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Aperiodic neural activity reflects metacontrol in task-switching

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"Metacontrol" refers to the ability to find the right balance between more persistent and more flexible cognitive control styles, depending on task demands. Recent research on tasks involving response conflict regulation indicates a consistent link between aperiodic EEG activity and task conditions that demand a more or less persistent control style. In this study, we explored whether this connection between metacontrol and aperiodic activity also applies to cognitive flexibility. We examined EEG and behavioral data from two separate samples engaged in a task-switching paradigm, allowing for an internal replication of our findings. Both studies revealed that aperiodic activity significantly decreased during task switching compared to task repetition. Our results support the predictions of metacontrol theory but contradict those of traditional control theories which would have predicted the opposite pattern of results. We propose that aperiodic activity observed in EEG signals serves as a valid indicator of dynamic neuroplasticity in metacontrol, suggesting that truly adaptive metacontrol does not necessarily bias processing towards persistence in response to every control challenge, but chooses between persistence and flexibility biases depending on the nature of the challenge.

Keywords Aperiodic neural activity, Neural noise, EEG, Cognitive flexibility, Task-switching, Metacontrol

While many other animals easily outperform humans with respect to various basic functions, including perception, memory, and motor performance, humans excel if it comes to the tailoring of their behavior to different and quickly changing circumstances. This great flexibility of human behavior is commonly ascribed to cognitive control, a concept that summarizes the various functions that allow agents to carry out different actions to the same stimuli, and vice versa[1]. The traditional concept of cognitive control emphasizes what is called willpower in everyday language: sticking to one's goal even under challenging circumstances, focusing on information related to this goal while ignoring distraction, and overcoming obstacles on the way[2,3]. Frontal areas, and the lateral prefrontal cortex in particular play a key role in orchestrating this persistence aspect of cognitive control[4–6]. However, there is increasing interest in another, in some sense opposite aspect of cognitive control as well. Indeed, sometimes it is more reasonable to give up one's current goal and trade it for a more realistic, more rewarding one, to consider the opportunities provided by task-unrelated information, and to carry out unanticipated actions. Arguments in favor of a more balanced, bilateral view on action control have been motivated in various ways, including functional considerations[7,8], neuroanatomical/neuropathological implications[9,10], and neurochemical modeling[11]. These arguments motivated the metacontrol model of Hommel and colleagues[12–14], which defines metacontrol as the function that serves to find the most adaptive balance between the poles of *persistence*, as reflected by willpower, and *flexibility*, which opens the processing stream for unpredicted events and unforeseen ideas and action alternatives.

Recent studies in electrophysiology have shown that aperiodic neural activity—non-oscillatory EEG signals—plays a significant role in cognitive control[15,16]. While the aperiodic aspect has often been dismissed as mere noise or an extraneous factor to be overlooked or adjusted[17], new findings indicate that this type of activity is consistently linked to various psychological functions, including arousal levels[18], task performance[19,20], and cognitive control mechanisms such as inhibition control[15]. Importantly, the power spectrum is characterized by a distribution where spectral power decreases as frequency increases[21–23]. The aperiodic dynamics can be reliably detected through the aperiodic exponent, which relates to the negative 1/f slope found in the logarithmic transformation of the power spectrum. This slope reflects the rate of decline in power across different frequencies[21]. Additionally, the aperiodic exponent may act as a measure of "neural variability," representing the brain's capability to adapt its neural activity according to the demands of the situation[24]. Shifts in aperiodic brain activity can indicate changes in the balance between excitatory and inhibitory neural mechanisms, commonly known as the "E/I ratio."

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According to Turri et al.[25], a higher exponent (which reflects a steeper spectrum) typically suggests a greater prevalence of inhibition over excitation, while a lower exponent indicates the contrary[26,27]. Increased excitation levels have been associated with a decrease in the synchronization of rhythmic brain activities and neuron firing, leading to a rise in neural noise that may hinder proper neural communication[28]. Recent efforts have succeeded in providing a direct neurophysiological indicator of metacontrol and changes therein. Both neural intraindividual variability in fMRI and aperiodic exponents of so-called FOOOF (fitting oscillations & one over f[21]) analyses derived from EEG data could be demonstrated to directly reflect the impact of task conditions with different control demands. For instance, participants increase their FOOOF exponent as soon a task-specific stimulus appears, and they do more so if that stimulus signals a condition that requires more persistence[29,30]. Along the same lines, individual FOOOF exponents obtained under resting-state conditions can successfully predict the FOOOF exponent and its temporal dynamics in another, entirely different persistence-heavy or flexibility-heavy task[29]. Hence, the demand for metacontrol persistence is accompanied by an increase of the FOOOF exponent, a measure of aperiodic brain activity, which can be taken to reflect the cortical noise level. This suggests that cortical noise is at least a systematic, indicative by-product of metacontrol changes, perhaps even the means of implementing metacontrol policies. Accordingly, relatively high exponents (indicating decreased "E/I ratio", more inhibition and likely less cortical noise) can be taken to indicate a metacontrol bias towards persistence, while relatively low exponents (indicating increased "E/I ratio", more excitation and likely more cortical noise) would indicate a metacontrol bias towards flexibility.

Even though the idea of using measures of aperiodic brain activity to assess cognitive control was motivated by the metacontrol approach[29,30], the available findings can also be accounted for by the more traditional control account[31–34]. Traditional accounts conceptualize cognitive control in terms of more or less, which implies a unipolar dimension ranging from maximal to minimal control. It has been assumed that control is related to the neural signal-to-noise (SNR) ratio, and that more control is associated with, or enabled by a higher ratio[35]. Consistent with this idea, our previous observations that experimental conditions calling for more and tighter control, like the rare Nogo trials in Zhang et al. and in Pi et al.[29,36], can be easily accounted for by traditional control models[31–34]. The metacontrol model would make the same prediction, so that our previous observations do not serve to distinguish between the two approaches.

The purpose of the present study was to find and test a condition that would be better suited to evaluate and compare predictions from the traditional control account and the metacontrol account: a condition that control theorists would consider particularly control-demanding, so that they would need to predict a relative increase of the aperiodic exponent (which would reflect decreased "E/I ratio", more inhibition and likely less cortical noise), but that from a metacontrol point of view would call for more flexibility, so that this approach would suggest a relative reduction of the aperiodic exponent (reflecting increased "E/I ratio", more excitation and likely more cortical noise). We considered that the switch condition in a task-switching paradigm might serve this purpose. In this paradigm, participants are presented with the same stimulus- and response-set in each trial, but sometimes the mapping of responses to stimuli changes—the task, that is. These changes may be unpredictable[37] or predictable[38], as in the present study. It is a common finding that the need to switch the task hampers performance, often rather drastically, as compared to trials in which the task of the previous trial can be repeated[39]. Even though the exact reason for why that might be the case, and how many components might contribute to the so-called task-switching costs, are still under discussion, there is widespread agreement that trials that require a task switch rely much stronger on cognitive control than trials in which the previous task can be repeated[39].

Indeed, control theories clearly assume that task switching requires more control resources and efforts than task repetition[37,38]. Accordingly, the switching condition should increase the amount of control and, if control is positively associated with an increase of the signal-to-noise ratio, cortical noise should be reduced, which in turn would be visible in an increased aperiodic exponent. The metacontrol model would also assume that switching conditions require control, but this control should not consist in a high or increased degree of metacontrol persistence (the traditional type of control), because persistence would result in sticking even more and longer with the previous task set. Rather, the system would need to open up, relax constraints, so to allow the new task set to be selected and established. If so, one would expect a *reduction* of persistence-type control and a stronger bias towards flexibility. This should come with an increase in cortical noise and, accordingly, with a decrease of the aperiodic exponent. To test this hypothesis, we analyzed two independent samples (Study 1 and Study 2), which were previously tested for other scientific aims. To get a better idea about the temporal dynamics of metacontrol changes, we compared two time windows per trial: a pre-trial window preceding the stimulus, which can be taken as a temporal (relative) baseline[29,36], and a within-trial window that was supposed to capture the stimulus-specific changes in metacontrol. The prediction of differences in the FOOOF exponents for switch and repeat trials referred to the within-trial time window.

So far, few studies studying scalp distributions of FOOOF exponents related to cognitive persistence indicated that the aperiodic exponents at frontal and central areas exhibited the most significant changes before and during the trial[30,40–42]. However, there are no theoretical reasons to believe that exponent effects are bound to particular loci. Metacontrol is commonly assumed to emerge from the interaction of the prefrontal and the striatal dopaminergic pathway[43–45], which means that changes in metacontrol are likely to be bound to neurochemical changes. As these changes should be expressed through dopaminergic pathways, which reach a very substantial part of the entire brain, effects of these changes are unlikely to be bound to tightly circumscribed brain areas. Hence, even though future research may identify spatial boundaries of exponent effects, which for instance may depend on the task and/or the employed strategies, we neither claim nor assume a particular locus of exponent effects, and we see no theoretical constraints for a possible region of interest. Accordingly, we were interested in the scalp distribution of FOOOF exponents, so to fine-tune our analyses to those sensors that were most sensitive to exponent effects, but we always started with a whole-brain analysis of exponents—a brain-wide

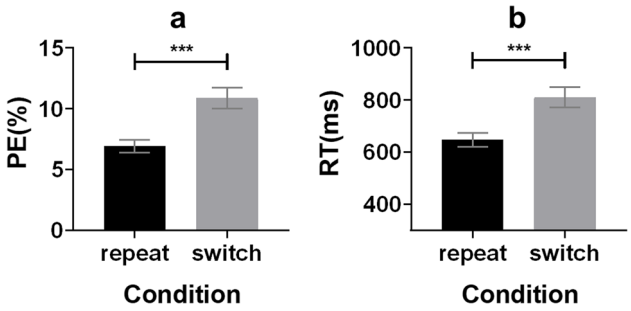


Figure 1. Behavioral data results. Panel **a**: Percent error (PE) results in different conditions. Panel **b**: Reaction time (RT) results in different conditions. Error bars represent standard errors of the mean (SEM), ***$p < 0.001$.

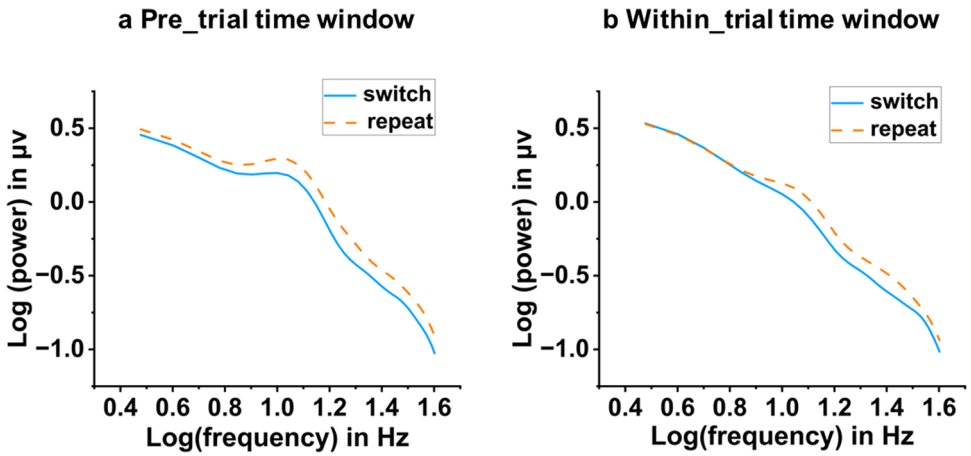


Figure 2. Log-log transformed power spectral densities averaged across electrodes and participants. Panel **a** shows PSDs in the pre-trial period; panel **b** displays PSDs in the within-trial period.

permutation cluster-based test. In other words, we predicted the direction of exponent effects, but not their cortical location.

Results

Study1

Behavioral results

Mean percent errors (PEs) and reaction times (RTs) are shown in Fig. 1. Paired-sample t-tests showed a significant difference between the repeat and switch condition on PE ($t_{(32)} = 8.25$, $p < 0.001$, $d = 1.43$, $BF_{10} > 1000$) and RT ($t_{(32)} = 9.42$, $p < 0.001$, $d = 1.64$, $BF_{10} > 1000$). The PE was significantly higher in the switch condition (10.87 ± 4.96) than in the repeat condition (6.90 ± 3.04). The RT in the switch condition was also significantly higher (810.39 ± 38.34) than in the repeat condition (647.05 ± 26.05).

Aperiodic exponents

The power spectral density (PSD) results in a log-log space are shown in Fig. 2 at the frequency from 3 Hz to 40 Hz for different experimental conditions in the within-trial period and the pre-trial period.

The results of the two-factor repeated measures ANOVA showed significant main effects of condition ($F_{(1, 32)} = 5.59$, $p < 0.05$, $\eta_p^2 = 0.15$, $BF_{10} > 1000$) and time window ($F_{(1, 32)} = 14.01$, $p < 0.001$, $\eta_p^2 = 0.31$, $BF_{10} > 1000$). The aperiodic exponent was significantly lower in the pre-trial time window (1.32 ± 0.27) than in the within-trial time window (1.37 ± 0.027), and significantly higher in the switch condition (1.35 ± 0.27) than in the repeat condition (1.34 ± 0.27). The interaction effect of time window × condition was also significant ($F_{(1, 32)} = 61.49$, $p < 0.001$, $\eta_p^2 = 0.66$, $BF_{10} > 1000$). Post hoc t-tests revealed that the relationship between repeat and switch trials reversed from the pre-trial to the within-trial period: whereas the aperiodic exponent was significantly higher in the switch condition (1.34 ± 0.26) than in the repeat condition (1.31 ± 0.25) during the pre-trial period ($t_{(32)} = 5.90$, $d = 1.03$, $p < 0.001$, $BF_{10} > 1000$), it was significantly lower in the switch condition (1.36 ± 0.26) than in the repeat condition (1.38 ± 0.26) during the within-trial period ($t_{(32)} = 4.06$, $p < 0.001$, $d = -0.71$, $BF_{10} = 96.40$). The aperiodic exponent is displayed in Fig. 3 across various time windows and conditions.

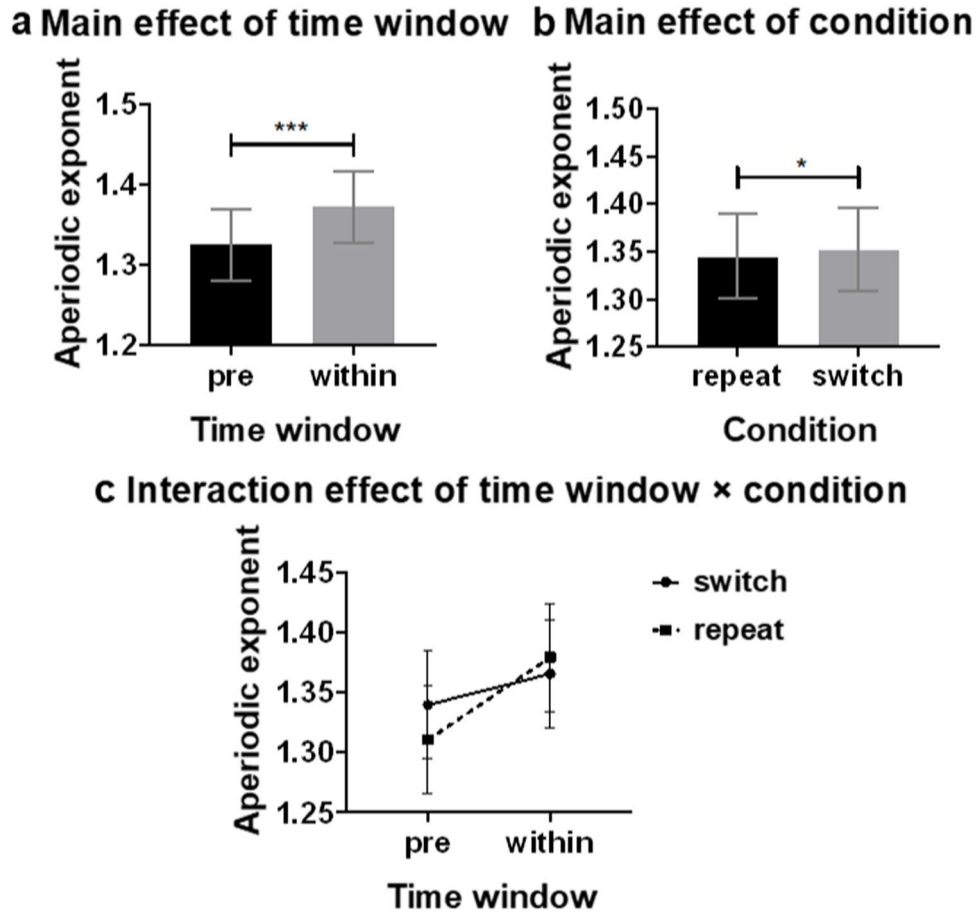


Figure 3. Aperiodic exponent results. Panel **a**: Aperiodic exponent in different time windows; Panel **b**: Aperiodic exponent in different conditions; Panel **c**: The interaction effect of time window × condition. Error bars represent SEM, $^{*}p < 0.05$, $^{***}p < 0.001$.

Cluster-based permutation test

The scalp distribution of the aperiodic parameters was tested using cluster-based permutation. A main effect of time window was found in the aperiodic exponent over frontal, central, posterior, temporal and occipital, areas (cluster 1: CZ, FCZ, FC1, CP1, FZ, F1, FC3, C3, CP3, P1, AFZ, AF3, $F_{(1,32)} = 48.75$, $P < 0.01$, $\eta_p^2 = 0.16$; cluster 2: AF7, FT7, T7, $F_{(1,32)} = 14.18$, $P < 0.05$, $\eta_p^2 = 0.49$; cluster 3: O1, FT9, TP9, $F_{(1,32)} = 46.90$, $P < 0.001$, $\eta_p^2 = 0.78$; cluster 4: P11, FC2, CP2, CPZ, F2, FC4, C4, CP4, P2, PZ, AF4, $F_{(1,32)} = 59.66$, $P < 0.001$, $\eta_p^2 = 0.18$; cluster 5: C6, CP6, $F_{(1,32)} = 20.28$, $P < 0.05$, $\eta_p^2 = 0.57$; cluster 6: O2, OZ, FT10, TP10, $F_{(1,32)} = 42.49$, $P < 0.01$, $\eta_p^2 = 0.65$). The main effect in the aperiodic exponent in different conditions was observed over frontal, central, temporal areas (cluster 1: AF7, FT7, T7, TP7, $F_{(1,32)} = 10.13$, $P < 0.05$, $\eta_p^2 = 0.31$; cluster 2: F2, FC4, C4, $F_{(1,32)} = 11.30$, $P < 0.05$, $\eta_p^2 = 0.45$; cluster 3: FC6, C6. CP6, $F_{(1,32)} = 24.54$, $P < 0.05$, $\eta_p^2 = 0.56$; cluster 4: AF8, FT8, T8, TP8, $F_{(1,32)} = 13.49$, $P < 0.05$, $\eta_p^2 = 0.32$). The interaction effects of time window × condition formed a cluster containing all electrode points ($F_{(1,32)} = 83.96$, $P < 0.01$, $\eta_p^2 = 0.051$). The scalp topography for the aperiodic exponent is shown in Fig. 4.

Study 2

Behavioral results

The behavioral results were comparable to Study 1, see Fig. 5. Analysis using paired-sample t-tests revealed a significant difference between the repeat and switch conditions in both PE ($t_{(42)} = 3.46$, $p < 0.01$, $d = 0.39$, $BF_{10} = 24.53$) and RT ($t_{(42)} = 6.31$, $p < 0.001$, $d = 0.56$, $BF_{10} > 1000$). PEs were significantly higher in the switch condition (6.35 ± 0.79) than in the repeat condition (4.62 ± 0.55). Similarly, RTs were significantly longer in the switch condition (808.65 ± 25.64) than in the repeat condition (721.32 ± 21.33).

Aperiodic exponents

Figure 6 shows the PSD results at the frequency of 3 Hz to 40 Hz for different experimental conditions in the within-trial period and the pre-trial period in a log-log space.

Results are visually depicted in Fig. 7, showcasing the aperiodic exponent across different time windows and conditions. The two-factor repeated measures ANOVA yielded significant main effects for both condition ($F_{(1, 42)} = 6.56$, $p = 0.014$, $\eta_p^2 = 0.135$, $BF_{10} < 0.01$) and time window ($F_{(1, 42)} = 148.44$, $p < 0.001$, $\eta_p^2 = 0.779$, $BF_{10} = 0.001$).

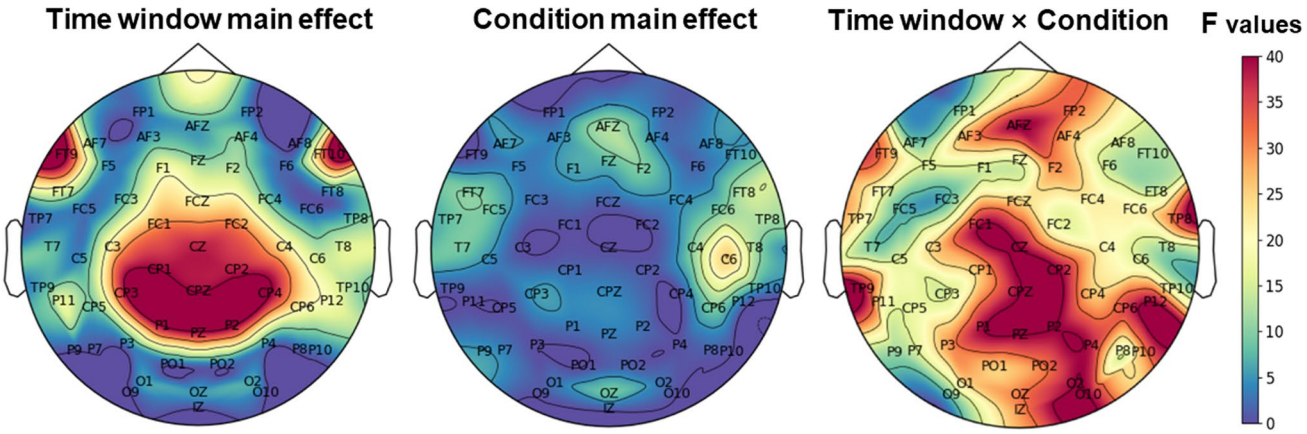


Figure 4. Scalp distributions of the aperiodic exponent. The figures show electrode sites with a significant main effect of time window, condition and the interaction effect of time window × condition, respectively, in the aperiodic exponent (this corresponds to the subheading in the figure). Colors indicate cluster-level summed F-values.

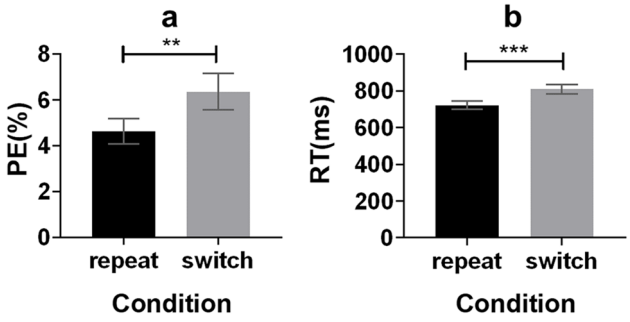


Figure 5. Behavioral results. Panel **a**: PE results in different conditions. Panel **b**: RT results in different conditions. Error bars represent SEM, ** $p < 0.01$, *** $p < 0.001$.

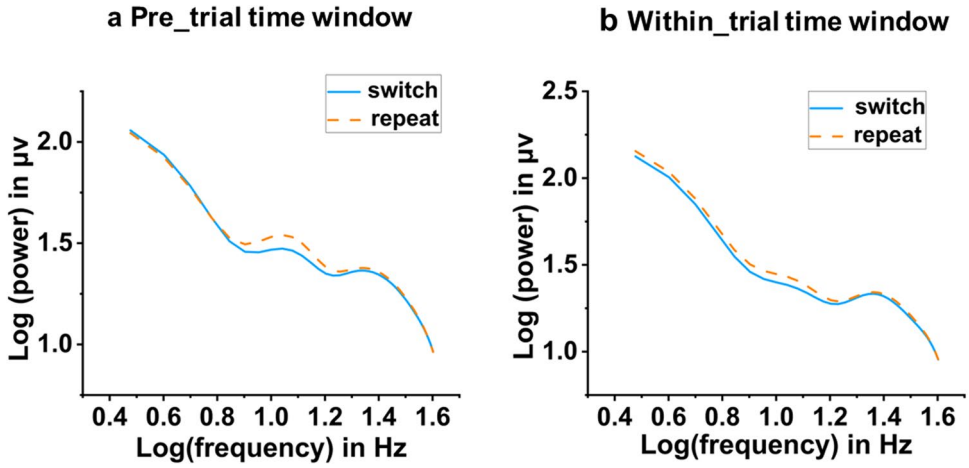


Figure 6. Log-log transformed power spectral densities averaged across electrodes and participants. Panel **a** shows PSDs in the pre-trial period; panel **b** displays PSDs in the within-trial period.

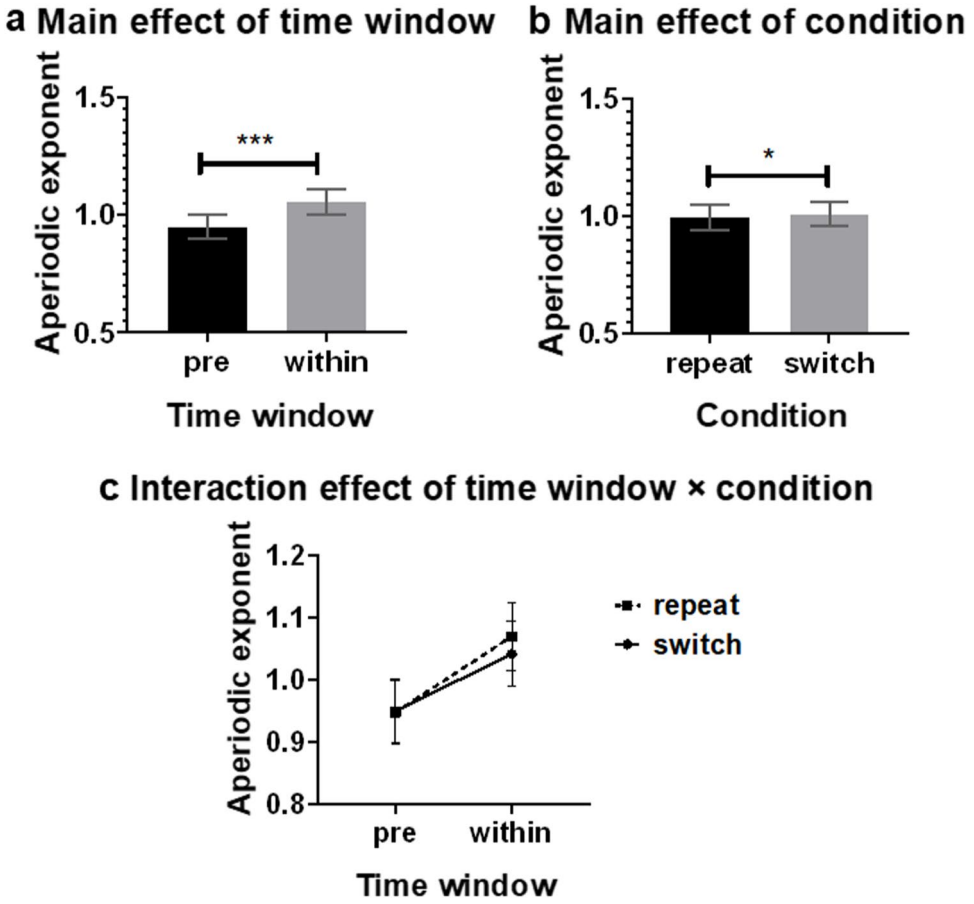


Figure 7. Aperiodic exponent results. Panel **a**: Aperiodic exponent in different time windows; Panel **b**: Aperiodic exponent in different conditions; Panel **c**: The interaction effect of time window × condition. Error bars represent SEM, $^{*}p < 0.05$, $^{***}p < 0.001$.

Furthermore, there was a significant interaction effect between time window and condition ($F_{(1, 42)} = 19.94$, $p < 0.001$, $\eta_p^2 = 0.322$, $BF_{10} = 250$). Post hoc analyses revealed a significant decrease in the aperiodic exponent in the switch condition (1.04 ± 0.35) as compared to the repeat condition (1.07 ± 0.36) during the within-trial period ($t(42) = 4.46$, $d = 0.11$, $p < 0.001$, $BF_{10} > 380.48$). However, no significant difference was observed between the two conditions in the pre-trial period.

To test for systematic differences between the two studies, we ran a three-factorial ANOVA on aperiodic exponents from both studies with study as a between-participant factor and time window and condition as within-participant factors. Unsurprisingly, this ANOVA also yielded a highly significant effect of time and of the interaction between time and condition, again showing that the exponent was higher in the within-trial than in the pre-trial period, and lower for task switches than task repetitions, but only in the within-trial period. However, two other significant effects involved the study factor (all other ps > 0.158): The first was an interaction between time and study ($F_{(1,74)} = 16.09$, $P = 0.001$, $\eta_p^2 = 0.18$), indicating that the increase of the exponent from the pre-trial to the within-trial period was more pronounced in Study 2 (0.95 vs. 1.06) than in Study 1 (1.32 vs. 1.37), where the pre-trial level of the exponent was also higher. The second was an interaction between condition and study ($F_{(1,74)} = 10.15$, $P = 0.008$, $\eta_p^2 = 0.12$). This reflected the fact that, averaged across the time factor, task switch showed a lower exponent than task repetition in Study 2 (0.99 vs. 1.01), but not in Study 1 (1.35 vs. 1.34). This in turn was due to that, in Study 2, the condition effect was even numerically absent in the pre-trial period (see Fig. 7), so that the overall condition effect was driven by the within-trial period only whereas, in Study 1, the pre-trial period showed a numerically reversed effect (see Fig. 3), which reduced and numerically reversed the overall condition effect. To get a better understanding of this pre-trial difference between the two studies, we subtracted the pre-trial scalp distribution of the exponent for Study 2 from this distribution for Study 1 (see Fig. 8). The distribution is clearly more homogeneous in Study 2, whereas Study 1 shows hotspots of sensors in central and prefrontal regions, as indicated in the difference plot (8c). We will consider possible reasons for this difference in the Discussion.

Cluster-based permutation test

An additional cluster-based permutation test was performed to examine the distribution of the aperiodic components on the scalp. A main effect of time window was found over the whole brain ($F_{(1,42)} = 164.73$,

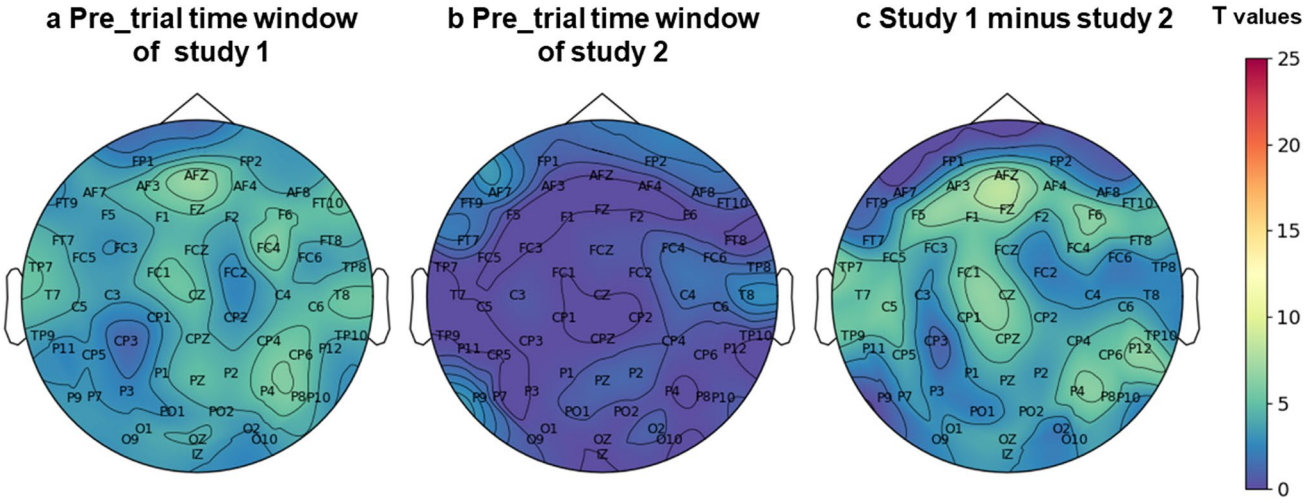


Figure 8. Scalp distributions of the aperiodic exponent in the pre-trial periods of (**a**) Study 1 and (**b**) Study 2, as well as (**c**) the difference plot (Study 1 minus Study 2). Colors indicate F-values.

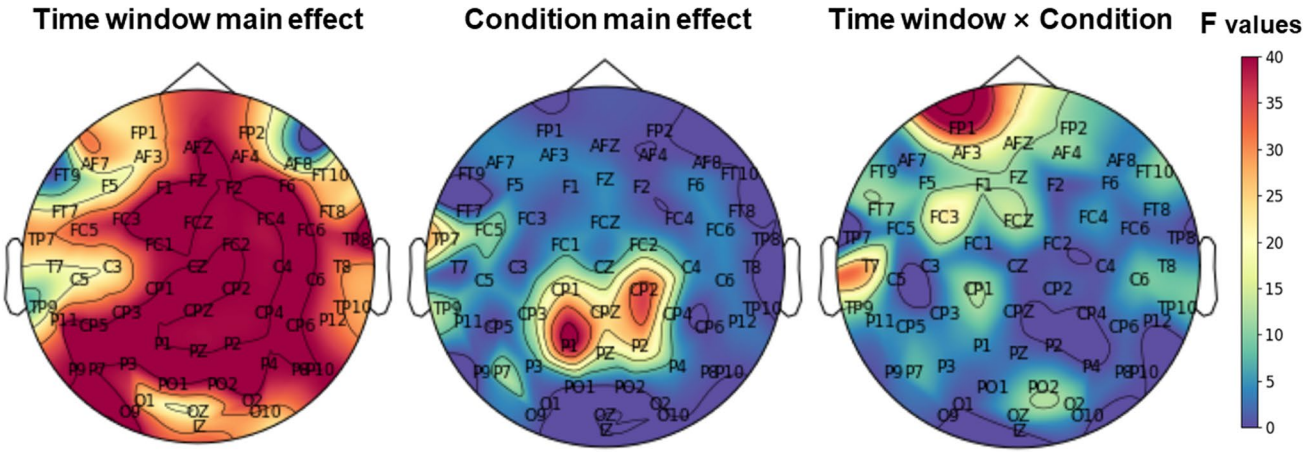


Figure 9. Scalp distributions of the aperiodic exponent. The figures show electrode sites with a significant main effect of time window, condition and the interaction effect of time window × condition, respectively, in the aperiodic exponent (this corresponds to the subheading in the figure). Colors indicate cluster-level summed F-values.

$P = 0.01$, $\eta_p^2 = 0.05$). The main effect in the aperiodic exponent in different conditions was observed over frontal and central areas (cluster 1: FC2, CP2, CPZ, CP1, $F_{(1, 42)} = 17.41$, $P < 0.05$, $\eta_p^2 = 0.39$; cluster 2: P2, PZ, P1, CP3 $F_{(1,32)} = 20.12$, $P < 0.05$, $\eta_p^2 = 0.43$). The interaction effects of time window × condition was observed over frontal areas (cluster: AF7, FP1 $F_{(1, 42)} = 27.07$, $P < 0.05$, $\eta_p^2 = 0.80$). The scalp topography for the aperiodic exponent is shown in Fig. 9.

Discussion

The aim of this study was to test and compare predictions from the traditional approach to cognitive control with predictions from metacontrol theory in a task-switching paradigm. Assuming that higher control demands would lead to an increase of the signal-to-noise ratio and, accordingly, to an increase of the aperiodic exponent in a FOOOF analysis (indicating decreased "E/I ratio", more inhibition and likely less cortical noise), the traditional approach[31–34] would predict that the more demanding task-switching condition should show a *higher* aperiodic exponent than the less demanding task-repetition condition (note that this prediction holds irrespective of whether proactive or reactive control is assumed[2]). Metacontrol theory[13,46], in turn, would agree that the task-switching condition is demanding, but it suggests that this demand does not translate into more persistence but, rather, into more flexibility. Accordingly, it would predict a *lower* aperiodic exponent for task switching than for task repetition. Our results support the prediction from metacontrol theory[13,46], but not the predictions from traditional control accounts[31–34].

There are two widely accepted ways to interpret the aperiodic activity seen in EEG signals, and these interpretations can coexist. The first perspective is related to the E/I ratio, which can be estimated from the slope of the EEG power spectrum[26,27]. A flatter slope suggests an increase in the E/I ratio, whereas a steeper slope implies a decrease in this ratio. A range of studies has highlighted the relationship between the E/I ratio and aperiodic activity. First, research by Lendner et al. demonstrated that the aperiodic exponent can be used as an indicator to distinguish various arousal states, showing elevated values during REM sleep compared to NREM sleep and NREM sleep compared to wakefulness[18]. These findings are corroborated by in vivo calcium imaging work by Niethard et al., which indicates that inhibitory activity prevails in cortical networks during REM sleep[47]. Second, research indicates that the aperiodic exponent rises during propofol anesthesia, resulting in a notable increase in inhibitory activity[24]. In contrast, during ketamine anesthesia, this exponent decreases, resulting in a relative boost in excitation[24]. These findings imply that tasks relying on cognitive persistence might correlate with a reduced E/I ratio, whereas tasks relying on cognitive flexibility could be associated with an increased E/I ratio. Hence, the findings of the current study may reveal a potential link between states of metacontrol and shifts in neural modulation, either towards strengthened inhibitory processes or heightened excitatory activity within the brain's circuitry.

An alternative perspective regarding aperiodic activity is often referred to as the "neural noise" interpretation. This approach proposes that the aperiodic exponent serves as an indicator of the amount of noise within the neural circuits that generate these signals[19,48]. Synchronized neural spiking activity results in a pronounced increase in the steepness of the 1/f slope, which is associated with an improved SNR in the nervous system. On the contrary, when spiking becomes desynchronized, the 1/f slope often appears less steep, leading to a lowered SNR[28,49]. Converging evidence from different studies support the assumption that variations in the 1/f slope found in EEG signals may serve as a potential indicator of "neural variability"[50,51]. That is, neural variability fluctuations are thought to contribute to the brain's ability to dynamically adjust its activity in response to the requirements of various tasks or situations[24]. Therefore, our results are in line with previous findings by Pi et al., Gao et al., Jia et al., and Zhang et al.[29,36,41,42], which suggest that different levels of neural variability are associated with varying states of metacontrol.

The main effects of the aperiodic exponent in different conditions were observed in particular over frontal and central areas. This fits with previous observations in metacontrol studies[29,41]. However, as emphasized in the introduction, there are good theoretical reasons to be skeptical about a strong connection between metacontrol effects and particular cortical loci. Given the widespread assumption that metacontrol is an emergent property of interactions between the prefrontal and the striatal dopaminergic pathway[43–45], effects are theoretically expected at least across all cortical areas that are affected, directly or indirectly, by these dopaminergic pathways—which holds for the largest part of the human brain. Accordingly, exponent analyses should consider wide cortical spaces, and it is possible that differences in terms of scalp distribution, such as between our two studies, reflect a mixture of theoretically relevant and theoretically irrelevant factors, such as sample size, experimental design, participant characteristics, tasks, and strategies.

Our theoretically motivated main interest was related to the sign of effect in the within-trial period, and here our predictions were clearly supported. Not only was the exponent lower in switch than in repeat trials, but this pattern was found in both studies, suggesting that this effect is replicable. Given that this effect is counter to predictions from traditional control theory, which would have predicted increases in persistence in the switch trials, this demonstration of the robustness of the effect was particularly important.

Even though we had no particular predictions for the pre-trial period, we found that the exponent effects differed between the two studies. In Study 2, the exponents for switch and repeated trials were statistically equivalent, which makes intuitive sense. Not so in Study 1, however, where the pre-trial interval showed the opposite effect of what was obtained in the within-trial interval. Given that the trial did not yet begin, why would there be a difference between the two conditions at all and why would it show the opposite pattern as the within-trial period? Considering the structure of the task makes this outcome more intuitive. Note that the relationship between the condition in the within-trial period and the preceding condition was not random but followed the instructed sequence of tasks. Accordingly, a task switch in the within-trial period would always be preceded by a task repetition. A task repetition in the within-trial period might be preceded by a task switch or a task repetition, but the probability that the preceding task was a task switch was higher for task repetitions than it was for task switches. If we thus assume that the previous trial was accompanied by a metacontrol state that tended to be the opposite of the metacontrol state in the present trial (which fits with the pattern shown in Fig. 8c), it is not unreasonable that the pre-trial period shows the opposite pattern than the within-trial period at all. What remains to be understood, however, is why this pattern occurred in Study 1 but not in Study 2. This might reflect a different strategy of the participants and/or a different speed to change metacontrol from one trial to the other. For instance, participants in Study 1 may have been (more) aware of the task rule, (better) anticipated the upcoming task-switching trial, and (more strongly) prepared to exert greater control. This could be associated with an increased aperiodic exponent. In any case, more research to clarify this issue will be necessary.

One significant limitation of this study is its correlational nature, which restricts the conclusions that can be drawn regarding the relationship between aperiodic activity and metacontrol. Specifically, while this study successfully identifies an association between aperiodic activity and metacontrol, it does not provide evidence for a cause-and-effect relationship. This means that we cannot conclude that changes in aperiodic activity directly influence metacontrol or vice versa. To address this gap, future studies that include causal manipulations of aperiodic activity are warranted. Exogenous modulations of metacontrol may involve techniques thought to affect individuals' cortical noise levels. One effective method is transcranial direct current stimulation (tDCS), a non-invasive brain stimulation technique that alters cortical excitability in the areas directly beneath the electrodes by making subthreshold adjustments to resting membrane potentials[52,53]. Indeed, studies have shown that anodal tDCS (atDCS) improves the SNR by increasing spontaneous neuronal firing and enhancing cortical

excitability in the targeted regions[54–56]. Therefore, using atDCS could help illuminate the relationship between metacontrol and aperiodic activity, as reflected in cortical noise levels.

Taken altogether, our findings provide new insights into the adjustment of cognitive control processes to task-induced challenges. In particular, they offer two major conclusions. First, they support assumptions that the regulation of cognitive control is targeting changes in the neural signal-to-noise ratio[35]. However, in contrast to traditional control theory[31–34], which suggests that all control challenges increase the ratio, we found evidence that some challenges can also reduce it. This finding fits with predictions from metacontrol theory[13,46], which suggests that it is the nature of the challenge that determines the direction of the adjustment. While it is true that many challenges call for stronger persistence, like flanker or Stroop tasks, some challenges, like the need to switch from one task to another, call for more flexibility. As persistence can be considered to benefit from a less noisy brain, flexibility is likely to benefit from more noise[36]. Accordingly, and this is our second conclusion, truly adaptive metacontrol does not necessarily bias processing towards persistence in response to every control challenge, but chooses between persistence and flexibility biases depending on the nature of the challenge. Which, indeed, fits with the very definition of metacontrol.

Methods

Study 1

Participants

Study 1 involved a reexamination of data that had been previously collected from a cohort of 50 participants for different scientific purposes[57]. Participants who did not complete the experiment for physiological reasons (i.e. cramps in neck or arm muscles, drowsiness, headaches) and technical problems during recording were excluded from data analysis ($n=10$). We also excluded participants with performance $<70\%$ in any session ($n=3$) and participants ($n=4$) who were unable to improve their performance above 70% even with the presentation of helping cues (see task design and statistical analysis below). As a result, the final sample for these and our present analyses consisted of 33 participants, including 15 males, with an average age of 24.56 ± 4.75 years. Each participant provided their written consent voluntarily and received compensation, either in the form of financial remuneration or course credits, for their participation. The research was conducted in compliance with the ethical guidelines set by the Ethics Committee of the Medical Faculty at TU Dresden, following the principles outlined in the Declaration of Helsinki.

Task design

A task-switching paradigm established by, and validated in various previous studies of the Dresden lab was used[58–61]. Participants were asked to follow specific rules while responding to a series of number stimuli. Each trial involved selecting one number from the set {1,2,3,4,6,7,8,9}. Out of these numbers, {1,3,4,7} were consistently presented in a large font size of 100, while the remaining numbers were displayed in a smaller font size of 50. The response for each stimulus in this experiment was determined based on three rules. These rules were referred to as NUM (short for the German word "numeric," meaning "numeric" in English), GER (short for "Geradzahligkeit," meaning "even(ess)" in English), and SG (short for "Schriftgröße," meaning "font size" in English). Participants had to make decisions regarding whether the stimulus was smaller or larger than 5 (NUM), the polarity of the number (GER), and the font size of the number (SG). Figure 10 (panel A) visually represents the pairings between different stimuli and their corresponding responses, organized according to each specific grammar.

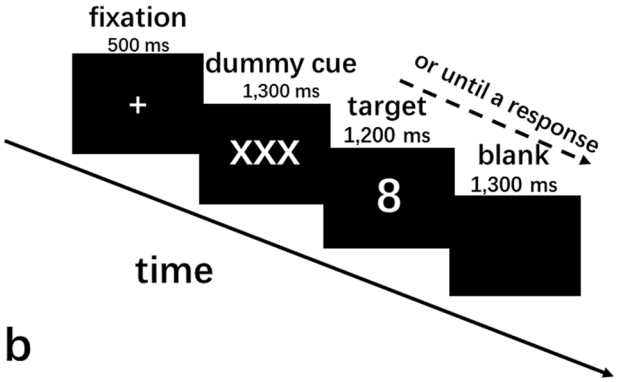
In the experiment, a predetermined sequence of rules was followed during the trials. This sequence involved the repetition of rules such as NUM-NUM-NUM-GER-GER-GER-SG-SG-SG-NUM- and so on. Participants were required to maintain this rule sequence in their working memory to guide the selection of appropriate responses. To facilitate the application of a rule to a presented target stimulus, a dummy cue in the form of "XXX" was used. This dummy cue served as a reminder for participants to retrieve the relevant rule and prepare for selecting the appropriate response. The sequence of events for each trial are shown in Fig. 1B. At the begin of the trial, a fixation cross appeared on the screen for 500 ms, followed by a dummy cue for 1,300 ms, the target stimulus for 1,200 ms, and a blank screen for 1,300 ms. Participants were instructed to respond as quickly and accurately as possible immediately after the stimulus appeared. Once a response was made, the trial ended promptly. In order to counteract any effects on pupil dilation caused by irrelevant brightness, suitable colors were chosen for both the stimuli (RGB code 168, 175, 77) and the background (RGB code 147, 170, 192), as illustrated in Fig. 1 (panel B). To help participants remembering the task rules, we implemented a reminder mechanism. Whenever participants made consecutive errors, they were provided with informative cues that explicitly conveyed the task rules. These cues were presented three times alongside feedback, with the aim of helping the participants regain their focus and understanding.

Procedure

To control the effects of daytime and caffeine (which may lead to performance enhancement), all participants were told to avoid the use of caffeinated beverages prior to the experiment and were tested at approximately 9:00 AM. Participants' psychological status was assessed via the Fatigue Scale for Motor and Cognitive Functions (FSMC)[62] and Beck's Depression Inventory (BDI)[63]. Following that, while EEG caps were set in place, participants were positioned in front of a 24-inch screen, situated 50–70 cm away. An initial brief version of the experiment, comprising 36 trials, was conducted to acquaint participants with the task. The main experiment commenced once participants had a comprehensive understanding of the task, encompassing a total of 2,880 trials and lasting approximately 2 h.

a

Response button	Specific rule	Numerical stimulus
Left button	NUM	smaller than 5 (1 2 3 4)
	GER	odd number (1 3 7 9)
	SG	small fontsize (2 6 8 9)
Right button	NUM	greater than 5 (6 7 8 9)
	GER	even number (2 4 6 8)
	SG	large fontsize (1 3 4 7)



b

Figure 10. The specific rules and procedure of the experimental paradigm. Panel **a** depicts the responses corresponding to numerical stimuli under different rules. Panel **b** shows the procedure of each trial. To enhance visibility, the colors of the stimuli and background have been changed to white and black. GER, even(ess); NUM, numeric; SG, font size.

Nineteen 10-sec intervals were implemented throughout the whole experiment for participants to relax the muscles in neck and arm/hand. The total trials were evenly distributed into four sessions, with each session containing a set of 720 trials.

EEG recording and processing

During the EEG data recording, a total of 60 Ag/AgCI equidistant electrodes were used, along with the BrainVision Recorder software package from Brain Products, Inc. The data was sampled at a rate of 500 Hz. The ground electrode was positioned at coordinates theta = 58, phi = 78, while the reference electrode was positioned at theta = 90, phi = 90. To preprocess the collected EEG data, the BrainVision Analyzer 2 software package was employed. The first preprocessing step involved downsampling the raw data to a frequency of 256 Hz. Then, an infinite impulse response (IIR) filter was applied to the downsampled data, with a range of 0.5 to 40 Hz. The slope of the filter was set to 48 dB/oct, and a notch filter was applied at 50 Hz to eliminate any interference from power line noise.

Following the filtering step, faulty channels that showed no activity or had high noise levels were identified and removed. Additionally, a new average reference was calculated and subtracted from the remaining channels to improve the data quality. To further enhance the quality of the data, manual inspection and artifact removal were performed. Independent component analysis (ICA) using the infomax algorithm was implemented, to enable the removal of common artifacts such as eye blinks and eye movements from the EEG data (average number of 6.21 ± 2.80 independent components were removed). Then, to interpolate discarded channels, spherical splines interpolation was applied.

Next, the pre-processed EEG data were split into fragments of 5500ms duration, with the stimulus onset serving as the locking point. Each fragment spanned from 2,500ms before to 3,000ms after the locking point. In order to identify and eliminate trials with residual artifacts, an automated artifact rejection method was applied, using the default criteria specified in BrainVision Analyzer 2. These criteria involved limiting the maximum voltage step to 30 mV/ms, allowing a maximum difference of 100 mV in intervals of 200 ms, and setting the minimum and maximum amplitude at 150 mV respectively. Additionally, the activity in any given 100ms interval should not fall below 0.5 mV. Trials with helping cues and feedback were excluded from the analysis. The trials were further categorized as either "switch" or "repetition" trials based on the presence or absence of a rule transition, respectively.

Parameterization of spectral data

We identified two specific time windows for analysis. The pre-trial period encompassed the time form −1000 ms to 0 ms before the stimulus. The within-trial period spanned form 0 ms to 1000 ms after the stimulus onset. Drawing from prior research[64], which incorporated a 0.25s Hamming window and a 50% overlap, the power

spectral density (PSD) for each frequency was computed using the 'pwelch' function in Matlab (version R2018a). These calculations were performed individually for each participant, electrode, condition (switch/repeat) and specific period (pre-trial/within-trial). The power spectra of the EEG were analyzed using the Python-based FOOOF toolbox version 1.0.0 (available at https://github.com/fooof-tools/fooof[21,65]). This toolbox decomposes the power spectrum into two components: aperiodic activity [*L(f)]* and periodic (oscillatory) activity [*Gn(f)*]. The FOOOF algorithm breaks down the power spectrum and expresses it as a linear combination of these two components, where *f* represents the frequency:

$$PSD\,(f) = L\,(f) + \sum_n G_n\,(f) \tag{1}$$

The PSD is achieved by merging the aperiodic element, denoted as *L(f)*, with a sum of n Gaussian distributions. To encompass the entire spectrum being analyzed, *L(f)*, which represents the aperiodic component, is fitted as a function spanning the complete range. The mathematical expression that describes the aperiodic component, *L(f)*, is defined as follows:

$$L\,(f) = b - \log\,[f^x] \tag{2}$$

In this mathematical expression, we encounter two variables. The first variable, denoted as 'b', represents the aperiodic offset, indicating the overall shift in broadband power. The second variable, represented by 'x', stands for the aperiodic exponent, which signifies the slope of the power spectrum line when plotted on a log-log scale. This expression also involves periodic components, often referred to as oscillatory components, which correspond to specific frequency regions where the power surpasses the aperiodic component. Each oscillatory element, known as a 'peak', can be characterized by a Gaussian function that requires three parameters for its definition. The Gaussian fitting can be encapsulated by the following model where the amplitude, center frequency, and bandwidth are the characteristics represented by 'αn', 'μn', and 'σn' respectively:

$$G_n\,(f) = \alpha_{\,n}\exp\left[-\frac{(f-\mu_{\,n})^2}{2\sigma_{\,n}^{\,2}}\right] \tag{3}$$

To fit the spectra within the frequency range of 3 to 40, specific settings were used: peak width limits of 2 to 8, a maximum of 8 peaks, a minimum peak height of 0.05, and a fixed aperiodic mode. This fitting process provided an aperiodic exponent value for each electrode, task condition, participant, session and period. The average R^2 value for the spectral fits across all 33 participants exceeded 0.97, indicating highly accurate fits. The study conducted by Zhang et al. highlighted the influence of index metacontrol states on the aperiodic exponent[30], while the impact on the offset was negligible. Accordingly, our analysis primarily focuses on examining the aperiodic exponent. We calculated this exponent for each participant and electrode using the aperiodic-only signal. Since we had no a-priori assumptions about the distribution of aperiodic neural activity across the scalp, we followed the recommendation of Hill et al. by utilizing the "global" exponent for our statistical analysis[66]. First of all, the average of the exponent values across 60 electrodes for each participant were calculated to observe the overall trend of variation at different periods. After that, an extra cluster-based permutation test was conducted to examine the distribution of the aperiodic components on the scalp. The non-parametric cluster-based permutation test is a statistical method proposed for analyzing high-dimensional EEG/MEG data. Its aim is to identify significant differences between electrodes while taking into account multiple comparisons[67]. This approach involves the formation of clusters based on the adjacency of thresholded sample-level F-values with an alpha level of 0.05[67]. MNE-python (https://mne.tools/stable/index.html) was used to perform the permutation cluster test. Permutation testing involves repeatedly shuffling the data labels (e.g., assigning different conditions to the data points) to create random permutations. The more iterations conducted, the closer the simulation results align with the real situation, leading to a more accurate estimation of the P-value. In general, the number of iterations should reach at least 1000 to yield more reliable results. The specified number of permutations (in our case, 1000) determines how many times the shuffling process is repeated. By generating a large number of permutations, the test can estimate the probability of observing the detected clusters by chance, even if there are no real differences between the conditions. This helps control for false positives (Type I errors). In line with this, the cluster-level statistics are calculated by summing the F-values within each cluster. To determine the significance of clusters, a Monte Carlo random sampling technique is employed, with 1,000 iterations and a significance level of 0.05.

Statistical analysis

This is a re-analysis of existing data from Yu et al.[57], who studied the impact of fatigue by dividing trials into four sessions. As our present study did not focus on fatigue, we omitted this factor in our analyses by averaging across sessions. SPSS 26.0 (IBM) was used to analyze the behavioral data, and the aperiodic exponent data. Percent error (PE) and mean reaction times (RTs) data from trials with correct responses were used as behavioral measures. The parameters for each condition (switch/repeat) were calculated for each participant. Then, paired-sample T-test was used to analysis the percent error and reaction time. A two-factor repeated measures ANOVA was conducted to analyze the data on aperiodic exponent. The factors included were condition (switch/repeat) and time window (pre-trial/within-trial). Greenhouse–Geisser correction was used to adjust the ANOVA results, and Bonferroni correction was applied to Post hoc tests. Finally, an extra cluster-based permutation was used to test the distribution of the aperiodic exponent on the scalp. All descriptive statistics include reporting the mean and the standard error of the mean (SEM).

Study 2

Participants

Study 2 used data from 45 healthy participants collected in a previous study for a different scientific aim[68]. Two participants were excluded due to strong artifacts. As a result, the present analyses consisted of 43 participants, including 21 males, with a mean age of 34.65 ± 10.48 years. All participants had normal or corrected-to-normal vision. For more information on the participants, see Petruo et al., 2017. The study and all procedures were approved by the Ethics Committee of the Technical University of Dresden.

Task design

Study 2 employed a task-switching paradigm based on previous work by Wolff et al. and Gajewski et al.[58,60]. This paradigm closely resembled the one used in Study 1, except for the following differences. Two conditions, cue block, and memory block, were introduced in this version. In the cue block, a specific rule cue was presented before each trial to indicate the rule to be followed. However, in the memory block, an "XXX" dummy cue replaced the informational cue. Participants were asked to memorize the sequence of response rules and to respond accordingly in the memory block. The stimulus materials, specific rules, response sequence and conditional division (switch & repeat) in the memory block were identical to the task in Study 1. In the memory block, the presentation of the fixation point marks the start of a trial. This fixation point was followed by a 1300 ms dummy cue, after which a target stimulus appeared. Participants were instructed to respond promptly and accurately based on the memorized rules. After the response, a feedback stimulus was presented with a delay of 500 ms, lasting 500 ms. Correct feedback was indicated by a positive sign and incorrect feedback by a negative sign. After 300 ms, the feedback stimulus disappeared and the next trial started. Upon three successive incorrect responses or no response within a 2500 ms interval, a written rule statement was shown on the screen, along with a clear task cue for the subsequent three trials to guide participants back on track. The memory block consisted of 198 trials. After every 33 trials, participants could take a short break and decide when to start the next 33 trials. Only data from correct trials in the memory block were included in the analysis. The specific rules and procedure are shown in Fig. 11. For more details on the task, see Petruo et al.[68].

Procedure

Participants were treated in accordance with the Declaration of Helsinki and informed consent was obtained before conducting measurements. They were then asked to complete demographic and self-report questionnaires, which included the German versions of the Beck Depression Inventory (BDI)[69] and the Fatigue Scale for Motor and Cognitive Functioning (FSMC)[62]. In addition, ta short and validated test for estimating premorbid IQ levels, the Multiple Choice Word Test-B (MWT-B)[70], was administered. The task was presented on a 20-inch CRT monitor with white numbers on a black background. The numbers were displayed 3 mm above

a

Response button	Specific rule	Numerical stimulus
Right button	NUM	smaller than 5 (1 2 3 4)
	GER	odd number (1 3 7 9)
	SG	small fontsize (2 6 8 9)
Left button	NUM	greater than 5 (6 7 8 9)
	GER	even number (2 4 6 8)
	SG	large fontsize (1 3 4 7)

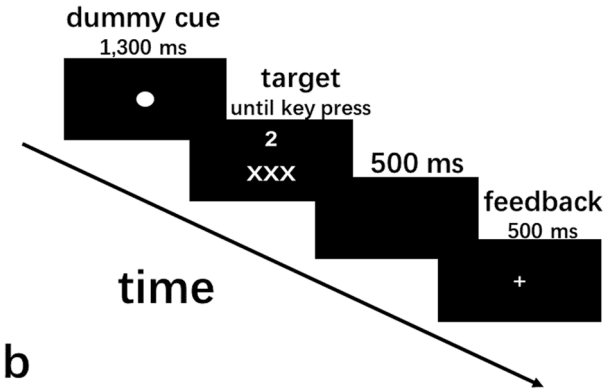


Figure 11. The specific rules and procedure of the memory block paradigm. Panel **a** depicts the responses corresponding to numerical stimuli under different rules. Panel **b** shows the procedure of each trial. To enhance visibility, the colors of the stimuli and background have been changed to white and black. GER, even(ess); NUM, numeric; SG, font size.

a 10 mm diameter white fixation cross, the size of which varied between task conditions (small = 7 × 10 mm; large = 12 × 18 mm). Prior to the formal test, participants were introduced to the task requirements and asked to complete 18 practice sessions to familiarize themselves with the stimuli and task rules. Once participants were confident in understanding the task requirements, they proceeded to the formal trial. All participants were encouraged to answer the questions as quickly and accurately as possible. At the end of the testing, participants were rewarded with €30.

EEG recording and processing

The settings for electrode points, sampling rate and electrode impedances during the acquisition of Study 2 were all consistent with Study 1. Data were manually pre-processed using the Brain Vision Analyzer application. The first step in data pre-processing was to manually inspect and remove visible technical artefacts. The data were then downsampled to 256 Hz using a bandpass filter (IIR zero phase shift Butterworth filter, order 8, time constant 0.32). After downsampling, Independent Component Analysis (ICA) was performed to remove recurrent artefacts such as eye movements, blinks and pulse artefacts. In this way, reconstructed EEG data were obtained. The data were then divided into 4000 ms starting 2000 ms before the lock point and ending 2000 ms after the lock point. An automated artefact rejection procedure was then applied to discard segments containing signal amplitudes greater than 150 μV, less than − 150 μV, periods of 200 ms with activity less than 0.5 μV, or amplitude differences greater than 80 μV for at least 100 ms. The next step was re-referencing using the current source density (CSD) transformation, which removes the reference potential by using the potential difference between the electrodes and the total potential difference of the surrounding electrodes. (parameters utilized: $n = 4$ splines, $m = 10$ Legendre polynomials, $\lambda = 1 \times 10^{-5}$). Subsequently, baseline corrections and mean calculations were made for each condition at the individual level before group means were calculated. Only correct trials were included in the analysis. As in Study 1, trials were further categorized into 'switch' and 'repeat' conditions.

Parameterization of spectral data

The procedure and specific parameterization of the spectral data was identical to Study 1. The average R^2 value for the spectral fits across all participants was 0.85, indicating a good fit.

Statistical analysis

The procedures for the analysis of the data were the same as in Study 1.

Data availability

The aggregated data analyzed in this article are available via the Open Science Framework: https://osf.io/hvf29.

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Declarations

Competing interests
The authors declare that they have no competing interests.

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