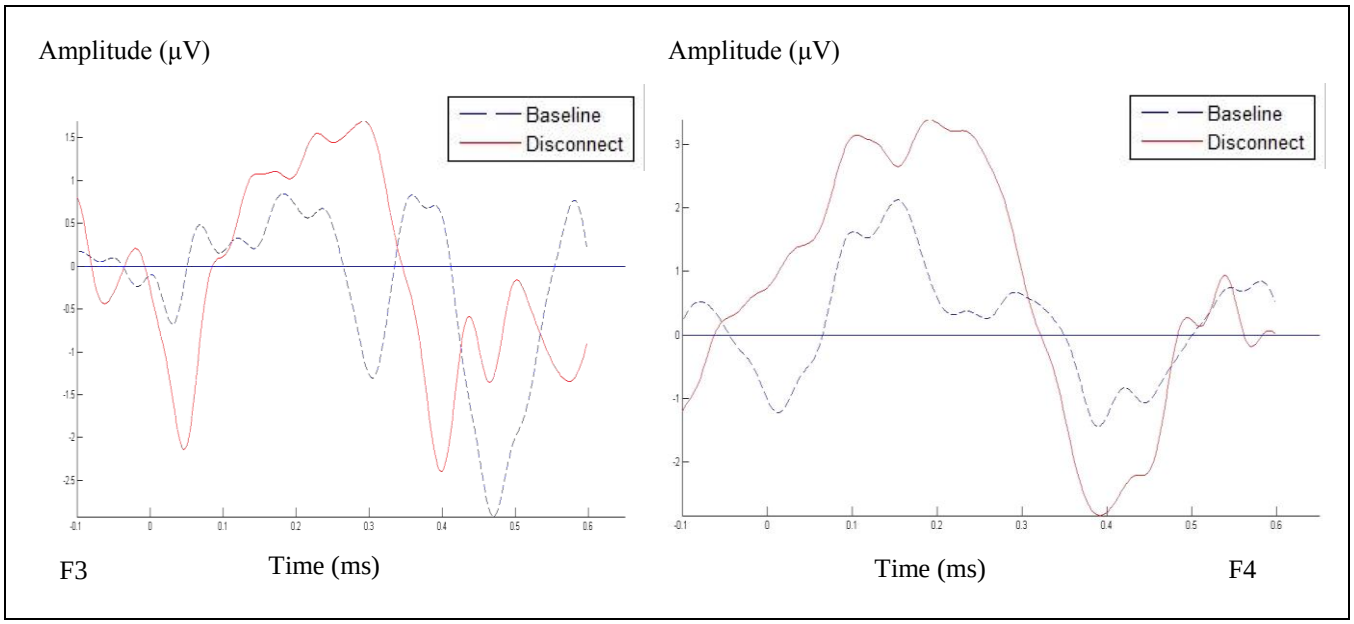

Fig 8 Grand-averaged event-related potentials (ERPs; averaged across 7 infants) elicited in baseline and disconnect phase in left and right frontal electrodes (F3, F4, according to International 10/20 system, negative plotted downwards)

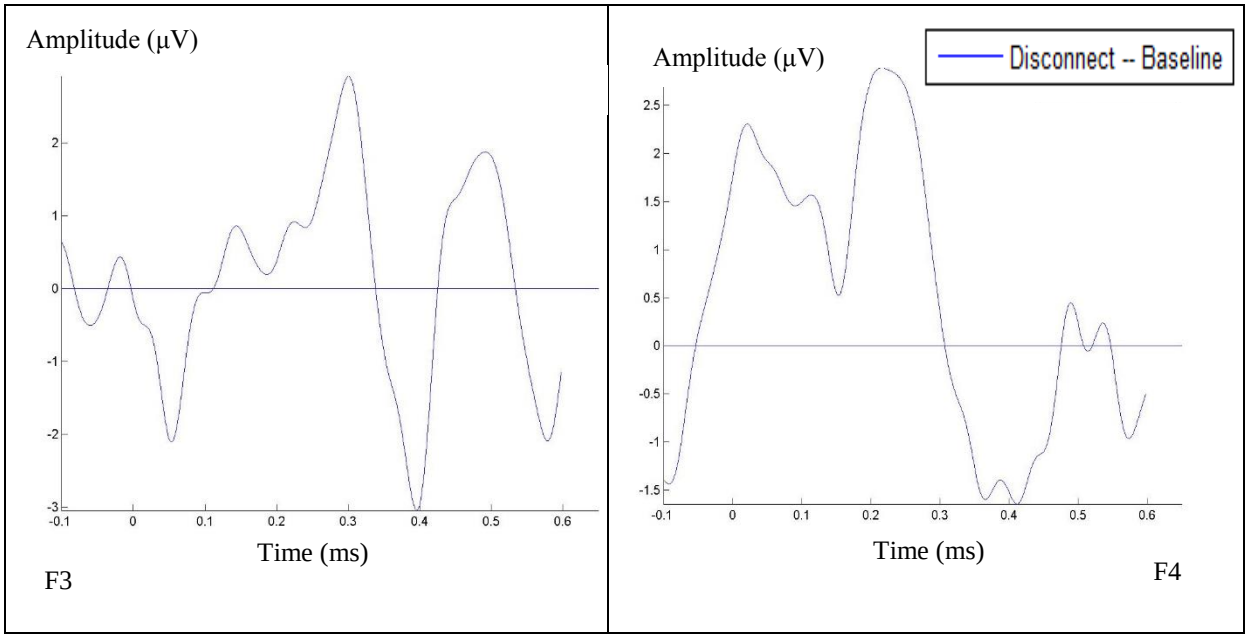

Fig 9. Grand-averaged of difference waves (in left/F3 and right/F4 frontal electrodes according to International 10/20 system, averaged across 7 infants), obtained by subtracting movement-locked ERP in baseline from movement-locked ERP response in disconnect phase, negative plotted downwards

It is possible that we did not reach significance due to our limited sample size. Therefore, to have more data to compare, we analyzed the data from F3 and F4 as one cluster, i.e. treating the average of mean amplitude at 200-300ms from both electrodes as one data, and compared between conditions. Prior to clustering the data, we performed dependent t-test for each electrode (F3 and F4) over 200-300ms within the same condition to ensure there was no significant difference between electrodes that might affect the clustered data. We confirmed there was no statistically significant difference between F3 and F4 in both baseline (F3, M=0.13, SD=2.8; F4, M=0.18, SD=2.8; t(6) = -0.56, p=0.59, uncorrected) and disconnect (F3, M=1.4, SD=2.6; F4, M=2.67, SD=4.1; t(6) = 0.117, p=0.91, uncorrected) phase.

The plot of movement-locked ERPs response in baseline and disconnect phase for the data cluster along with the difference wave was shown in Figure 10. A more prominent positivity over 200-300ms could be seen on the plots. To validate this, we performed two-tail dependent t-test on the data cluster and found a significant difference in baseline ERPs mean amplitude (M=0.158, SD=2.69) and disconnect ERPs mean amplitude (M=2.08, SD=3.59); t(13) = -2.4, P<0.05. The mean difference value between two conditions confirmed that ERPs mean amplitude in disconnect

phase was larger compared to baseline. Therefore, it seems that the ERP response at the latency where we expected MMN to appear was more positive in the disconnect phase relative to baseline phase.

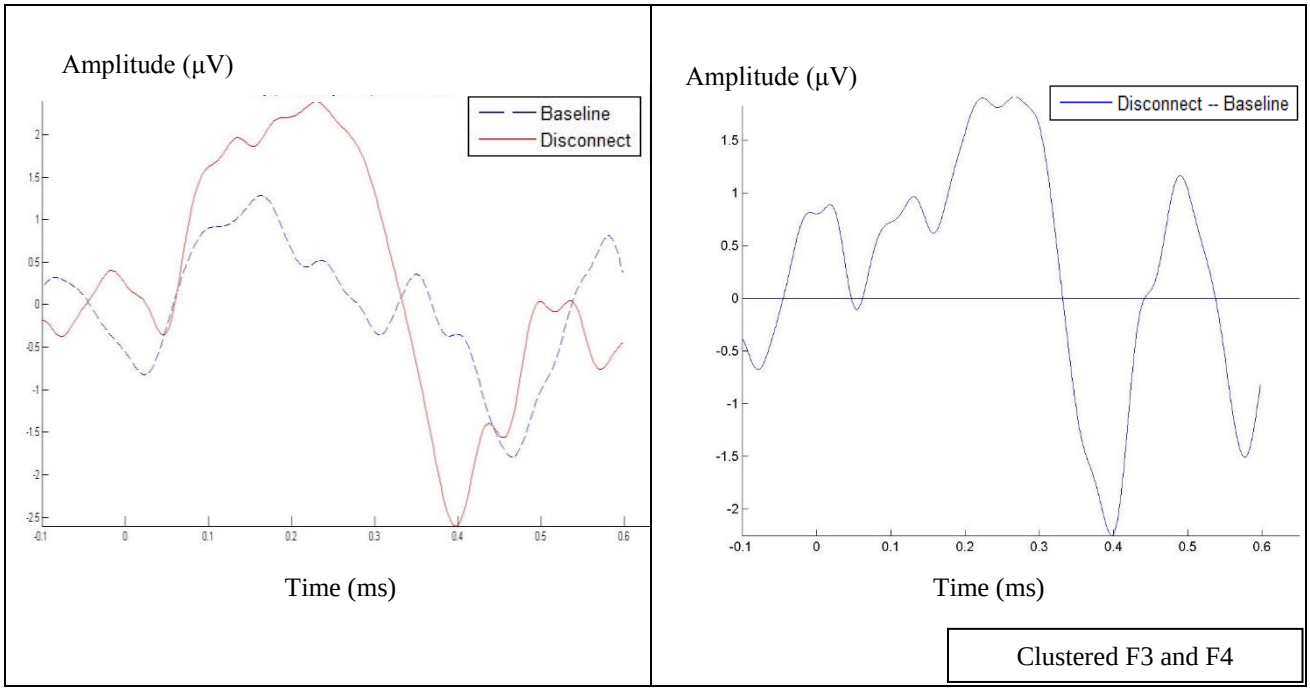

Fig 10. Grand-averaged of event-related potentials on clustered electrodes F3 and F4 (ERPs; averaged across 7 infants) elicited in baseline and disconnect phase (left) and difference waves between conditions (right), negative plotted downwards

Aside from the positivity observed at 200-300ms, there was also some peaks in the difference wave over several time ranges. The most prominent one seems to be a negative deflection around 400ms. We explored more of these peaks by performing the same routine as we did with the 200-300ms latency. We did a separate two-tail dependent t-test on data from F3 and F4 within each condition, for each latency prior to clustering. We confirmed there was no statistically significant difference between F3 and F4 in baseline for latency 100-200ms (F3, M=0.4, SD=2.51; F4, M=1.6, SD=2.13; t(6) = -0.87, p=0.41), 300-400ms (F3, M=0.15, SD=3.86; F4, M= -0.29, SD=3.42.13; t(6) = 0.55 , p=0.6), 400-500ms (F3, M= -1.77, SD=3.77; F4, M= -0.7, SD=3.18; t(6) = -1.0 , p=0.35), and 500-600ms (F3, M= -0.29, SD=2.54; F4, M=0.6, SD=2.97; t(6) = -0.58, p=0.584). We also found no statistically significant difference in disconnect for latency 100-200ms (F3, M=0.86, SD=3.03; F4, M=3.02, SD=2.7.1; t(6) = -1.4, p=0.2), 300-400ms (F3, M= -0.32, SD=3.6; F4, M=-1.3, SD=4.71; t(6) = 0.45, p=0.67), 400-500ms (F3, M= -1.4, SD=6.17; F4, M= -1.4, SD=4.68; t(6) = 0.17 ,

p=0.87), and 500-600ms (F3, M= -0.93, SD=7.07; F4, M=0.28, SD=4.17; t(6) = -0.5, p=0.63). However we did find a statistically significant difference between F3 and F4 in disconnect phase over latency 0-100ms (F3, M= -0.93, SD=7.07; F4, M=0.28, SD=4.17; t(6) = -3.08, P<0.05).

Therefore, we clustered data only for the latency that was confirmed to have no difference between electrodes within the same condition. We found no significant difference between conditions in latency 300ms-400ms (baseline, M= -0.07, SD=3.5; disconnect, M= -0.82, SD=4.06; t(13) = -0.79, p=0.444), 400ms-500ms (baseline, M= -1.23, SD=3.4; disconnect, M= -1.21, SD=5.27; t(13) = -0.01, p=0.991); 500ms-600ms (baseline, M= 0.16, SD=2.7; disconnect, M= -0.33, SD=5.6; t(13) = 0.28, p=0.78). To test whether there was statistically significant difference between conditions over latency 0-100ms, we performed a two-tail dependent t-test on data from F3 and F4 separately. We found no significant difference for F3 between conditions (t(6) = 0.84, p=0.43). We did find a significant difference for F4 between conditions (t(6) = -0.25, p<0.05). However, this significance did not hold after FDR correction for multiple comparisons problem (F3, p=0.433; F4, p=0.09).

4. Discussion

This study aimed to address the issue on whether young infants build an internal (causal) model of how their actions cause effects, and, therefore experience sense of agency. We investigated 3-to-4-month old infants in terms of their mismatch response to omission of own action's effect. The infants were presented with static visual display for 2 minutes (i.e. baseline) followed by 3.5 minutes audio-visual stimuli triggered by movement (i.e. connect), and finished with another 2 minutes of static visual display (i.e. disconnect) while their EEG was recorded. The infants' number of movement and movement frequency across phases was measured in order to replicate results from previous literature (Rovee-Collier, et al., 1978; Watanabe & Taga, 2006, 2009, 2011). The ERP response to movement in baseline and disconnect phase was measured and analyzed to investigate the occurrence of violation of expectation during disconnect phase and, based on that result, to infer the presence of an internal (causal) model. We would address results from the behavioral analysis followed by results

from the ERP analysis in the following discussion.

The behavioral experiment conducted in this study has been done in several other studies, each with a slightly different response parameter (Rovee-Collier, et al., 1978; Watanabe & Taga, 2006, 2009, 2011). However, each study reported a similar data pattern in terms of infant's response to a stimulus. For instance, Rovee-Collier (1978) found increased in infants' behavioral response to the mobile movement when the mobile was connected to the infants' limb relative to the phase before. They also found an additional increased during the period after the connection linking infants' movement and the mobile was cut. In accord with their findings, we expected to find an increased movement response across the phases (i.e. from baseline to connect to disconnect). An analysis of the number of movements in consecutive 30s time bins showed a slight increase across conditions. However, statistical analysis revealed that the increase was not significant. Although this is quite surprising, this inconsistency might be due to the difference in the experiment duration. Rovee-Collier and colleagues (1978) did the analysis based on the total score of movement response within phases, which lasted longer than our experiment (3 min baseline, 15 min connected, 5 min disconnect). Longer duration would allow more movement to be sampled in the total score. Moreover, we used a slightly different set up (hanging mobile toy in the previous studies versus a computer display in our experiment), type of movement feedback (movement of the hanging mobile toy versus movement with sound of the computer display), and infant's position (lying down in a crib versus sitting in a child seat).

In addition to number of movement, we analyzed the difference in the movement frequency across phases. Watanabe and Taga (2006, 2009, and 2011) reported increased in movement velocity (based on 3D position of the limb) during the period when infants' limb movement followed by the mobile movement. Consistent with their findings, we observed a significant difference in the movement frequency (per second) between baseline and disconnect phase as well as between connect and disconnect phase. However, in contrast to what was found in Watanabe and Taga (2006), we did not find a significant difference between baseline and connect phase.

This might be due to difference in the nature of measurement used in their study. They conducted the statistical analysis based on the total of 2-min mean velocities within each phase, and used data from the last 2-min for the connect phase (which lasted 6 min in total). Whereas in this study, we performed the statistical analysis based on the movement frequency obtained by dividing the total number of movement within each phase by the duration of the phase (which lasted differently for each phase). They also obtained the mean velocities based on infants' limb position, which was not analyzed in this study.

Moreover, in contrast to Rovee-Collier and colleagues (1978) and Watanabe and Taga (2006, 2009), we did not find any significant limbs effect, both in terms of number of movement and movement frequency, indicating that there was no difference in all limbs movement throughout all phases. This inconsistency might be due to the difference in the way the infants were positioned during the experiment. In our experiment, infants were placed in sitting position in a child seat (with safety belt on), which might limit the overall limbs movement.

Nevertheless, the increased movement frequency from connect to disconnect was expected. One way to interpret this data pattern is that infants learn the connection between their movement and the stimulus feedback during the connect phase and the increased might occur due to infants' behavioral response to the omission of the audio-visual feedback. Alternatively, this might also due to infants' increased fuzziness after being in the experimental set up for a period of time. However, this is unlikely because we did see a mismatch response elicited by movement in the disconnect phase (see below). We did not expect to see such mismatch response if the increased movement frequency was only due to increased fuzziness.

Although it may seem unexpected to find significant difference in movement frequency that was not accompanied by significant difference in number of movement, this might be explained by a difference between baseline and disconnect duration among participants (similar number of movements in the disconnect phase as compared to baseline but in a shorter period of time). Despite the fact that we did not

find a significant result with regard to the number of movements, we did observe a difference in terms of movement frequency. However, we could not make any inference regarding internal model or sense of agency based only on this data pattern. Therefore, we did the main analysis on the ERP data.

The ERP (mismatch response) was determined as the response locked to infants' movement in baseline and disconnect phase. The ERP epochs were 800ms in duration, starting 200ms before and ending 600ms after the onset of the movement. Our hypothesis was that if the model building did occur during the connect phase, and infants built the causal model linking their action to the stimulus changes, we expected to find a larger mismatch response in the disconnect phase compared to the baseline period where the model was not present yet. Based on MMN characteristic as a negative component of the waveform elicited by sudden changes in stimulation (Garrido et al., 2009), we expected a larger mismatch response to present as a more negative response in the disconnect phase.

In contrast to what we expected, the analysis of the movement-locked ERP response revealed that there was a significant difference in the latency around 200-300ms with a positive difference wave, indicating mean amplitude in disconnect phase was more positive relative to baseline. Despite the fact that the significance was only reached when the mean amplitude data from the F3 and F4 electrodes was clustered (whereas, for instance, Trainor and colleagues (2003) found it for the individual electrode as well), visual observation of ERPs plot confirmed that this positivity is detected in both F3 and F4.

Although this result partially matched our hypothesis, the reported positive direction of the difference wave was the opposite of our a priori hypothesis. Previous literature described MMN as the mismatch response to deviant stimuli in 3-month-old infants that was found at 200-300ms in negative direction (Alho et al., 1997; Alho & Cheour, 1997). Nevertheless, consistent with what was found by Trainor and colleagues (2003), it is possible to interpret the positivity as a mismatch response as well. This supported by several other studies that also reported increases in positivity to stimulus

deviance in younger infants (Dehaene-Lambertz & Baillet, 1998; Dehaene-Lambertz and Dehaene, 1994; Morr et al., 2002).

As Trainor et al., (2003) suggested, it is also possible that overlapping components, such as the positive slow wave, obscured the MMN response. However, as it is also elicited in response to stimulus deviance, this positive slow wave could also be interpreted as a mismatch response. As a matter of fact, Ho and colleagues (2009) noted that there were two types of mismatch response in young infants consistently reported in several studies, namely a negative response similar to adult MMN (e.g. He, Hotson & Trainor, 2007; Hirasawa, Kurihara, & Konishi, 2003; Trainor et al, 2001; and Trainor et al., 2003) and an increase in the slow positive wave dominating young infants' ERP responses (e.g. He et al., 2007; Cheour et al., 1999; Trainor et al., 2001; and Trainor et al., 2003). There is an ongoing debate on whether these positive slow waves are an early form of either P1 or P2, or whether they are unrelated to any component found in adults. Nevertheless, the report on this slow positivity consistently reveals that infants show mismatch responses to a violation of expectation as shown by a larger (more positive) response to deviant relative to standard stimuli.

Our hypothesis was that if the infants have a sense of agency over their movement and its effect, they would build expectations for upcoming events based on that model. We predicted that if this expectations was violated, the mismatch response would occur during the disconnect phase, which was analogue to stimulus deviance. Accordingly, we expected to see differences in the movement-locked ERPs of disconnect phase relative to baseline. Therefore, the observed larger frontal positive slow wave in the disconnect phase might indicate that infants in this study did experience a violation of expectation. Furthermore, since the ERP was time-locked to the movement, we might interpret the mismatch response as the infants' expectations of their movement effect. In other words, the infants' expectation was built based on an internal model linking a movement with an effect, and the violation of expectation provides evidence for the presence of internal model building capability.

Despite our effort to carefully design this experiment, there were some limitations in

this study. Movement, which brings about EEG artifacts, is inherent in the nature of our experiment. We minimalized the confounding effect by performing the analysis on the movement-locked ERPs and obtaining the difference wave by subtracting two datasets of movement-locked response, meaning that in both conditions there was a movement present right at the onset of the trial. However, there may be some additional movement variability present the ERPs data. Due to technological limitations, we do not have information about the characteristics of the movements (e.g. velocity). There may be characteristic differences across conditions that could influence the ERP result. Furthermore, since there is variability of the number of movements and movement frequency across participants, this resulted an unbalanced number of trials across phases. Due to our limited sample size, we were lenient in setting the inclusion criterion for the ERP analysis. Accordingly, we included all participants if they had at least 5 valid trials in each phase (as described in Method section). The difference in number of trials per participant could have an influence on the result. Nevertheless, we based our analysis on the mean amplitude of ERPs response. According to Luck (2005), the mean amplitude is not biased by an increase in noise level. Hence, it is justifiable to compare mean amplitude measurements from waveforms based on different number of trials. Finally, a larger sample size is necessary to ensure a robust result and to perform a more rigorous analysis.

Further investigation might be done by applying different preprocessing parameter (such as implementing different filter) to test if there is an MMN response obscured by the positive slow wave. In addition, it might be useful to correlate the behavioral data pattern of each participant with their respective ERP response to investigate whether difference in movement activity (thus self-exploration) in the connect phase affects the magnitude of the mismatch response. Last, performing time-frequency analysis on the EEG data during the connect phase might also provide additional information on sense of agency in infancy.

Conclusion

The results of the current analyses suggest that infants experienced violation of expectation in response to the omission of their movement effect. This indicated that

infants built an internal (causal) model linking their movement with its effect, which suggest the presence of sense of agency. To our knowledge, these findings are novel as this is the first study that relates the disconnect phase in relation to sense of agency, and combines mobile paradigm with EEG recordings.

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Reference

Alho, K., & Cheour, M. (1997). Auditory discrimination in infants as revealed by the mismatch negativity of the event-related brain potential. *Developmental Neuropsychology*, *13*(2), 157–165. doi:10.1080/87565649709540675

Angulo-Kinzler, R. M., & Horn, C. L. (2001). Selection and memory of a lower limb motor-perceptual task in 3-month-old infants. *Infant Behavior and Development*, *24*(3), 239–257. doi:10.1016/s0163-6383(01)00083-2

Baldeweg, T. (2007). ERP repetition effects and mismatch Negativity generation. *Journal of Psychophysiology*, *21*(3-4), 204–213. doi:10.1027/0269-8803.21.34.204

Basirat, A., Dehaene, S., & Dehaene-Lambertz, G. (2014). A hierarchy of cortical responses to sequence violations in three-month-old infants. *Cognition*, *132*(2), 137–150. doi:10.1016/j.cognition.2014.03.013

Bayne, T and Levy, N. "The feeling of doing: deconstructing the phenomenology of agency," Disorders of Volition, pp. 49–68, 2006.

Bayne, T. (2008). The Phenomenology of agency. *Philosophy Compass*, *3*(1), 182–202. doi:10.1111/j.1747-9991.2007.00122.x

Bendixen, A., SanMiguel, I., & Schröger, E. (2012). Early electrophysiological indicators for predictive processing in audition: A review. *International Journal of Psychophysiology*, *83*(2), 120–131. doi:10.1016/j.ijpsycho.2011.08.003

Čeponien, R., Kushnerenko, E., Fellman, V., Renlund, M., Suominen, K., & Näätänen, R. (2002). Event-related potential features indexing central auditory discrimination by newborns. *Cognitive Brain Research*, *13*(1), 101–113. doi:10.1016/s0926-6410(01)000933

Cheour, M., Čeponien, R., Hukki, J., Haapanen, M.-L., Näätänen, R., & Alho, K. (1999). Brain dysfunction in neonates with cleft palate revealed by the mismatch negativity. *Clinical Neurophysiology*, *110*(2), 324–328. doi:10.1016/s1388-2457(98)00005-4

Dehaene-Lambertz, G., & Baillet, S. (1998). A phonological representation in the infant brain. *NeuroReport*, *9*(8), 1885–1888. doi:10.1097/00001756-199806010-00040

Dehaene-Lambertz, G., & Dehaene, S. (1994). Speed and cerebral correlates of syllable discrimination in infants. *Nature*, *370*(6487), 292–295. doi:10.1038/370292a0

Friston, K. (2012). Prediction, perception and agency. *International Journal of Psychophysiology*, *83*(2), 248–252. doi:10.1016/j.ijpsycho.2011.11.014

Gallagher, S. (2000). Philosophical conceptions of the self: Implications for cognitive science. *Trends in Cognitive Sciences*, *4*(1), 14–21. doi:10.1016/s1364-6613(99)01417-5

Garrido, M. I., Kilner, J. M., Stephan, K. E., & Friston, K. J. (2009). The mismatch negativity: A review of underlying mechanisms. *Clinical Neurophysiology*, *120*(3), 453–463. doi:10.1016/j.clinph.2008.11.029

Geangu, E. (2008). Notes on self awareness development in early infancy. *Cognition, Brain, Behavior*, *XII*(1), 103–113. Retrieved from http://www.devpsychology.ro/wp-content/uploads/2010/01/y4t7x_Geangu_ccc_mar2008.pdf

Guilaume, P. (1926). *L'imitaion chez l'enfant* (Imitation in the child) , Paris: Alcan

Haggard, P., & Chambon, V. (2012). Sense of agency. *Current Biology*, *22*(10), R390–R392. doi:10.1016/j.cub.2012.02.040

He, C., Hotson, L., & Trainor, L. J. (2007). Mismatch responses to pitch changes in early infancy. *Journal of Cognitive Neuroscience*, *19*(5), 878–892. doi:10.1162/jocn.2007.19.5.878

He, C., Hotson, L., & Trainor, L. J. (2009). Maturation of cortical mismatch responses to occasional pitch change in early infancy: Effects of presentation rate and magnitude of change. *Neuropsychologia, 47*(1), 218–229. doi:10.1016/j.neuropsychologia.2008.07.019

Heathcock J.C., Bhat A.N., Lobo M.A., Galloway J.C. (2005). The relative kicking frequency of infants born full-term and preterm during learning and short-term and long-term memory periods of the mobile paradigm. *Physical Therapy 85*(1), 8-18.

Hirasawa, K., Kurihara, M., & Konishi, Y. (2002). The relationship between mismatch negativity and arousal level. Can mismatch negativity be an index for evaluating the arousal level in infants? *Sleep Medicine, 3*, S45–S48. doi:10.1016/s1389-9457(02)00164-8

Kalnins, I. V., & Bruner, J. S. (1973). The coordination of visual observation and instrumental behavior in early infancy. *Perception, 2*(3), 307–314. doi:10.1068/p020307

Kelso, J. A. S. (2016). On the self-organizing origins of agency. *Trends in Cognitive Sciences*, *20*(7), 490–499. doi:10.1016/j.tics.2016.04.004

Kurtzberg, D., Vaughan, H. G., Kreuzer, J. A., & Fliegler, K. Z. (1995). Developmental studies and clinical application of mismatch Negativity. *Ear and Hearing*,*16*(1), 105–117. doi:10.1097/00003446-199502000-00008

Leppänen, P. H. T., Guttorm, T. K., Pihko, E., Takkinen, S., Eklund, K. M., & Lyytinen, H. (2004). Maturational effects on newborn ERPs measured in the mismatch negativity paradigm. *Experimental Neurology*, *190*, 91–101. doi:10.1016/j.expneurol.2004.06.002

Lewis, M., Sullivan, M. W., & Brooklyn-Gunn, J. (1985). Emotional behaviour during the learning of a contingency in early infancy. *British Journal of Developmental Psychology, 3*, 307-316

Luck, S. J. (2014). *An introduction to the event-related potential technique*. Cambridge, MA: Bradford Books.

Morr, M. L., Shafer, V. L., Kreuzer, J. A., & Kurtzberg, D. (2002). Maturation of mismatch negativity in typically developing infants and preschool children. Ear and Hearing, 23, 118–136.

Näätänen, R., & Alho, K. (1997). Higher-order processes in auditory-change detection. *Trends in Cognitive Sciences*, *1*(2), 44–45. doi:10.1016/s1364-6613(97)01013-9

Rochat, P., & Hespos, S. J. (1997). Differential rooting response by neonates: Evidence for an early sense of self. *Early Development & Parenting*, *6* (3–4), 105–112.

Rochat, P., & Striano, T. (1999). Emerging self-exploration by 2-month-old infants. *Developmental Science*, *2*(2), 206–218. doi:10.1111/1467-7687.00069

Rochat, Philippe, & Striano, T. (2000). Perceived self in infancy. *Infant Behavior and Development*, *23*(3-4), 513–530. doi:10.1016/s0163-6383(01)00055-8

Rovee-Collier, C. K., Morrongiello, B. A., Aron, M., & Kupersmidt, J. (1978). Topographical response differentiation and reversal in 3-month-old infants. *Infant Behavior and Development*, *1*, 323–333. doi:10.1016/s0163-6383(78)80044-7

Schulz, L. E., Gopnik, A., & Glymour, C. (2007). Preschool children learn about causal structure from conditional interventions. *Developmental Science, 10*(3), 322–332. doi:10.1111/j.1467-7687.2007.00587.x

Siqueland, E. R., & Delucia, C. A. (1969). Visual reinforcement of non-nutritive sucking in human infants. *Science, 165*, 221-232

Todd, J., Michie, P. T., Schall, U., Ward, P. B., & Catts, S. V. (2012). Mismatch negativity (MMN) reduction in schizophrenia—Impaired prediction-error generation, estimation or salience? *International Journal of Psychophysiology*, *83*(2), 222–231. doi:10.1016/j.ijpsycho.2011.10.003

Trainor, L. (2012). Predictive information processing is a fundamental learning mechanism present in early development: Evidence from infants. International Journal of Psychophysiology. *83*, 256-258

Trainor, L., McFadden, M., Hodgson, L., Darragh, L., Barlow, J., Matsos, L., & Sonnadara, R. (2003). Changes in auditory cortex and the development of mismatch negativity between 2 and 6 months of age. *International Journal of Psychophysiology*, *51*(1), 5–15. doi:10.1016/s0167-8760(03)00148-x

Trainor, L.J., Samuel, S.S., Galay, L., Hevenor, S.J., Desjardins, R.N., Sonnadara, R., (2001). Measuring temporal resolution in infants using mismatch negativity. NeuroReport 12, 2443–2448.

Wacongne, C., Changeux, J. ., & Dehaene, S. (2012). A neuronal model of predictive coding accounting for the mismatch Negativity. *Journal of Neuroscience*, *32*(11), 3665–3678. doi:10.1523/jneurosci.5003-11.2012

Watanabe, H., & Taga, G. (2006). General to specific development of movement patterns and memory for contingency between actions and events in young infants. *Infant Behavior and Development*, *29*(3), 402–422. doi:10.1016/j.infbeh.2006.02.001

Watanabe, H., & Taga, G. (2009). Flexibility in infant actions during arm- and leg-based learning in a mobile paradigm. *Infant Behavior and Development*, *32*(1), 79–90. doi:10.1016/j.infbeh.2008.10.003

Watanabe, H., & Taga, G. (2011). Initial-state dependency of learning in young infants. *Human Movement Science 30*, 125-142.

Winkler, I., Karmos, G., Näätänen, R., (1996). Adaptive modeling of the unattended acoustic environment reflected in the mismatch negativity event related potential. Brain Research 742, 239–252.

Wolpert, D. M., Ghahramani, Z., & Jordan, M. (1995). An internal model for sensorimotor integration. *Science*, *269*(5232), 1880–1882. doi:10.1126/science.7569931

Zaadnoordijk, L., Hunnius, S., Meyer, M., Kwisthout, J., & van Rooij, I. (2015). *The developing sense of agency: Implications from cognitive phenomenology*. . doi:10.1109/devlrn.2015.7346126