# It's not just my fault: Neural correlates of feedback processing in solo and joint action

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Abstract

People often coordinate their actions with others' in pursuit of shared goals, yet little research has examined the neural processes by which people monitor whether shared goals have been achieved. The current study compared event-related potentials elicited by feedback indicating joint errors (resulting from two people's coordinated actions) and individual errors (resulting from one's own or another person's observed actions). Joint errors elicited a reduced feedback-related negativity (FRN) and P3a relative to own errors, and an enhanced FRN relative to observed errors. In contrast, P3b amplitudes did not differ between joint and individual errors. These findings indicate that producing errors together with a partner influences neural activity related to outcome evaluation but has less impact on activity related to the motivation to adapt future behavior.

*Keywords*: action monitoring, joint action, interpersonal coordination, event-related potentials, FRN

Efficient and flexible behaviour often requires that people monitor the outcomes of their actions to determine whether their goals have been achieved. Research investigating the cognitive and neural mechanisms underlying action monitoring (see Ullsperger, Danielmeier, & Jocham, 2014, for a review) has focused predominantly on how people monitor *individual* action outcomes, i.e., outcomes of a single person's actions. However, humans are highly social beings; a significant portion of our behavioural repertoire is obtained through social interactions that require sensitivity to the outcome of both our own and others' actions. Recent studies have begun to unravel the impact of social context on action monitoring, typically by examining how people process *observed* individual outcomes in tasks that require them to monitor their own and another person's performance on successive trials (Koban & Pourtois, 2014). Little research has examined action monitoring in social contexts that require people to actively coordinate their actions with each other in pursuit of a *joint* action outcome (e.g., scoring a goal in hockey as a result of multiple players passing the puck back and forth; see Sebanz, Bekkering, & Knoblich, 2006). Joint outcomes present a challenge for the action monitoring system, as each person involved has only partial control over the outcome despite actively adapting their own actions to other people's. Furthermore, joint outcomes often take the form of external feedback that provides information about the joint performance as a whole and is available only at the end of the entire shared action sequence. Nevertheless, people are typically able to identify errors that have been committed, attribute them to a single person or to the group as a whole, and adapt their future behaviour accordingly.

Researchers have identified several event-related potential (ERP) components associated with evaluating individual action outcomes and adapting ongoing behaviour based on external feedback. The feedback-related negativity (FRN) is an anterior, negative-going ERP that peaks ~250 ms after feedback (Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997). FRN amplitudes are larger following negative feedback indicating an unfavourable outcome (e.g., an error) compared to positive feedback (Walsh & Anderson, 2012). The FRN is thought to reflect an initial evaluation of the outcome as better or worse than expected (Nieuwenhuis, Holroyd, Mol, & Coles, 2004) or as simply unexpected (Ullsperger, Fischer, Nigbur, & Endrass, 2014). The FRN is often followed by a P3, a positive-going potential with two sub-components: an earlier, anterior P3a and a later, posterior P3b. The P3a is thought to reflect an orienting response related to the initial evaluation of stimuli as task-relevant (Polich, 2007). The P3b is thought to reflect internal decision-making processes that facilitate appropriate behavioural responses to task-relevant stimuli (Nieuwenhuis, Aston-Jones, & Cohen, 2005).

Given the ubiquity of joint actions in everyday life and the need to establish healthy brain functioning during social interactions to better understand social disorders such as psychopathy (Brazil et al., 2011; de Bruijn, 2012), it is critical to examine how the neural mechanisms underlying action monitoring are modulated by the social context in which actions are performed. The current study compared ERPs elicited by jointly committed errors to ERPs elicited by individual errors committed by oneself or another person. We predicted that joint errors would elicit reduced ERP amplitudes relative to one's own errors but enhanced ERP amplitudes relative to another person's errors, based on previous work showing that reduced control over action outcomes reduces both FRN and P3 amplitudes (e.g., Li, Han, Lei, Holroyd, & Li, 2011; Li et al., 2010; Yeung, Holroyd, & Cohen, 2005) and that observing errors elicits reduced FRN amplitudes compared to producing them (e.g., Bellebaum, Kobza, Thiele, & Daum, 2010; Yu & Zhou, 2006).

## Method

**Participants**

Twenty two adults (8 male, 4 left-handed, mean age = 24.23, *SD* = 3.12) participated in the study. Participants were recruited in pairs without regard for specific gender combinations. Of the 11 pairs, 4 pairs consisted of two females, 1 pair consisted of two males, and 6 pairs were mixed-gender. All participants provided written informed consent according to procedures reviewed by the medical ethics committee at Radboud University Nijmegen. Participants were compensated with €30 for their participation.

**Design and Procedure**

In order to compare ERPs elicited by individual vs. jointly committed errors, we employed a sequence production task that could be performed either alone or in coordination with a partner. Specifically, participants were asked to produce sequences of 4 or 6 tones that matched the pace set by an initial series of isochronous clicks (see Figure 1). Participants produced the tone sequences in two settings: individual and joint. In the individual setting, each member of the pair produced the tone sequences alone while the other member of the pair sat quietly beside them. Each sequence in the individual setting therefore elicited ERP responses to *own* action outcomes for the participant who produced the tone sequences, and to *observed* action outcomes for the participant who observed the other person produce the tone sequences. In the joint setting, the two participants alternated button presses so as to produce the tone sequences together. Sequences produced in the joint setting elicited ERP responses to *joint* action outcomes for both participants.

During the experiment, participants sat next to each other on the same side of a table. A computer screen was centered between them, approximately 80 cm from the edge of the table. Each participant had a Logitech Gamepad F310 game controller aligned with their right hand, approximately 20 cm from the edge of the table. The game controllers were modified to include pressure sensitive buttons (2 cm diameter) that registered presses without providing auditory feedback. Each button press triggered a 1000 Hz sinusoidal tone (100 ms duration; 20 ms rise/fall time; sound pressure level 70 dB). The initial series of isochronous clicks was produced in a snare drum timbre. Tones and clicks were presented via speakers placed on either side of the computer screen. Stimuli were presented using Presentation software (Neurobehavioral Systems, Inc.; Albany, CA), which also recorded participants' button presses.

Participants were fitted with EEG caps and then performed two practice trials (one for the individual setting and one for the joint setting) during which the experimenter controlled the presentation of the events that comprised a trial and explained the task. Participants then completed a training phase (two blocks of 18 trials) and a test phase (12 blocks of 30 trials) with the timing described below. At the beginning of each block, instructions presented on the computer screen indicated the sequence length and whether participants were to produce the tones alone or together. Blocks alternated between individual and joint settings, the order of which was held constant through both the training and test phases and was counterbalanced across pairs. Sequence length was either 4 or 6 tones, held constant for a given block and randomly determined with the constraint that half of the blocks in each setting were of length 4 and the other half of length 6. The person producing the sequence (individual setting) or the first tone in the sequence (joint setting) was randomly determined on each trial with the constraint that one participant produced or started the sequence on half of the trials and the other participant produced or started the sequence on the other half of the trials.

Each trial consisted of the following sequence of events, shown in Figure 1. A cue indicating which person was to produce the tone sequence (individual setting) or the first tone in

the sequence (joint setting) appeared on a black computer screen for 2000 ms. The cue consisted of a cartoon face with two arms, one of which was colored red to indicate that the person on that side of the table should produce or begin the sequence. A white fixation cross then appeared and remained on the screen until the last tone of the sequence was produced. Three pacing clicks were presented at 500 ms intervals (beginning 500 ms after the onset of the fixation cross). Participants were instructed to produce the tone sequence while maintaining the pace set by the clicks. After the last tone was produced, a black screen was presented for 700 ms, followed by feedback indicating whether the participants had successfully maintained the required pace. The feedback was presented for 700 ms, after which a black screen was presented for 700 ms before the next trial began. The feedback consisted of a green or red Euro symbol indicating correct or incorrect performance, respectively. Participants were told that they would each gain or lose €0.05 from the initial fee of €25 per person for every sequence they produced with correct or incorrect timing, respectively, regardless of who produced or started the sequence on a given trial. In reality, all participants were paid €30 at the end of the experiment.

The accuracy of participants' performance on each trial was determined based on the average interonset interval (IOI) produced by the participant(s) on that trial. A trial was deemed correct if the average IOI fell within a window around 500 ms. The window size was set to 60 ms at the beginning of the experiment (i.e., sequences were considered correct if the average IOI fell within 500 ± 30 ms). At the end of each block, the window size increased by 10 ms if more than 30% of the sequences in the block were produced with incorrect timing. Otherwise, the window size decreased by 10 ms (until the minimum window size of 20 ms was reached). The window size was adjusted separately for the individual and joint setting blocks. Only the last 12 of the 18 training trials that comprised the first block in each setting were included in the first window size adjustment. This procedure resulted in mean error rates of 28.08% (*SD*=8.17%) in the individual setting and 32.42% (*SD*=7.08%) in the joint setting (before outliers were removed as described in the Data Processing section). The difference in error rates between conditions was not significant, $t(10) = 1.89$, $p = .089$.

**EEG Acquisition**

EEG was recorded using 32 active electrodes (Acticap, Brain Products GmbH, Germany) per participant, arranged according to an extended version of the 10-20 system at Fz/3/4/7/8, FCz/1/2/5/6, Cz/3/4, CPz/1/2/5/6, Pz/3/4/7/8, Oz/1/2, and T7/8. All electrodes were referenced to the left earlobe. Vertical and horizontal eye movements were monitored using bipolar electrooculography (EOG) electrodes positioned above and beneath the right eye and at the outer canthi of both eyes. Impedance was kept below 10 kΩ. EEG and EOG signals were amplified within a bandwidth of 0.05-100 Hz and digitized with a sampling frequency of 1000 Hz.

**Data Processing**

For the behavioural data, inter-tap intervals (ITIs) were calculated between participants' taps, starting with the interval between the last pacing tone and the first tap. Trials (279/3960 = 7.05%) were excluded from analysis if they included one or more ITIs that was greater or less than 3 standard deviations from the mean of all intervals produced by a given pair. This left an average of 20.64 (*SD*=10.37) error trials and 64.32 (*SD*=13.60) correct trials produced by one participant or the other in the individual setting, and an average of 22.86 (*SD*=7.46) error trials and 59.50 (*SD*=7.94) correct trials started by one participant or the other in the joint setting.[1] See the Supplementary Material for an analysis of IOIs by setting and accuracy.

EEG data processing was performed off-line using Brain Vision Analyzer software (V2.01.3931, Brain Products GmbH, Germany). EEG data were re-referenced to the mean of

both earlobe electrodes. Ocular artifacts were removed using Independent Component Analyses (Jung et al., 2000). The data were filtered using a high- and low-pass filters of 0.05 Hz (24 dB/oct) and 30 Hz (24 dB/oct) to remove slow drifts and excessive noise, respectively. The corrected EEG data were segmented into epochs from 200 ms before to 700 ms after feedback onset. Individual trials were removed if they contained further artifacts induced by head, body, or arm movements, as indicated by a difference exceeding 100 μV between the maximum and minimum value within a segment. One participant's data were removed from analysis because artifact rejection left fewer than three error trials in the individual setting

**Data Analysis**

We compared ERP responses to own, joint, and observed errors. Average EEG waveforms were calculated separately for each participant and error type. The 200 ms before feedback onset was used as the baseline period. Difference waves were computed on individual averages by subtracting the EEG waveforms elicited by correct feedback from the waveforms elicited by error feedback (see Figure S1 in the Supplementary Material for non-differenced waveforms). The FRN was calculated on this difference wave using a peak-to-peak analysis (Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003; Pfabigan, Alexopoulos, Bauer, & Sailer, 2011; Picton et al., 2000) in which the mean voltage amplitude over a 10 ms window around the positive peak that preceded the FRN was subtracted from the mean voltage amplitude over a 10 ms window around the FRN peak. The FRN peak was defined as the most negative peak between 180-360 ms after feedback onset, and the preceding positive peak was defined as the most positive peak between 100-220 ms after feedback onset. Peaks were identified separately for each participant and error type with the aid of voltage scalp topographies. This analysis was conducted on electrodes FCz and Cz, where FRN amplitudes were maximal in both the current study (see Figure 2) and previous FRN studies (e.g., Miltner et al., 1997). FRN amplitudes were compared across error types by a one-way repeated-measures ANOVA.

The FRN was followed by a frontocentral P3a component and a subsequent parietocentral P3b component. The P3a was defined as the mean amplitude of the difference wave from 360-390 ms after feedback onset at electrodes Fz, FC1, and FC2. The P3b was defined as the mean amplitude from 430-560 ms after feedback onset at electrodes Pz, CP1, and CP2. Electrode sites were chosen based on previous studies on the P3a and P3b and the locations of their maximal amplitudes in the current study (see Figure 3). To confirm that the two subcomponents had distinct time windows and scalp distributions, we compared the mean amplitudes with a 2 (interval: early [360-390 ms], late [430-560 ms]) x 2 (location: frontocentral [Fz, FC1, FC2], parietocentral [Pz, CP1, CP2]) x 3 (error type: own, joint, observed) repeated measures ANOVA.

Finally, we checked for attentional effects by examining the posterior P1 and N1 components evoked by the feedback stimulus, as the amplitudes of these components depend on the level of visual attention that is allocated to a stimulus (Luck & Kappenman, 2012). We measured the mean amplitude of the P1 component from 110-140 ms after feedback onset at electrodes P7 and P8, and the mean amplitude of the N1 component from 150-180 ms after feedback onset at electrodes P7 and P8. We compared the mean amplitudes using a 3 (condition: own, joint, observed) x 2 (outcome: correct, error) ANOVAs for each component. All ANOVAs were Greenhouse-Geisser corrected where appropriate, and post-hoc comparisons were conducted with paired-samples t-tests.

## Results

Figure 2 shows that own errors elicited larger FRN amplitudes than joint errors, which elicited larger FRN amplitudes than observed errors. The ANOVA on FRN amplitudes

confirmed a significant effect of error type, $F(2, 40) = 22.22$, $p < .001$. Post-hoc tests confirmed that FRN amplitudes were significantly larger for own errors compared to joint errors, $t(20) = 2.77$, $p = .012$, and compared to observed errors, $t(20) = 5.34$, $p < .001$. FRN amplitudes were also significantly larger for joint errors compared to observed errors, $t(20) = 5.13$, $p < .001$. There were no differences in the latencies of the FRN peak or the preceding positive peak by error type (see Supplementary Material for details).

Figure 3 shows the mean P3 amplitudes elicited by each error type at each combination of location and time interval. The ANOVA on mean amplitudes showed a significant location by interval interaction, $F(1, 20) = 40.69$, $p < .001$, such that mean amplitudes were larger at frontocentral compared to parietocentral sites for the early interval, $t(20) = 3.22$, $p = .004$, whereas mean amplitudes were larger at parietocentral compared to frontocentral sites for the late interval, $t(20) = 3.97$, $p = .001$. This confirms that there were indeed two distinct subcomponents: an early component that peaked frontocentrally (the P3a) and a later component that peaked parietocentrally (the P3b). The ANOVA also showed a main effect of error type, $F(2, 40) = 4.73$, $p = .014$, which was qualified by an error type by location interaction, $F(2, 40) = 4.76$, $p < .014$, and an error type by location by interval interaction, $F(2, 40) = 6.52$, $p = .004$. All other main effects and interactions were non-significant, $F$s < 1.03, $p$s > .35. To further examine the 3-way interaction, we conducted 2 (location) x 3 (error type) ANOVAs separately for the P3a (early interval) and the P3b (late interval).

The ANOVA on P3a amplitudes confirmed a main effect of location, $F(1, 20) = 10.34$, $p = .004$, such that mean amplitudes were larger at frontocentral than parietocentral sites. The ANOVA also revealed a main effect of error type, $F(2, 40) = 5.95$, $p = .005$, and an error type by location interaction, $F(2, 40) = 9.12$, $p = .001$. The left panel of Figure 3c shows that at frontocentral sites, where the P3a was maximal, own errors elicited larger amplitudes compared to joint errors, $t(20) = 2.67$, $p = .015$, and compared to observed errors, $t(20) = 3.99$, $p = .001$, whereas amplitudes elicited by joint and observed errors did not differ, $t(20) = 1.17$, $p = .26$. At parietocentral sites, differences between error types were smaller, such that own errors elicited larger amplitudes than observed errors, $t(20) = 2.16$, $p = .043$, but the difference between own and joint errors was not significant, $t(20) = 1.85$, $p = .079$, nor was the difference between joint and observed errors, $t(20) = 0.44$, $p = .66$.

The ANOVA on P3b amplitudes revealed only a main effect of location, $F(1, 20) = 15.73$, $p = .001$, confirming that mean amplitudes were larger at parietocentral compared to frontocentral sites. There was no main effect of error type, $F(2, 40) = 2.03$, $p = .16$, or error type by location interaction, $F(2, 40) = 0.65$, $p = .53$.

Finally, the ANOVAs on mean P1 and N1 amplitudes showed no main effects or interactions, all $p$s > .05. Thus, participants initially attended equally the feedback stimulus irrespective of the person(s) who had produced the sequence and the correctness of their effort.

## Discussion

The current study compared ERP responses to feedback indicating that joint errors (resulting from two people's coordinated actions) or individual errors (resulting from a single person's own or another person's observed actions) had been committed. Differences between joint and individual errors were largest for the FRN, the amplitude of which was largest in response to a person's own errors, reduced for jointly committed errors, and further reduced for another person's observed errors. The P3a also differentiated between joint and individual errors, such that P3a amplitudes were larger for own errors compared to both jointly-committed and observed errors. In contrast, P3b amplitudes did not differentiate between joint and individual

errors. These findings indicate that producing an error together with a partner influences neural activity related to outcome evaluation (the FRN and P3a) but has less impact on activity related to adapting future behavior (the P3b).

The reduced FRN elicited by joint compared to own errors corroborates previous research showing that FRN amplitudes are reduced for action outcomes that are partly contingent on another person's passively observed action (Li et al., 2010) or on a computer's random selection (Martin & Potts, 2011; Yeung et al., 2005) compared to action outcomes that are fully contingent on a person's own action. The current findings are the first to show that the FRN is reduced for action outcomes that are partly contingent on another person's actions even when both people must actively and continuously adapt to each other's actions to achieve a desired outcome. The finding that FRN amplitudes are reduced when people share control over an action outcome is also consistent with previous research showing reduced FRN amplitudes when people believe that task outcomes are not controllable compared to controllable (Li et al., 2011).[2] The current findings also add to previous reports that the FRN is reduced for observed compared to own action outcomes (e.g., Bellebaum et al., 2010; Fukushima & Hiraki, 2009; Koban, Pourtois, Bediou, & Vuilleumier, 2012; Leng & Zhou, 2010; Yu & Zhou, 2006). In most of these studies, participants performed tasks independently and each was rewarded based on their own task performance; only in Koban et al. (2012) did participants' independent outcomes result in a combined reward for both participants. The current findings extend the pattern of self-other differences in outcome processing to a context in which participants' outcomes not only resulted in combined rewards but were also the product of active interpersonal coordination.

One possible explanation for the modulation of FRN amplitudes in the current study is that shared control reduced the affective value of joint errors relative to own errors, and likewise the absence of control reduced the affective value of observed errors relative to own and joint errors. Some researchers have argued that action outcomes that are less contingent on a person's actions have reduced affective value compared to outcomes that are fully contingent on the person's actions (Yeung et al., 2005). Moreover, the affective value of action outcomes modulates the FRN in both social and non-social contexts (see Koban & Pourtois, 2014, for a review). A second possibility is that shared control altered the subjective probability of correct vs. incorrect outcomes. Outcomes that are less objectively or subjectively probable elicit larger FRNs (Walsh & Anderson, 2012). In the current study, the objective probability of correct vs. incorrect outcomes was equated between individual and joint settings through the use of an adaptive window to determine accuracy. However, people often experience a greater subjective probability of positive outcomes than is warranted by the objective probability (e.g., Hajcak, Moser, Holroyd, & Simons, 2007; Miller & Ross, 1975). People are also more optimistic about their own performance compared to other people's (Krueger, 1998). Thus, participants in the current study may have expected more correct outcomes for self-produced sequences compared to joint and observed sequences, rendering errors more unexpected and therefore eliciting larger FRNs in the former conditions (see Li et al., 2010, for a similar argument). Further work is needed to determine whether and to what extent the affective value and subjective probability of action outcomes are modulated by acting in coordination with a partner.

The P3a has received relatively less attention than the FRN in the action-monitoring literature. Most researchers treat the P3 that follows the FRN as a unitary component, although recent work indicates that the P3a and P3b subcomponents are differentially sensitive to the valence and magnitude of reward feedback (West, Bailey, Anderson, & Kieffaber, 2014). In the current study, P3a amplitudes were larger for own errors compared to joint or observed errors.

Given previous research indicating that the P3a reflects an attentional response related to the initial evaluation of a stimulus as task-relevant (Polich, 2007), this finding suggests that people may allocate more attentional resources to, and evaluate as more task-relevant, outcomes over which they have full control compared to outcomes over which they share or have no control. The larger P3a amplitudes elicited by own compared to joint errors in the current study is consistent with West et al.'s (2014) finding that both FRN and P3a amplitudes were larger when Blackjack players experienced negative outcomes that could be attributed primarily to their own actions compared to outcomes that could be attributed in part to the dealer's actions. The lack of difference between P3a amplitudes elicited by joint and observed errors suggests that the P3a may be particularly sensitive to differences between a person's own action outcomes and outcomes that can be partly or fully attributed to other people.

Previous research examining how control over action outcomes influences the P3 has focused primarily on later activity at more posterior electrodes (i.e., Cz, CPz, and Pz; Li et al., 2010; Yeung et al., 2005; Martin & Potts, 2011). This work has shown that P3 amplitudes are larger for outcomes that are fully contingent on a person's own actions compared to outcomes that are partly or not at all contingent on the person's actions (e.g., Li et al., 2010; Yeung et al., 2005). The current study revealed no significant differences in later, more posterior (i.e., P3b) amplitudes across conditions, despite the fact that own errors were fully contingent on the participant's actions whereas joint and observed errors were not. Although this may seem contradictory to previous findings, it might instead highlight differences in the cognitive and neural processes elicited by performance monitoring compared to gambling tasks. The current paradigm allowed participants to adapt their behaviour after errors in order to influence subsequent outcomes, whereas the gambling tasks used in previous studies did not. In the current paradigm, both own and joint errors signaled a need to adapt future behaviour, regardless of the degree of control participants had over the error. In addition, observed sequences were randomly intermixed with self-produced sequences, which may have encouraged participants to learn from their partners' mistakes as well as their own. The lack of differences between conditions is in line with the notion that P3b amplitudes reflect participants' motivation to adapt future behaviour (Nieuwenhuis et al., 2004), although it may be worth noting that there were numerical differences in P3b amplitudes that scaled with control over action outcomes. It is therefore possible that differences between joint and individual errors could be evident in P3b amplitudes if an alternative experimental design was implemented.

## Conclusion

The neural processes involved in monitoring multiple people's coordinated actions have just begun to be investigated. Although previous work has examined how people monitor each person's individual outcomes within a joint action (e.g., the individual tones that comprise a musical duet; Loehr, Kourtis, Vesper, Sebanz, & Knoblich, 2013; see also Picton, Saunders, & Jentzsch, 2012), the current study is the first to investigate the effects of interpersonal coordination on monitoring and evaluating action outcomes. We show that producing errors together with another person reduces neural activity related to outcome evaluation but has less impact on neural activity associated with the motivation to adapt future behaviour. These findings further our understanding of how people monitor and evaluate joint action outcomes to ensure that shared goals are achieved, and may have important implications for investigations of pathological conditions that entail social neurocognitive deficits such as psychopathy and autism.

## Footnotes

[1]Data were initially analyzed separately by which participant started the sequence in the joint setting. Because there were no differences based on who started the joint sequence in any of the analyses, all *p*s > .25, we collapsed across starting person in all analyses reported here.

[2]It is possible that sharing control over the action outcome may have induced participants to feel shared responsibility for the outcome. Indeed, control and responsibility often go hand in hand; for example, Li et al.'s (2011) manipulation of people's beliefs about control affected their feelings of responsibility, which correlated with FRN amplitudes. We use the term shared control to capture the fact that participants produced the action sequences together, while remaining agnostic with respect to their feelings of responsibility.

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## Figure Captions

*Figure 1*. Schematic illustration of the sequence production task in the individual and joint settings. Following instructions and fixation, participants heard a series of isochronous pacing clicks (illustrated by eighth note symbols) and then produced a sequence of tones (illustrated by combined button press and eighth note symbols, labeled P1 and P2 for Participants 1 and 2, respectively). After producing the last tone, participants received feedback indicating whether the sequence they produced matched the pace set by the isochronous clicks.

*Figure 2*. (a) Grand-average difference waves for each error type at electrode FCz, time-locked to feedback onset, and the scalp voltage distribution of the grand-average difference wave within the time window of analysis of the FRN. (b) Mean peak-to-peak amplitudes of the FRN for each error type.

*Figure 3*. (a) Grand-average difference waves for each error type at electrode Fz, time-locked to feedback onset, and the current source density of the grand-average difference wave within the time window of analysis of the P3a. (b) Grand-average difference waves for each error type at electrode Pz, time-locked to feedback onset, and the current source density of the grand-average difference wave within the time window of analysis of the P3b. (c) Mean amplitudes for each error type by location and time interval.

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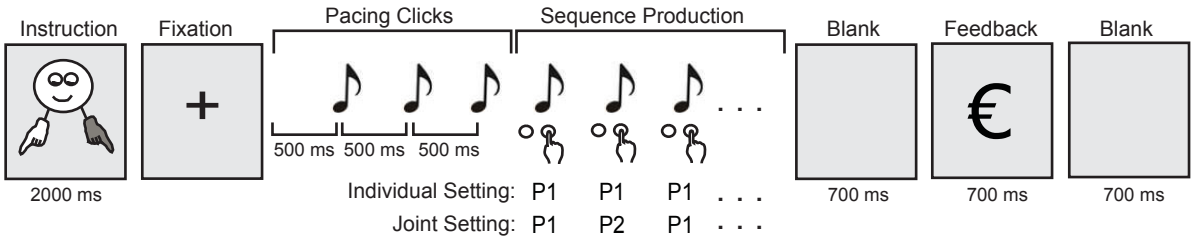
Instruction
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500 ms 500 ms 500 ms
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Individual Setting: P1 P1 P1 . . .
Joint Setting: P1 P2 P1 . . .

Figure 1

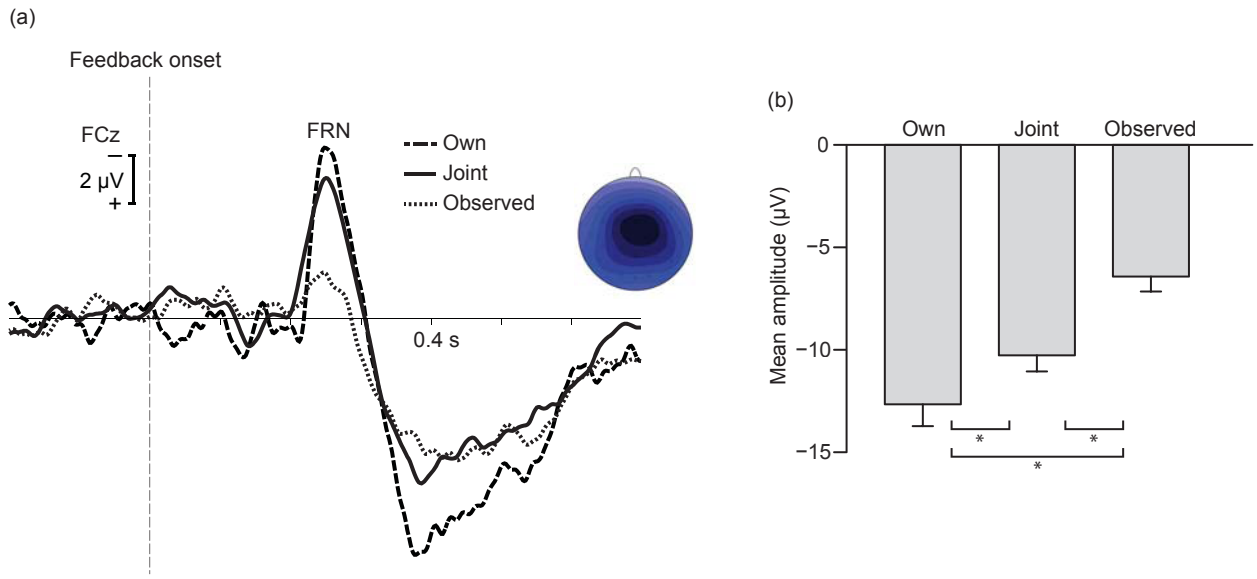
(a)
Feedback onset
FCz
2 μV
FRN
Own
Joint
Observed
0.4 s
(b)
Own
Joint
Observed
0
−5
−10
−15
Mean amplitude (μV)
*
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Figure 2

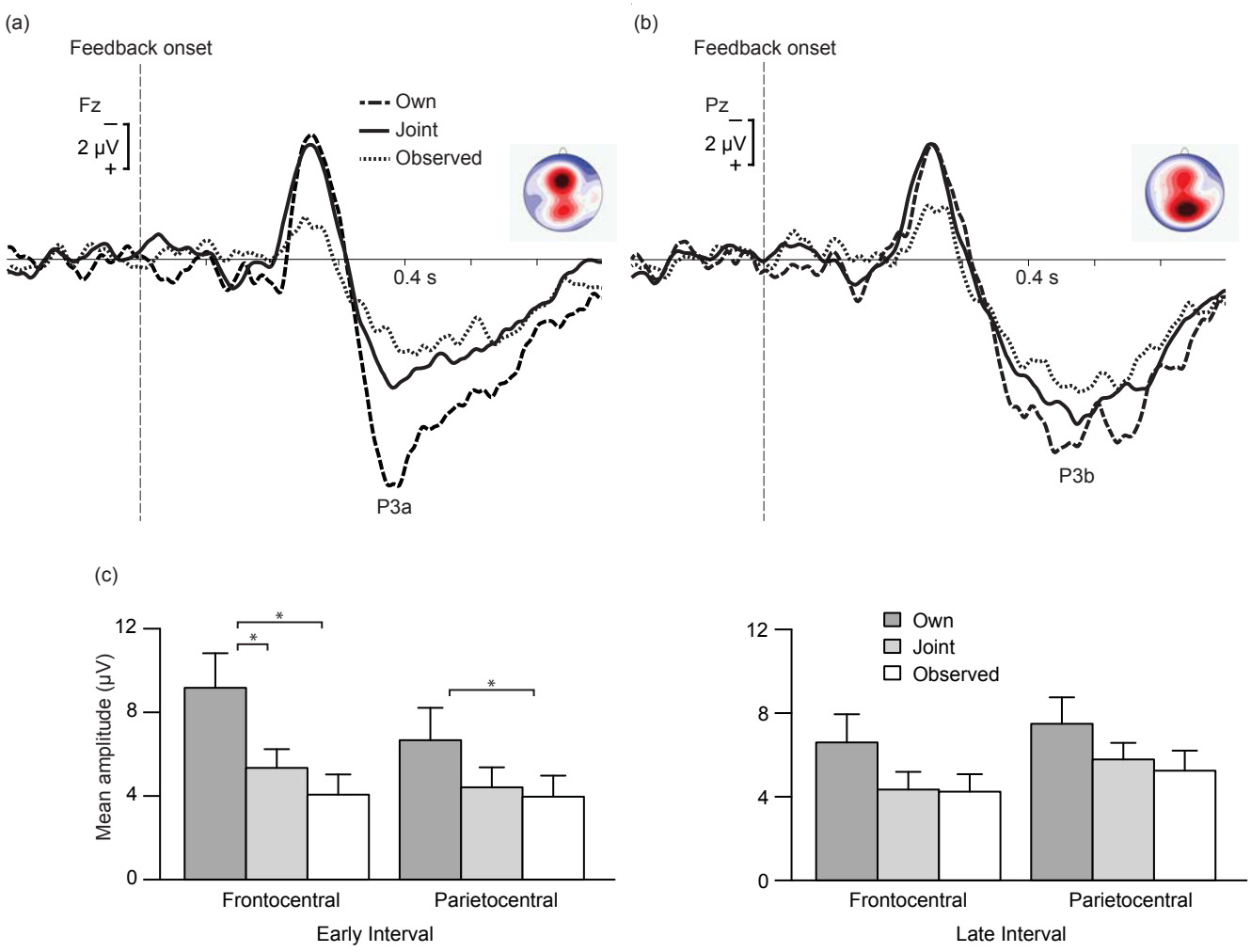
(a)
Feedback onset
Fz
2 μV
Own
Joint
Observed
0.4 s
P3a
(b)
Feedback onset
Pz
2 μV
0.4 s
P3b
(c)
Mean amplitude (μV)
12
8
4
0
*
*
*
Frontocentral
Parietocentral
Early Interval
Own
Joint
Observed
Frontocentral
Parietocentral
Late Interval

Figure 3

# Supplementary Material

## Behavioral Data

We examined participants' performance in the individual and joint settings by calculating the absolute IOI error (defined as the absolute difference between the mean IOI of each sequence and the required interval of 500 ms) for each trial. We compared the mean absolute IOI error for correct and error trials in the individual and joint settings using a 2 (setting) by 2 (accuracy) repeated-measures ANOVA.

The mean absolute IOI errors (± *SD*) were 8.84 ± 2.97 ms and 11.29 ± 5.64 ms for correct trials in the individual and joint settings, respectively, and 30.64 ± 14.25 ms and 35.70 ± 14.11 ms for error trials in the individual and joint settings. An ANOVA confirmed a main effect of accuracy, $F(1, 10) = 57.10$, $p < .001$, and indicated a trend toward slightly larger errors in the joint compared to the individual setting, $F(1, 10) = 4.47$, $p = .061$. The interaction was not significant, $F(1, 10) = 1.84$, $p = .20$.

## ERP Latencies

ANOVAs comparing the latencies of the FRN peak and the preceding positive peak across error types revealed no significant differences. For the FRN peak, mean latencies (± *SD*) were 259.7 ± 34.2 ms, 252.0 ± 21.6 ms, and 252.1 ± 40.2 ms for own, joint, and observed errors, respectively, $F(2, 40) = 0.52$, $p = .60$. For the positive peak that preceded the FRN, mean latencies (± *SD*) were 165.9 ± 40.9 ms, 165.4 ± 24.7 ms, and 161.9 ± 35.0 ms for own, joint, and observed errors, respectively, $F(2, 40) = 0.16$, $p = .85$.

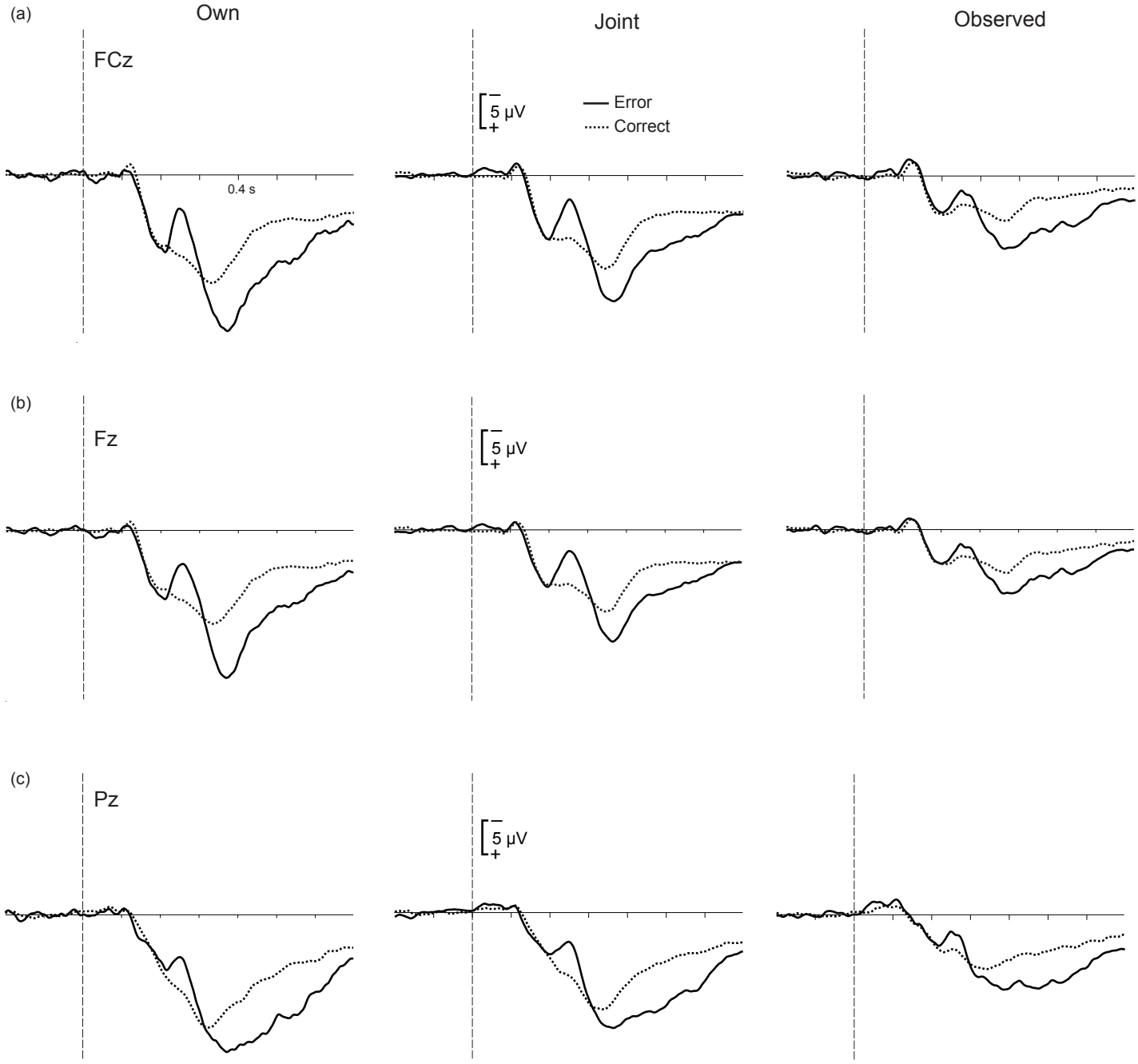


*Figure S1.* ERP waveforms elicited by error and correct feedback at electrodes (a) FCz, (b) Fz, and (c) Pz for each error type.