Dynamic Adjustments of Cognitive Control: Oscillatory Correlates of the Conflict Adaptation Effect

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Abstract

■ It is a prominent idea that cognitive control mediates conflict adaptation, in that response conflict in a previous trial triggers control adjustments that reduce conflict in a current trial. In the present EEG study, we investigated the dynamics of cognitive control in a response-priming task by examining the effects of previous trial conflict on intertrial and current trial oscillatory brain activities, both on the electrode and the source level. Behavioral results showed conflict adaptation effects for RTs and response accuracy. Physiological results showed sustained intertrial effects in left parietal theta power, originating in the left inferior parietal cortex, and midcentral beta power, originating in the left and right (pre)motor cortex. Moreover, physiological analysis revealed a current trial conflict adaptation effect in midfrontal theta power, originating in the ACC. Correlational analyses showed that intertrial effects predicted conflict-induced midfrontal theta power in currently incongruent trials. In addition, conflict adaptation effects in midfrontal theta power and RTs were positively related. Together, these findings point to a dynamic cognitive control system that, as a function of previous trial type, up- and down-regulates attention and preparatory motor activities in anticipation of the next trial. ■

INTRODUCTION

Cognitive control refers to the human ability to flexibly switch between different thoughts and actions in response to changing task demands and internal needs. In particular, it refers to the ability to detect and resolve conflicting response tendencies by switching from automatic to controlled response modes in a context-dependent matter, thus enabling humans to intentionally carry out an appropriate but weak response in the face of inappropriate but prepotent responses (Miller & Cohen, 2001). In the last decade, mechanisms of conflict resolution between appropriate and inappropriate responses have been subject to intensive research. Using response conflict tasks like the Stroop task (Stroop, 1935), the Eriksen flanker task (Eriksen & Eriksen, 1974), the Simon task (Simon & Small, 1969), and the response-priming task (Rosenbaum & Kornblum, 1982), it has been shown that, on the single-trial level, the amount of response interference in a current trial crucially depends on the presence or absence of response conflicts in previous trials (e.g., Gratton, Coles, & Donchin, 1992). In these tasks, cognitive control is assumed to regulate conflict between task-relevant and task-irrelevant response activations and, in particular, to mediate conflict adaptation on the single-trial level, in that conflict in trial $n - 1$ triggers control adjustments that reduce conflict in trial n (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001).

Behavioral work provided ample evidence that congruency effects on a current trial can be modulated by the congruency on the previous trial (for a review, see Egner, 2007). In the response-priming task, for instance, the response to a target (e.g., a left-hand response) is faster and less error prone when the target is preceded by a (task-irrelevant) prime that activates the correct response (e.g., a left-hand response) as compared with a prime that activates an incorrect response (e.g., a right-hand response), typically resulting in a congruency effect (e.g., Fröber & Dreisbach, 2012; Kunde & Wühr, 2006; Kunde, 2003; Vorberg, Mattler, Heinecke, Schmidt, & Schwarzbach, 2003; Klotz & Neumann, 1999). Importantly, in this task, the current trial congruency effects for RTs and response accuracy are smaller after incongruent than after congruent trials, reflecting a conflict adaptation effect (Kunde & Wühr, 2006; Kunde, 2003). Following the view that cognitive control mediates conflict in the response conflict task, the detection of conflict in trial $n - 1$ is assumed to trigger control adjustments that down-regulate the influence of the prime on the processing of the target in trial n (Kunde & Wühr, 2006).

Prior work on the physiological basis of conflict adaptation has shown that the ACC is a hub region in the processing of response conflict (Botvinick, Cohen, & Carter, 2004; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Imaging studies, for instance, have shown that ACC activity in currently incongruent trials depends on previous trial congruency in various response conflict

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tasks (e.g., Kerns, 2006; Kerns et al., 2004; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). In particular, ACC activation is reduced on conflict trials following conflict trials as compared with conflict trials following nonconflict trials (Kerns et al., 2004). ERP studies have further demonstrated that the midfrontal N200 and N400, both originating in ACC, index response conflict and conflict adaptation (e.g., Clayson & Larson, 2011; Forster, Carter, Cohen, & Cho, 2011; Larson, Kaufman, & Perlstein, 2009; Hanslmayr et al., 2008). Moreover, recent work on brain oscillations has shown that midfrontal theta power (4–8 Hz), originating in ACC, relates to the processing of response conflict in various tasks (Cavanagh, Zambrano-Vazquez, & Allen, 2012; Nigbur, Cohen, Ridderinkhof, & Stürmer, 2012; Pastötter, Berchtold, & Bäuml, 2012; Cohen & Cavanagh, 2011; Nigbur, Ivanova, & Stürmer, 2011; Pastötter, Hanslmayr, & Bäuml, 2010; Hanslmayr et al., 2008). Going beyond this recent work, in this study, we examined whether theta power in currently incongruent trials depends on previous trial congruency in the response-priming task.

Prominent models of cognitive control assume that ACC monitors for conflict in trial $n - 1$ and after detection sends signals to the dorsolateral PFC, which implements control adjustments to reduce conflict in trial n (Mansouri, Tanaka, & Buckley, 2009; Carter & van Veen, 2007; Botvinick et al., 2001). Regarding the dynamics of cognitive control, it has been suggested that control adjustments may be implemented in a preparatory way, during intertrial intervals, between trial $n - 1$ and trial n. For instance, theoretical work suggested that conflict adaptation may involve learning and memory processes that maintain information about experienced conflict within and across trials (Mansouri et al., 2009; Botvinick et al., 2001). In addition, recent physiological work showed that postconflict control adjustments may alter attention and motor activities in an anticipatory manner (Compton, Huber, Levinson, & Zheutlin, 2012; Horga et al., 2011). Consistently, in a recent imaging study using the Simon task, examination of postconflict adjustments in blank trials following incongruent trials revealed increased brain activations in the left prefrontal and the left parietal cortex. These increased blank trial activations were related to conflict-induced brain activations in ACC and the left pre-SMA in incongruent trials (Horga et al., 2011). Moreover, recent EEG work by Compton and colleagues, examining alpha oscillations around 12 Hz in intertrial intervals (ITIs) in the Stroop task, showed that intertrial alpha power is modulated by previous trial type, with larger alpha power increases after incongruent trials than after congruent ones (Compton et al., 2012) and larger increases after neutral trials than after incongruent ones (Compton, Arnstein, Freedman, Dainer-Best, & Liss, 2011). These findings support the view that postconflict control adjustments are implemented in a preparatory way. In this study, we extended the prior work by investigating whether conflict-induced adjustments of intertrial brain oscillations, that is, brain oscillations between trial $n - 1$ and trial n, are related to behavioral and physiological conflict adaptation effects in trial n.

An EEG study is reported in which we examined the oscillatory correlates of sequential conflict adaptation using the response-priming task that has proven useful to study response priming and response conflict in prior work (Pastötter et al., 2010, 2012). In this task, response conflict arises when a correct motor response to a target (e.g., a left-hand response) is preceded by a spatial cue that primes an alternative motor response (e.g., a right-hand response). Focusing on current trial processing, in the prior work, we showed that conflict processing as reflected by a theta power increase in ACC can constrain response priming as reflected by a beta power (15–25 Hz) decrease in the motor cortex, slowing response processing and lowering response errors within the same trial (Pastötter et al., 2010). In the present experiment, we examined the effects of conflict adaptation on both intertrial and current trial brain oscillations. On the behavioral level, we predicted sequential conflict adaptation in terms of reduced congruency effects following incongruent trials. In the EEG data, this sequential conflict adaptation should be reflected in midfrontal theta power, with lower conflict-induced theta power in incongruent trials after incongruent trials than after congruent ones. Following the view that cognitive control mediates conflict adaptation in an anticipatory way (Horga et al., 2011; Mansouri et al., 2009), previous trial type was expected to affect intertrial theta and beta brain oscillations indexing attention and motor control (Cheyne, 2013; Sauseng, Hoppe, Klimesch, Gerloff, & Hummel, 2007). In particular, effects on intertrial brain oscillations were expected to predict the conflict adaptation effect in midfrontal theta power. Finally, conflict adaptation effects in midfrontal theta power and behavior were expected to be related in incongruent trials.

METHODS

Participants

Twenty-five students (17 women, 8 men) at Regensburg University, Germany, with a mean age of 22.1 years (SD = 3.2 years) participated in the study. All participants gave written informed consent, reported normal or corrected-to-normal vision, and were paid €12 for participation. With three exceptions, all participants were right-handed. No participant reported any history of neurological disease. The study was conducted in accordance with the Declaration of Helsinki.

Materials and Procedure

Participants viewed a computer screen from a distance of 150 cm and were instructed to maintain fixation on a centrally located gray fixation cross throughout the

whole experiment and to not move the eyes. The screen background was black and displayed two gray square outline boxes (2.5° × 2.5°) centered 6.5° above and below fixation at all times (Figure 1A). Two-hundred sixty-four (88%) of the 300 single trials were cued trials in which, after an ITI of variable duration (2300–2700 msec), the cue, a brightening of one of the two square boxes, was presented for 100 msec, at either location with equal probability. After a variable delay of 50–150 msec, the target, a red cross (1.25°), was presented within one of the two boxes. In half of the cued trials, cue and target occurred at the same spatial position (congruent trials, C trials hereafter), and in the other half, cue and target occurred at opposite spatial position (incongruent trials, I trials hereafter). Cue–target onset asynchrony ranged from 150 to 250 msec. Intermixed with the cued trials, 36 (12%) uncued trials were run in which no cue was presented and the preceding ITI was extended by 100 msec. Uncued trials were included to ensure that participants were highly attentive to both cue and target presentation.

Participants responded to the targets' spatial location. Two response keys were marked on the computer keyboard, one on the upper left side and one on the upper right side. Participants were instructed to press the left key with the index finger of their left hand for targets that appeared above fixation and to press the right key with the index finger of their right hand for targets that appeared below fixation. They were told to not respond to the cues. Targets remained on the screen until participants made a response or 1000 msec elapsed, followed by the ITI. Both speed and accuracy were stressed in the instruction. No feedback on speed or accuracy was provided.

Order of congruent trials, incongruent trials, and uncued trials was randomized within the constraints that none of these three trial types were repeated more than four times in a row and that target location changed after at least five successive trials. Furthermore, the frequency of congruent trials following congruent trials (CC), congruent trials following incongruent trials (IC), incongruent trials following congruent trials (CI), and incongruent trials following incongruent trials (II) was counterbalanced across participants. Presentation and randomization were done with E-Prime software (v1.1.4, Psychology Software Tools, Sharpsburg, PA).

Analysis of Behavioral Data

Mean median RTs and mean error rates were analyzed based on a 2 × 2 design with the within-subject factors of Previous Trial Type (C, I) and Current Trial Type (C, I). For analysis of RTs, mean medians were analyzed to reduce the influence from outliers in the single-trial data (Miller, 1991); only trials for which responses on both the current and the previous trial were correct were included. For analysis of error rates, only commission errors were included in the analysis; omission errors were

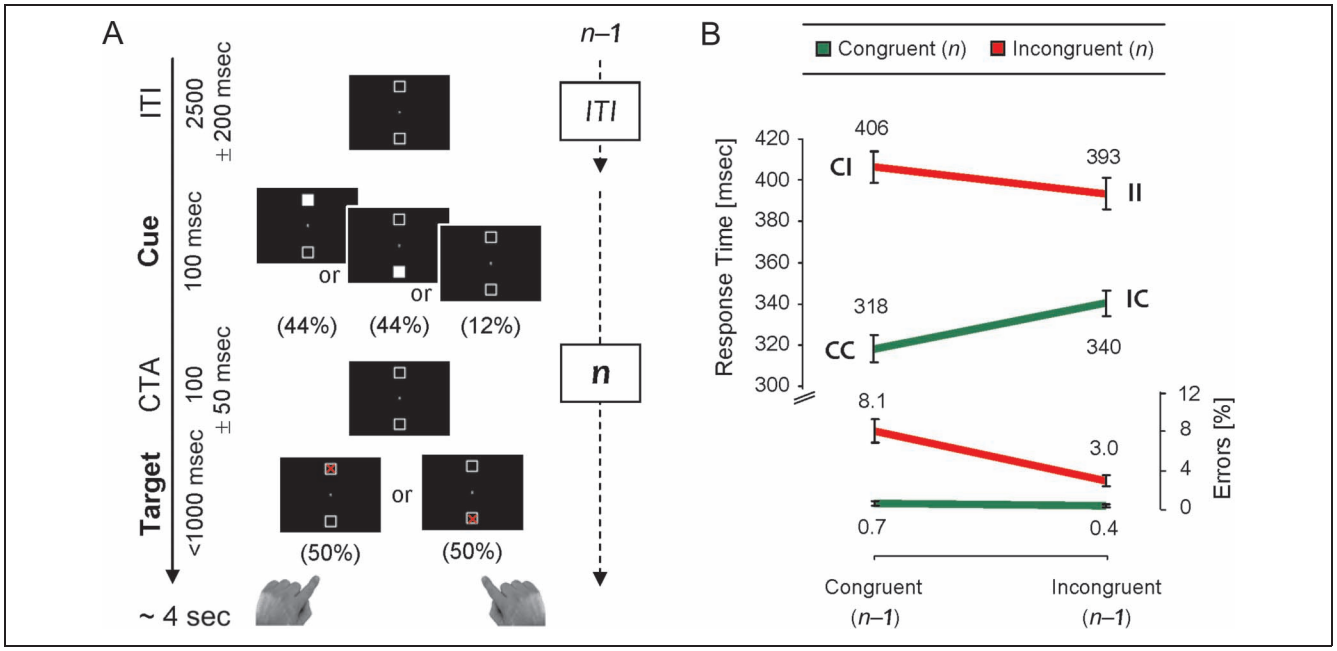

Figure 1. Experimental procedure and behavioral results. (A) Following a previous trial (trial $n - 1$) and an ITI of variable duration, in the current trial (trial n), participants responded to the location of a target, a red cross, presented in one of two vertically aligned square boxes with the index finger of their left hand (to targets above fixation) or right hand (to targets below fixation). Targets were validly or invalidly cued by brightening of one of the two square boxes shortly before target presentation or uncued. (B) Both RT and error rates showed a reliable conflict adaptation effect, that is, differences in RT and error rates between currently incongruent and currently congruent trials were larger after congruent trials than after incongruent ones. Error bars represent standard errors of the mean.

infrequent and were not included. For errors, single-subject data were arcsine-transformed to improve normality and homogeneity of variance. To avoid estimation problems, transformation was improved by replacing data points with 0% response errors with $(1/4n)$, where n represents the number of observations (Mosteller & Youtz, 1961). Analyses of left- versus right-hand responses showed that this factor did not influence the main findings in RT, error rate, intertrial effects, and current trial theta power (all ps $> .05$); therefore, this factor was not further analyzed, and data were collapsed over both hands instead. In uncued trials, mean median RT was 421 msec ($SE = 8.0$) and mean error rate was 0.4% ($SE = 0.2$); because of the small number of uncued trials, physiological data of the uncued trials were not analyzed.

Recording and Preprocessing of EEG Data

EEGs were recorded from 61 equidistant active electrodes mounted in an elastic cap (ActiCAP, Montage 10, Brain Products, Gilching, Germany). ActiCAP with its active electrode system enables fast electrode placement and low electrode skin impedance because of amplification circuitry built into the electrodes, boosting the signal and reducing the noise. Electrode skin impedance was kept below 20 kΩ. Vertical and horizontal eye movements were recorded from two additional channels. Electrode Cz served as common reference. Signals were digitalized with a sampling rate of 500 Hz and amplified between 0.15 and 100 Hz with a notch filter at 50 Hz, removing line noise (BrainAmpMR plus, BrainVision Recorder, v1.20, Brain Products).

EEG recordings were rereferenced off-line against average reference and EOG corrected by using calibration data and generating individual EOG artifact coefficients (Ille, Berg, & Scherg, 2002), as implemented in the BESA Research software package (v5.3.7, BESA Software, Gräfelfing, Germany). Remaining artifacts were marked by careful visual inspection. EEG data were segmented into epochs ranging from −1000 msec preceding target onset in trial $n - 1$ to 1000 msec following target onset in trial n. Segments containing artifacts, segments with RTs outside the range of 100–1000 msec in trial n, segments with response errors either on trial n or on trial $n - 1$ were excluded from further analysis. Finally, for each single subject, numbers of CC, IC, CI, and II segments that went into analysis were matched to be equal by randomly selecting, for each of the four conditions, exactly the number of segments available in the condition with the smallest number of remaining segments. Random selection of trials did not affect the results, as suggested by three additional independent samplings of the original data set. Across participants, mean number of artifact-free segments for each of the four conditions (CC, IC, CI, II) was 41.7 ($SD = 5.6$), with a range from 34 to 53 segments per condition and subject.

Spectral Analysis of EEG Data

EEG data were transformed into the time–frequency domain using a complex demodulation algorithm as implemented in BESA Research (v5.3.7; see Hoechstetter et al., 2004). The algorithm consists of a multiplication of the time domain signal with a complex periodic exponential function, having a frequency equal to the frequency under analysis, and subsequent low-pass filtering. The low-pass filter is a finite impulse response filter of Gaussian shape in the time domain, which is related to the envelope of the moving window in wavelet analysis. The data were filtered in a frequency range from 2 to 30 Hz. Time resolution was set to 78.8 msec (FWHM), and frequency resolution was set to 1.42 Hz (FWHM). Time–frequency data were exported in bins of 50 msec and 1 Hz.

Event-related power (Pfurtscheller & Aranibar, 1977) was examined by calculating percentage of power decrease and percentage of power increase following target presentation in trial $n - 1$, during the ITI, and following target presentation in trial n in relation to a prestimulus baseline interval, which was set from −750 to −250 msec before target onset in trial $n - 1$ for all analyses. Power data were collapsed from 4 to 8 Hz in the theta frequency range and from 15 to 25 Hz in the beta frequency range. Examination of individual time–frequency plots showed that these frequency ranges were suitable for all participants.

A priori, theta and beta power were analyzed both with and without subtracting the evoked signal. As it turned out, the conclusions were the same with and without subtraction. The present results are based on analyses without subtraction. We do not report on detailed alpha findings because, in the 400-msec interval following target onset, alpha activities were superimposed by dominant theta power.

Statistical Analysis of Time–Frequency Data

To control for the problem of multiple comparisons, cluster-based random permutation analyses were calculated to test the significance of differences between conditions at multiple electrode sites (Maris & Oostenveld, 2007), as implemented in BESA Statistics (v1.0). For conflict adaptation analysis, we examined conflict-induced theta power, that is, the difference in theta power between C and I trials, averaged over time from 200 to 400 msec following target onset in trial n, as a function of previous trial type. For analyses of intertrial effects, we examined theta and beta power, both averaged over time from −1500 to −500 msec before target onset in trial n, as a function of previous trial type.

In each permutation analysis, a paired t test on the difference in averaged time–frequency data between conditions was calculated for each electrode site. Clusters were identified by considering only those (at least five) contiguous electrode sites (with maximum distance of

45 mm between neighboring sites, resulting in an average of 4.87 neighbors per electrode site) that fell below a p value of .01 in the t test. For each cluster, the sum of t values of the single electrodes was calculated as a test statistic. In each permutation, this statistic was repeated for shuffled data in which data were randomly reordered across conditions and the cluster with the highest sum of t values was kept. By these means, a null distribution was created from 5000 permutations, and the critical p value for the empirically derived cluster was calculated.

Averaged over electrodes of significant clusters, two-way repeated-measures ANOVAs on current trial theta power were calculated as a function of Previous Trial Type (C, I) and Current Trial Type (C, I). For correlational analyses, nonparametric Spearman correlations were calculated across participants.

Source Analysis

To localize neuronal sources of effects from the electrode level, a multiple-source beamformer was used, as implemented in BESA Research (v5.3.7). The multiple-source beamformer is a modified version of the linearly constrained minimum variance vector beamformer (Gross et al., 2001) that allows to image sources of differences in event-related power between conditions in user-defined time–frequency ranges. Cluster-based random permutation analyses and plotting of sources were done with BESA Statistics (v1.0). In each permutation analysis on the source level, paired t tests on the differences in the averaged time–frequency data between conditions were calculated for each voxel (voxel size in Talairach space was set to 7 mm). Clusters were identified by considering adjacent voxels that fell below a p value of .01 in the t test; no minimum number of voxels in a cluster was set. The test statistic was the sum of t values of all voxels in a cluster. In each analysis, 5000 random permutations were run. Anatomic labeling of neuronal sources was feasible using the WFU Pickatlas (v2.0) software toolbox (Maldjian, Laurienti, Kraft, & Burdette, 2003).

RESULTS

Behavioral Results

RTs

Mean median RTs are shown in Figure 1B. A two-way ANOVA with the factors of Previous (C vs. I) and Current Trial Type (C vs. I) revealed main effects of both Previous, $F(1, 24) = 7.8, p < .01$, and Current Trial Type, $F(1, 24) = 202.9, p < .001$. Responses were faster on trials following Ctrials than following I trials (362 msec vs. 367 msec), and responses were faster on current C trials than on current I trials (329 msec vs. 400 msec).

More importantly, the ANOVA also revealed an interaction between the factors of Previous and Current Trial Type, $F(1, 24) = 91.3, p < .001$, indicating sequential conflict adaptation in RT. In particular, C trials were answered faster following C trials than following I trials (318 msec vs. 340 msec; $t_{24} = 8.7, p < .001$), and conversely, I trials were answered faster following I trials than following C trials (406 msec vs. 393 msec; $t_{24} = 5.3, p < .001$). The current trial congruency effect, that is, the difference in RTs between I and C trials, was larger after C trials (CI–CC: 88 msec) than after I trials (II–IC: 54 msec), but significant in both conditions ($t_{24} = 16.3, p < .001$; $t_{24} = 10.3, p < .001$).

Response Errors

Error data by and large mirrored the RT data (Figure 1B). A two-way ANOVA with the factors of Previous (C vs. I) and Current Trial Type (C vs. I) revealed main effects of both Previous, $F(1, 24) = 30.1, p < .001$, and Current Trial Type, $F(1, 24) = 42.8, p < .001$. Responses were more error prone on trials following C trials than following I trials (4.4% vs. 1.7%), and responses were less error prone on current C trials than on current I trials (0.6% vs. 5.5%).

Moreover, the ANOVA revealed an interaction between the two factors, $F(1, 24) = 31.1, p < .001$, indicating sequential conflict adaptation in accuracy. Whereas accuracy in C trials was not influenced by congruency in the previous trial (0.7% vs. 0.4%; $t_{24} < 1$), responses on I trials were more error prone following C trials than following I trials (8.1% vs. 3.0%; $t_{24} = 5.5, p < .001$). The current trial congruency effect, that is, the difference in response errors between I and C trials, was larger after C trials (CI–CC: 7.4%) than after I trials (II–IC: 2.6%), but significant in both conditions ($t_{24} = 6.9, p < .001$; $t_{24} = 4.6, p < .001$). However, given the very low error rate on C trials, these results should be interpreted with caution.

Physiological Results

Conflict Adaptation Effect in Theta Power

Our first step in the analysis of the physiological data was to examine whether current trial theta power showed a conflict adaptation effect. On the electrode level, a cluster-based permutation analysis of conflict-induced theta power, that is, the difference in theta power between I and C trials (from 200 to 400 msec following target onset in trial n) as a function of previous trial type (CI–CC vs. II–IC) was calculated. This analysis revealed a significant mid-frontal electrode cluster ($p < .001$; Figure 2A) showing lower conflict-induced theta power after I trials (II–IC) than after C trial (CI–CC), thus indicating a conflict adaptation effect in trial-averaged theta power. On the source level, this conflict adaptation effect in theta power was localized to a cluster of voxels extending on the left hemisphere from the left cingulate gyrus in Brodmann's area 24 (Montreal Neurological Institute [MNI] coordinates: $x = -18, y = -20, z = 42$; Figure 2B) to the pre-SMA.

Figure 2C shows time–frequency spectrograms of (significant) differences in conflict-induced oscillatory power

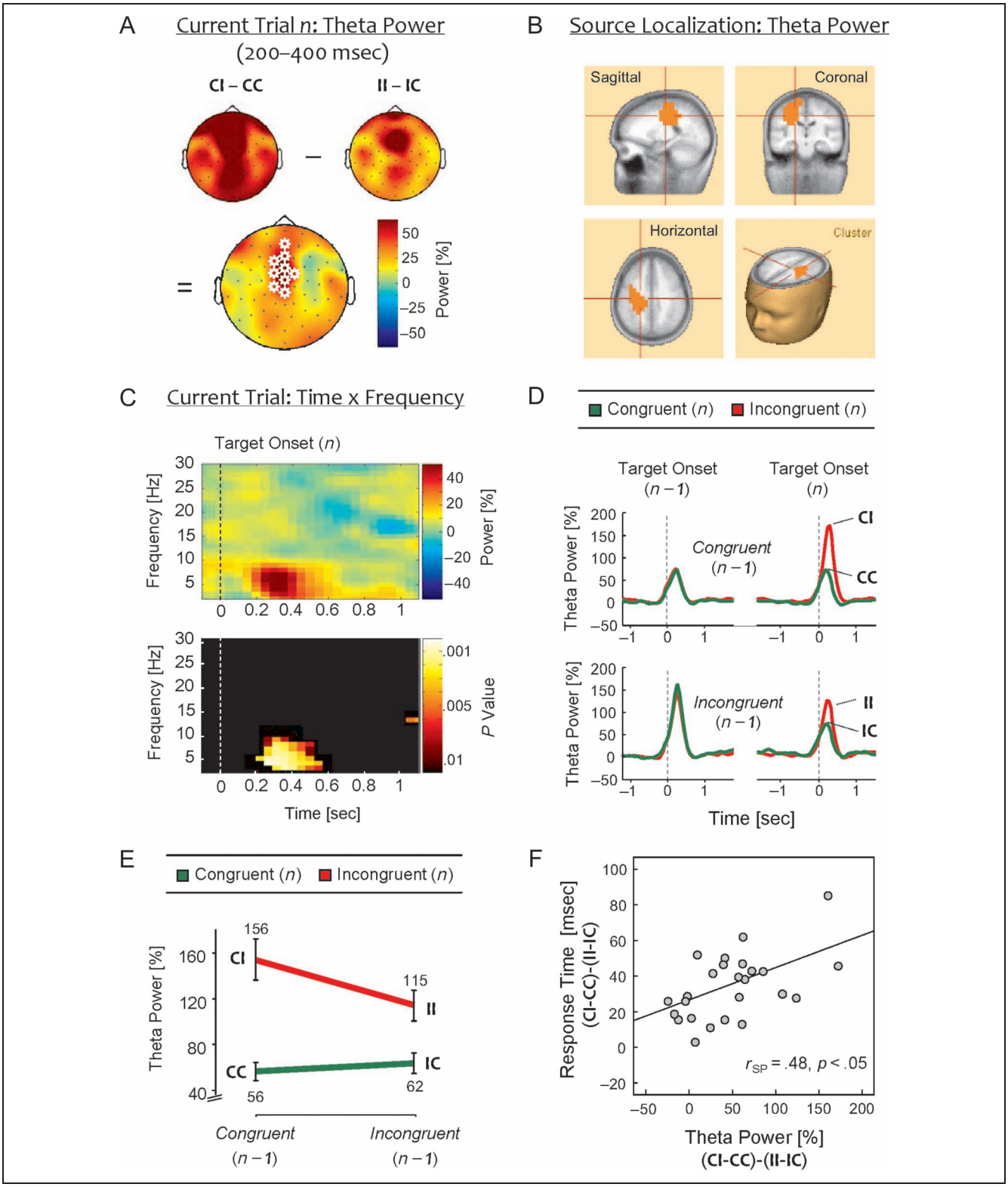

Figure 2. Conflict adaptation effect in theta power. (A) Topographies of the conflict adaptation effect in midfrontal theta power, that is, the difference in theta power between currently incongruent and congruent trials (from 200 to 400 msec following target onset in trial *n*) as a function of previous trial type (CI–CC vs. II–IC). Warm color coding indicates power increases; cold color coding indicates power decreases. (B) The conflict adaptation effect in theta power was localized to the left ACC (extending to the left pre-SMA); sagittal slice seen from the left, coronal slice seen from the back, horizontal slice seen from the top. (C) Time–frequency spectrograms of (significant) power differences, averaged over electrodes of the midfrontal cluster and time-locked to target onset in trial *n*. (D) Time courses of midfrontal theta power as a function of previous and current trial type, time-locked to target onsets in trials $n - 1$ and *n*. (E) Midfrontal theta power from 200 to 400 msec following target onset in trial *n* as a function of previous and current trial type; error bars represent standard errors of the mean. (F) Brain–behavior correlation between the physiological conflict adaptation effect in midfrontal theta power and the behavioral conflict adaptation effect in RT.

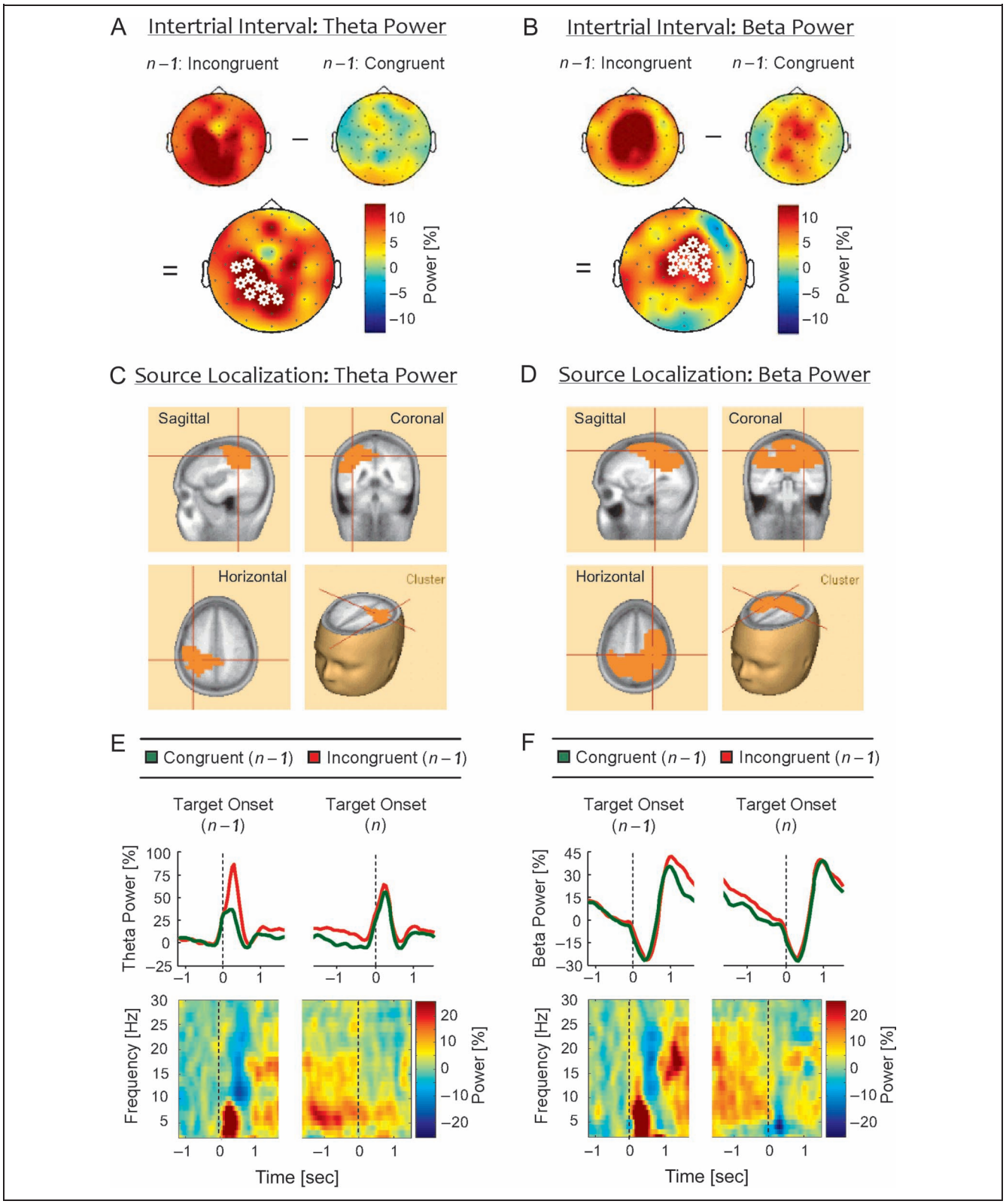

Figure 3. Intertrial effects in theta and beta power. (A, B) Topographies of the intertrial theta and beta power effects, that is, theta and beta power from −1500 to −500 msec before target onset in trial *n* as a function of previous trial type. Warm color coding indicates power increases; cold color coding indicates power decreases. (C, D) The intertrial effect in theta power was localized to the left parietal cortex; the intertrial effect in beta power was localized to the left and right (pre)motor cortex: sagittal slice seen from the left, coronal slice seen from the back, horizontal slice seen from the top. (E) Time courses of left parietal theta power as a function of previous trial type, time-locked to target onsets in trials $n - 1$ and n; time–frequency spectrogram of power differences, averaged over electrodes of the left parietal cluster. (F) Time courses of midcentral beta power as a function of previous trial type, time-locked to target onsets in trials $n - 1$ and n; time–frequency spectrogram of power differences, averaged over electrodes of the midcentral cluster.

as a function of previous trial type [(CI–CC)–(II–IC)], averaged across electrodes of the midfrontal cluster. Figure 2D shows time courses of theta power as a function of previous and current trial type, averaged across electrodes of the midfrontal cluster and time-locked to target onsets in trials $n - 1$ and n. These figures show that the conflict adaptation effect in the midfrontal cluster was restricted to early theta power.

An ANOVA on mean theta power from 200 to 400 msec following target onset in trial n, averaged across electrodes of the midfrontal cluster, with the factors of Previous Trial Type (C vs. I) and Current Trial Type (C vs. I) showed a main effect of Previous Trial Type, $F(1, 24) = 26.7$, $p < .001$, indicating that theta power was higher after C trials than after I trials (106% vs. 88%), and a main effect of current trial type, $F(1, 24) = 45.0$, $p < .001$, indicating that theta power was lower in C trials than in I trials (59% vs. 135%; Figure 2E). The analysis also showed an interaction between the two factors, $F(1, 24) = 21.2$, $p < .001$, reflecting sequential conflict adaptation. Indeed, conflict-induced theta power was higher after C trials (CI–CC: 100%) than after I trials (II–IC: 53%), but significant in both conditions ($t_{24} = 7.5$, $p < .001$; $t_{24} = 4.6$, $p < .001$). Importantly, previous trial type affected theta power in I trials (CI: 156% vs. II: 115%; $t_{24} = 8.7$, $p < .001$), but not in C trials (CC: 56% vs. IC: 62%; $t_{24} = 1.3$, $p = .20$).

Correlational brain–behavior analysis showed a positive correlation between the physiological conflict adaptation effect in midfrontal theta power and the behavioral conflict adaptation effect in RT ($r = .48$, $p < .05$; Figure 2F), indicating that participants with a larger conflict adaptation effect in theta power also showed a larger conflict adaptation effect in RT. Moreover, separate analyses for I and C trials showed that this positive correlation primarily arose from a positive brain–behavior correlation in I trials. Indeed, the conflict adaptation effect in midfrontal theta power was positively related to the sequential modulation of RTs in I trials (CI–II; $r = .57$, $p < .01$), but not in C trials (CC–IC; $r = -.19$, $p = .37$).

Intertrial Effects in Theta and Beta Power

Our next step was to examine whether theta and beta power showed intertrial effects arising from cognitive control adjustments. On the electrode level, cluster-based permutation analyses of theta and beta power from −1500 to −500 msec before target onset in trial n as a function of previous trial type (C vs. I) revealed a left-parietal electrode cluster for intertrial theta power ($p < .001$; Figure 3A) and a midcentral electrode cluster for intertrial beta power ($p < .001$; Figure 3B), both showing higher oscillatory power ITIs following I trials than following C trials.

On the source level, beamformer analysis localized the intertrial effect in theta power to the left inferior parietal lobule in Brodmann's area 40 (MNI coordinates: $x = -45$, $y = -40$, $z = 52$; Figure 3C), and the intertrial effect in beta power to the left and right (pre)motor cortex in Brodmann's areas 4/6 (approximate MNI coordinates: $x = -30/30$, $y = -30$, $z = 55$; Figure 3D). Time courses and time–frequency spectrograms of power as a function of previous trial type (C vs. I), combined for each of the two clusters' electrodes and time-locked to target onsets in trials $n - 1$ and n, are shown in Figure 3E and F. Correlational analysis showed that the two effects were positively related ($r = .40$, $p < .05$), indicating that participants with a larger intertrial effect in left-parietal theta power also showed a larger intertrial effect in midcentral beta power.

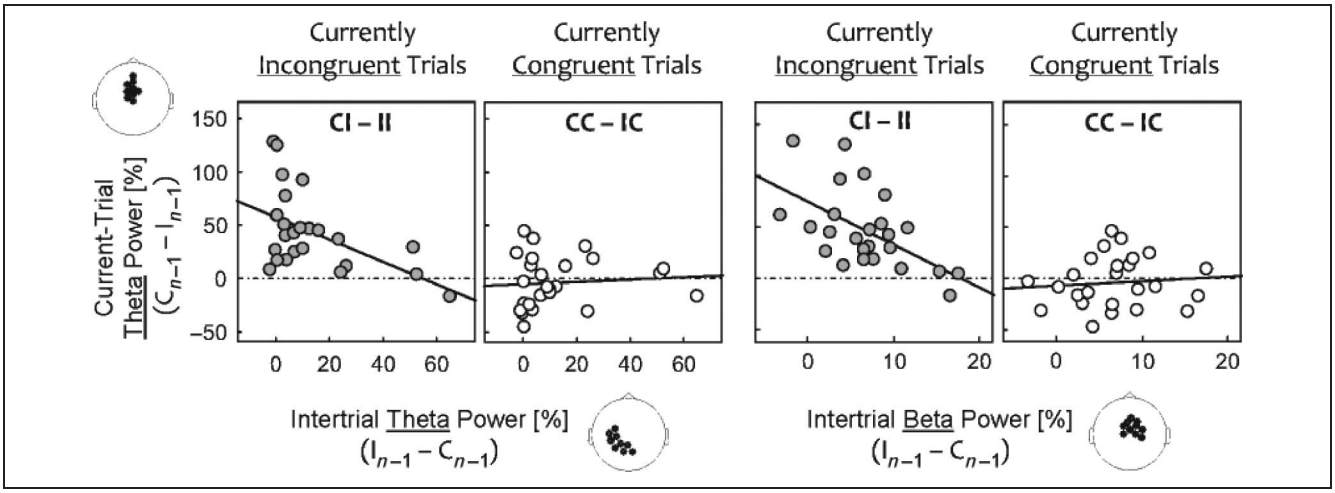

Figure 4. Relationships between intertrial effects and current trial processing. Intertrial effects in both left parietal theta power and midcentral beta power, with higher power following incongruent trials than following congruent ones, were negatively related to current trial conflict processing, as indexed by the previous trial type effect on midfrontal theta power from 200 to 400 msec following target onset in currently incongruent trials (CI–II). No such relationship was observed for currently congruent trials (CC–IC). Scatterplots with gray dots depict the significant correlations (ps < .05).

Relating Intertrial Effects to Current Trial Processing

Our last step was to examine whether intertrial effects in left parietal theta and midcentral beta power were related to the sequential modulation of response conflict, as indexed by the previous trial type effect on midfrontal theta power in I trials (CI vs. II), across participants. Correlational analyses showed that intertrial effects in both left parietal theta and midcentral beta power were related to the sequential modulation of response conflict in I trials (Figure 4). That is, participants with larger intertrial effects in both theta and beta power showed a smaller conflict adaptation effect in midfrontal theta power (ITI theta: $r = -.47, p < .05$; ITI beta: $r = -.53, p < .01$), indicating that large individual effects in high intertrial theta and beta power may relate to enhanced (but less effective) adjustment efforts in low-control than in high-control participants after I trials. No such relationship was observed for C trials (ITI theta: $r = .18, p = .39$; ITI beta: $r = .13, p = .54$).

DISCUSSION

This study examined conflict adaptation effects in the response-priming task, both on the behavioral and the physiological level. On the behavioral level, the present results show conflict adaptation effects in RT and response accuracy, both characterized by a reduced current trial congruency effect after I trials. For RTs, the conflict adaptation effect was mediated by both faster RTs in I trials and slower RTs in C trials following I trials. For response accuracy, previous trial type affected I trials but not C trials. Because of possible floor effects, however, the conflict adaptation effect in response accuracy should be interpreted with caution. Together, these results are well in line with prior work on behavioral conflict adaptation in the response-priming task (Kunde & Wühr, 2006; Kunde, 2003).

Importantly, on the physiological level, the present results show a conflict adaptation effect in midfrontal theta power that was localized in a cluster of voxels extending on the left hemisphere from ACC to the pre-SMA, characterized by a main effect of current trial congruency, with more theta power in I trials than in C trials, that was modulated by previous trial congruency, with smaller current trial congruency effects following I trials. The main effect of current trial congruency is in line with prior work showing that conflict processing is related to ACC activity (e.g., Kerns, 2006; Liu, Banich, Jacobson, & Tanabe, 2004; van Veen & Carter, 2002; Botvinick et al., 1999), pre-SMA activity (e.g., Horga et al., 2011; Isoda & Hikosaka, 2007; Matsumoto, Matsumoto, Abe, & Tanaka, 2007; Huettel, Mack, & McCarthy, 2002), and midfrontal theta power (e.g., Cavanagh et al., 2012; Nigbur et al., 2011, 2012; Pastötter et al., 2010, 2012; Hanslmayr et al., 2008). Going beyond prior work, this study demonstrates a reliable conflict adaptation effect in trial-averaged midfrontal theta power, mediated by a previous trial type effect on theta power in I trials, but not in C trials. Moreover, a positive correlation between conflict adaptation effects in midfrontal theta power and RT arose, largely driven by a high correlation between the conflict adaptation effect in theta power and the previous trial type effect on RTs in I trials, but not in C trials. Consistent with these findings, Cohen and Cavanagh (2011) recently reported conflict adaptation-like regression effects on the single-trial level when relating single-trial theta measures to single-trial RTs in the Eriksen flanker task. However, in this prior work, Cohen and Cavanagh failed to find a conflict adaptation effect on trial-averaged theta power, indicating that the effect may not be easily found in tasks in which response conflict is typically confounded with stimulus conflict, such as the Eriksen task and the Stroop task (Hommel, 2011; Kornblum, Hasbroucq, & Osman, 1990).

In line with the preparatory system view of cognitive control (Compton et al., 2011, 2012; Horga et al., 2011; Mansouri et al., 2009), analyses of the ITI revealed effects of previous trial type on intertrial brain oscillations, both in the theta and the beta frequency range. Indeed, sustained intertrial differences were found to be present in left parietal theta power, localized in the left inferior parietal cortex (IPC), and midcentral beta power, localized in the left and right (pre)motor cortex, with higher theta and beta power after I than after C trials. Correlational analyses showed that, first, the two intertrial effects were positively related to each other across participants and, second, both intertrial effects were negatively related to conflict adaptation, as indexed by midfrontal theta power in I trials. Control adjustments in the ITI thus were functionally related to the processing of conflict in trial *n*.

The intertrial effect in theta power was localized in the left IPC. This area is consistent with both prior imaging work on conflict adaptation (Horga et al., 2011; Durston et al., 2003; Carter et al., 2000) and recent brain oscillation work on attention and motor behavior (Perfetti et al., 2011; Green & McDonald, 2008). Carter et al. (2000), for instance, showed that both ACC and IPC activities relate to conflict processing in the Stroop task. However, whereas ACC activity was transient and returned to baseline before the next trial, IPC activity remained high until the next trial. Durston et al. (2003) further showed that IPC activity increases as the number of preceding incongruent trials increases in the Eriksen task. Imaging data thus points to a functional role of the IPC in the allocation of attentional resources. Consistently, parietal (IPC) theta power has been shown to index initiation of attentional control (Green & McDonald, 2008) and movement planning (Perfetti et al., 2011), and (more central) theta power baseline adjustments have been shown to relate to controlled speed–accuracy tradeoff (Pastötter et al., 2012) and the influence of negative feedback on behavioral conflict adaptation in the Eriksen task (van Steenbergen, Band, & Hommel, 2012). On the basis of this prior work and following recent theoretical suggestions that sustained theta synchronizations may index the allocation of

attentional resources in visuomotor processing (Sauseng et al., 2007) and decision-making (Womelsdorf, Vinck, Leung, & Everling, 2010), the present intertrial theta effect can be interpreted in terms of proactive adjustments of attentional control.

The intertrial effect in beta power was localized in the left and right (pre)motor cortex. Following the prominent idea that beta oscillations index cortical motor processing, with increases of beta power reflecting motor inhibition and decreases of beta power reflecting release of motor inhibition (Jenkinson & Brown, 2011; Pfurtscheller & Lopes da Silva, 1999), the present intertrial effect might reflect adjustments of motor control (Cheyne, 2013). In particular, the observed increase of intertrial beta power following incongruent trials might indicate motor inhibition to prevent premature motor activation after being misled by the cue on the preceding incongruent trial (Wendt, Luna-Rodriguez, Reisenauer, Jacobsen, & Dreisbach, 2012). Thereby our results add to the growing literature, showing that beta baseline modulations in the prestimulus or ITI affect motor processing and RTs. Specifically, it has been shown that beta baseline activities can be modulated by different factors, including response uncertainty, response inhibition, and controlled speed–accuracy tradeoff (Pastötter et al., 2012; Tzagarakis, Ince, Leuthold, & Pellizzer, 2010; Pastötter, Hanslmayr, & Bäuml, 2008). Adding to this literature, this study shows that previous trial context also is a factor that affects motor processing by modulating beta baseline activities. The present intertrial beta effect can be interpreted in terms of proactive adjustments of motor control.

Both the behavioral and physiological results are well in line with the view that cognitive control mediates conflict adaptation in the response-priming task (Kunde & Wühr, 2006; Kunde, 2003). In the literature, however, there exists an ongoing debate whether sequential modulations in response conflict tasks should rather be explained by repetition priming and episodic retrieval (Notebaert, Gevers, Verbruggen, & Liefooghe, 2006; Hommel, Proctor, & Vu, 2004; Mayr, Awh, & Laurey, 2003; for a review, see Egner, 2007). Prior work by Kunde and Wühr (2006) addressing this debate in the response-priming task showed that sequential modulations in RT and response errors are the same regardless of whether stimuli and responses are repeated in subsequent trials or not, suggesting that cognitive control and conflict adaptation play a major role in this task. However, from this prior work, it still cannot be excluded that the present effects in brain oscillations may have been mediated (to some extent) by repetition priming and episodic retrieval. This is because this study and the study by Kunde and Wühr differed in various aspects, including material, prime duration, prime–target interval, and, most importantly, number of cues and targets. Future work may examine possible modulations of repetition priming and episodic retrieval on brain oscillations in the response-priming task by controlling for repetitions and alternations of stimuli and responses.

Regarding the exact time point at which cognitive control occurs in the response priming task, the present results support the view that cognitive control adjustments can already be initiated in the ITI, biasing attention and motor processes in an anticipatory manner (see also Coste, Sadaghiani, Friston, & Kleinschmidt, 2011; Horga et al., 2011; Mansouri, Buckley, & Tanaka, 2007). This is not to say that our results contradict the recent view that conflict adaptation in trial n may be because of carryover effects from within-trial control adjustments in trial $n - 1$ (Scherbaum, Fischer, Dshemuchadse, & Goschke, 2011). Such within-trial control adjustments, regulating conflict "on the fly" in trial n have been shown for different tasks, including the response-priming task, and have been suggested by both prior behavioral and computational work (Appelbaum, Smith, Boehler, Chen, & Woldorff, 2011; Pastötter et al., 2010; Davelaar, 2008; Goschke & Dreisbach, 2008; Brown, Reynolds, & Braver, 2007; Taylor, Nobre, & Rushworth, 2007; see also Paus, Koski, Caramanos, & Westbury, 1998, for a regulatory theory of ACC function). However, our data provide evidence for preparatory control adjustments that cannot be ascribed to carryover effects alone. In particular, previous trial type effect on beta power was found to be much larger in the ITI than in the preceding conflict trial. In addition, the previous trial type effects on intertrial and within-trial theta power were localized to different brain areas. Thus, the present results suggest that, beyond possible carryover effects from the previous trial, additional preparatory control adjustments in the ITI promote sequential conflict adaptations in the response-priming task.

To conclude, consistent with the preparatory view of cognitive control, this study demonstrates a reliable conflict adaptation effect in trial-averaged midfrontal theta power that is predicted by intertrial brain oscillations. Indeed, sustained intertrial effects in left parietal theta power and midcentral beta power, likely reflecting preparatory adjustments of attention and motor control, predicted current trial response conflict as indexed by midfrontal theta power in currently incongruent trials. These findings point to a dynamic, preparatory cognitive control system that, depending on previous trial context, up- and down-regulates attention and motor activities in anticipation of the upcoming trial.

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