Title: **Learning to synchronize: midfrontal theta dynamics during reversal learning**

Abbreviated title: **Midfrontal theta during reversal learning**

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Abstract

In recent years, several hierarchical extensions of well-known learning algorithms have been proposed. For example, when stimulus-action mappings vary across time or context, the brain may learn two or more stimulus-action mappings in separate modules, and additionally (at a hierarchically higher level) learn to appropriately switch between those modules. However, how the brain mechanistically coordinates neural communication to implement such hierarchical learning, remains unknown. Therefore, the current study tests a recent computational model that proposed how midfrontal theta oscillations implement hierarchical learning via the principle of binding by synchrony (Sync model). 64-channel EEG signal was recorded while 27 subjects performed a probabilistic reversal learning task. In line with the Sync model, post-feedback theta power showed a linear relationship with negative prediction errors, but not with positive prediction errors. This relationship was especially pronounced for subjects with better behavioral fit (measured via AIC) of the Sync model. Also consistent with Sync model simulations, theta phase-coupling between midfrontal electrodes and temporo-parietal electrodes was stronger after negative feedback. Our data suggest that the brain uses theta power and synchronization for flexibly switching between task rule modules, as is useful for example when multiple stimulus-action mappings must be retained and used.

Significance Statement

Everyday life requires flexibility in switching between several tasks. A key question in understanding this ability is how the brain mechanistically coordinates such switches. The current study tests a recent computational framework (Sync model) that proposed how midfrontal theta oscillations coordinate activity in hierarchically lower task-related areas. In line with predictions of this Sync model, midfrontal theta power was stronger when rule switches were most likely (strong negative prediction error), especially in subjects who obtained a better model fit. Additionally, also theta phase connectivity between midfrontal and task-related areas was increased after negative feedback. Thus, the data provided support for the hypothesis that the brain uses theta power and synchronization for flexibly switching between tasks.

Switching between tasks is key to function in a complex and rapidly changing environment. For instance, when at the pub with friends, behavior is likely guided by different social rules than at work. However, when the boss suddenly walks into the pub, this might require to flexibly switch between these two sets of social rules. Importantly, an empirically valid model that explains how the human brain mechanistically deals with such switches, remains lacking.

In experimental settings, this cognitive flexibility is typically tested in a reversal learning setup (Izquierdo, Brigman, Radke, Rudebeck, & Holmes, 2017). Here, agents must learn task rules consisting of collections of stimulus-action mappings. During the task, these rules are regularly reversed. One popular framework to explain performance during reversal learning tasks is the Rescorla-Wagner model (RW; Rescorla & Wagner, 1972; Widrow & Hoff, 1960). Here, on every trial, obtained reward is used to update the value of active stimulus-action mappings. By learning fast, the agent can flexibly deal with changes in task rules. However, when feedback is probabilistic (e.g., Cools, Clark, Owen, & Robbins, 2002), this approach experiences difficulties. Specifically, a high learning rate will lead agents to "chase the noise" introduced by probabilistic feedback. In contrast, a low learning rate increases robustness against noise, but decreases flexibility on rule switches. Thus, some have proposed that learning rate should be adaptive (e.g., Bai, Katahira, & Ohira, 2014; Behrens, Woolrich, Walton, & Rushworth, 2007; Silvetti, Vassena, Abrahamse, & Verguts, 2018). In this adaptive learning rate (ALR) proposal, agents track rule switches by comparing an estimate of reward probability to the received reward. Consistently high prediction errors indicate that the underlying rule has changed, and learning rate should be increased. More fundamentally however, irrespective of learning rate flexibility, both the RW and ALR framework assume that, on every rule reversal, old information is overwritten. Especially for more complex problems, this is inefficient; as is demonstrated by the problem of catastrophic forgetting in artificial neural networks (French, 1999)).

To overcome catastrophic forgetting, separate mapping collections for every task rule may be stored (Saez, Rigotti, Ostojic, Fusi, & Salzman, 2015; Wilson, Takahashi, Schoenbaum, & Niv, 2014). However, this poses the agent with a new problem of keeping track which task rule is currently relevant. Recent fMRI research focusing on this hierarchical approach toward reversal learning has pointed to

midfrontal cortex as responsible neural structure for keeping track of the current task rule (Wilson et al., 2014). However, how midfrontal cortex mechanistically coordinates neural communication in switching between task rules, remains an open question.

This question was recently addressed by a novel computational framework of hierarchical learning (Verbeke & Verguts, 2019). As proposed in (Wilson et al., 2014), this model retains separate mappings for every task rule. It keeps track of rule reversals by calculating prediction error. In order to guide neural communication between areas holding the appropriate mappings, the model relies on binding by synchrony (BBS; Fries, 2005, 2015; Gray & Singer, 1989; Womelsdorf et al., 2007) in theta frequency (4-8 Hz). Specifically, midfrontal theta oscillations synchronize neuronal activity along task-relevant pathways. Thus, task-relevant neurons can communicate and learn, while stability is achieved in currently irrelevant pathways.

The current study empirically tests this model, further on referred to as Sync model (Fig 1B). For this purpose, the Sync model is fitted on data of subjects performing a probabilistic reversal learning paradigm, and empirically compared to alternative models (Bai et al., 2014; Rescorla & Wagner, 1972). Then, Sync model simulations provided several predictions for EEG measured while subjects performed this task, specifically in theta frequency (model-driven EEG predictions). First, a linear relationship between midfrontal theta power and negative prediction errors was predicted, especially in subjects with good behavioral Sync model fit. Second, a peak of midfrontal theta power was predicted for data locked to rule switches. Third, phase coupling between midfrontal and posterior electrodes was predicted to be stronger after negative feedback.

Materials and Methods

Materials

The experiment was run on a Dell Optiplex 9010 mini-tower running PsychoPy software (Peirce et al., 2019). Electrophysiological data were recorded using a BioSemi ActiveTwo system (BioSemi, Amsterdam, Netherlands) with 64 Ag/AgCl electrodes arranged in the standard international 10–20 electrode mapping (Jasper, 1958), with a posterior CMS-DRL electrode pair. Two reference electrodes were positioned at the left and right mastoids. Eye movements were registered with a pair of

electrodes above and below the left eye and two additional electrodes at the outer canthi of both eyes. EEG signals were recorded at a sampling rate of 1024 Hz.

Models were fitted using the differential evolution method of the SciPy (version 1.4.1) package in Python (version 3.7.6). Other behavioral analyses were done using R software (R Core Team, 2017). The electrophysiological data were preprocessed in MATLAB R2016b (The MathWorks Inc., 2016) using an EEGLAB preprocessing pipeline (Delorme & Makeig, 2004). Also for simulations of the Sync model MATLAB R2106b was used.

Code and Data Accessibility

All code used to provide the results described in the current paper is provided at https://github.com/CogComNeuroSci/PieterV_public/tree/master/Reversal_learning. At publication, also the data will be made freely accessible at https://osf.io/wt36f/.

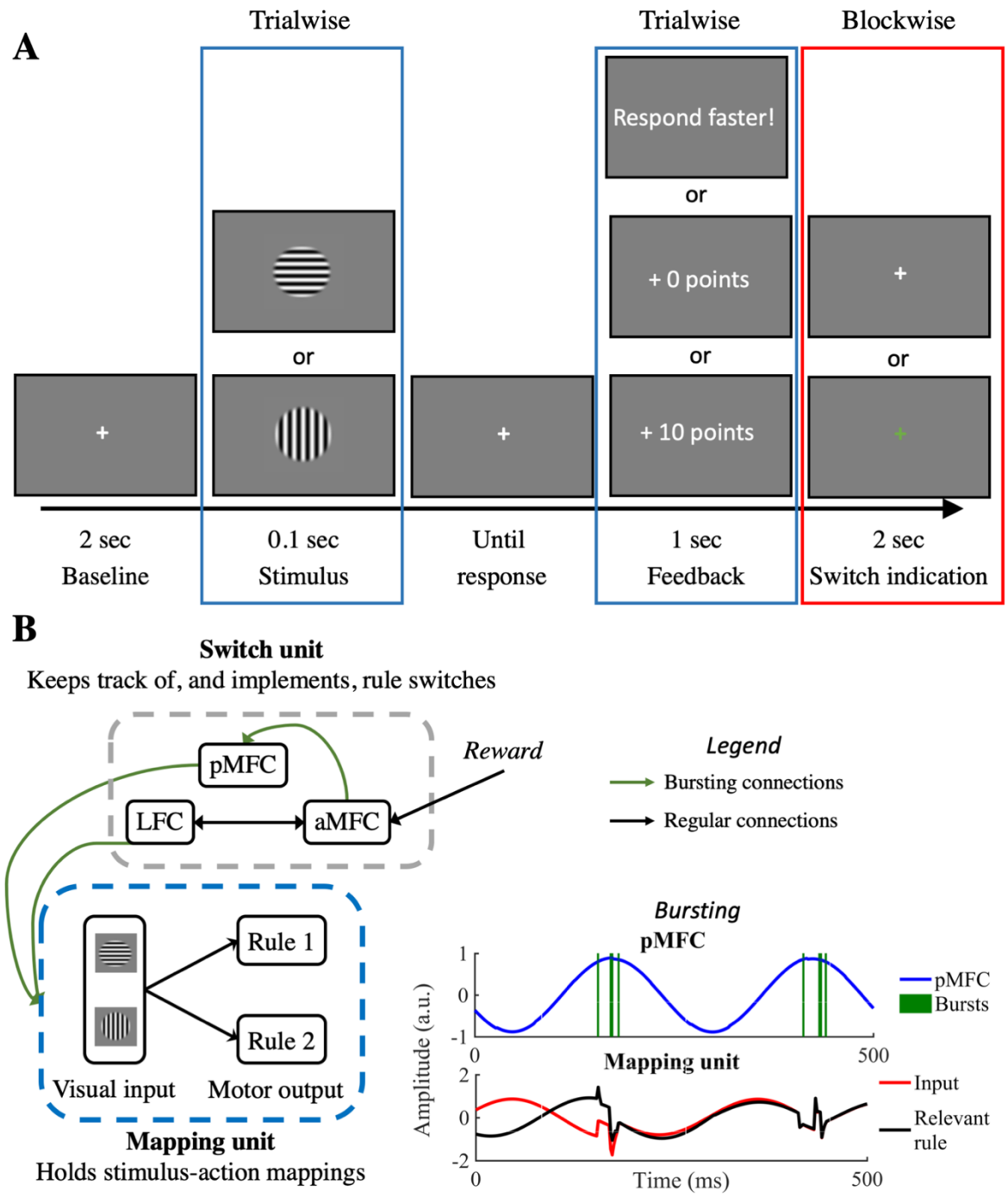


Fig 1. Methods. *A: The task*. The time course of one trial in the experimental paradigm is shown. Elements highlighted by a blue rectangle, such as the presented stimulus and feedback, are manipulated on a trial-by-trial basis. Elements highlighted by the red rectangle are manipulated blockwise. Here, the fixation cross after feedback was green in one experimental block (half of all trials). In this reporting block, subjects had to press space during this period if they thought the rule had switched. *B: The Sync model*. The left diagram represents a schematic overview of the Sync model. In the lower right corner, a detailed illustration shows how bursts originating in the

pMFC synchronize task-relevant areas in the Mapping unit (see (Verbeke & Verguts, 2019) for detailed explanation).

Experimental Task

Both the model (27 simulations) and human subjects (N = 27) performed a probabilistic reversal learning task (see Fig 1A). Agents had to learn task rules consisting of two stimulus-action mappings which were regularly reversed during the task. Every trial started with a centrally presented white fixation cross for 2 seconds. Then the stimulus was presented for a period of 100 milliseconds. This stimulus was a centrally presented circular grating with a raised-cosine mask and a size of 7 visual degrees. The grating was either vertically or horizontally oriented. After stimulus presentation, the screen turned blank until response. Responses were given by pressing the 'f'- (left) or 'j'-key (right) on an azerty keyboard. In task rule 1, the horizontal stimulus mapped to a left response and the vertical stimulus to the right response; this was reversed for task rule 2. During the task (480 trials), 15 rule switches were introduced. These rule switches occurred at random (uniform distribution from 15 to 45 trials after the previous task switch). After response, feedback was presented in the center of the screen. This feedback consisted of '+10 points' for rewarded trials, '+0 points' for unrewarded trials or 'Respond faster!' when response times (RT) were more than 1 second. After feedback, the fixation cross appeared again for another 2 seconds. Crucially, the experiment was divided into two experimental blocks (240 trials each). In one block, the reporting block, the post-feedback fixation cross was presented in green. During this period, subjects were instructed to press the space bar if they thought the task rule had switched. The purpose of this approach was to obtain an indication of when the subject reached his or her own 'Switch threshold', as happens in the Sync model. This was only done during one block, so critical changes due to this difference in task structure could be checked. The order of the two blocks was counterbalanced across subjects. In between blocks, as well as three times within a block, subjects were allowed a small break. This break could only occur if there was no rule switch within 10 trials from the break.

Human Testing Procedure

34 subjects participated in this study, 7 subjects were removed because of either technical problems with the EEG recording (4) or an inability to give a correct response on more than 2/3 of the trials (3), resulting in $N = 27$. Subjects were told they would receive €25 for their participation, with a possibility to earn up to €3 extra reward depending on their performance.

Before starting the task, the subject had to go through two short practice sessions with gratings that were tilted 45° to the left or to the right relative to a vertical line. In the first practice session, the subject performed 30 trials with only one task rule. Here, the goal was to let the subject get acquainted with the general paradigm and learn a task rule through probabilistic feedback. Subjects were only allowed to continue to the second practice session if they performed above chance level (50%) and could report the correct task rule to the experimenter. If not, they performed this practice session again. In the second practice session, subjects performed 60 trials of the task with 3 rule switches and with the post-feedback green fixation cross (as in the reporting block). In this session, subjects pressed the space bar to indicate a task switch and received feedback for each press. The press was considered correct if subjects responded within 10 trials from the actual rule switch. They were allowed to continue to the next task if they were able to perform above chance level and had at least 1 correct indication of a rule switch. After successfully performing both practice sessions, subjects performed 480 trials of the actual task.

Behavioral Analyses

To check for differences between the reporting block (green fixation cross) and the non-reporting block (see Experimental Task and Fig 1A), paired t-tests were performed for both accuracy and RT, depending on experimental block. In order to deal with the skewed distribution of RT, the natural log of RT was used for all analyses. Additionally, trials with too late responses were excluded for both behavioral and EEG analyses.

Model Analyses

Model formulation

More extensive analyses of behavioral data were done with a model-based approach. Current work aims to test the Sync model (Verbeke & Verguts, 2019), but two baseline models were fitted as well. In the following section, we first provide a conceptual overview, followed by a more detailed description of all three models. Then, we describe how model fit was evaluated.

An overview of model architecture is provided in Fig 1B. The RW and ALR model are restricted to only the Mapping unit (with one rule module). The RW model (Rescorla & Wagner, 1972) had a constant learning rate while the ALR model (Bai et al., 2014), was implemented with an adaptable learning rate. The Sync model consists of two units, the Mapping and Switch unit. The Mapping unit contains a classic network with 2 layers (visual input and motor output). Here, weights are adapted with the RW algorithm (Widrow & Hoff, 1960). In the Sync model, 4 nodes (2 for each response option) at the motor output layer, are divided in 2 rule modules, one for each task rule. Hence, as in (Wilson et al., 2014), the Mapping unit holds separate stimulus-action mappings for each task rule. In addition, a Switch unit forms a hierarchically higher network modeled after primate prefrontal cortex. This Switch unit keeps track of switches in task rule. Specifically, the Switch unit consists of the lateral frontal cortex (LFC), posterior medial frontal cortex (pMFC) and anterior midfrontal cortex (aMFC). Here, the LFC holds pointers (Botvinick et al., 2001) that indicate which rule should be synchronized in the Mapping unit. Since BBS implements gating, allowing efficient communication between synchronized nodes and inefficient communication between non-synchronized nodes (Fries, 2005, 2015), the agents' behavior will be guided by the synchronized rule. This synchronization process is then executed by the binding by random bursts principle (Springer & Paulsson, 2006; Verguts, 2017; Zhou, Chen, & Aihara, 2005). In the Sync model, a theta-frequency-paced signal produced in the pMFC is responsible for sending these bursts (see (Verbeke & Verguts, 2019; Verguts, 2017) for details). The aMFC contains a neural network (for simplicity not shown in Fig 1B) that is adapted from previous work (Silvetti, Seurinck, & Verguts, 2011). Here, again RW learning is employed but on a hierarchically higher level. More specifically, the aMFC learns an expected reward (V) for the currently used rule module (see equation (4)). Which rule module is currently used, is extracted from activation in the LFC. The expected reward is then compared to an external reward signal (*Rew*; Reward in Fig 1B) in order to

compute prediction errors. The negative prediction error signal is propagated to both the Accumulator neuron (within the aMFC neural network) and to pMFC. A single negative prediction error increases (via bursting) the power of the theta signal in pMFC (bursting connection in Fig 1B; see (Verbeke & Verguts, 2019) for details). Instead, the Accumulator neuron evaluates the prediction error signal on a slower time scale, and thus requires multiple prediction errors before activation in the Accumulator neuron reaches its Switch threshold (see equation (5)). When this happens, it signals the need for a switch to the LFC. Correspondingly, the LFC will change the signal to the Mapping unit, and synchronize another rule module. In sum, bursts received by the Mapping unit are the result of a cooperation between LFC and pMFC. Here, the pMFC determines the intensity of theta bursts while the LFC determines which task rule in the Mapping unit is susceptible to the bursts. For further details see (Verbeke & Verguts, 2019).

All nodes in the visual input and motor output layer of the Mapping unit as well as the pMFC are oscillatory nodes. In line with previous work (Verguts, 2017), oscillatory nodes contain neuronal triplets. In the pMFC, which executes top-down control by sending bursts, activity oscillates at a theta (6 Hz) frequency, in line with suggestions of previous empirical work (Cavanagh & Frank, 2014; Womelsdorf, Johnston, Vinck, & Everling, 2010). Different from our previous modelling work, a theta frequency was used in the Mapping unit (see Discussion) as well. Since bursts lead to a significant increase of power, a radius parameter (r_{min}) is implemented in order to attract power back to baseline after a burst. Since continuously high pMFC power is computationally suboptimal and empirically implausible (Holroyd, 2016), power in the pMFC was attracted towards a smaller radius, $r_{\min} = .50$. How fast oscillations decay to baseline is determined by a damping parameter (D) which was set to $D = .30$ in the Mapping unit. Since the pMFC not only receives burst but also sends them, a slower decay $D = .01$ was implemented here to allow a sufficient activity window (~ 500 ms/3 theta cycles) for bursts to be sent. In order to reduce model complexity, no oscillations were used in the LFC and aMFC. For a full description of model dynamics see (Verbeke & Verguts, 2019).

We turn now to the more detailed descriptions. On every trial, the RW and ALR models respond with a probability based on the following softmax rule:

$$p(a) = \frac{e^{Q(s,a)/\tau}}{\sum e^{Q(s,a_i)/\tau}} \quad (1)$$

in which $Q(s,a)$ is the value of a given stimulus-action pair (s, a). τ is the temperature parameter which determines how strongly the subject explores different actions. On every trial, $Q(s,a)$ is updated based on the rule:

$$Q(s,a)_{j+1} = Q(s,a)_j + \alpha * (Rew - Q(s,a)_j) \quad (2)$$

in which α is the Mapping learning rate and *Rew* is the reward received by the agent.

In the ALR model (Bai et al., 2014), the learning rate changes in an adaptive manner. Here, the Mapping learning rate is updated on every trial by

$$\alpha_{j+1} = \eta * \left|Rew - Q(s,a)_j\right| + (1 - \eta) * \alpha_j \quad (3)$$

in which η determines how strongly the learning rate is influenced by the current difference between *Rew* and *Q* (lower-level prediction error).

The third model is the Sync model. In order to implement BBS, on every trial multiple time steps were simulated in which oscillations occurred. Here, motor nodes accumulate activation over time. The motor node with the maximal accumulated activation over time, was considered as the model response. Values of stimulus action pairs (*Q*) in each rule module (R) are updated similar to equation (2). As described above, this model has an additional Switch unit which adds a hierarchical learning algorithm on top of the RW (fixed learning rate) model in the Mapping unit. This Switch unit evaluates whether there was a rule switch. More specifically, it learns a value for every rule module by

$$V(R)_{j+1} = V(R)_j + \alpha_{high} * (Rew - V(R)_j) \quad (4)$$

in which α_{high} is the higher order Switch learning rate. The absolute difference between the expected value $V(R)$ in equation (4) and the obtained *Rew* (i.e., the unsigned prediction error) is accumulated in the Accumulator neuron (A) via

$$A_{j+1} = \begin{cases} \gamma * A_j + (1-\gamma) * \left|Rew - V(R)_j\right| & if\ \left(Rew - V(R)_j\right) < 0 \\ \gamma * A_j & otherwise \end{cases} \quad (5)$$

Here, γ is the Cumulation parameter which determines how strongly the Accumulation neuron is affected by a single prediction error. While a low Cumulation parameter causes the agent to strongly weigh single prediction error and therefore regularly switch between rule modules, a high Cumulation parameter implements a more conservative approach. Since switches are only required when negative feedback occurs, the Accumulator neuron is selective for negative prediction errors. When several negative prediction errors occur in succession, this Accumulator neuron will reach a Switch threshold which is set to .5, and the model will switch to another rule module (R) in the Mapping unit.

For behavioral data fitting only, the full Sync model was simplified by introducing a hard gating process between task rules instead of BBS and a response selection mechanism similar to (1). This allowed to skip the loop of 1500 timesteps every trial, which was needed to simulate oscillations. We refer to this model as the behavioral Sync (bSync) model.

Model evaluation

For each subject, the goodness of fit of these three models was compared by using three measures. The log-likelihood (LL)

$$LL = \sum_{j=1}^{J} a_j * ln\ (p(a_j = 1)) + (1 - a_j) * \ln\ (p(a_j = 0)) \quad (6)$$

in which $p(a)$ is the probability of the given action (see equation (1)) and J represents the number of trials. The AIC uses this LL but includes a penalty for the number of parameters (k) that were used in the model:

$$AIC = 2 * k - 2 * LL \tag{7}$$

From this AIC, AIC weights ($wAIC$) can be derived which allows to make a relative comparison between the model fit of the three different models. These wAIC values are computed as

$$wAIC = \frac{e^{-\frac{1}{2}\Delta AIC_m}}{\sum_{m=1}^{M} e^{-\frac{1}{2}\Delta AIC_m}} \tag{8}$$

in which M is the number of models that are compared (3) and

$$\Delta AIC_m = AIC_m - \min(AIC) \tag{9}$$

Here, min(AIC) is the lowest AIC value out of the three models for that subject. Thus, Equation (8) results in a wAIC value for each model. The sum of all three wAIC values is 1, and if all three models fit the data equally well, wAIC equals .33 for all three models.

Simulations

In order to provide hypotheses for EEG data, 27 simulations of the full Sync model were performed. For all simulations, the same parameter values were used. These parameter values were sampled from the estimated parameter distributions of the bSync model so that overall accuracy of model simulations (M = 78.00%, SD = 1.30) closely resembled accuracy of subjects (M = 76.80%, SD = 4.91). This resulted in a Mapping learning rate (α) of .8, a Switch learning rate (α_{high}) of .1 and a Cumulation parameter (γ) of .3. The full Sync model did not use a Temperature (τ) parameter but the synchronization procedure introduces noise which also introduces some randomness in behavior. The

Switch threshold was always fixed to .5. Since the current model does not provide RTs, a period of 500 ms was used as trial period in which the visual layer received stimulation. Thereafter, 1500 ms of inter-trial interval was simulated in order to provide a post-feedback period that could be analyzed in the same way as the empirical data. All other aspects of the task, such as the frequency and timing of rule switches, were the same for the model as for the human subjects.

Power Analyses

Time-frequency decomposition was performed on the excitatory neuron within the neuronal triplet of the model's pMFC node in the model. Complex Morlet wavelets were used for frequencies between 2 and 48 Hz defined in 25 logarithmically spaced steps. For each frequency, between 3 and 8 cycles were used, also defined in 25 logarithmically spaced steps. Power was extracted as the squared absolute value of the time-frequency decomposed signal. In order to locate activity that was specific to feedback processing, the difference between power in trials with negative feedback and trials with positive feedback was computed. For simplicity, we selected the 2.5% most positive values as cluster of interest. This cluster contained one group of data points in theta frequency and approximately 250-500 ms after feedback. On every trial, the mean power in this cluster was computed and entered in the consecutive analyses. Since a negative prediction error in the model increases activity of the pMFC, we performed a linear regression of cluster power with prediction error as the independent variable. To test our first hypothesis, that this relationship was specific to negative prediction error, a second regression model was used that also included the interaction between prediction error and reward. The second hypothesis states that because negative prediction errors are strongest at the moment of a rule switch, a peak of post-feedback theta power should be found when we lock data to rule switches. To investigate this, we extracted power from the model cluster in trials within a 31-trial window around the rule switch (-15 to +15). The time course (one data point for each of the 31 trials) that resulted after averaging over all (15) rule switches and all (27) simulations, was then used as a regressor in a linear regression with data from the empirical clusters.

Phase Analyses

Our third hypothesis stated that phase coupling between pMFC and model nodes in the Mapping unit was stronger after negative feedback. Specifically, theta power in the model pMFC increases after negative prediction error. When there is sufficient power in the pMFC, it will increase synchronization in the Mapping unit (posterior/lateral task-related regions, e.g., pre-motor or visual areas). For this purpose, the pMFC uses binding by random bursts (Verguts, 2017). Here, the pMFC will at specific phases, send bursts to the Mapping unit. Thereby it will shift the phase of neurons in the Mapping unit (see Verbeke & Verguts, 2019 for details). This leads to phase shifts in these lower pre-motor or visual task-related areas, and a short period of phase-alignment between these task-related areas in the Mapping unit and pMFC. Phase was extracted in all model nodes by taking the angle of the Hilbert transform of the raw signal. For simplicity the model was implemented without inter-areal delays. Furthermore, in contrast to analyses on the empirical EEG data (see equation (10)), control for volume conduction was not needed, so the regular phase locking value (PLV; Lachaux, Rodriguez, Martinerie, & Varela, 1999) was computed between the model pMFC and the nodes in the motor layer of the Mapping unit. This PLV was then averaged over all 4 motor nodes and the time period included in the power cluster (~250-500 ms post feedback).

EEG Analyses

Preprocessing

The data were re-referenced offline to the average of the mastoid electrodes. Breaks or other offline periods were manually removed. Particularly noisy electrodes were interpolated between neighboring electrodes on all timesteps. For three subjects one electrode was interpolated, for another three subjects we had to interpolate two electrodes; because of a bridge, one subject needed interpolation for five posterior electrodes. Additionally, activity was band-pass filtered between 1 and 48 Hz in order to remove slow drifts and line noise of 50 Hz. Eyeblinks and other motor-related noise components were removed through EEGLAB independent component analysis (ICA). After ICA-removal, the data was epoched, once locked to feedback onset and once to stimulus onset. The epochs based on stimulus onset were used to extract baseline activation, which was -1500 to -500 ms relative to stimulus onset. This baseline activity was subtracted from all epochs. After epoching, on average 7.5% of epochs were

removed by applying an amplitude threshold of -500 to 500 mV and an improbability test with 6 standard deviations for single electrodes and 2 standard deviations for all electrodes, as described in Makoto's preprocessing pipeline (Makoto, 2018). Before time-frequency analyses, data was also downsampled to 512 Hz.

Time-frequency Decomposition

Time-frequency decomposition was based on code from (Cohen, 2014). Similar to model analyses, complex Morlet wavelets were used for frequencies between 2 and 48 Hz defined in 25 logarithmically spaced steps. For each frequency, between 3 and 8 cycles were used, also defined in 25 logarithmically spaced steps.

Power Computation

A baseline correction was applied to the power estimates for each subject, electrode and frequency separately, based on the average baseline activity (-1500 ms to -500 ms from stimulus onset) across all 480 trials. Finally, the baseline-corrected data underwent a decibel conversion. Before final analyses, also trials with late responses were removed from the data.

Power Cluster Analyses

Similar to model analyses, we were interested in activity selective for feedback. Hence, a contrast between Z-scored power in trials with negative feedback and trials with positive feedback was computed. On these values, a non-parametric clustering procedure was applied (Maris & Oostenveld, 2007). The distribution of statistics was computed. On each side of the distribution (two-sided test), the 1% most extreme values were entered into the clustering analysis. From these, we clustered adjacent neighbors in the channel, frequency and time domains. To calculate our cluster-level statistic, we multiplied the number of items (i.e., (channel, frequency, time) points) in the cluster with the largest statistic of that cluster. A significance threshold of 5% was imposed on the subsequent non-parametric permutation test with 1000 iterations. Clusters that survived this permutation test were taken into the consecutive analyses. As an exploratory analysis, we also extracted mean cluster statistics for each subject, and ran a Spearman rank correlation of these statistics with wAIC of the bSync model obtained in the behavioral model fitting procedure.

Midfrontal Theta Power and Prediction Error

The Sync model uniquely yields specific EEG predictions, to which we now turn. To test the first model-driven EEG hypothesis of a relation between theta power and prediction errors, we first extracted a measure of prediction error for every subject on every trial by simulating the bSync model. This measure of prediction error was then used in a linear mixed effects models as a predictor for the mean Z-scored power of every cluster that was selective for feedback. Here, a random intercept for every subject was included and a fixed slope. Because the Sync model predicted different relationships for positive prediction errors and negative prediction errors, also the interaction between prediction errors and reward was tested. Additionally, in order to explore whether the wAIC influenced the interaction between prediction errors and reward, also a three-way interaction between prediction error, reward and wAIC was tested. More specifically, three regression models were fitted: One in which only prediction error was included as regressor, one in which both prediction error and the interaction between prediction error and reward were included, and finally a third model in which the main effect, the two-way interaction, and an extra three-way interaction between prediction error, reward and wAIC was included. These regression models were then compared via ANOVA.

Rule Switch Locking

A second model-driven EEG hypothesis considers theta power locked to the moment of a rule switch. For this analysis, EEG data of 31 trials around the rule switch (-15 to +15 trials, including the rule switch trial itself) were extracted. On these trials, the mean power for every cluster selective for feedback was computed. This data was then again averaged over all switches, giving us a trial-to-trial time course of mean cluster-power from -15 trials before rule switch to 15 trials after rule switch for every subject. On each time point, a 99.84% confidence interval (CI) was computed based on a Bonferroni correction for multiple comparisons (100-(5/31)). This confidence interval was compared to a baseline power. Baseline power was computed based on the mean power in this cluster, averaged over all trials that were more than 15 trials removed from the rule switch.

As the rule switch trial, we considered both the actual rule switch and the subjective indication of a rule switch. Hence, power close to a rule switch was compared with the mean power of trials that

were far from the rule switch. When the confidence interval did not include the baseline value, power on this trial was considered as significantly deviating from baseline. Additionally, we aimed to investigate the similarity between the data pattern predicted by the model and the empirical data. For this purpose, data from the model simulations (see above for details) was used as a linear regressor for the empirical data. Also for this hypothesis, an extra analysis was performed to investigate whether wAIC had an influence on the observed effect. Here, we extracted subject data on trials of which cluster power significantly deviated from baseline and used this data as a dependent variable in a linear regression with wAIC.

Midfrontal-posterior Phase-coupling Analyses

For the third model-driven EEG hypothesis, we considered all midline electrodes (10) as seed and other electrodes (54) as receiver in the phase connectivity analyses. Because we were interested in phase-locking related to rule modules conveying the correct response, all data was lateralized with respect to the correct response. All data ipsi-lateral to the correct response was brought to the left electrodes; all contra-lateral data was brought to the right electrodes. The iPLV (Bruña, Maestú, & Pereda, 2018) was computed between all midline electrodes and all lateral electrodes for every time point in the feedback-locked data. This iPLV measure was computed by the following equation

$$iPLV = \left| \frac{1}{n}\sum_{t=1}^{n} Im(e^{-i(\Delta\varphi_t)}) \right| \tag{10}$$

which computes the average phase angle (φ) difference over trials (t). By only looking at the imaginary (Im) part of this phase angle difference, phase differences of zero are eliminated. Hence volume conduction effects are excluded, because such volume conduction effects are represented in zero-phase differences (Bruña et al., 2018; Nolte et al., 2004). Again, a non-parametric cluster algorithm was performed on the contrast between iPLV for trials with negative versus positive feedback. For this analysis, only data of one midline electrode was used. More specifically, we checked on which of the 10 midline electrodes the mean contrast in the theta frequency (4-8 Hz) reached a maximum. This was

in the FCz electrode, hence only iPLV between FCz and all lateral electrodes were entered in the clustering algorithm. As for power, an exploratory analysis was performed in which we extracted mean cluster statistics for each subject, and ran a Spearman rank correlation of these statistics with wAIC of the bSync model obtained in the model fitting procedure.

Results

Behavioral Data

A paired t-test confirmed that there were no significant differences between the experiment block in which subjects had to indicate when a task switch happened or when they did not have to indicate this (see Materials and Methods for details), neither in accuracy ($t(26) = .029$, $p = .977$), nor in RT ($t(26) = -1.290$, $p = .208$).

Model Analyses

The distribution of all fitted parameter values for each model is given in Fig 2A. Simulations of the models with these parameter values allowed us to estimate a learning curve that illustrates how accuracy increases after rule switches (Fig 2B). Goodness of fit measures are summarized in Table 1. Here, log-likelihood was highest (best) for the bSync model, lowest for the ALR model, with the RW model in between. When a penalty for model complexity was applied (AIC, wAIC), the RW and bSync models performed approximately equal. Importantly, wAIC results indicated significant differences across individuals. As illustrated in Fig 2C, subjects could be roughly divided into three groups based on the wAIC of the bSync model. In one group (8 subjects), the wAIC were significantly smaller (better) for the bSync model ($M =. 12$, $SD = .026$) than for the RW model ($M = .78$, $SD = .027$). A second group (7 subjects) showed wAIC values that were approximately equally strong for the bSync ($M = .44$, $SD = .036$) as for the RW model ($M = .50$, $SD = .032$). In a third group (12 subjects), the bSync model showed wAIC that were significantly higher for the bSync model ($M = .64$, $SD =. 027$) than for the RW model ($M = .32$, $SD = .026$).

Model	Mean LL	SD LL	Mean AIC	SD AIC	Mean wAIC	SD wAIC
RW	-208.08	.07	420.16	.13	.51	.20
ALR	-209.31	.05	424.63	.10	.05	.02
bSync	-206.35	1.11	420.70	2.22	.44	.22

Table 1. Goodness of fit measures. Results of log-likelihood (LL), AIC and wAIC computations over subjects are shown for each of three models. For LL and wAIC, high values indicate a better fit, while for AIC a low value indicates a good fit.

Three parameters of the bSync model showed a significant correlation with wAIC (Fig 2D). These parameters were the Switch learning rate (*rho* = -.761, $p < .001$), the Cumulation parameter (*rho* = -.708, $p < .001$), and the Temperature parameter (*rho* = -.497, $p = .008$). There was no significant correlation with the Mapping learning rate (*rho* = -.145, $p = .468$). Additionally, a correlation test between accuracy and wAIC revealed that the bSync model fitted significantly better for subjects with a lower accuracy (*rho* = -.510, $p = .007$).

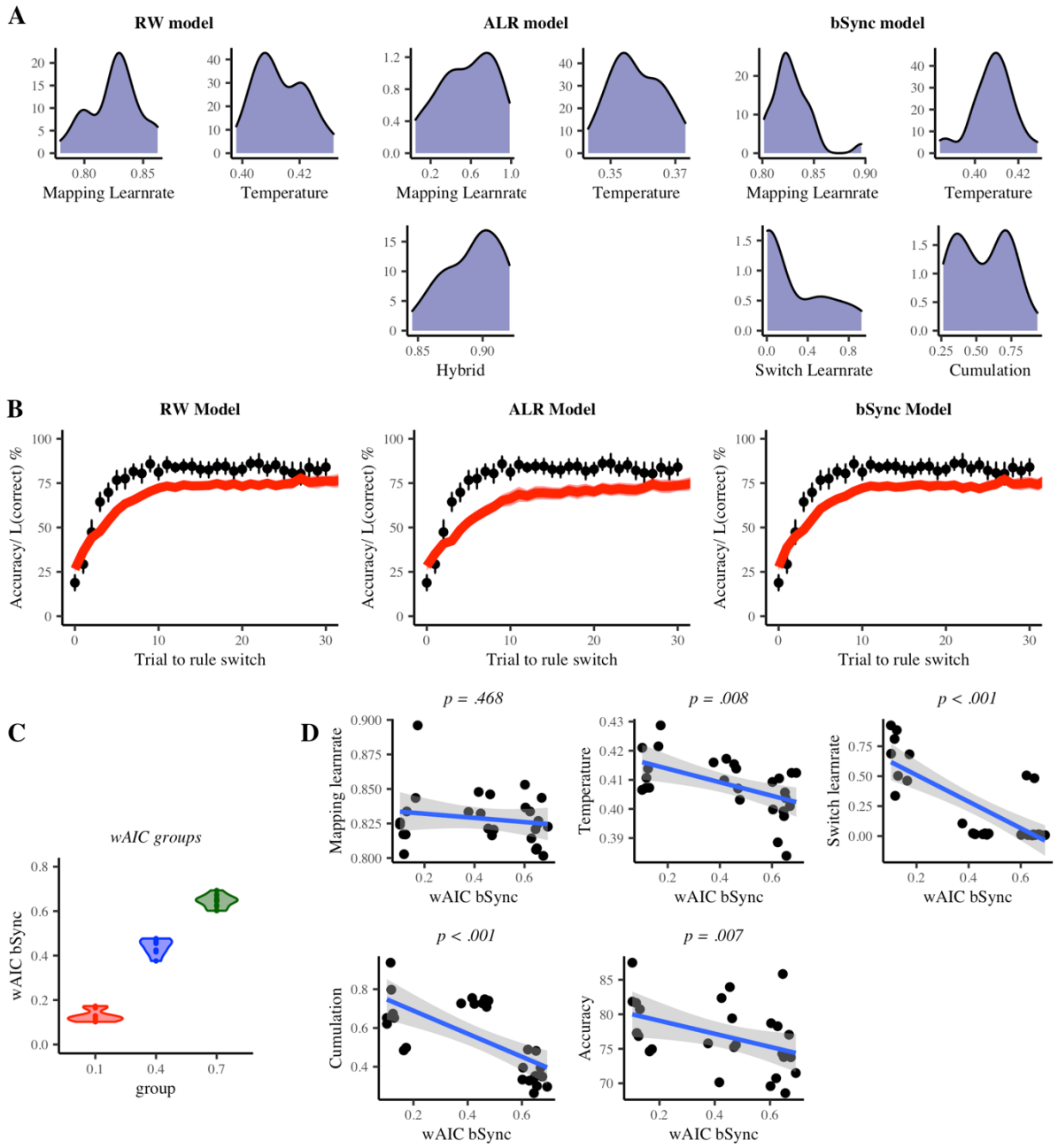


Fig 2. Model comparison. *A: Parameter distributions*. Distributions of fitted parameter values are shown for each model. *B: Learning curve fit*. Black dots represent the mean accuracy data over all subjects. The error bars show the 95% confidence intervals. The red line illustrates the mean Likelihood of data that was simulated with the fitted parameter values. The shade represents the 95% confidence interval. *C: wAIC groups*. This figure illustrates how wAIC values can be roughly divided in three groups (colors). *D: Correlation plots*. Correlations are shown between wAIC of the bSync model and all parameters of the bSync model. In the lower middle plot, also the correlation between wAIC and task accuracy is shown.

EEG and Model Data

Power Cluster Analyses

Cluster analysis on post-feedback power revealed three significant clusters that were selective for feedback processing (Fig 3). All three clusters appeared between 0 and 750 ms from feedback onset. As was predicted by the Sync model (Fig 3A), one of these clusters was in the theta frequency range (~ 4-8 Hz) and located on midfrontal electrodes (Fig 3B, D). This theta cluster showed more power when feedback was negative than when it was positive. Additionally, we found two clusters located on the posterior channels. One of these clusters was in the delta frequency (< 4 Hz; Fig 3B, E), the other cluster was located in the alpha-frequency range (~ 8-15 Hz; Fig 3B, C). Both the delta and alpha cluster showed less power for negative feedback than for positive feedback. No correlation between the power contrast of a cluster and subjects' wAIC for the bSync reached significance.

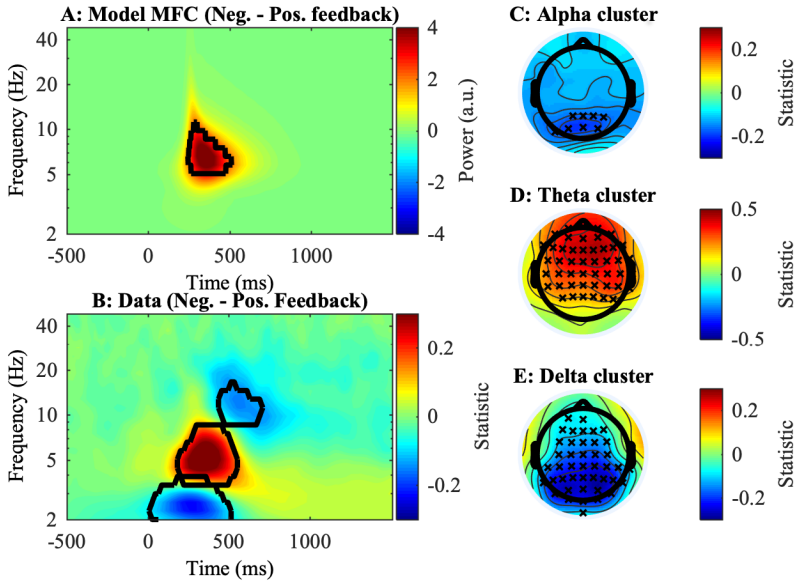


Fig 3. Power results. *A-B: Time-Frequency plots of contrast (Negative – Positive feedback)*. Significant clusters are indicated by the black contour line. *A:* Contrast of power in the model pMFC. *B:* Contrast for Z-scored power in the human data, averaged over all 64 electrodes. *C-E: topographical plots of clusters found in the human data*. Crosses indicate channels where the cluster statistic reached significance.

Midfrontal Theta Power and Prediction Error

We next consider the first of three model-driven EEG hypotheses. We first perform statistical analysis on the Sync-model simulated data (Fig. 4A). Theta power in the Sync model data was best

predicted by the regression model that included an interaction between reward and prediction error ($F(1, 11980) = 22133$, $p < .001$). Hence, there was a significant main effect of prediction error ($F(1, 11980) = 742962$, $p < .001$, $\beta = -4.99$) and a significant interaction of prediction error and reward ($F(1, 11980) = 22133$, $p < .001$, $\beta = 4.48$). Thus, as predicted, the model cluster showed a negative linear relationship with negative prediction error, and no linear relationship with positive prediction error (Fig 4A).

For power in the empirical theta cluster, the regression model including the interaction between prediction error and reward fitted significantly better than the regression model with only prediction error as regressor ($\chi^2(1, N = 27) = 110$, $p < .001$). Additionally, the regression model including the three-way interaction between prediction error, reward and wAIC fitted significantly better than the regression model with only the two-way interaction ($\chi^2(2, N = 27) = 20.74$, $p < .001$). Here, all effects reached significance. Hence, there was a main effect of prediction error ($\chi^2(1, N = 27) = 1299$, $p < .001$, $\beta = -.79$) and an interaction of prediction error with reward ($\chi^2(1, N = 27) = 110$, $p < .001$, $\beta = .65$). Additionally, there was a significant interaction between prediction error, reward and wAIC ($\chi^2(2, N = 27) = 20.900$, $p < .001$). As can be observed in Fig 4B these results indicated a significant negative linear relationship between power and negative prediction error, which was stronger for subjects with a high wAIC (i.e., better behavioral fit of the Sync model); and an absence of linear relationship between power and positive prediction error which did not differ significantly for wAIC (Fig 4B). Interestingly, the three-way interaction was significant in the unrewarded (negative prediction error) trials ($\beta = -.89$, $p < .001$) but did not reach significance in the rewarded (positive prediction error) trials ($\beta = .44$, $p = .077$).

For exploratory purposes, we investigated the same regression models in the delta and alpha clusters. In the delta cluster, the difference in regression model fit between the regression models without and with the prediction error-reward interaction term did not reach significance ($\chi^2(1, N = 27) = 3.490$, $p = .062$). However, the regression model that also included the three-way interaction between prediction error, reward and wAIC fitted significantly better than the regression model with no

interaction terms ($\chi^2(3, N = 27) = 9.27$, $p = .026$). Here, the main effect of prediction error was significant ($\chi^2(1, N = 27) = 580$, $p < .001$, $\beta = .45$). The interaction between prediction error and reward did not reach significance ($\chi^2(1, N = 27) = 3.490$, $p = .062$, $\beta = -.07$). Also the three-way interaction term did not reach significance ($\chi^2(2, N = 27) = 5.830$, $p = .054$). However, if the interaction was considered separately for rewarded trials ($\beta = .61$, $p = .018$) and unrewarded trials ($\beta = -.50$, $p = .033$), both reached significance. As can be observed in Fig 4C, this meant that there was a positive linear relationship between power and prediction error for both positive and negative prediction error (Fig 4D). For subjects with low wAIC, the slope in unrewarded trials seemed similar to the one in rewarded trials, while for subjects with high wAIC, an inverse effect of the theta cluster was observed in which there was a flat(ter) slope in unrewarded trials but a steeper slope in rewarded trials.

In the alpha cluster, the regression model with the two-way interaction term showed a significantly better fit than the regression model without interaction ($\chi^2(1, N = 27) = 224$, $p < .001$). When the three-way interaction was added, it did not lead to a significantly better regression model ($\chi^2(2, N = 27) = .35$, $p = .841$). Here, a significant main effect of prediction error ($\chi^2(1, N = 27) = 142$, $p < .001$, $\beta = .85$) and a significant interaction between prediction error and reward ($\chi^2(1, N = 27) = 226$, $p < .001$, $\beta = -1.38$) were observed. The three-way interaction between prediction error, reward and wAIC was not significant ($\chi^2(2, N = 27) = .360$, $p = .833$). As is shown in Fig 4D, power in the alpha cluster exhibited a positive linear relationship for negative prediction error but a negative linear relationship with positive prediction error. These effects did not differ with respect to wAIC.

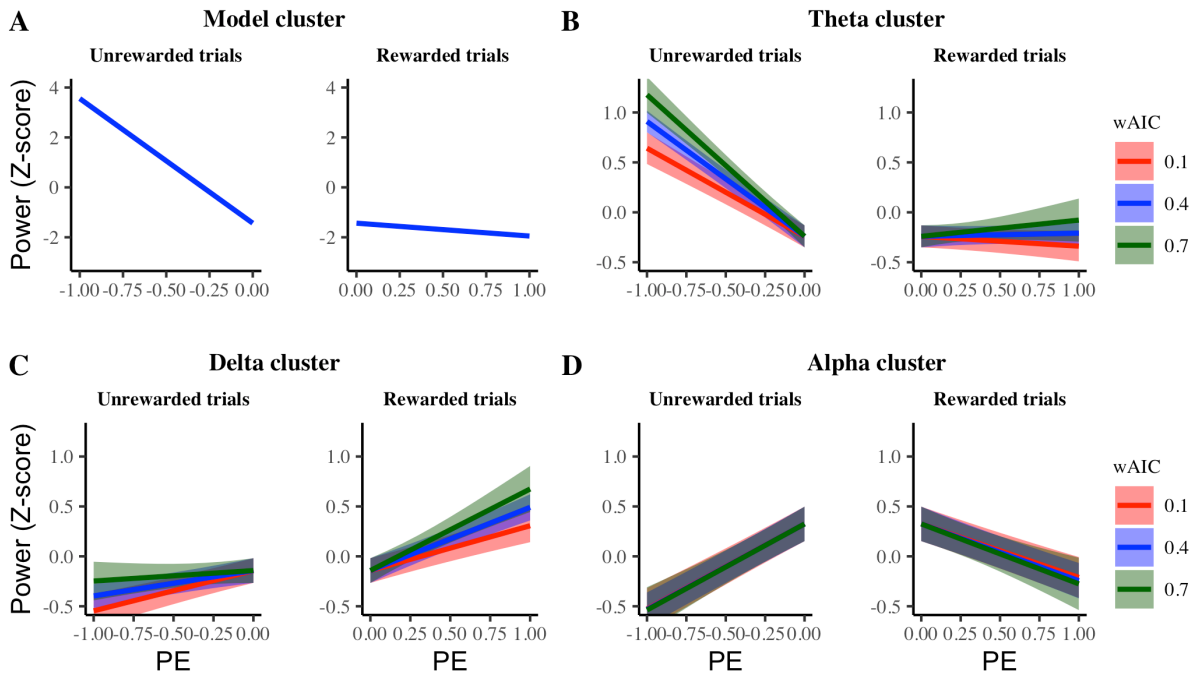


Fig 4. Result of linear regression between power and prediction error (PE) in all clusters. Lines indicate slopes and the shades represent 95% confidence intervals. The Model cluster (A) aimed to predict empirical data from the theta cluster (B).

Rule Switch Locking

For the second model-driven EEG hypothesis, power from the data clusters was extracted in trials within a 31-trial window from the rule switch (-15 to +15). In all clusters, one trial was observed that significantly deviated from baseline power. In the theta cluster (Fig 5A), only the exact trial of the rule switch (0) was significant above baseline (CI99.84 [-2.059, .256], *baseline* = -2.340). Linear regression of the data time course (across 31 trials) on the Sync model time course showed a significant effect ($F(1, 835) = 20.510$, $p < .001$, $R^2_{adj} = .023$, $\beta = .31$). In the delta cluster (Fig 5B), only the exact trial of the rule switch (0) was significantly below baseline (CI99.84 [-2.450, -1.265], *baseline* = -1.201). Linear regression of the data time course on the Sync model time course revealed a significant correlation ($F(1, 835) = 7.360$, $p = .007$, $R^2_{adj} = .008$, $\beta = -.18$). For the alpha cluster (Fig 5C), again one trial was significantly below baseline (CI99.84 [-6.275, -3.603], *baseline* = -3.584). Notably, this was the trial after the rule switch (+1). Moreover, when data was locked to the moment where subjects indicated the rule switch (Fig 5D), alpha power reaches a minimum at this exact moment (CI99.84 [-

7.675, -3.686], *baseline* = -3.584). Also in the alpha cluster, the linear regression of the power on the Sync model pattern reached significance with a negative slope ($F(1, 835) = 32.720$, $p < .001$, $R^2_{adj} = .037$, $\beta = -.65$).

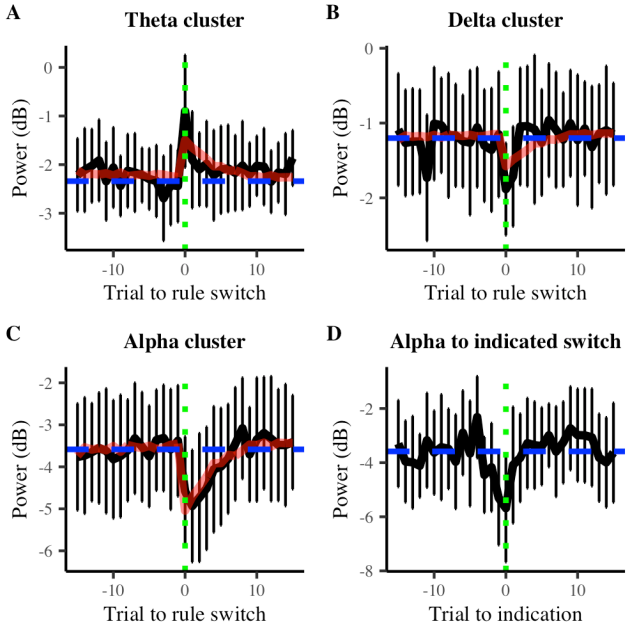


Fig 5. Power locked to rule switch. Black lines show the mean power. Error bars show the 99.84% confidence interval (Bonferroni correction). The horizontal blue dashed line represents baseline power and the vertical green dotted line indicates the moment of the rule switch. The red line visualizes the result of linear regression between the Sync model and human data. *A-C* show data locked to the moment of the actual rule switch. *D* shows data of the alpha cluster locked to the moment when subjects indicated they noticed the task switch.

Power at the peak trials (0 for theta and delta, +1 for alpha) was extracted and added to a linear regression with wAIC as predictor. This revealed no significant effects for the theta ($F(1, 25) = .004$, $p = .948$, $R^2_{adj} = -.040$, $\beta = -.10$) or delta cluster ($F(1, 25) = .680$, $p = .417$, $R^2_{adj} = -.012$, $\beta = .66$). However, the effect of wAIC did reach significance in the alpha cluster ($F(1, 25) = 7.220$, $p = .013$, $R^2_{adj} = .193$, $\beta = 4.17$). Fig 6 sheds light on how activity in the alpha cluster differed depending on wAIC. For illustrative purposes, subjects were divided in three groups of low, middle and high wAIC. For each group, the data pattern of alpha activity was plotted, once locked to the real rule switch and once locked

to the indication of a rule switch (Fig 6). Here, it is observed that the alpha pattern is mainly driven by subjects that have a low wAIC (i.e., good fit) for the bSync model.

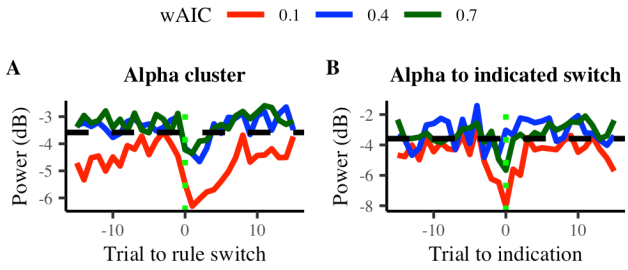


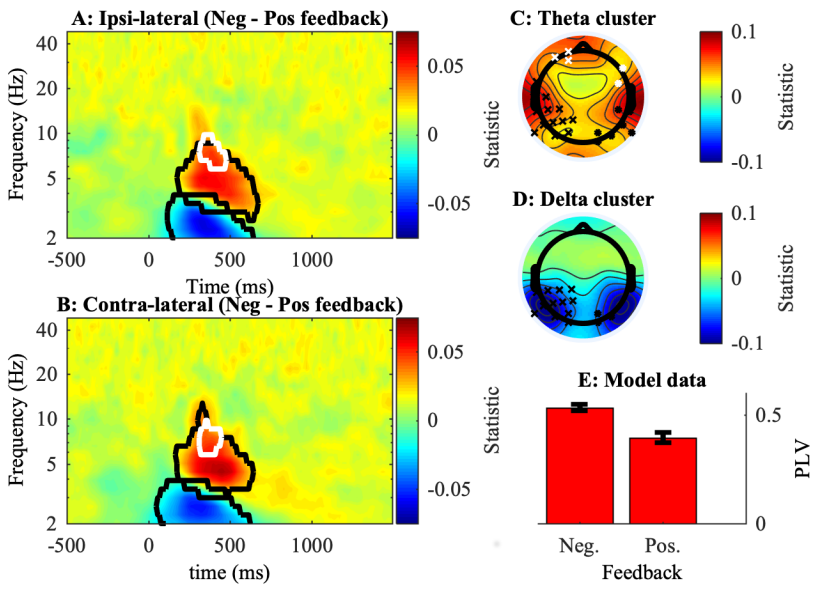
Fig 6. Power locked to rule switch for different wAIC. Data patterns are shown for different wAIC values (colored lines). The horizontal black dashed line shows the baseline power over all subjects and the vertical green dotted line indicates the moment of the rule switch (A) or indication of rule switch (B).

Midfrontal-posterior Phase-coupling Analyses

We next turn to our third model-driven EEG analysis concerning an increase of phase coupling between midfrontal and posterior electrodes after negative feedback. As previously described, this coupling is induced by bursts that are sent from pMFC to posterior areas in the Mapping unit. Since pMFC power is stronger after negative feedback, also the number of bursts and the amount of phase-coupling is increased.

Here, non-parametric cluster analyses on the phase-locking data (phase-locking with midfrontal electrodes; Fig 7) revealed six significant clusters that were selective for feedback (for details see Materials and Methods). These clusters were located in the theta (4; Fig 7A, B, C) or delta (2; Fig 7A, B, D) frequency band. In the theta frequency band, two clusters were located at temporal electrodes; two other clusters were located on more lateral/anterior frontal electrodes. In the delta frequency band, both clusters were located on posterior electrodes. In line with the results of Sync model simulations (Fig 7E), the theta clusters showed an increase in phase-locking after negative feedback. This was the case for both the ipsilateral and contralateral electrodes. The delta clusters show the inverse pattern of the theta cluster. Here, phase-locking was stronger after positive feedback than after negative feedback in both the ipsi- and contralateral cluster. As in the power analyses, we also explored whether the phase-

543 locking contrast in each cluster correlated with the subjects' wAIC for the bSync model. None of these
544 correlations reached significance.



545

546 **Fig 7. Phase-locking results.** *A-B: Time-Frequency plots of contrast (Negative – Positive feedback).* Significant
547 clusters are indicated by the black or white contour line. The black line represents posterior clusters in C and D
548 while the white line represent the frontal clusters in C. A: Contrast of iPLV averaged over all ipsi-lateral
549 electrodes. B: Contrast of iPLV averaged over all contra-lateral electrodes. *C-D: Topographical plots of clusters.*
550 Data was averaged over all time points and frequencies that were included in the respective contours of A and B.
551 Channels where cluster statistic reached significance are marked by crosses or dots. The left channels (crosses)
552 present ipsi-lateral electrodes and the right channels (dots) present contra-lateral electrodes. Again, the white color
553 was used to distinguish the frontal clusters from the temporal clusters.

554

555 **Discussion**

556 The current study aimed to gain insight in neural mechanisms that allow humans to flexibly
557 adapt to changes in the environment. For this purpose, 27 healthy human subjects were tested on a
558 probabilistic reversal learning task while EEG was recorded. On behavioral level, three models of
559 increasing hierarchical complexity were compared. A first model, the RW model, updated the value of
560 stimulus-action mappings on a trial-by-trial basis with a fixed learning rate. In a second model, the ALR
561 model, this approach was extended by employing an adaptable learning rate, allowing the ALR model
562 to be flexible in adapting to rule switches (fast learning rate) but to also be robust to noise evoked by

probabilistic feedback (slow learning rate). The third, Sync, model implemented modularity to retain task-specific mappings. It employs hierarchical learning to determine when to switch between rule modules. This approach obviates the need to relearn mappings on each rule switch. No evidence was found for the ALR model, while for some people the RW fit best, and for others the Sync model.

Simulations of the Sync model allowed formulation and testing of three model-driven EEG hypotheses. The first hypothesis considers the relation between midfrontal theta power and prediction errors. In the Sync model, prediction errors are used to evaluate how much control should be exerted. Here, the level of control is represented by theta power in pMFC. Since only negative prediction errors inform about possible rule switches, the Sync model increased control after negative prediction errors but not after positive prediction errors. Empirical data supported this hypothesis. A linear relationship between prediction error and power in the theta cluster was observed for unrewarded trials (negative prediction error) but not for rewarded trials (positive prediction error). Moreover, this effect was stronger for subjects that fitted better with the Sync model. Since prediction errors are strongest at the moment of a rule switch, a second model-driven hypothesis stated that theta power would peak at rule switches. Again, this hypothesis was supported by empirical data. Moreover, simulated power significantly predicted power in the empirical theta cluster. In a third model-driven hypothesis, the Sync model predicted that phase connectivity would be increased after negative feedback. Here, six significant clusters were found. Four of them were in theta frequency range and showed the pattern predicted by the Sync model. Two of these clusters were located on posterior-temporal electrodes, which is roughly in line with our prediction of motor and visual areas.

Several hypotheses remain to be tested. First, as was briefly mentioned in the Materials and Methods section, previous modeling work used a gamma frequency in the Mapping unit instead of a theta frequency. This frequency was currently changed because empirical work provides strong evidence for within-frequency (theta-theta) coupling (Cavanagh, Cohen, & Allen, 2009; Clouter, Shapiro, & Hanslmayr, 2017; Nigbur, Cohen, Ridderinkhof, & Stürmer, 2011) during cognitive tasks, in addition to cross-frequency coupling. We thus also studied within-frequency coupling empirically. Nevertheless, future work, using MEG or more invasive measurements, should also study the role of

cross-frequency (theta-gamma) coupling. Second, the limited spatial resolution of EEG did not allow testing the prediction that different task rules are implemented by synchronizing different task-relevant modules.

Several extensions can be made to the model as well. For instance, while for the reversal learning task of the current study it was sufficient to use prediction error to determine when to make a binary switch, a more sophisticated approach might apply in everyday life, where contextual cues allow navigating a vast map of tasks and rules. One way to address this issue is by adding second level (contextual) features which allow the LFC to (learn to) infer which of multiple task modules should be synchronized. Additionally, scalability of the Sync model is limited by how modularity was implemented in the Mapping unit. Here, none of the task rule 1 mappings are shared with task rule 2. Such a strict division of task mappings is optimal when those mappings are orthogonal. However, when some (but not all) of the mappings can be generalized between tasks, the current approach does not allow agents to transfer knowledge across contexts. As has been addressed in previous work (Collins & Frank, 2013; Gershman, Blei, & Niv, 2010), a more sustainable way is to construct modules of mappings that are shared between tasks. Instead of having to learn each new task from scratch, this approach allows agents to transfer knowledge from one task to another. Future work should explore whether these more complex hierarchical learning algorithms can be integrated in the Sync model.

Since the current task did not contain (contextual) cues to infer the appropriate mappings, the Sync model relied on prediction errors to estimate the hidden state (task rule) of the environment. This is in line with previous ideas that reinforcement learning and more specifically prediction errors signaled by dopamine are used to infer so called belief states (Gershman & Uchida, 2019; Starkweather, Babayan, Uchida, & Gershman, 2017; Wilson et al., 2014). Instead, non-hierarchical models (such as RW and ALR) only use prediction errors to adjust value of lower-level mappings.

Building on suggestions of previous work (Piray, Dezfouli, Heskes, Frank, & Daw, 2019), current study illustrated how individual differences in model fit can be leveraged to address cognitive questions. Here, three groups could be distinguished. One group of subjects aligned with the RW model, a second group aligned with the Sync model and in a third group, no distinction could be made between

the RW and Sync model. Interestingly, subjects with lower accuracy fitted better with the Sync model. This is consistent with previous work (Verbeke & Verguts, 2019) which illustrated that modularity as employed by the Sync model is only beneficial if the learning problem is sufficiently complex. Furthermore, despite previous work showing a good behavioral fit of ALR-type models (Bai et al., 2014; Behrens et al., 2007; Silvetti, Seurinck, & Verguts, 2013), fit of the ALR model in the current study was consistently low over all subjects. Crucially, previous studies typically included long stable trial blocks in which the task rule did not change. In these periods it was beneficial to decrease learning rate. In contrast, the current task applied frequent rule switches in rapid succession, favoring tonically high learning rates. Thus, future work should investigate whether subjects flexibly employ the RW, Sync or ALR framework depending on the structure and complexity of the task.

The Sync model implements modularity via neural oscillations between task-relevant areas. This concords with a growing interest in the function of neural oscillations for a wide variety of cognitive functions, such as visual attention (e.g., Gray & Singer, 1989; Han, Lee, & Choi, 2019; Jensen, Bonnefond, & VanRullen, 2012), working memory (e.g., Hsieh, Ekstrom, & Ranganath, 2011; Hsieh & Ranganath, 2014; Lisman & Idiart, 1995), cognitive control (e.g., Cavanagh & Frank, 2014; Oehrn et al., 2014) and declarative learning (Ergo, De Loof, & Verguts, 2020). According to the BBS hypothesis (Fries, 2005, 2015), all these cognitive functions require in some way the binding of several stimuli, dimensions or features. Current work described how oscillations, and more specifically synchronization, might be relevant in hierarchical learning. Here, BBS is employed to flexibly bind mappings that are relevant for a certain task rule.

On anatomical-functional level, current work builds on suggestions from previous work that pMFC closely cooperates with LFC (Cavanagh, Frank, Klein, & Allen, 2010; Kondo, Osaka, & Osaka, 2004; Mac Donald, Cohen, Stenger, & Carter, 2000) to exert hierarchical control over lower-level motor processes (Alexander & Brown, 2015; Badre & Nee, 2018; Holroyd & McClure, 2015; Koechlin, Ody, & Kouneiher, 2003). In the Sync model, LFC signals which rule modules should be synchronized. This is in line with previous theoretical work describing LFC as containing task demands (Botvinick et al., 2001) and empirical work that found strong communication between LFC and pMFC in cognitive tasks (Cavanagh et al., 2010; Kondo et al., 2004; Mac Donald et al., 2000). Also in line with previous

empirical work (Boorman, Behrens, Woolrich, & Rushworth, 2009; Wilson et al., 2014), the model aMFC is responsible for keeping track of the relevant task rule. Additionally, consistent with previous fMRI work (e.g., Aben, Calderon, den Bussche, & Verguts, 2020; Hare, Schultz, Camerer, O'Doherty, & Rangel, 2011), the current study also found increased coupling between midfrontal cortex and task-related areas when more control was needed (negative feedback). Importantly, while this fMRI work showed detailed networks of connectivity, current study described how this connectivity works at algorithmic level.

To sum up, we have demonstrated how the brain might employ synchronization in order to bind task-relevant areas for hierarchical and efficient task processing. To achieve this, we used a combination of EEG, computational modelling, individual differences, and behavioral analysis. We believe that such an approach might reveal how and whether more complicated tasks can be implemented via synchronization as well, in which contexts, and in which individuals.

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