Midfrontal conflict-related theta-band power reflects neural oscillations that predict behavior

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Running head: Conflict-related theta is non-phase-locked

Acknowledgements. Thanks to Irene van de Vijver and Jessika Buitenweg for help programming the tasks and collecting data, and to Rasa Gulbinaite for help with the statistical power analyses. MXC is funded by a VIDI grant from the Netherlands Organization for Scientific Research (NWO). Thanks also to John Allen for helpful comments on the manuscript.
Abstract

Action monitoring and conflict resolution require the rapid and flexible coordination of activity in multiple brain regions. Oscillatory neural population activity may be a key physiological mechanism underlying such rapid and flexible network coordination. EEG power modulations of theta-band (4-8 Hz) activity over the human midfrontal cortex during response conflict have been proposed to reflect neural oscillations that support conflict detection and resolution processes. But it has remained unclear whether this frequency-band-specific activity reflects neural oscillations or non-oscillatory responses (i.e., event-related potentials; ERPs). Here we show that removing the phase-locked component of the EEG did not reduce the strength of the conflict-related modulation of the residual (i.e., non-phase-locked) theta power over mid-frontal cortex. Furthermore, within-subject regression analyses revealed that the non-phase-locked theta power was a significantly better predictor of the conflict condition than was the time-domain phase-locked EEG component. Finally, non-phase-locked theta power showed robust and condition-specific (high vs. low-conflict) cross-trial correlations with reaction time, whereas the phase-locked component did not. Taken together, our results indicate that most of the conflict-related and behaviorally relevant mid-frontal EEG signal reflects a modulation of ongoing theta-band oscillations that occurs during the decision process but is not phase-locked to the stimulus or to the response.
Response conflict occurs when competing responses are activated but only one can be selected, and elicits a cascade of cognitive processes including conflict detection, action selection, selective suppression, and post-response monitoring. These processes (among others) are referred to as cognitive control, and rely critically on structures along the medial frontal wall, including the anterior cingulate cortex and surrounding medial frontal cortical areas. A commonly held view is that frontal midline structures detect activations of competing actions and recruit other brain regions to help resolve the conflict and avoid errors (Miller and Cohen, 2001; Ridderinkhof et al., 2004; Carter and van Veen, 2007).

In human electrophysiology, response conflict and cognitive control are often studied using event-related potentials (ERPs), defined as the time-domain average voltage deflection that is phase-locked to external events. During correct trials in conflict tasks, there is an enhancement of a negative-going ERP deflection that peaks around 200-300 ms after stimulus onset, called the “N2,” in high-conflict compared to low-conflict trials (Botvinick et al., 2001; Yeung et al., 2004; Folstein and Van Petten, 2008; Hughes and Yeung, 2011). The N2 can be observed after stimulus onset and prior to response, suggesting that it reflects conflict processing that occurs between stimulus detection and response execution. Other studies have reported that response conflict modulates EEG activity in the theta (4-8 Hz) frequency range between stimulus onset and manual response (Wang et al., 2005; Cohen et al., 2008; Womelsdorf et al., 2010; Cohen and Cavanagh, 2011; Cavanagh et al., 2012; Nigbur et al., 2012).

The midfrontal theta modulations and the ERP signatures such as the N2 tend to occur at similar times, however, the relationship between these two mid-frontal conflict-related EEG signatures is not well understood. It has been argued that brief modulations of low-frequency EEG power do not reflect actual neural oscillations, but rather transient ERP components (Yeung et al., 2004), which tend to have dominant power in the low frequency range. Disambiguating the ERP from neural oscillations has important implications for understanding the neural mechanisms of cognitive control: Establishing that conflict-related mid-frontal theta power reflects neural oscillations would set the stage for linking this human EEG signature to neurophysiological mechanisms implicated in information processing and large-scale network functioning, including mechanisms thought to underlie top-down attention and sensory-motor decision-making (Fries, 2005; Miller and Wilson, 2008; Donner and Siegel, 2011; Akam and Kullmann, 2012; Siegel et al., 2012). In contrast, the
neurophysiological mechanism that produces the N2 is not established, nor are there prevailing theories that link the N2 to neurophysiology.

Here, we tested the hypothesis that conflict-related midfrontal EEG modulations primarily reflect a modulation of ongoing theta-band oscillations, which is neither phase-locked to stimulus onset nor to response, and, therefore, cannot be attributed to the ERP. To identify oscillations, we applied two criteria that have been commonly applied in sensory and motor physiology: The modulation of time-frequency power should be (1) band-limited and (2) non-phase locked to stimulus or response (Pfurtscheller and Lopes da Silva, 1999; Tallon-Baudry and Bertrand, 1999; Donner and Siegel, 2011). We analyzed two existing EEG datasets (N=40) and compared non-phase locked theta-band power to the ERP, in terms of condition modulations at the within-subject and across-subjects levels, within-subjects correlations with behavior, and statistical power.

We found a robust non-phase-locked and band-limited midfrontal theta-band power modulation that was not affected by removing the phase-locked part of the signal (the ERP). Furthermore, the non-phase-locked theta power modulation was more closely linked to the conflict manipulation and to its effects on reaction time compared to the ERP. We conclude that non-phase-locked theta oscillations in the human medial frontal cortex are a sensitive marker of response conflict during decision-making tasks. These neural oscillations may play a crucial mechanistic role in orchestrating the online monitoring and adaptation of task performance during tasks that elicit response conflict.

### Materials and Methods

#### Participants

Forty subjects from the University of Amsterdam psychology undergraduate program volunteered in exchange for course credit or money (€14). Subjects had normal or corrected-to-normal vision and no reported history of psychosis, brain disease, or psychiatric illness, and were self-reported right-handed. The local ethics committee at the University of Amsterdam approved both experiments, and subjects provided informed consent prior to the start of the experiment.

#### Task design
We pooled data from two separate conflict tasks. All data analyses were identical for both experiments. Details of both experimental designs are published elsewhere, and are briefly described below (see Figure 1a for an overview of events and timing of each experiment). Experiment 1 (Cohen and Ridderinkhof, 2013) was a Simon task, in which a circle was presented in the left or the right visual hemifield. Subjects were instructed to respond, as quickly as possible, to the color of the stimulus while ignoring its location. Conflict occurs when the response hand is opposite to the visual presentation hemifield. Four colors were used, two mapped to the left hand and two mapped to the right hand. All manipulations were pseudo-randomized, as was the order of color and stimulus presentation side, which avoids stimulus repetition effects (Wuhr and Ansorge, 2005). Each subject performed 1500 trials. Experiment 2 (Cohen, 2011) was an auditory-visual Simon task. A separate group of 20 subjects that did not participate in Experiment 1 responded according to the physical location of a visual stimulus while ignoring the simultaneous auditory beep that was presented to each ear via in-ear EEG-compatible air tubes, or vice-versa (attended features were cued in separate blocks). Subjects pressed a button with the left hand for stimuli on the left, and with their right hand for stimuli on the right. Conflict is introduced when, e.g., the stimulus is in the left hemifield while the tone is played in the right ear. Each subject performed 2400 trials over two sessions.

We use the term “high conflict” to indicate trials in which the irrelevant task feature was associated with the opposite response as the relevant task (e.g., when the tone was played in the right ear but the subject had to respond with the left hand), and we use the term “low conflict” to indicate trials in which both task dimensions were associated with the same response hand. Although the conflict may arise due to different factors from the two experiments (response-spatial location vs. bimodal integration), several previous studies suggest that the conflict- and error-related computations performed by MFC are independent of input and output modalities (Holroyd et al., 1998; Riesel et al., 2013).

The purpose of pooling data from two separate but similar tasks was to increase statistical power. Statistical power is increasingly discussed as a major limiting factor in interpreting and generalizing results from cognitive neuroscience experiments (Button et al., 2013). Typical EEG studies include 10-20 subjects. Increasing our sample size to 40 ensured robust group-level statistics and increases confidence about the findings and their generalizability to other studies. In the Results section, we report the post-hoc statistical power of several effects. These were
computed using the *G*power 3.1 software, which is freely available online (Faul et al., 2007). We use the term “statistical power” to refer to the probability of correctly rejecting the null hypothesis (that is, 1-β, where β is the probability of making a Type II error), and “power” to indicate EEG time-frequency power.

**EEG acquisition and preprocessing**

EEG data were acquired at 2048 Hz (512 Hz in Experiment 2) from 64 channels placed according to the international 10-20 system using BioSemi equipment (see www.biosemi.com for hardware details). Additional electrodes were placed to acquire horizontal eye movements and electromyographic signals from the thumbs. Electrodes were placed on both earlobes to serve as offline average reference. Offline, EEG data were high-pass filtered at 0.5 Hz and then epoched from -1 to +1.5 seconds surrounding each trial. All trials were visually inspected and those containing EMG or other artifacts not related to blinks were manually removed. Independent components analysis was computed using the eeglab toolbox for Matlab (Makeig et al., 2004), and components containing blink/oculomotor artifacts or other artifacts that could be clearly distinguished from brain-driven EEG signals were subtracted from the data.

The following trials were removed prior to analyses: Error and post-error trials, trials with RTs shorter than 200 ms or longer than three standard deviations above each subject’s median RT, trials with partial errors (details below), trials with horizontal eye movements indicating saccades away from fixation, and the first trial following each rest period or instruction change. Partial errors are when subjects made the correct response but twitched the muscle corresponding to the incorrect response, and elicit a qualitatively distinct pattern of brain oscillatory dynamics compared to pure correct responses (Cohen and van Gaal, in press). We identified a trial as containing a partial error if the Z-transformed derivative of the EMG signal exceeded two standard deviations between stimulus onset and the button press, and if the peak of the EMG response was more than two times the largest peak from -300 ms to stimulus onset. Trials with horizontal eye movements were defined as those in which the HEOG (low-pass filtered at 20 Hz) exceeded 5 standard deviations to either direction relative to pre-stimulus baseline activity. Finally, conditions were trial- and RT-matched within-subject, such that the condition with the fewest trials was identified, and RT-matched trials from all other conditions were selected. This procedure minimizes the possibility of condition differences in EEG activity resulting from differences in trial count, signal-to-noise ratio (which depends on trial count) or global differences in time-on-task.
(Cavanagh, Cohen, and Allen, 2009; Cohen and Cavanagh, 2011). After pre-processing, there were on average 274 (standard error: 18.8) trials per condition per subject used in the data analyses.

We also compared the results with and without applying a surface Laplacian (also sometimes called current source density or current scalp density) to the preprocessed single-trial EEG data. The surface Laplacian is a spatial band-pass filter that attenuates activity from distant sources, or activity that is highly spatially distributed and temporal coherent (Cohen, 2014). Comparing EEG results with and without the surface Laplacian (Figures 3 and 4) provides support for the notion that the EEG-measured conflict modulations of theta power were due to a focal and source with a likely radially oriented dipole in medial frontal cortex, rather than being due to volume conduction from other brain regions.

**EEG analyses**

We defined the ERP as the phase-locked component of trial-related EEG modulations, as obtained by averaging across trials in the time-domain.

EEG data were decomposed into their time-frequency representation via wavelet convolution performed in the frequency domain (note that the convolution theorem guarantees that the following method is equivalent to time-domain convolution). The power spectrum of the EEG (obtained from the fast-Fourier-transform) signal was multiplied by the power spectrum of complex Morlet wavelets \( e^{i2\pi ft} e^{-t^2/(2\sigma^2)} \), where \( t \) is time, \( f \) is frequency, which increased from 2 to 60 Hz in 30 logarithmically spaced steps, and \( \sigma \) defines the width of each frequency band, set according to \( n/(2\pi f) \) where \( n \) is the number of wavelet cycles, and increased from 3 to 10 in logarithmic steps), and the inverse fast-Fourier-transform was then taken. Note that this was done on the single-trial data, not the ERP. From the resulting complex signal (the analytic signal), an estimate of frequency-band-specific power at each time point was defined as the squared magnitude of the result of the convolution \( (\text{real}[z(t)]^2 + \text{imag}[z(t)]^2) \). All power values in the time-frequency representation were normalized to the average pre-stimulus baseline power at each frequency band. We used a decibel (dB) transform for normalization (dB power = \( 10\times\log_{10}[^\text{power/baseline}] \)). The baseline power was computed as the average power across all experiment conditions, from -300 to -100 ms pre-stimulus. A pre-stimulus baseline period was used for both stimulus-locked and for response-locked analyses.
The non-phase-locked power was obtained in the following way. First, we computed the ERP (the time-domain trial-average), and then subtracted the ERP from the time-domain EEG signal on each trial (Kalcher and Pfurtscheller, 1995). This was done separately for each condition, electrode, and subject. After this single-trial subtraction of the ERP, the time-frequency dynamics were extracted as described in the previous paragraph. Figure 1 shows an overview of the procedure for one subject. We did not apply a low-pass filter on the ERPs, for two reasons. First, trial averaging itself acts as a low-pass filter, particularly with an average of 274 trials per condition/subject. Second, low-pass filtering removes a valid part of the signal, and thus would involve less signal removal when computing the non-phase-locked power. Thus, applying a low-pass filter would invite the risk of differences between phase-locked and non-phase-locked activity being due to the low-pass filtering of the ERP.

The method described in the previous paragraph assumes that the ERP is both amplitude- and shape-invariant on each trial (that is, that the ERP on each trial is the same as the trial-averaged ERP). We therefore tried an alternative method of subtracting the non-phase-locked activity while assuming only that the ERP is shape-invariant, but not amplitude-invariant. This was done by scaling the amplitude of the ERP on each trial according to the normalized dot product between the ERP and the single-trial EEG trace (Donner et al., 2008). Thus, the amplitude of the ERP can vary on each trial, although its shape remains the same. Results were similar to when subtracting the unweighted ERP, and are thus not shown here. The assumptions underlying single-trial ERP subtraction are discussed further in the Discussion section. Phase-locked power was computed by subtracting the non-phase-locked from the total power.

Cross-trial within-subject regression

Typically in cognitive neuroscience tasks, data are averaged over all trials within-subject and then statistics are performed at the group level. This procedure increases signal-to-noise, but also ignores within-subject variability. We thus tested whether EEG features (time-frequency power and single-trial ERP) were reliable predictors of low- vs. high-conflict trials within-subjects. Therefore, we applied a within-subject cross-trial logistic regression analysis. The following procedure was conducted separately for each time point or time-frequency point, for each subject. We first constructed a linear model over trials: \( \beta = (X^T X)^{-1} X^T D \), where \( X \) is the design matrix (one column for the intercept and one column for the condition labels, dummy-coded to 0 and 1 for low- and high-conflict), \( X^T \) and \( \cdot^{-1} \) indicate the matrix transpose and inverse, and \( D \) is the vector of data.
from all trials at that time or time-frequency point. The $\beta$ term is vector of regression coefficients that describe the relationship between the data and the design matrix. For each subject, the conflict-$\beta$ coefficient (the first coefficient is the intercept and is not of interest here) was binarized as 1 if it indicated that increased power over trials at that time-frequency point predicted a high-conflict trial, and 0 if increased power predicted a low-conflict trial. Thus, at the group level, a value of 90% indicates that for 90% of subjects, the within-subject regression over trials predicts that more time-frequency power or ERP amplitude predicts a high-conflict condition at that time or time-frequency point. Results were tested against 0.5 using one-sample Mann-Whitney-U (a.k.a. Wilcoxon rank sum test) nonparametric t-tests, and were considered significant if they exceeded a threshold of $p<0.001$. This analysis complements the typical group-level analyses because it incorporates the cross-trial, within-subject variance that is typically ignored in group-level analyses.

**Cross-trial EEG-RT correlations**

The purpose of this within-subjects analysis was to test whether the EEG time- or time-frequency-domain features were related to trial-varying task performance, as measured through RT. For each subject, EEG data were correlated with RT over trials at each time point or time-frequency point. This produced a time-course or time-frequency map of correlation coefficients for each subject. Correlation coefficients were then tested against zero at the group level, after first being Fisher-Z transformed. Spearman correlations were used because power is non-normally distributed.

**Statistics**

Statistical analyses of the EEG data relied on non-parametric permutation testing, correcting for multiple comparisons across all time-frequency space, or across all time points. At each of 1000 iterations during permutation testing, the effect (all conditions vs. baseline, or high- vs. low-conflict trials) time-course or time-frequency map was multiplied by -1 for a random subset of subjects. Note that this is equivalent to randomly swapping the order of the condition subtraction (A-B vs. B-A), and provides an appropriate test against the null hypothesis of no condition differences (Maris and Oostenveld, 2007). Next, a t-statistic was calculated as $\frac{X}{\sigma \sqrt{n}}$, where $X$ is the effect of interest, $\sigma$ is the standard deviation at each time point (across subjects), and $n$ is the number of subjects (40). From each null-hypothesis iteration, the maximum and minimum t-values from the entire map were stored; thus, after 1000 iterations a distribution of minimum and maximum t-values expected
under the null hypothesis could be created. In the true t-statistic map, any pixel with a t-value greater than or less than 99.999% of the null-distribution t-values was considered a significant effect. This corresponds to a p-value of 0.001, correcting for multiple comparisons across all time points or time-frequency points. For the correlation results, we used a p-value threshold of 0.01 (also correcting for multiple comparisons over time-frequency space, or over time points). A conservative threshold was used because we had many subjects and relatively small variances.

**Results**

**Behavioral results**

Behavioral performance for experiment 1 has been reported previously (Cohen and Ridderinkhof, 2013). Briefly, the expected conflict effect (longer RTs and higher error rates) was observed in the high-conflict compared to the low-conflict condition (mean/standard error RTs for high-/low-conflict trials: 503/10 and 466/14 ms; mean/standard error accuracy for high-/low-conflict trials: 88/2 and 96/5%). The conflict effect in experiment 2 has not been reported before (the Cohen 2011 paper focused on errors vs. correct trials). RTs for high-conflict and low-conflict trials were 359 ms and 319 ms (standard errors: 10 ms and 7 ms; t_{19}=10.78, p<0.001). Accuracy for high-conflict and low-conflict trials was 85.5% and 98.4% (standard error: 1.5% and 0.4%; t_{19}=10.7, p<0.001).

**EEG dynamics during sensory-motor task performance**

Task performance was associated with a number of well-known EEG signatures reflecting the (1) encoding of the visual stimulus in occipito-parietal cortex, (2) conflict processing in medial frontal cortex, and (3) response preparation in (pre-)motor cortex (Pfurtscheller and Lopes da Silva, 1999; Tallon-Baudry and Bertrand, 1999; Donner et al., 2009; Cohen and Cavanagh, 2011; Donner and Siegel, 2011). These signatures were most evident in the three pre-defined electrode groups highlighted in Figure 2, and each of them comprised non-phase-locked (upper panels) and phase-locked (i.e., ERP) features (upper panels).

The brief (50 or 200 ms, see Figure 1a) visual stimuli elicited a phase-locked (ERP) response over visual cortex (Figure 2b, lower panel), which was largely confined to the <8 Hz range and accompanied by non-phase-locked (“induced”) suppression of alpha-band (8-15 Hz) power (Figure
2A, upper panel). As expected, both of these stimulus-related components were more closely aligned to the stimulus compared to the response (compare left and middle panels in Figure 2A). Motor response preparation, on the other hand, was associated with two movement-selective components evident in the EEG lateralization over the motor cortices (i.e., contralateral vs. ipsilateral to the response hand): a phase-locked enhancement of contralateral low-frequency power (Figure 2C, lower panel) and a non-phase-locked suppression of contralateral beta-band (10-20 Hz) power (Figure 2C, upper panel). As expected, both of these response-related components were more closely aligned to the motor response than to the stimulus onset (compare middle and left panels in Figure 2C).

Finally, electrodes over midfrontal cortex exhibited both phase-locked and non-phase-locked components of power modulations, which were about equally well aligned to stimulus onset and the motor response (compare middle and left panels in Figure 2B). There were two significant midfrontal non-phase-locked components: a power enhancement in the theta-band (4-8 Hz) and a power suppression in the beta-band (15-30 Hz). The theta-band power modulation was specific to midfrontal electrodes; the beta-band suppression may reflect distributed activity from the motor cortices (see topographical map in top right panel of Figure 2C).

To quantify how much of the time-frequency power is non-phase-locked, we computed the percent of the total power that is phase-locked (computed as 100*(total—non-phase-locked)/total). This was computed within time-frequency windows that were selected based on the conflict effect (see below; this window was selected to be orthogonal to possible differences between total and non-phase-locked power). For the stimulus-locked analysis from 300-600 ms and 4-8 Hz, 84.6% of the total power was non-phase-locked (standard deviation 9.9%, range 60.7% to 96.5%). For the response-locked analysis from -200 to 0 ms and 4-8 Hz, 80.6% of total power was non-phase-locked (standard deviation 36.0%, range 25.9% to 98.2%). In the rest of this paper, we show that this non-phase locked theta power modulation specifically relates to conflict processing and is a good predictor of task condition and RT.

**Conflict modulation of non-phase-locked midfrontal EEG power**

Theta power (peak at 6.5 Hz) increased after stimulus onset and around the time of response, for high-conflict trials compared to low-conflict trials (Figure 3). We next computed the post-hoc statistical power of the theta-band conflict effect (200-600 ms, 4-8 Hz). With 40 subjects and
assuming an alpha of 0.001, the effect size was 1.25 for the total power and 1.29 for the non-phase-locked power (condition difference means/stds.: 0.5506/0.4262 and 0.5949/0.4368, and intervariable correlations: 0.9254/0.9105 for total and non-phase-locked, respectively), and the statistical power (1-\(\beta\)) was >0.99. Indeed, with this effect size, even 18 subjects would have produced a statistical power of around 0.9, which is generally considered a large effect.

There were no statistically significant differences between the conflict effect in the total-power analysis versus that in the non-phase-locked analysis. The analyses presented in Figure 3 demonstrate that the conflict effect in the EEG signal is driven by non-phase-locked activity, and is therefore unchanged when removing the ERP from the data. This pattern of effects was consistent across both experiments (results not shown).

We quantified the percentage of the conflict effect that was contained in the non-phase-locked part of the data using the procedure and time-frequency windows reported in the previous section. We report results only for electrode FCz, partly because it was the electrode that showed the maximum task-related and conflict-related theta power modulations (see Figure 3) and partly because previous EEG studies on conflict processing focus mainly on this electrode. For the stimulus-locked analysis, this was 108.7% (standard deviation 38.6%), and for the response-locked analysis this was 120.5% (standard deviation 61.2%). Percentages above 100% indicate that some of the phase-locked signal can obscure part of the conflict modulation (this can also be observed in the slightly blue regions in the time-frequency plots in the bottom row in Figure 3a).

There was little qualitative change in the topographical or the time-frequency characteristics of the theta-band power after applying the surface Laplacian, in particular for the theta-band effects (Figure 3c-d). The delta-band (<4 Hz) modulation by conflict was attenuated by the surface Laplacian. This demonstrates that the conflict modulation of theta power is not driven by distant or distributed sources, and is consistent with a neural source in medial prefrontal cortical regions close to the skull, underlying electrode FCz (the surface Laplacian would have attenuated activity from deep sources such as the anterior cingulate).

**Conflict modulation of the midfrontal phase-locked EEG component (ERP)**

The ERP at midfrontal electrode FCz (Figure 4a) appeared qualitatively similar to previous reports of conflict-related ERPs (e.g., Yeung et al., 2004). The ERPs for high-conflict vs. low-conflict trials
were significantly different around 400 to 600 ms in the stimulus-locked ERPs, and around -150 to 0 in the response-locked ERPs (Figure 4a). In the time period in which an N2 was visually apparent (~175-250 ms), there was no statistically significant condition difference when correcting for multiple comparisons across time points. However, a post-hoc t-test at the time of the N2 peak revealed a statistically significant effect (250 ms; t_{39}=-2.84, p=0.007, uncorrected for multiple comparisons, but see below for statistical power analysis). Topographically, the ERPs diverged from the theta power. The condition-averaged ERP was maximal over central posterior sites rather than midfrontal sites, and the conflict modulation showed a spatially multi-peaked central-parietal distribution, rather than identifying a single source (Figure 4b). Statistical power analyses on the stimulus-locked ERP components revealed low statistical power for the N2 component (condition difference mean/std. at 250 ms: -0.9466/2.1056, inter-variable correlation: 0.8993, effect size=-0.4495, power=0.26) and high statistical power for the later P3-type component (condition difference mean/std. from 380-550 ms: 1.8026/1.1884, effect size=1.5168, power=1.0). Note that for the N2 component, with the present effect size and alpha of 0.001, a sample size of over 100 would be required to obtain a statistical power of 0.9, which is generally considered a large effect.

In contrast to the theta power results, the ERP was strongly affected by applying the surface Laplacian. This can be seen in Figure 4c-d both in the overall shape of the ERPs and in the topographical distributions. When data appear qualitatively different before vs. after the surface Laplacian, one can interpret this as demonstrating that the activity was not generated by brain tissue beneath the electrode, but rather, by deep or distant sources that were volume-conducted, or by large and distributed cortical networks that are highly temporally coherent (Cohen, 2014). Importantly, the topography of the conflict modulation of the ERP was qualitatively distinct from that of the theta power.

Our results so far indicate that the conflict-related midfrontal theta power and ERP components are two distinct, superimposed physiological phenomena that overlap in time during conflict processing. We next quantified whether these two EEG manifestations were correlated with each other over subjects, using bivariate Spearman’s correlation. Importantly, we also computed partial correlations, holding constant the conflict modulation on the ERP at P9 in the same time window as used for the N2 modulation. P9 was selected because it is far away from FCz and showed no conflict effect (t_{39}=0.47, p=0.63). The idea here is that if the N2 and the theta power are simply different ways of measuring the same midfrontal neural process, they should be highly correlated, and thus
partialing out the non-significant conflict modulation at P9 should have no effect on the FCz N2-theta correlation. In contrast, if the N2 and theta power reflect distinct midfrontal neural processes, any correlation between them may simply reflect non-specific factors that affect the EEG signal (e.g., skull thickness and electrode impedance); thus, partialing out P9 variance should reduce the N2-theta correlation. It is important to take these general and potentially mediating factors into account, otherwise correlations across subjects (Harper et al., 2013) or across conditions based on subject averages (Hajihosseini and Holroyd, 2013) could be misleading.

For the stimulus-locked ERP, we used the N2 time period rather than the later and more statistically significant period because most ERP research on response conflict focuses on the N2; thus, the N2 is the most theoretically relevant component. For the theta power effect, we selected the same time-frequency window that we used to compute the percentage of non-phase-locked power. There was a significant negative correlation between the N2 conflict effect and theta power (Spearman’s r=-0.37, p=0.017) in the stimulus-locked analyses, which became non-significant when partialing out ERP effects at P9 (r=-0.28, p=0.08). For correlations in the response-locked analyses, we selected -25 ms for the ERP, and -200 to 0 for the theta-band power. There was a non-significant trend towards a positive correlation (Spearman's r=0.28, p=0.075), which became even weaker when partialing out shared variance with the ERP from P9 at the same time window (r=0.25, p=0.11).

In sum, the conflict effects in the ERP and in the non-phase-locked theta power appear to reflect distinct components of the EEG signal. The reduction in correlation when partialing out ERP variance at electrode P9 suggests that any shared variance between the N2 and theta power reflects non-specific individual EEG signal factors, perhaps including skull thickness, electrode impedance, or overall levels of cortical activation.

Within-subject cross-trial regression from midfrontal EEG components

Thus far, all of our analyses have involved first averaging over all trials within each subject, and then performing group-level analyses. However, it is also useful to determine how well the phase-locked and non-phase-locked parts of the EEG relate to the single-trial data within each subject. Thus, we performed two sets of within-subjects analyses to quantify the link between the different EEG signatures and conflict conditions and behavioral performance.
The first set of analyses was based on within-subject logistic regressions to predict the condition of each trial (low- or high-conflict) based on the single-trial non-phase-locked time-frequency power or on the single trial time-domain EEG signal (the “single-trial ERP”). Results from midfrontal electrode FCz are shown in Figure 5. Values indicate the percentage of subjects for whom the cross-trial regression indicated that increased power was associated with high-conflict compared to low-conflict trials. In the time-frequency regression, highest performance was seen in the theta band, peaking at around 400 ms and 6.5 Hz for the stimulus-locked analyses, and at the response time and 6.5 Hz for the response-locked analyses. Average regression performance was significantly greater than chance (p<0.001; see gray patches in line plots of Figure 5) for theta power from around 175 to 750 ms in the stimulus-locked analysis, and from around -450 to +200 ms in the response-locked analyses.

Averaged regression performance based on the single-trial ERP (that is, the time-domain EEG signal on each trial) was significant (p<0.001; see green patches in line plots in Figure 5) from around 400 to 550 ms in the stimulus-locked analysis and around -50 ms in the response-locked analyses.

Averaged regression performance was significantly greater for theta compared to the ERP (p<0.001; see thick red bar under line plots in Figure 5) from around 150 to 750 ms in the stimulus-locked analysis, and from around -425 to +50 ms in the response-locked analysis. Overall, these findings indicate that both theta power and the ERP can be used to predict the conflict condition at the within-subject level, and that the non-phase-locked theta power significantly out-performs the ERP as a statistical predictor.

Correlating cross-trial reaction time with midfrontal EEG components

The second set of within-subject cross-trial analyses linked the EEG data to conflict processing by means of cross-trial correlations with behavior. Here the idea was that if conflict-related midfrontal activity reflects neural mechanisms of detecting and resolving conflict, then trial-to-trial fluctuations in midfrontal activity should predict trial-to-trial fluctuations in task performance as measured by RT. Although fluctuations in RT may reflect a variety of processes including attention, motivation, and time since the last rest-break, differences in brain-RT correlations across conflict conditions should result in a subtraction of these general factors, thus isolating conflict-specific processes (Cohen and Cavanagh, 2011).
We computed cross-trial correlations between time-frequency power and RT separately for the total power and the non-phase-locked power. Significant correlations across conditions were seen in the delta-theta bands from around 500-1000 ms in the stimulus-locked analyses, and in the theta band from around -500 to -100 ms in the response-locked analyses (Figure 6a-b). There were significant increases in the correlation coefficients for high- compared to low-conflict trials in the theta band in both stimulus- and response-locked analyses. In none of the analyses were there significant differences in correlation coefficients between total vs. non-phase-locked power.

Statistical power analyses on the difference in Fisher-Z-transformed correlation coefficients between high and low-conflict conditions for total power (using an alpha of 0.01 and a time-frequency window of 4-8 Hz, 350-450 ms) revealed a mean/std. of 0.0513/0.0843, an effect size of 0.6085, and a statistical power of 0.8659. The statistical power for the same time-frequency window in the non-phase-locked power-RT correlations was 0.6168.

We next computed within-subject cross-trial correlations between RTs and the single-trial ERPs (the time-domain EEG signal on each trial). The single-trial ERPs significantly correlated with RTs in both conditions from around 200 to 900 ms in the stimulus-locked plots, and from around +80 to +600 ms in the response-locked plots (Figure 6c-d). However, there were no significant differences in correlation coefficients between high- and low-conflict trials. In a subsequent analysis, we quantified the extent to which the EEG on each trial resembled the trial-averaged ERP (see Methods). These single-trial weights were then correlated with RT within subject. At the group level, the correlation coefficients were weakly but reliably different from zero for both conditions (low-conflict trials, mean $r=-0.059, T_{39}=-3.87, p=0.0004$; high-conflict trials, mean $r=-0.068, T_{39}=-3.98, p=0.0002$). However, the condition difference in correlation coefficients was not significant across subjects ($T_{39}=-0.49, p=0.623$). Note that it is difficult to interpret the sign of a correlation coefficient with an ERP because its polarity is related to the orientation of the dipole. In contrast, the sign of a correlation coefficient with band-specific power has a clear interpretation, because trial-varying fluctuations in power are not dependent on dipole orientation.

**Discussion**

**Midfrontal EEG components and conflict processing**
Two electrophysiological signatures of response conflict that are most commonly studied are the 
N2 (van Veen and Carter, 2002; Yeung et al., 2004; Gajewski et al., 2008) and midfrontal theta 
power (Cavanagh et al., 2012; Nigbur et al., 2012). So far, it has remained unclear how these two 
signatures are related to each other. In some papers, authors conflate the two as if they measure the 
same neurocognitive process, but this has been purely speculative and not based on quantitative 
assessments. Some authors have suggested that midfrontal theta-band activity during cognitive 
control tasks may be the result of applying filters to a transient ERP waveform (Yeung et al., 2007), 
whereas others have shown that a substantial portion of the EEG activity time-locked to errors is 
non-phase-locked (Trujillo and Allen, 2007), and seems to correlate only weakly with the error-
related negativity (Cavanagh et al., 2012). Those previous studies focused on the error-related-
negativity, and it was not clear whether the conflict-related EEG signal also comprises largely non-
phase-locked activity. Furthermore, the functional significance of the ERP vs. the non-phase-locked 
activity with respect to task performance has not been previously investigated. Nor was their 
statistical robustness evaluated in terms of within-subject dynamics or statistical power. Other 
studies that directly compared ERP and time-frequency dynamics underlying cognitive control 
processes are more difficult to interpret. For example, Harper and colleagues (Harper et al., 2013) 
computed the time-frequency dynamics from the ERP, thus ignoring the single-trial data; 
furthermore, the authors did not control for possible global effects as we did with the partial 
correlation approach. Another study attempted to compare the N2 to midfrontal theta (Hajihosseini 
and Holroyd, 2013), but this comparison is difficult to interpret because the data were averaged 
over subjects per task and condition, thus using only six data points to compute correlation 
coefficients (each data point corresponded to one task/condition, and one data point is an outlier 
that seems to drive the effect, see Figure 3 in their paper). Furthermore, the tasks across which data 
were compared were qualitatively distinct (“oddball” infrequent event detection vs. valenced 
feedback during learning), and it is not reasonable to assume that a brain oscillation speed reflects 
the same high-level psychological processes in all tasks.

Here, we show that (1) a major component (> 80%) of task-related theta power modulations over 
mid-frontal cortex is time-locked but not phase-locked to stimulus onset or response (the specific 
modulation by conflict condition was ~100% non-phase-locked); (2) this non-phase-locked theta 
power reflected conflict conditions more reliably than the phase-locked EEG component in within-
subject regression analyses; and (3) non-phase-locked theta power also showed robust and 
condition-specific (high vs. low-conflict) single-trial correlations with reaction time, whereas the
phase-locked EEG component did not. These findings suggest that most of the task-relevant EEG signal during response conflict reflects frequency band-limited non-phase-locked theta-band oscillations within the medial frontal cortex (see also Nigbur et al., 2012). Specifically, our findings suggest a more important functional role of midfrontal frontal theta-band oscillations in conflict monitoring and top-down control of sensory-motor decision-making than the typically studied ERP correlates of response conflict, the N2 and subsequent P3.

Modulations of EEG power have been interpreted as reflecting oscillations if they are band-limited and non-phase-locked to an external stimulus or motor movement (Pfurtscheller and Lopes da Silva, 1999; Tallon-Baudry and Bertrand, 1999; Donner and Siegel, 2011) (but see David et al., 2006 for a more complex distinction between "evoked" and "induced" neural activity). The conflict-related midfrontal theta activity clearly meets these criteria. The conflict-related changes in power were spectrally localized to the theta band (with a distinct spectral peak at 6.5 Hz), and they remained nearly unchanged when subtracting the ERP (using two different methods for subtracting the ERP: assuming amplitude invariance over trials, or assuming shape-invariance and fitting the amplitude of the ERP to each trial separately). Consequently, our findings are consistent with conflict-related theta power modulations reflecting non-phase-locked oscillations.

There are several explanations for why conflict-related theta activity has previously been suspected to reflect the ERP: Both the theta power increase and the ERP occur at roughly the same time, the increase in theta power relative to the pre-stimulus baseline lasts a few hundred milliseconds, and ERPs are generally dominated by low frequency activity overlapping with (though typically extending beyond) the theta-band. Our present findings decouple the theta-band modulations from the ERP, suggesting that these two EEG signatures reflect distinct neural processes. This is consistent with previous reports of weak and often non-significant correlations between ERP and time-frequency measures of conflict and feedback learning (Cohen et al., 2007; Cavanagh et al., 2012), and is consistent with the conflict modulation primarily affecting power and not inter-trial phase clustering (that is, the phase-locked part of the signal; Nigbur et al., 2012). It remains to be determined precisely what neurocognitive processes are measured by the ERP versus the non-phase-locked theta, although it is clear that discussing the two as if they were different reflections of the same neural process is inappropriate. In the present dataset (comprising two experiments), the theta-band activity, compared to the ERP, was more closely linked to trial-varying task performance as measured by reaction time (Figures 6), and was a more robust statistical predictor
of single-trial conflict condition (Figure 5). In contrast, the N2 did not out-perform the time-frequency representation in any link to behavior or in statistical robustness.

The brevity of the conflict modulation of theta power likely reflects the phasic nature of some aspects of cognitive control processes in response conflict tasks, in particular, those top-down control processes that are maximally engaged between the stimulus and response. Invasive recordings have shown that stimulus- and task-related modulations of neural oscillations in other frequency bands tend to occur in brief bursts, both in the visual cortex (Gray and Singer, 1989) and in motor cortex (Murthy and Fetz, 1992). Further, sensory-motor decision processes are associated with brief bursts of non-phase-locked oscillatory long-range synchronization between frontal and parietal cortex (Pesaran et al., 2008). Our findings suggest that the medial frontal cortex produces brief bursts of theta-band oscillations during top-down control. These bursts are time-locked—but not phase-locked—to stimulus onset and response, and have a duration of a few (about 2-4) cycles of the theta rhythm. It is likely that these transient theta dynamics serve as a communication medium to coordinate long-range networks, as demonstrated by several findings linking inter-regional theta-band phase synchrony to cognitive control and related processes (Cavanagh et al., 2009; Cohen, 2011; Polania et al., 2012; Phillips et al., 2013).

Defining the ERP as the phase-locked time-domain component of the EEG, our findings are clearly inconsistent with the notion that the conflict-related frontal theta power results from filtering the ERP. Our definition of the ERP as comprising phase-locked activity relies on the assumption that the ERP exists at the single-trial level and has roughly the same time course on each trial (although we allowed the amplitude to vary on each trial and this did not qualitatively affect the results). One might still argue that an ERP with a random latency jitter on each trial would produce a band-limited and non-phase-locked time-frequency feature. However, this “moving-target” interpretation of the ERP simply side-steps the issue by making the ERP an unquantifiable phenomenon that is difficult to disprove or use in single-trial analyses. Furthermore, this account does not provide a compelling neurophysiologically plausible mechanism for the theta-band signal that remains after subtracting the phase-locked ERP. In contrast, accepting that the theta-band conflict modulation reflects brain oscillatory processes provides a number of compelling interpretations, links to animal physiology and computational studies, and new hypotheses, as we outline in the next section. We stress here that our interpretations and conclusions concern only the conflict-related EEG modulations; the extent to which other ERP components may or may not exist on single trials, and
how they might be related to oscillatory mechanisms, is a separate matter that should be addressed for each ERP component (e.g., Makeig et al., 2002). Indeed, it is not reasonable to assume that all ERP components are generated by the same neurophysiological mechanisms.

**Implications and conclusions**

Our results have several important implications. The first implication is that they establish a direct link between high-level studies of conflict processing in human decision-making and in-vivo and in-vitro neurophysiology studies of oscillations. Neural oscillations are evident across many spatial and temporal scales of the brain—from membrane potentials of individual neurons to intracortical field potentials to large-scale scalp EEG measurements (Varela et al., 2001; Donner and Siegel, 2011; Siegel et al., 2012). The underlying biophysical cellular and network mechanisms of oscillations are increasingly well understood (Wang, 2010), and these oscillations may provide the scaffolding for flexible local and long-range neural communication in large-scale brain networks (Fries, 2005; Miller and Wilson, 2008; Akam and Kullmann, 2012; Siegel et al., 2012). Specifically, medial prefrontal regions act as a key node in a network that monitors on-going behavior for potential conflicts, and engages other brain systems to perform online and goal-directed behavior adaptations. We speculate that theta-band oscillations reflect the medium of communication within this network (Cavanagh et al., 2009; Cohen, 2011; Cohen et al., 2011).

The second implication of our findings is to set the stage for testing the causal role of theta-band oscillations in conflict processing. For example, if theta-band oscillations underlie conflict monitoring and resolution, then exogenously boosting theta-band oscillations should boost conflict task performance (in contrast, it is difficult to image how to boost an ERP without a task). Furthermore, if theta-band oscillatory synchronization is a mechanism by which information can be integrated over large-scale brain networks (Cohen, 2011), then entraining large-scale brain networks in the theta band should improve performance on cognitive control tasks. In fact, this was recently demonstrated in humans (Polania et al., 2012). Finally, if the increase in non-phase-locked theta power is brief because cognitive control requirements are brief, then the duration of the theta power increase should correlate with the duration of time in which cognitive control remains active.

The third implication of our findings is that they clearly highlight the utility of spectral analysis of EEG data in the study of conflict processing. Even if one is not interested in brain oscillations or
neurophysiological mechanisms, from signal-processing and statistical perspectives, analysis approaches that are strongly linked to task conditions and behavioral performance (non-phase-locked theta dynamics) should be preferred over analysis approaches that are weakly or non-significantly correlated with task conditions and behavior (ERP), and that have little neurophysiological interpretability.

In conclusion, we demonstrated that the human EEG signature of response conflict is centered over midfrontal topographical regions and reflects theta-band activity that is timelocked—but not phase-locked—to stimulus onset and response. These findings indicate that conflict effects in human medial frontal cortex primarily reflect non-phase-locked neural theta-band oscillations. These oscillations may facilitate coordination of neural processing in the brain’s sensorimotor pathways to support efficient decision-making.
References


Figure 1. Overview of task timing and illustration of total vs. non-phase-locked activity from one subject, electrode Oz (177 trials). Note that the tone in experiment 2 was played into the ear (in this example, the right ear); there was no visual depiction of the tone location on the screen. Time=0 in the time-frequency plots refers to stimulus onset. Panel B shows total activity (the unadulterated data from all trials; this is the typical analysis approach), panel C shows non-phase-locked power (also sometimes called “induced” activity), which was created by subtracting the ERP from single trials. This procedure annihilates the ERP (by definition) while leaving the non-phase-locked power intact. Panel D shows the phase-locked power, defined as the difference between total and non-phase-locked power. The ERP is overlaid (y-axis scaling is arbitrary).

Figure 2. Time-frequency power plots for three electrode groupings covering three major regions involved in visual-motor decision processing: parietal/occipital (electrodes PO7, PO8, P7, and P8; panel A, and see white/black electrodes on topographical maps), midfrontal (electrode FCz; panel B), and sensory-motor (C3-C4 or C4-C3, depending on the response hand used in each trial; panel C). For each panel, time-frequency plots show non-phase-locked (top) and phase-locked (bottom; defined as the difference between the non-phase-locked and the total power, as in Figure 1) condition-averaged power, shown separately for stimulus-locked (left) and response-locked (right) data. Topographical maps show spatial distribution of selected time-frequency windows. Data in this figure were scalp-Laplacian transformed.

Figure 3. The conflict effect (time-frequency power of high-conflict trials vs. low-conflict trials) on time-frequency theta-band power. Results are shown for stimulus- and response-locked analyses, separately for the spatially unfiltered data (panels A and B) and for the surface Laplacian data (panels C and D). “stim.” is stimulus onset, and “resp.” is the time of button press. Note that the theta-band time-frequency characteristics and topographical distributions are qualitatively very similar with and without the Laplacian.

Figure 4. ERP results. Panels A and C show grand-averaged ERPs from electrode FCz, and panels B and D show the topographical maps of ERPs from time windows corresponding to statistically
significant condition differences in the FCz ERPs (see black regions in panels A and C). Note that the ERP waveforms are qualitatively different before vs. after applying the surface Laplacian, and that the topographical distributions are qualitatively distinct from the theta-band power shown in Figure 3. Note also that each topographical map has its own color scaling.

**Figure 5.** Results from within-subject logistic regression analyses, shown for electrode FCz. Based on EEG time-frequency power or time-domain amplitude, a logistic regression was applied to classify each trial as being low-conflict or high-conflict. Beta coefficients were binarized for each subject and averaged. 0.5 indicates chance-level performance, values above 0.5 indicate that greater power/amplitude predicts high-conflict trials, and values below 0.5 indicate that greater power/amplitude predicts low-conflict trials. Panels A and B show results from stimulus-related and response-related analyses, respectively. Results was tested at p<0.001 against 0.5 using non-parametric t-tests (dark gray and green bars), and for the theta accuracy against the ERP accuracy (red bar on bottom of plot).

**Figure 6.** Correlation coefficients (y-axis or color scale) over time or time-frequency between single-trial EEG data and reaction time (RT), plotted separately for stimulus-locked (panels A and C) and response-locked (panels B and D) data. Black contours in panels A and B outline regions of statistical significance. Difference maps between total and non-phase-locked power are not shown because there were no significant differences in any of the four plots. In panels C and D, gray patches indicate significant time-domain EEG-RT correlation coefficients. Although the correlation coefficients across subjects differed significantly from zero when averaging over both conditions, there were no differences in correlation coefficients between high- and low-conflict trials (hence, the gray patches are drawn to the average of the two conditions).
A) Trial timing for experiment 1 (left) and experiment 2 (right)

B) Total power

C) Non-phase-locked power

D) Phase-locked power
A) Lateral occipital/parietal electrodes (P7/PO7/P8/PO8)

Stimulus-locked

Response-locked

Non-phase-locked

Phase-locked

100-500 ms, 8-12 Hz

B) Midfrontal electrode (FCz)

Stimulus-locked

Response-locked

20-600 ms, 4-8 Hz

C) Contralateral-ipsilateral motor electrodes (C3 or C4)

Stimulus-locked

Response-locked

400-700 ms, 10-20 Hz
A) No spatial filter: Time-frequency power from FCz

B) No spatial filter: Topographical maps of theta (4-8 Hz) power

C) Scalp Laplacian filter: Time-frequency power from FCz

D) Scalp Laplacian filter: Topographical maps of theta (4-8 Hz) power
A) No spatial filter: ERPs from FCz

B) No spatial filter: topographical maps

C) Scalp Laplacian spatial filter: ERPs from FCz

D) Scalp Laplacian spatial filter: topographical maps

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<th>Condition average</th>
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A) Stimulus-related

B) Response-related

-200 stim 200 400 600 800
Time (ms)

Frequency (Hz)

-200 resp 200 400 600
Time (ms)

Average binarized t-coefficient

0.2 0.4 0.6 0.8

A) Stimulus-locked time-frequency correlations

B) Response-locked time-frequency correlations

C) Stimulus-locked ERP correlations

D) Response-locked ERP correlations