Can Occipital Alpha Neurofeedback Influence LTRCs and Deterministic ERPs without Critical Branching?

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Abstract: Critical branching is a theoretical interaction in-between simple units, such as neuronal elements of the human brain. Zhigalov, Kaplan, and Palva (2016, *Clin. Neurophysiol.*, 127(8), 2882–2889) revealed that neurofeedback flash stimulation locked to the phase of high-amplitude occipital alpha influences stimulus-locked occipital averages in the alpha-band. This feedback also influences the power scaling of long-range temporal correlations in alpha-band amplitude fluctuations. Seemingly, neurofeedback influences critical branching alongside there being an interaction between ongoing neuronal activity and evoked responses. However, the causal relations between these neuronal long-range temporal correlations, sustained attention, and any avalanche dynamics are called into question. Further, uncorrected concerns include false discovery rate and an objective mathematical error in the precedent (Palva et al., 2013, *Proc. Nat. Acad. Sci. U.S.A.*, 110(9), 3585–3590). An alternative set of illustrative mathematical principles offers a preliminary fit to the effects in the data. That is, neurofeedback influences the deterministic contribution to the single-trial event-related potentials, which each flash evokes, separately from the oscillatory alpha gain that those flashes cause. Accordingly, distinct principles of this neurofeedback-related exponential occipital oscillatory alpha gain and deterministic event-related potential generation produce micro-behaviours with macroscale consequences: neurofeedback causally influences power-scaling of long-range temporal correlations without critical branching.

Subjects: Neurons and Cognition (q-bio.NC); Adaptation and Self-Organizing Systems (nlin.AO); Biological Physics (physics.bio-ph). Keywords: Closed-loop stimulation; deterministic Event-Related Potentials (ERPs); electroencephalography (EEG); critical neuronal dynamics; long-range temporal correlations (LTRCs); mathematical model.

1.1. Introduction

In a neurofeedback investigation, Zhigalov et al. (2016) refer to criticality analyses alluding to self-organised criticality: a theory inconclusively supported by the neuroscientific data. This term self-organised criticality, or just criticality, is often misconstrued as a property of a physical system, whereas self-organised criticality is, rather, a theory of the interactions in-between multiple simple elements in a physical system. This theory assumes systems of such elements tend to criticality: Inter-elemental interactions,
termed critical branching, poise that system in a critical state between responding in
two ways. In a critical state, a system can respond either through linear local inter-
actions or through a higher-amplitude nonlinear propagation of the response via a
widespread network of elements. That nature of interaction between elements leading
to such a system, poised in what is called a critical state, is termed critical branching.

This theory originated in the peer-reviewed articles on systems such as accumu-
lating piles of long-grain rice self-organising into such a critical state. In turn the
theory became widely popularised across several domains, including systems neuro-
science, now spreading to clinical neurophysiology. Characteristics of a system in a
critical state derived by criticality analyses include $1/f$ spectra, avalanche dynamics,
and fractal self-similarity in the scale-invariance of power-law scaling of long-range
temporal correlations (LRTCs).

1.2. Fluctuations of sustained attention, LRTCs, and avalanche dynamics

Poignant is Zhigalov et al.’s departure from a criticality interpretation that relates neu-
nral LRTCs to arguably conscious phenomena such as fluctuations of sustained atten-
tion: Previously, analysing MEG-derived time-series, Palva et al. (2013) not only iden-
tified such neuronal LRTCs, but also an avalanche dynamics – another characteristic
of criticality. Investigating power-scaling of LRTCs, during a stimulus detection task,
neuronal exponents correlated positively not only with behavioural exponents but also
with neuronal exponents at rest. These behavioural LRTCs concern periods of attention
when the participant correctly identifies a stimulus at threshold punctuated by periods
of inattention. Incorrect performance defines this inattention. Thus power-scaling of
those LRTCs shows that the extent of fluctuation on one timescale determines that on
others – psychologically, there is a power-scaling of fluctuations of sustained atten-
tion. Measures of the amplitude and duration of highly nonlinear neuronal avalanches
during the task correlate negatively with both behavioural and neuronal LRTC expon-
ents. Nevertheless, those avalanche measures did not significantly predict behaviour
independently from neuronal exponents (Palva et al., 2013). These findings call into
question the role of avalanches as a cause for transitions between attention and inat-
tention: there is no such avalanche (Private communication, Alexander Y. Zhigalov,
2015). Thus, self-organised criticality, encompassing an avalanche dynamics, cannot
drive fluctuations of sustained attention.

1.3. Analytical concerns about the precedent (Palva et al., 2013)

Concerns include the objectively incorrect mathematical expression of the parcella-
tion strategy computation (Private communication, Muriel Lobier, 2015). That is, Palva
and colleagues’ in-house pipeline confused real and imaginary parts of complex num-
bers whilst calculating similarities of phase between time series in measures such as
the phase-locking value (PLV) and the distinct imaginary PLV (Siebenhühner et al.,
2016; Korhonen et al., 2014). This confusion has consequences for the fidelity and infi-
delity measures (Korhonen et al., 2014) used in the parcellation strategy of Palva et al.
(2013) (Private communication, Muriel Lobier, 2015), the data from which Zhigalov et al.
(2016) cite as motivating their study. While this objective error might be trivialised
(Private communication, Jaakko M. Palva, 2015), placing further weight on the results
of Palva et al. (2013) in motivating and interpreting new investigations such as that
of Zhigalov et al. (2016) requires errata that demonstrate Palva et al. (2013)’s results
go unaffected by this objective error. A further concern that could also necessitate an
erratum to Palva et al. (2013)’s germane study is the elevated false discovery rate of
the in-house data pipeline (Private communication, Hugo Eyherabide, 2015). These
analytical concerns are prominent inasmuch that Palva et al. (2013) associate scale-
free behavioural dynamics and MEG source-level dynamics motivating Zhigalov et al.’s
assertions that closed-loop neurofeedback, such as that of Zhigalov et al. (2016), when neuroanatomically targeted could offer insights into pathological conditions. However, inferences drawn about the characterisation of Palva et al.’s seemingly psychologically relevant sources remain questionable thus warranting cautious reconsideration of the potential for this targeted neurofeedback that Zhigalov et al. propose.

1.4. Neurofeedback influences power-scaling of LRTCs without a reported avalanche dynamics
Zhigalov et al.’s eyes-closed rest procedure demonstrates neurofeedback alters LRTCs of scalp-measured EEG time-series in the alpha-band. That neurofeedback relied on a flash 12.5 msec after alpha oscillations’ peaks exceed a certain value (Fig. 1A). As depicted by the triangles denoting the time of flashes following those high-amplitude peaks, that 12.5 msec is roughly one eighth (0.125) of a 0.1 sec alpha oscillation. In contrast to Palva et al., Zhigalov et al. do not present an avalanche dynamics that would occur if neurofeedback influenced critical branching. If a subset of the hallmarks of self-organised criticality need be present for critical branching to be operational, then surely the conceptual integrity of the notion is subject to question: A critical branching with an avalanche dynamics (Palva et al., 2013) would be theoretically distinct from that seemingly without such dynamics (Zhigalov et al., 2016).

1.5. ERPs and consciousness
Zhigalov et al. claim the neurofeedback to be unconscious. In the auditory domain, ERP componentry such as the auditory mismatch negativity (Näätänen and Winkler, 1999; Campbell et al., 2007; Ruby et al., 2007; Morlet and Fischer, 2014) is generated in a manner classically considered unconscious and unmodulated by attention (for recent debate, see Campbell, 2015; Wiens et al., 2016). Nevertheless, auditory onsets of which participants are unconscious due to physical characteristics of the stimulation do not elicit the N1 and P2 deflections of auditory ERPs (Lightfoot and Kennedy, 2006). Do onsets of visual stimuli that are subliminal due to characteristics of the stimulation have the capacity to elicit ERPs unconsciously? Flashes at a 50% luminance detection threshold elicit ERPs but only on trials when participants detect those flashes (Busch et al., 2009): Trials on which participants’ performance revealed they were not subjectively conscious of a flash did not elicit an ERP. Zhigalov et al. reveal alpha-band ERPs, which are influenced by neurofeedback, because participants are visually conscious of the flashes seen: That is, the experimenter adjusted the luminance so that participants, with their eyes closed, were aware of the flashes through their eyelids. On balance, in complex paradigms it is possible for stimuli to elicit visual ERP componentry (e. g., Lyyra et al., 2010; Sysoeva et al., 2015) even without conscious awareness of the content of the stimulus that is otherwise supra-threshold in isolation. However, it is hard to imagine how the paradigm of Zhigalov et al. (2016) engendered participants to be unconscious of flashes that they could see. No neurofeedback-related changes in the visibility of the flashes through the participant’s eyelids are reported. The point is that physical characteristics of a stimulus need to be in principle accessible to perceptual consciousness to elicit ERPs. Luminance increments arguably summon attention when a luminance increment defines the to-be-attended stimulation (Yantis and Hillstrom, 1994). Accordingly, consciously perceived luminance increments involuntarily capture attention.

1.6. Higher amplitude ongoing alpha in the baseline of neurofeedback treatment than in the sham control
Delivering the neurofeedback flash at a fixed interval from the alpha peak causes ongoing alpha to represent more strongly in the pre-stimulus baseline. That is, the neurofeedback treatment epochs were phase-locked to a particular point in the alpha cycle.
Fig. 1. A simulated noisy amplitude-modulation of a noisy period-modulated 10 Hz harmonic oscillator, with neurofeedback at (A) 0.5π to π (B) eliciting complexes of overlapping flash-evoked deterministic ERP alpha-band waveforms and exponential oscillatory amplitude gain in the O2 recording of alpha-band, \( \alpha_t \), during the first neurofeedback block \( B_1 \). Effective neurofeedback succeeds high-amplitude oscillations not exceeding a physiological maximum, \( \theta_{\text{max}} \). Composite ERP-oscillatory waveforms trigger flash volleys until the influence of physiological sources, including this exponential gain, rise over a physiological maximum or, fall below a calibrated minimum, \( \theta_{\text{min}} \), ceasing the contribution of oscillatory gain. The Supplementary Material defines this model. (C) Exponential gain is stronger on the third neurofeedback block \( B_3 \). (D) Grand-averaged \(< \alpha_t >\) time-locked to flashes exhibits both pre- and post-flash differences between neurofeedback blocks as flashes occur in volleys due to partially overlapping ERP complexes and oscillatory gain spindles. Neurofeedback block influences (E) LRTC Detrended Fluctuation Analysis neurofeedback mean exponents (block 3: \( \beta_B > \) block 1: \( \beta_{1B} \)) exceeding surrogate data exponents (\( \beta_{S3} \) and \( \beta_{S1} \)).
Fig. 2. Phase-locking of stimulus presentation to the alpha oscillation can cause constructive averaging influencing the representation of ongoing alpha in the pre-stimulus baseline. Different schematic epochs of alpha-band EEG, each at a different set of discrete time-points, $t$, contain ongoing oscillations $O_t$, simplified to $2 \sin(2 \pi f t + \varphi)$, such that $f_t \sim N(10, 1)$ is constant within an epoch, added to the deterministic ERP, $E_t$, as detailed in Supplementary Materials, with only one flash-elicited deterministic ERP evoked per epoch. (A) The phase $\varphi \sim N(0, 1)$ of $O_t$ can be inconsistent at corresponding time-points relative to different stimulus onsets in each simulated sham control epoch, $C_n$, as regularly in counter-phase at corresponding time-points in different epochs. (B) Contrastingly, the phase, $\varphi = (7 \pi)/8$, of $O_t$ is broadly consistent at corresponding time-points in each simulated neurofeedback treatment epoch, $T_n$, and very rarely in counter-phase. This difference in the distribution of ongoing alpha oscillation phase between simulated sham $C$ and neurofeedback $T$ is best gleaned by considering at $t = 0$ the less uniform phase of epoch waveforms in A than in B. In line with Zhigalov et al.'s Fig. 4, ongoing alpha oscillations are thus less strongly represented in the pre-stimulus baseline of averages of 800 epochs, $\langle O_t + E_t \rangle$, time-locked to (C) control sham epochs $C$ then for (D) treatment neurofeedback epochs $T$. 

On the sham control trials, there was no such phase-locking, alpha cycles appearing in epochs starting at multiple phases with flash onset. As such, in averaging, alpha cycles were of very similar phase in all neurofeedback treatment epochs averaged. Contrastingly, although alpha oscillations can also be in-phase between some of the sham control epochs, the phase of these alpha cycles of these control epochs tends to differ more than between neurofeedback epochs. After averaging, the alpha waveform phase-locked to the onset is thus apparent in neurofeedback not sham averages of Zhigalov et al.’s Fig. 4. As schematised in Fig. 2, this ongoing alpha thus considerably averages-out on sham trials.

1.7. Long-term effects of neurofeedback in the pre-stimulus baseline
This ongoing occipital alpha, phase-locked to the flash, is higher in amplitude on the third neurofeedback block than on other blocks. As simulated in Fig. 1A–C, if flashes and the subsequent alpha augment occurred in volleys, effects of neurofeedback from the flashes beforehand not only influence the post-stimulus ERP but also, more subtly, the pre-stimulus baseline (Fig. 1D). Zhigalov et al. adopt the notion that there is an interaction between ongoing neuronal activity and evoked responses rather than their linear summation. A corollary is that ERPs are an amplitude modulation of non-zero mean oscillations in the ongoing EEG (Nikulin et al., 2007).

1.8. Simulations with an alternative micromodel
Rather, higher ERP amplitudes due to occipital alpha contamination phase-locked to flashes support another model: noise and ongoing EEG oscillations summate linearly with an invariant ERP signal to each stimulus of a given class (Ivannikov et al., 2009). Building on this notion of linear summation, in simulations with the micromodel detailed in Supplementary Material, occipital alpha measurements derive from addition of the neurofeedback-modulated deterministic contribution of single-trial flash-evoked ERPs to ongoing alpha oscillations. These measurements also derive from a further additive neurofeedback flash-evoked gain in the amplitude of those oscillations. Neurofeedback influences simulated power-scaling (Fig. 1E) of LRTCs (Fig. 1B–C) without the inclusion of a critical branching assumption. This micromodel is phenomenological, an illustrative set of mathematical principles offering parameters (Table 1 of Supplementary Material) of a preliminary fit to Zhigalov et al.’s data. There is further potential to optimise this fit. The principles of the micromodel comment on the effects simulated. Whilst critical branching might be considered a mechanism, the observed effects do not warrant the conclusion that these effects influence such a mechanism. These effects cannot reveal the biological mechanisms of the effects and as such, although feasible, the principles of the micromodel offered in Supplementary Material are not specified at a mechanistic level of detail. Rather, as the implementation demonstration-proof that critical branching is not a necessary mechanism, the micromodel reveals effects on the, seemingly critical, power-scaling of LRTCs: This micromodel uses random Gaussian noise generated with a computer algorithm instead of a critical branching process. For a related perspective that power-scaling of LRTCs in itself is not a sufficient condition to assume critical branching is in operation, please see Botcharova et al. (2015). The micromodel offered here does make unprecedented predictions testable with the dataset of Zhigalov et al. For instance, predictions include that neurofeedback flashes would occur in volleys (Fig. 1A–C).

1.9. Concluding remarks
Zhigalov et al.’s findings neither conclusively support a theory of critical branching between neuronal elements nor deny stimulus-evoked ERPs theoretically deterministic characteristics. In light of the foregoing issues, a further concern is that the clinical
potential of this approach to neurofeedback that Zhigalov et al. implied remains empirically undetermined.

1.10. Conflict of interest statement
The author has no potential conflicts of interest to disclose.

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Supplementary Material

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2. Simulation model

The simulation model (Eq. 1) assumes that measures of an EEG time-series in the alpha-band, \( \alpha_t \), at discrete time-points \( t \) comprise the addition of ongoing neuronal oscillations, \( O_t \), to the deterministic contribution to single-trial flash-evoked ERPs, \( E_t \), thence to neurofeedback flash-evoked gain in the amplitude of those oscillations, \( G_t \), all of which are contaminated by the linear addition of additive Gaussian white noise \( n_t \sim \mathcal{N}(0,v) \), \( v \) being the variance of zero-mean noise controlled by multiple physiological and non-physiological factors. These noise factors correlate neither with these contributions to alpha oscillation generation nor with ERPs:

\[
\alpha_t = O_t + E_t + G_t + n_t
\]  

In the following, each of the first three terms in this simulation model are introduced, leading into the basic dynamics and operation of that model. The principles introduced foreshadow how the simulation model comments upon the averaged data and the power-scaling of long-range temporal correlations (LRTCs) in the continuous alpha-band data of Zhigalov et al. (2016). The discussion now turns to the first term of the model.

2.2. Ongoing oscillations

Ongoing oscillations, \( O_t \), are amplitude modulations of a harmonic oscillator, \( \sin(2\pi ft+\pi) \), of a frequency \( f \) (Eq. 2) that the influence of thalamic and neocortical pacemakers on occipital neuronal elements determines, for instance via shunting inhibition (Scheeringa et al., 2011). The period of those ongoing oscillations, determined by multiple factors, has a Gaussian distribution such that \( f_t \sim \mathcal{N}(10,1) \) with a mean of 10 Hz and a variance set to 1 for definiteness. Multiple physiological factors related to the amplitude-modulation of ongoing alpha-band oscillations produce multiplicative zero-mean Gaussian white noise \( m_t \sim \mathcal{N}(0,u) \) with variance \( u \). In turn, a smoothing of the absolute value of this physiological multiplicative noise, \( p_t \), (Eq. 3) amplitude-modulates each oscillation. This process produces a smooth random modulation of the ongoing oscillation that is typically low but occasionally high (Eq. 2).

\[
O_t = p_t \sin(2\pi ft + \pi)
\]  

\[
p_t = \frac{\sum_{t-g}^{t} |m_t|}{10}
\]  

The neuronal underpinnings of long-range temporal correlations in the alpha-band could be amongst the multiple physiological factors that determine \( m_t \sim \mathcal{N}(0,u) \).
However, the presence of strong power-scaling of LRTCs in resting-state alpha amplitude modulation without neurofeedback or stimulation, is unnecessary as a ground condition: Without such strong power-scaling initially present, it is possible to simulate the influence of neurofeedback flashes on averaged waveforms time-locked to those flashes. It is also possible to simulate how neurofeedback causes power-scaling of LRTCs as well as influencing that power-scaling. Here, \( m_t \sim \mathcal{N}(0, u) \), rather, simplifies to a random matrix of Gaussian white noise with the constraint that values are identical within an oscillation but do not predict the values on subsequent oscillations. However, smoothing (Eq. 3) does render the amplitude modulation of oscillations predictable on the time-scale of 10 oscillations, ca. 1 s. The resulting ongoing oscillations, \( O_t \), contaminated by additive noise unrelated to alpha oscillations, \( n_t \sim \mathcal{N}(0, v) \), suffice for occasional alpha peaks of \( \alpha_t \), at the peak of \( O_t \), to exceed an individually calibrated threshold \( \theta_{\text{min}} \) even without neurofeedback stimulation.

2.3. Deterministic ERPs

Each flash then evokes the theoretically deterministic contribution to a single-trial ERP waveform with, accordingly, an invariant time-course of these deterministic ERPs. Turning to this second \( E_t \) term of the model, these deterministic ERP waveforms derive empirically from the waveforms in Zhigalov et al. (2016)’s Fig. 4. These deterministic ERP waveforms follow the time-course of the grand-averaged ERP to the first set of sham blocks’ grand-averaged O2 ERP from 0 to 250 msec post-stimulus. In this simulation, in response to each flash, each deterministic ERP, \( w_t \), thus lasts 250 msec. With deterministic ERPs elicited by flashes in rapid succession, deterministic ERPs elicited by a single prior flash or several prior flashes can overlap the deterministic ERP to the most recent flash. When such overlap occurs, deterministic ERPs add together constructively in the ongoing time-series, \( w_t \). There is an emergent property of such overlapping deterministic ERPs with inter-flash intervals near to 100 msec, as would occur with stimuli presented 12.5 msec after the peak of consecutive alpha oscillations. That is, the refracted P1 to the most recent flash adds to the refracted P2 that the preceding stimulus elicited. Flashes, which are phase-locked to consecutive alpha oscillations that are relatively stable in period, which \( f_t \sim \mathcal{N}(10, 1) \) engenders in (Eq. 2), thus give rise to complexes of overlapping deterministic ERPs. The schedule of stimulation evokes these deterministic ERPs. The time-series, \( E_t \), thus contains these deterministic ERPs and their complexes. \( E_t \) is otherwise set to zero without a recent flash.

While the time-course of deterministic ERPs is invariant, exposure to neurofeedback influences the amplitude of the deterministic generation of the componentry of those ERPs. Relatively longstanding factors caused by exposure to neurofeedback – for instance, drowsiness, learning, and changes in sustained or selective attention to flashes – lead to increases in the blockwise strength coefficient, \( s \), as a function of neurofeedback block number. While these factors may operate gradually over a matter of minutes, the model emulates their contribution blockwise. This blockwise increment in strength, \( s \), augments the amplitude of the contribution of the componentry of deterministic ERPs \( w_t \) to \( E_t \) (Eq. 4). Accordingly, the amplitude of each deterministic ERP \( w \) weighted by \( s \) is higher during later, neurofeedback blocks, during which an assumption is that neurofeedback has more influence:

\[
E_t = sw_t
\]  

(4)

2.4. Neurofeedback-related exponential occipital oscillatory alpha amplitude gain

On a shorter-term time-scale, for each effective flash, a neurofeedback flash-related gain in the amplitude of the subsequent oscillation, \( G_{\text{f}} \), increases exponentially according to the number of flashes occurring during preceding consecutive alpha peaks
It is to this $G_t$ term of the simulation model that the discussion now turns. The exponent of this gain is the number of preceding effective flashes, $l$, on consecutive oscillations weighted by a gain coefficient, $g$, determining the increasing potency of each such consecutive flash to increment gain on the next oscillation. Accordingly, independent of influences on deterministic ERPs, relatively longstanding factors caused by exposure to neurofeedback – as might include drowsiness, learning, and changes in sustained or selective attention to flashes – lead to a modulation of this gain that varies on a block-by-block basis. While these factors may operate gradually over a matter of minutes, the model implements their contribution blockwise. Increments in a block-wise coefficient, $b$, thus also increase the flash-related exponential gain, $G_t$, in the next alpha oscillation as neurofeedback becomes more effective from block-to-block:

$$G_t = b e^{gl} \sin(2\pi ft + \pi)$$  \hfill (5)

2.5. Model dynamics and operation

Having introduced the terms of the model, this discussion turns to the model’s dynamics and operation. Neurofeedback flashes occur between $0$ to $\pi$ of oscillations of $-\pi$ to $\pi$ radians of the oscillation, $\sin(2\pi ft + \pi)$, i.e., during the peak rather than the trough of an alpha oscillation (Fig. 1A) as in Zhigalov et al. (2016)’s neurofeedback blocks. Consequently, there is a reduction in the occipital haemodynamic response to such visual stimuli (Scheeringa et al., 2011). Whilst limiting the occipital haemodynamic response to stimulation, an assumption is that such flashes during alpha peaks (Fig. 1A) tend to increase occipital alpha power on the subsequent oscillation. As specified in Eq. 5, for each such effective flash, $l$ thus increments by 1. Each such effective neurofeedback flash thus increases gain, $G_t$, elevating the probability that the peak amplitude of the next oscillation of $\alpha_t$ will exceed a calibrated threshold, $\theta_{min}$, at the peak of $O_t$. In this way, flashes occur in volleys triggered by alpha spindles (Fig. 1A–C) rising in amplitude over this threshold according to an exponential tendency. When $\alpha_t$ reaches a primarily physiologically determined maximum, $\theta_{max}$, $l$ returns to 0 such that the gain $G_t$ is also 0 for the subsequent oscillation. The triggered neurofeedback flash is thus ineffective. In turn, that gain does not contribute to the amplitude of that oscillation, tending to bring the reciprocal neurofeedback between a spindle of oscillatory gain, complexes of deterministic ERPs, and a volley of flashes to a gradual closure. These dynamics apparent for neurofeedback block 1 in Fig. 1B are more prominent for neurofeedback block 3 in Fig. 1C. Whilst increases in oscillatory gain occur on the cycle following an effective flash, each flash elicits a deterministic ERP – whether effective or ineffective. In the simulated dynamics of $\alpha_t$, such ineffective flashes occur (Fig. 1B–C) more frequently on the later block 3.

Multiple factors producing physiological noise, $p_t$, related to the strength of amplitude modulation or potentials from multiple unrelated sources of physiological or non-physiological noise $n_t$, can together occasionally cause $\alpha_t$ at the peak of the oscillation to fall below the pre-calibrated minimum, $\theta_{min}$, for triggering a new flash. $\alpha_t$ can fall below this threshold even when there is an increase in oscillatory gain, $G_t$. Falling below this $\theta_{min}$ threshold, at the peak of $O_t$, also brings the reciprocal interaction between neurofeedback flashes, the spindle of oscillatory gain, and deterministic ERPs to a closure. Accordingly, as simulated in Fig. 1B–C, volleys of neurofeedback flashes can thus end without an ineffective flash.

2.6. Preliminary parameter fits for simulating Zhigalov et al.’s dataset

A variety of dynamics is available within the model’s parameter space, and these parameters might be tuned to fit individual data. Having introduced the terms, dynamics and operation of the model, the discussion now turns to the simulation of Zhigalov et al. (2016)’s data. Table 1 overleaf details parameters for the simulation...
depicted in Fig. 1 that centres on fitting the grand-averaged O2 waveform from neurofeedback blocks 1 and 3 (Zhigalov et al., 2016). In this simulation of nine first neurofeedback blocks and nine corresponding third neurofeedback blocks, blocks of 20 minutes each, i.e., 180 minutes of $\alpha_t$, the model's parameters thus serve in the emulation of alpha-band $\alpha_t$ data at O2 for nine representative participants.

| Parameter                                                                 | Symbol | Value     |
|---------------------------------------------------------------------------|--------|-----------|
| Variance of alpha oscillation-unrelated additive Gaussian white noise, such that $n_t \sim \mathcal{N}(0, v)$ | $v$    | 4.00      |
| Variance of alpha oscillation-related multiplicative Gaussian white noise, such that $m_t \sim \mathcal{N}(0, u)$ | $u$    | 2.31      |
| Mean frequency of the harmonic alpha oscillator, such that $f_t \sim \mathcal{N}(10, 1)$ | $f$    | $1.00 \times 10$ |
| Calibrated threshold for a neurofeedback flash                            | $\theta_{\text{min}}$ | 8.04 |
| Physiological maximum of EEG                                             | $\theta_{\text{max}}$ | $2.01 \times 10$ |
| Neurofeedback block 1 blockwise deterministic ERP strength coefficient modulation | $s_{B_1}$ | $9.10 \times 10^{-1}$ |
| Neurofeedback block 3 blockwise deterministic ERP strength coefficient modulation | $s_{B_3}$ | 1.19 |
| Neurofeedback increase in occipital oscillatory alpha gain exponent per effective flash | $g$ | $6.6 \times 10^{-2}$ |
| Neurofeedback block 1 blockwise occipital oscillatory alpha gain modulation | $b_{B_1}$ | 1.00 |
| Neurofeedback block 3 blockwise occipital oscillatory alpha gain modulation | $b_{B_3}$ | 2.70 |

500 msec epochs of $\alpha_t$ with a 250 msec pre-stimulus baseline for each participant were binned separately for the first and third neurofeedback blocks collapsing across epochs time-locked to effective and ineffective flashes, averaged, and these sim-
ulated individual weighted-average waveforms then grand-averaged in an unweighted manner. The primary goal of the simulation was to model averaged data from such simulated individual participants for which an actual grand-averaged O2 waveform was available (Fig. 4 of Zhigalov et al., 2016). The simulated grand-averaged waveform of amplitudes $\langle \alpha_t \rangle$ as a function of time post-stimulus onset met this goal to the extent depicted in Fig. 1D.

2.7. Simulated ascending alpha gain spindles and deterministic ERPs
Spindles of exponentially ascending alpha gain, additive to deterministic ERP complexes, and the consequent reciprocal volleys of neurofeedback flashes became more prevalent on the later blocks, when neurofeedback becomes more influential. In turn, the averaged simulated alpha-band amplitude $\langle \alpha_t \rangle$ (Fig. 1D) increased from early to later neurofeedback blocks. This increase occurs not only after the stimulus but also before the stimulus given that neurofeedback flashes in volleys also occur in the pre-stimulus baseline.

2.8. Simulated LRTC power-scaling for neurofeedback compared to surrogate and rest blocks
In this simulation, stimulation volleys, deterministic ERP complexes, and spindles of increasing oscillatory alpha gain in $\alpha_t$ impact the power-scaling of LRTCs (Fig. 1E), as Zhigalov et al. (2016) reveal. Detrended Fluctuation Analysis, variously known as DFA (Peng et al., 1994; Hardstone et al., 2012), yields exponents for simulated data from neurofeedback blocks 1 and 3. Respectively, these exponents have a mean $\beta_{B_1}, 0.600, 95\%$ Confidence Intervals (CI) $[0.585, 0.641]$, and a mean $\beta_{B_3}, 0.633, 95\%$ CI $[0.621, 0.665]$. These mean exponents are higher than the corresponding mean exponents of the surrogate data. That is, for the counterpart phase-randomised data, these surrogate exponents have a lower mean $\beta_{S_1}, 0.532, 95\%$ CI $[0.519, 0.569]$, and mean $\beta_{S_3}, 0.554, 95\%$ CI $[0.537, 0.600]$. For neither set of neurofeedback blocks does the range of power-scaling exponents overlap that of the corresponding surrogate data. These neurofeedback block exponents are higher because the surrogate data do not contain the temporal structure emergent in the simulated neurofeedback blocks.

There is further support for this notion that deterministic ERP complexes and spindles of increasing oscillatory alpha gain introduce the temporal structure reflected by the LRTC: These mean power-scaling exponents of the phase-randomised surrogate data reside more within the lower range of the simulated resting-state $\alpha_t$ mean exponent, $\beta_{R}, 0.503, 95\%$ CI $[0.483, 0.560]$, without stimulation. For neither neurofeedback block does the range of power-scaling exponents overlap that for the corresponding surrogate data. Given stimulation does not occur in this resting-state control, simulation of such resting-state data relies on a subset of the terms of Eq. 1. These terms include ongoing alpha-band oscillations $O_t$, into which the smoothing of Eq. 3 introduces autocorrelational temporal structure. That resting-state data also include noise, $n_t$, unrelated to those oscillations. This resting-state control data, regardless of said smoothing, exhibits the power-scaling exponent of a near-random process. The higher range of neurofeedback exponents thus overlapped less with the range of the power-scaling surrogate data exponents than the near-random range of simulated resting-state exponents overlapped with that of surrogated data exponents.

Parenthetically, in departure from Palva et al. (2013) and Zhigalov et al., the DFA convention of Hardstone et al. (2012)’s Neurophysiological Biomarker Toolbox (https://www.nbtwiki.net/) utilised for the simulation data here yielded positive rather than negative $y$-values for each time window on the log-log plot denoting the (mean) log of the fluctuation function $\langle \log_{10}(F(\Delta t)) \rangle$. Exponents, the positive slopes of the log-log plot $\beta$ in Fig. 1E for the simulation data, are thus loosely comparable with Zhigalov et al. (2016)’s similarly positive slopes $\beta$ for actual data on such log-log plots.
This influence of neurofeedback block on the power-scaling of LRTCs, shown comparing simulated neurofeedback blocks with surrogate data (Fig. 1E), need not require critical branching, as Zhigalov et al. (2016) imply. Rather, this simulated influence of neurofeedback on LRTCs relies on a long-term dynamics emergent from flash-elicited complexes of deterministic ERPs and accumulating exponential oscillatory alpha gain spindles due to closed-loop flash neurofeedback stimulation during consecutive alpha oscillation peaks. The micro-behaviours (Fig. 1A) produced by mechanisms of exponential occipital oscillatory alpha gain and deterministic ERP generation have macroscale consequences influencing the power-scaling of LRTCs (Fig. 1B–C, 1E).

2.9. Simulating block-to-block changes in LRTC power-scaling

This discussion now turns to the block-to-block change in the influence of neurofeedback block on LRTCs. As apparent in the slopes of Fig. 1E, this change increases the power-scaling exponent of LRTCs’ exponents from $\beta_{B_1}$ to $\beta_{B_3}$: In Zhigalov et al.’s data, the mean $\beta_{B_1}$ (0.695) on the neurofeedback block 1 is near-identical to the mean exponent from the corresponding first sham block. By contrast, mean $\beta_{B_3}$ is higher on neurofeedback block 3 (0.671) than on the third sham block (0.647). As an aside, it is worth considering that significant changes in mean $\beta$, denoting power-scaling of LRTCs, are visible in the actual data (Zhigalov et al., 2016, Fig. 1B) from the first (0.695) to third sham blocks (0.647). This block-to-block change in temporal structure is neither attributable to neurofeedback nor to changes in the influence of deterministic ERPs nor to oscillatory alpha gain.

The model’s assumptions, centred on neurofeedback, are agnostic to these relatively longstanding confounding changes, apparent in the sham, which led to an attenuation in the power-scaling exponents of LRTCs from the first to the third neurofeedback block in the actual data. Candidate explanations of these longstanding changes might include the consequences of continued mere exposure to flashes on temporal structure rather than neurofeedback effects per se.

Given the actual $\beta_{B_1}$ was near-identical on the first set of neurofeedback and the first set of sham blocks, the empirically motivated simulation control, circumventing the confounding influence of these longstanding changes, should concern an unconfounded effect of neurofeedback block ($\beta_{B_3} > \beta_{B_1}$). This effect should, for definiteness, be as strong as the difference between the actual sham LRTC exponent on block 3 (mean: 0.647) and the actual neurofeedback LRTC exponent on block 3 (mean: 0.671), i.e., 0.024. The model surpasses this criterion (Fig. 1E), with an increase in mean LRTCs’ exponents for neurofeedback blocks 1 and 3 of 0.033. That is, the simulation model thus takes a tenable stance on how neurofeedback on the later third block boosts the power-scaling exponents of LRTCs in a way that the neurofeedback on the first block does not.

On balance, neurofeedback has more influence on the later block 3, as apparent in the averaged waveforms (Fig. 1D), because of a faster rise in exponential oscillatory alpha gain, as well as an additive increase in the amplitude of deterministic ERP componentry visible in Figs. 1C–D. On the later block, the simulations results reveal spindles of oscillatory gain superposed upon higher-amplitude deterministic ERPs, yielding volleys of reciprocal neurofeedback stimulation with a higher proportion of ineffective flashes in those volleys due to $\alpha_t$ more regularly exceeding the physiological maximum, $\theta_{max}$. When that maximum is thus exceeded, the ineffectiveness of those flashes to yield an increase in alpha only occasionally brings a neurofeedback stimulation reciprocal volley to an immediate closure. This ineffective flash still elicits a deterministic ERP, which is higher in amplitude than on the first block. That deterministic ERP, insensitive to whether the flash was effective or ineffective in producing an increase in oscillatory gain, reduces the chances of the next few peaks of $\alpha_t$, at the peaks of $O_t$, falling below the calibrated minimum $\theta_{min}$ for triggering a flash.
Just as when comparing neurofeedback blocks to surrogate data, considering neurofeedback blocks 1 and 3, the block-to-block change in power-scaling of LRTCs (Fig. 1E) need not require critical branching. Rather, the blockwise changes in long-term dynamics are emergent from several factors operating during the third block of closed-loop flash neurofeedback stimulation. These factors are flash-elicited complexes of higher-amplitude deterministic ERPs and the more rapidly accumulating exponential oscillatory alpha gain spindles, both of which occur during closed-loop stimulation upon consecutive alpha oscillations; that alpha thus accumulating up unto a physiological maximum.

2.10. Concluding remarks
In assessment, the micromodel simulates aspects of alpha-band continuous EEG (as depicted in Fig. 1A–C), alongside power-scaling of LRTCs (Fig. 1E), as well as the averaged data from neurofeedback blocks (Fig. 1D). These averages resemble Zhigalov et al.’s grand-averaged waveforms. This simulation not only relies upon flash-related exponential increases in oscillatory alpha gain but also relies upon deterministic ERPs (Ivannikov et al., 2009) that add constructively into complexes.

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