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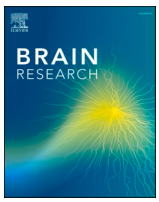

Considerations towards a neurobiologically-informed EEG measurement of sleepiness

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A B S T R A C T

Sleep is a daily experience across humans and other species, yet our understanding of how and why we sleep is presently incomplete. This is particularly prevalent in research examining the neurophysiological measurement of sleepiness in humans, where several electroencephalogram (EEG) phenomena have been linked with prolonged wakefulness. This leaves researchers without a solid basis for the measurement of homeostatic sleep need and complicates our understanding of the nature of sleep. Recent theoretical and technical advances may allow for a greater understanding of the neurobiological basis of homeostatic sleep need: this may result from increases in neuronal excitability and shifts in excitation/inhibition balance in neuronal circuits and can potentially be directly measured via the aperiodic component of the EEG. Here, we review the literature on EEG-derived markers of sleepiness in humans and argue that changes in these electrophysiological markers may actually result from neuronal activity represented by changes in aperiodic markers. We argue for the use of aperiodic markers derived from the EEG in predicting sleepiness and suggest areas for future research based on these.

We sleep because we have been awake for too long, and sleepiness is the biological signal that we need to sleep. That wakefulness drives sleepiness is an intuitive point, and one that is supported by theorical and empirical work on the mechanics of the homeostatic sleep drive, or Process S (Åkerstedt et al., 2004; Borbély et al., 2016; Borbély and Achermann, 1999; Dijk and Kronauer, 1999; Kronauer et al., 2007; Strogatz, 1987). Models of sleep regulation demonstrate that the control of sleep timing and intensity is determined by a combination of homeostatic, circadian and other (e.g., ultradian) factors. In terms of the homeostatic component of sleep regulation, the broad assumption is that, as we progress through hours of wakefulness, there is an accumulation of sleep need, which must relate to the accumulation of some neural by-product of wakeful activity and/or information processing (Lazarus et al., 2019; Thomas et al., 2020). Further, whatever the neural marker of sleep need is, it leads to worse performance in cognitive tasks (Belenky et al., 2003; Jewett and Kronauer, 1999). Sleep regulation models have led to advances in our understanding of the nature of sleep and, importantly, how sleep relates to cognition and performance. Despite this, there are several areas which have not been clarified; the clarification of which will help identify electroencephalographic (EEG) markers of Process S and help determine both the nature of sleep and sleepiness. First, how best to measure Process S with the EEG – there are numerous markers which have been proposed over decades of research (Shen et al., 2006), and very little attention has been paid to experimentally validating these against one another. Second, what these EEG markers are measuring, whether it be Process S directly, a by-product of wakeful activity or compensatory mechanisms resisting sleep. Third, how subjective sleepiness (self-rated perceptions of the current level of tiredness) or sleep propensity (self-rated perception of sleep onset likelihood) map onto objective, EEG-based measures of Process S. Fourth, *when* the optimal measurement of Process S in the EEG should occur – whether resting-state or task-related measurements are more optimal in this regard. Fifth, how and if circadian factors should be accounted for, and; sixth, how global or local one should expect an EEG-derived marker of sleepiness to be. It is acknowledged that the neural mechanisms linking Process S and sleepiness are not well understood and neither are the interoceptive signals that signify sleepiness. Mapping these will help further our understandings with potential benefits for the management of sleep disorders (especially when individuals are sleepy but do not experience sleepiness, as in obstructive sleep apnoea (Pak et al., 2019) and human performance. In this review, we will discuss the established literature on the use of EEG in measuring Process S, suggest the aperiodic component of the EEG as a novel, neurobiologically informed measure of sleepiness (see Fig. 1 for a schematic overview) and discuss

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the implications of this for our understanding of how to measure sleepiness in the EEG, and of sleep as a vital biological phenomenon.

Recent research suggests that human sleep is a strongly local phenomenon (Alfonsa et al., 2022; Ferrara and De Gennaro, 2011; Hung et al., 2013; Siclari and Tononi, 2017; Tsai et al., 2014; Vyazovskiy et al., 2011). Despite this, sleepiness and the measurement thereof is rarely considered a local phenomenon; studies have generally assumed that an EEG-derived sleepiness metric will be uniform across the cortex, and thus researchers have restricted themselves to using a frontal electrode or region of interest. The use-dependency and local nature of sleep has been a recent topic of interest in the literature and has been demonstrated in human and animal studies, as well as patient studies using intracranial EEG. These studies have demonstrated that sleep-specific EEG microstate phenomena can appear in the hippocampus during pre-sleep behavioural wakefulness and alongside 'awake' brain regions (Sarasso et al., 2014). While these results may indicate an opportunistic, wakeful consolidation process, due to the hippocampal locations sampled, other studies support the concept of local sleep by demonstrating use-dependent qualities of sleep EEG phenomena across cortex (Huber et al., 2013; Huber et al., 2006; Snipes et al., 2022). This is highlighted in classic arm immobilisation studies (Huber et al., 2006), in which the inability to use an arm (the arm being restrained in a sling),

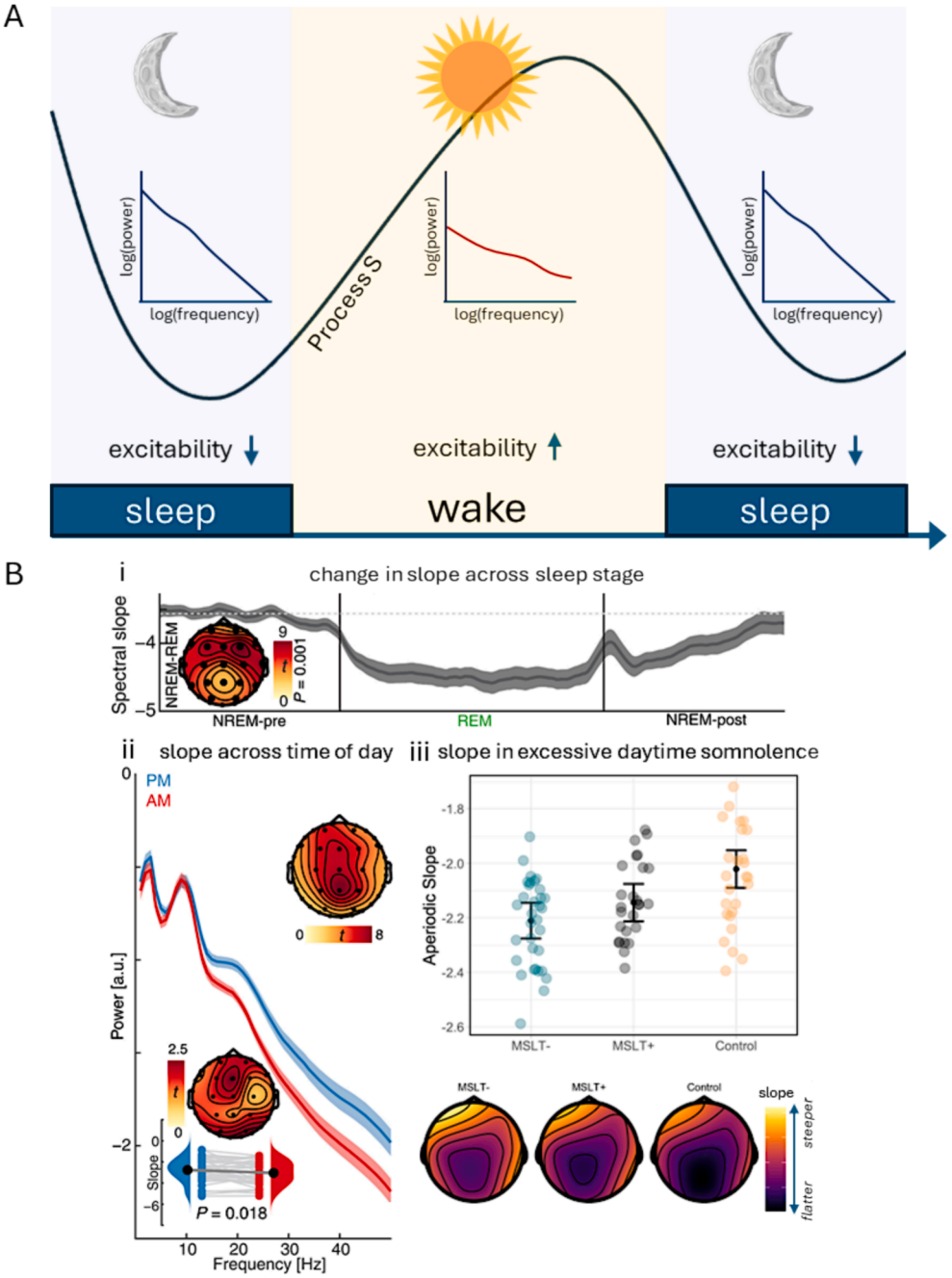

Fig. 1. Schematic overview of the relationship between the aperiodic slope and the accumulation of sleep need. (A) Process S is reflected by the decrease in sleep need across sleep, followed by the accumulation of sleep need during wake. During sleep, neuronal excitability decreases, reflected in a steeper aperiodic slope, while during wake, neuronal excitability increases, resulting in a flatter aperiodic slope. **(B)** Experimental evidence demonstrating sleep-related differences in the aperiodic slope. **(i)** change in the aperiodic slope across NREM and REM sleep stages[46]. **(ii)** time of day differences in the aperiodic slope, with the slope being flatter (i.e., increased high-frequency activity/neuronal excitability) in the afternoon relative to the morning[46]. **(iii)** Preliminary data from our group indicating differences in the aperiodic slope between those with and without excessive daytime somnolence (top) and the topographic differences in the magnitude of the slope (bottom).

results in less synaptic potentiation over the contralateral motor cortex and leads to a reduction in slow wave activity (SWA) over the same region in subsequent sleep. Results such as these highlight two important observations: sleep EEG appears to reflect local wakeful processes – sleep is therefore local (Ferrara and De Gennaro, 2011); and further, the sleep EEG appears to reflect the burden of information processing, learning and plasticity during previous wakefulness (Huber et al., 2013; Tononi and Cirelli, 2014). It is reasonable to put forth that sleepiness, as accrued sleep need in the individual, should follow these same considerations.

If sleep is both local and use-dependant, then so too is sleepiness. This has been demonstrated in the work of Snipes and colleagues (Snipes et al., 2022), who assessed both task and regional specificity of increased theta power as a marker of homeostatic sleep need in well-rested and sleep deprived individuals. It was observed that sleep deprivation led to the expected increases in theta power, but that these were generally over non-task related scalp regions, and that increased theta power did not relate to sustained or preserved task performance. These results are of interest in the context of older, magnetic resonance imaging (MRI) findings, which demonstrated compensatory frontal activation in preserving behavioural performance in a declarative memory task in conditions of sleep loss (Drummond et al., 2000). The major difference between these bodies of work being that previous studies found that non-task related brain regions were active in conditions of sleep loss, and this was related to preserved performance, whereas newer studies have noted that topographical activation appears to be unrelated to performance. That is, while there is indeed data to support local and use-dependent sleepiness, it is presently unclear as to where over the topography of the scalp measurement should be made to gain accurate measurement of current sleep need, given that the relationship between topography and objective sleep need may not be obvious and unrelated to the maintenance of performance (Snipes et al., 2022; Drummond et al., 2000; Balkin, 2011; Snipes et al., 2023). This issue is likely exacerbated by the field's present understanding of the functional significance of previously delineated EEG markers of sleepiness.

It is unclear what we are measuring when we try to quantify an objective marker of sleepiness. Not only are region-based differences in brain responses to sleepiness presently poorly understood, currently there is no data to help inform researchers as to whether a given marker of sleepiness is a measure of sleepiness in and of itself, or some compensatory process secondary (and naturally opposed to) to sleepiness, such as mechanisms to support continued functioning and information processing under the burden of sleepiness. Thus, while oscillatory markers have been shown to vary as a function of time awake and to tag the behavioural effects of sleep loss (Snipes et al., 2023), it is unclear what, precisely, is being measured to cause these effects. This is particularly relevant in the light of recent approaches and existing data, which suggest that sleepiness is the consequence of information processing in neural circuits (Snipes et al., 2022; Tononi and Cirelli, 2014). An answer to the functional meaning of established EEG-based markers of sleepiness would have important implications for our understanding of the nature of sleepiness and sleep in the brain. The evidence on this point is, however, currently mixed.

There is evidence to suggest that EEG-derived measures of sleepiness may measure the phenomenon directly – i.e., they may relate in some manner to neuromodulatory activity involving adenosine (Benington and Heller, 1995), BDNF (Kuhn et al., 2016) or chloride (Alfonsa et al., 2022) accumulation and can be separated from the effects of the circadian system (Cajochen et al., 2002). There is also evidence that such markers may be indirect measures and function through tagging compensatory processes (Snipes et al., 2022; Drummond et al., 2000). It is possible that both models may be correct in so far that homeostatic, use-dependent and circadian factors may interact to produce sleepiness, and therefore a neural marker may measure all or some of these processes in combination. This proposal is consistent with recent views which consider sleep need to arise from information processing and learning-related plasticity processes (Tononi and Cirelli, 2014). Taking such an approach may allow for a satisfying neurobiological basis for the phenomenon of sleepiness, while at the same time incorporating data around local, use-dependency related findings (Huber et al., 2013; Huber et al., 2006); however, it remains an open question as to whether and how circadian factors may be directly linked to plastic processes (Frank, 2012; Frank and Cantera, 2014), which would be important for this line of thought and stands as a testable idea for this proposed hypothesis on the nature of sleepiness. Of similar importance is the discovery of the optimal marker of these (combined or otherwise) processes.

Despite a lack of clarity as to what is (or should be) measured in the EEG to gauge sleepiness, a number of proposed markers have been published in the past three decades. Experimental support has been found for a role of all canonical frequency bands of brain activity as modulated by, or markers of, sleepiness (Aeschbach et al., 1997; Aeschbach et al., 1999; Cajochen et al., 1995; Dumont et al., 1999; Finelli et al., 2000; Kalauzi et al., 2012; Vyazovskiy and Tobler, 2005; Åkerstedt and Gillberg, 1990). A summary of these findings is presented in Table 1. There have also been findings in support of other elements of the EEG in relation to sleepiness, such as event-related potential (ERP) component amplitude changes (Hoedlmoser et al., 2011), microstates and phase locking values between various regions/frequencies (Comsa et al., 2019), informational complexity markers (Andrillon et al., 2020; Höhn et al., 2024) and aperiodic indices (Lendner et al., 2020; Lendner et al., 2023). While these markers can be used to measure sleep need, it is unclear as to their precise relationship with the phenomenon and they cannot inform us on the nature of homeostatic sleep need in humans, perhaps other than to suggest that sleepiness may be represented in a diffuse manner in the brain, at least in terms of objective measurement via the EEG. This is curious; if sleepiness is a local and use-dependent phenomenon arising from information processing, it might be expected that its effects would be more circumscribed and restricted in terms of the bandwidths and functions which are impacted. One intuitive solution to this is the idea that sleepiness impacts a single, fundamental function within the brain, which has subsequent, knock-on effects throughout the neural architecture, and could thus result in the discovery of numerous EEG markers of sleepiness.

One such possible underlying variable, which would influence all EEG measures that may reflect sleepiness is the excitation/inhibition (E/I) balance of neural firing, as measured by the aperiodic exponent of the EEG (Donoghue et al., 2020; Gao et al., 2017). The aperiodic neural signal has been suggested to be an objective marker of vigilance states and has been shown to differentiate between wakeful, NREM, REM and anaesthetised brain states (Lendner et al., 2020). Further, aperiodic measures can be neatly integrated into existing findings regarding the effects of sleepiness and prolonged wakefulness on the brain. For instance, time awake has been observed to relate to net increases in neural excitation (Huber et al., 2013; Kuhn et al., 2016; Vyazovskiy et al., 2008), and this is indexed by the aperiodic component of the EEG (Donoghue et al., 2020; Gao et al., 2017; Ahmad et al., 2022). Importantly, the aperiodic exponent is also related to the power distribution seen in the EEG, thus potentially linking it with, or potentially explaining, the established findings on low frequency EEG activity as modulated by sleepiness — changes in the aperiodic slope can shift band power estimates, but moreso, changes in the EEG due to aperiodic activity can be falsely attributed to non-existent oscillatory dynamics (i.e., there may be no actual oscillatory component above and beyond the aperiodic element of the EEG), as shown by Donoghue and colleagues (Donoghue et al., 2020). A particular demonstration of this detail was the analysis of resting-state alpha power recorded between younger and older adults, which broadly indicated that a failure to account for aperiodic activity has led to an overestimation of the effects of ageing on resting-state alpha power (Donoghue et al., 2020). In such a manner, not accounting for aperiodic activity in the measurement of sleepiness may have led to false positives, or overestimation of EEG effects. Thus,

Table 1
Narrative summary of oscillatory EEG findings related to sleep need.

Study	*n*	Intervention	Length (Hrs)	EEG Band	EEG Frequency (Hz)	Measurement Site	Key Finding(s)
Åkerstedt and Gillberg, 1990	8	CR	12	alpha, theta	8–12, 4–7.9	central, occipital	Positive relationship with subjective sleepiness
Cajochen et al., 1995	9	CR	40	theta/alpha	6.25–9	central	Positive relationship with subjective fatigue (VAS)
Aeschbach et al., 1997	9	CR	40	delta, theta, alpha	0.75–9.0	central	Global increasing trend with time awake, trough in evening
				alpha	9.25–12.0		Circadian modulation
				sigma, beta	12.25–25.0		Positively related to time awake
Aeschbach et al., 1999	19	CR	37–42	delta, theta, alpha	0.25–9.0	central	Positively related to time awake
				theta	4.25–8.0		Minimum 1 h after the onset of melatonin secretion
				alpha	10.25–13.0		Relationships with subjective alertness, melatonin, body temp
				sigma, beta	13.25—20.0		Positively related to time awake
Dumont et al., 1999	14	CR	38	delta, theta	2.00–7.75	central, occipital	Positive relationship with subjective sleepiness
				alpha	8.00–10.75		Positive relationship with core body temperature
				alpha, sigma	11.00–14.75		Positive relationship with subjective sleepiness
				beta	18.00–24.75		Positive relationship with subjective sleepiness
Finelli et al., 2000	8	CR	40	theta	5–8	global	Positive relationship with SWA power
Vyazovskiy and Tobler, 2005	14	SD	6	theta	5–7		Positive relationship with SWA power
				delta, low theta	1.5 – 4, 5 – 6.5		Positively related to time awake
Hoedlmoser et al., 2011	20	SD	24	delta, theta	1–4	global	Positively related to time awake
				P1 amplitudes			Negatively related to time awake
				delta/theta PLI	1–4,4–8		Negatively related to time awake
Comsa et al., 2019	16	SR	−1 to −2	alpha, theta	9.5–10.5, 5–6 Hz	global	Tracks progression from responsiveness to unresponsiveness

Note: CR = constant routine; SD = sleep deprivation; SR = sleep restriction; subjects in Vyazovskiy & Tobler, (2005) were rats; values for intervention length in Comsa et al. (2019) represent hours less than usual amount of sleep.

aperiodic metrics are linked to both brain state and between-population differences and should be accounted for in fully understanding electrophysiological activity. Whether established findings in terms of the EEG-based measurement of sleepiness can be recast in the light of aperiodic measures remains an important question for future research.

The aperiodic slope may be relevant for understanding sleepiness, in that it may resolve discrepancies around local and use-dependent aspects of sleep and sleepiness, either directly, or through an influence on oscillatory EEG markers of sleepiness. Topographic data indicate the existence of a flattening of the aperiodic slope from posterior to anterior scalp locations, broadly indicating a shift towards increased excitability in frontal regions of the brain (Donoghue et al., 2020); this is in line with the common frontal measurements used in sleepiness/EEG research to date. Further, the use of aperiodic measures can be informed by established work using single-unit readings of basic neuronal functions as markers of sleep need in humans (Thomas et al., 2020). Broadly, Process S dynamics can be read from neuronal firing rates and off periods, and this allows for the calculation of local estimates of Process S, as well as the averaging of these to approximate global Process S values. From this arises the idea that the transition to sleep may occur when a sufficient number of local areas exceed a threshold, leading to a global (i.e., behavioural) sleep state. These results should be extended to the study of subjective and objective metrics of sleepiness and sleep propensity. Such an endeavour is timely and practical, given that the same logic demonstrated in Thomas et al. (Borbély et al., 2016), can also be applied to aperiodic measurement of sleepiness, and may move the field away from ubiquitous frontal site measurement as a preferred site of measurement of sleepiness using EEG in established research, and the theoretical importance of non-REM SWA as a marker of sleep intensity. Frontal sites are necessarily impacted by aperiodic activity, as is SWA, and thus, there may be a latent factor here of aperiodic factors present in established data on the EEG-based measurement of sleepiness, and there may have been so for some time. Classic studies (Cajochen et al., 1995; Åkerstedt and Gillberg, 1990) should therefore be reproduced with an intent to disentangle oscillatory and aperiodic EEG components as they may relate to sleep need and subjective sleepiness, as well as to extend the established literature, for example, by determining the existence of circadian effects in aperiodic measures, and whether these are important factors to consider in using EEG to measure current sleep need in the awake individual.

There is currently no direct evidence of circadian effects in the aperiodic signal, although a relationship is likely, based on the interrelationships between aperiodic slope, PSD oscillatory power and the circadian modulation of neuronal activity (Frank, 2012; Frank and Cantera, 2014). It should be noted that early studies using the continuous running paradigm (Cajochen et al., 2002; Cajochen et al., 1995) noted both circadian and homeostatic modulations of low-frequency EEG power – in particular, in the alpha, theta and beta bands, and other studies also note circadian effects in delta and elements of SWA (Lazar et al., 2015). Subjective reports on experienced sleepiness tracked, but did not precisely match, these relationships. There is a substantial correlation between alpha power and the aperiodic slope, and between alpha and theta power (Donoghue et al., 2020), and this may be of particular interest in the current context, given that alpha and theta power have been seen as major markers of the accumulation of sleep need (Chia et al., 2021; Kovrov et al., 2018). This shared explanatory power may suggest circadian effects in aperiodic networks. Further, animal studies using invasive EEG have provided evidence that links circadian factors with the E/I balance of neuronal activity, although these findings need to be replicated in humans with sensor-level measurements and are not without inconsistencies. For example, Vyazovskiy et al. (Vyazovskiy et al., 2008) found that cortical evoked responses in sleep deprived mice were altered as a function of prior sleep/wake history, independently of circadian influences. These observations are in contrast to those of Bridi and colleagues (Bridi et al., 2020), who noted modulations of E/I balance across 24hr cycles, consistent with circadian effects. Regardless, both Vyazovskiy and Bridi note a function of sleep in upregulating inhibition (or LTD) in cortical circuits, and that local effects were present, in that E/I balance changes across circadian phases were not consistent across cortical areas. Thus, animal models exist that link the neuronal behaviours measured via the aperiodic slope with sleep and potentially, with the objective

measurement of sleepiness in humans. Future research is needed to determine the extent to which these classic findings may replicate in humans with scalp-measured aperiodic activity, as opposed to invasive EEG. Future research is also needed to determine if there is a latent factor of circadian phase in both the measurement of aperiodic slope, and the relationship between this and sleepiness.

It should also be noted that recent works in the area of neuromodulators and sleep need may enable an interesting analysis with the potential to inform the ideas presented in this review. Standard approaches have focussed on the role of adenosine, which is generally considered the major somnogen in the brain (Lazarus et al., 2019). With increasing time spent awake, adenosine accumulates and inhibits the inhibition of sleep promoting networks to initiate NREM sleep (Pace-Schott and Hobson, 2002). Recent works have provided further detail on this idea by examining the role of intracellular chloride (as a modulator of neuronal excitability) in linking prior sleep/wake history with subsequent sleep need and sleep electrophysiology (Alfonsa et al., 2022). Broadly, this influence may occur through the modulation of synaptic inhibition. Alfonsa et al. found that wakefulness in mice led to greater depolarisation in pyramidal neuron synaptic $EGABA_A$ (i.e., weaker inhibition). These shifts were found to be related to increased SWA in NREM sleep, a canonical marker of homeostatic sleep need. Depolarized $EGABA_A$ may enhance the recruitment of voltage-gated potassium ions and this may contribute to the oscillatory activity associated with sleep. It is striking that the mechanisms and outcomes noted here (i.e., neuronal E/I balance and electrophysiological markers of sleep need) are shared between this work and our ideas around the aperiodic component of the EEG. The two could be seen as potentially complimentary and could mutually inform one another. Future work is needed to assess the impacts of neuromodulators on the aperiodic EEG in general, and doing so may provide an important validation or disproval of the aperiodic signal as a biomarker of homeostatic sleep need in humans.

If a relationship can be convincingly delineated between aperiodic indices and sleepiness, it will provide a relatively straightforward, objective and neurobiologically-informed measure of sleepiness. The direct mapping of the aperiodic component to behavioural states is a relatively new development. Importantly, recent experimental work in sleep disordered patients has demonstrated that the aperiodic component of the EEG can be used to classify insomnia patients from non-insomniac controls (Andrillon et al., 2020). This can be seen as an applied extension of the work of Lendner et al. (Lendner et al., 2020; Lendner et al., 2023), who found that aperiodic measures could be used to differentiate between wakeful and sleep states, as well as between different sleep stages within the individual. Further, in line with predictions made in the Synaptic Homeostasis Hypothesis (Tononi and Cirelli, 2014) and experimental data supporting the theory (Vyazovskiy et al., 2008; Vyazovskiy et al., 2009), neuronal excitability has been shown to reduce across a night of sleep, and sleep deprivation was shown to reduce the homeostatic regulation of neuronal excitability. Thus, aperiodic measures of brain activity may not only be useful in quantifying sleepiness; they may do so by tagging fundamental and basic neural properties. Aperiodic measures may also be capable of differentiating clinical and non-clinical cases based on this fundamental property, and, as such, be important tools in furthering basic research into sleep and sleep disorders involving disruption of the homeostatic sleep drive.

While the investigation and application of the aperiodic component of the EEG has great potential in better understanding the brain, it should be acknowledged that the measurement of these components is a relatively recent development in cognitive neuroscience. It is generally assumed that the best practices for recording the aperiodic signal are the same as those for EEG in general. The precise EEG bandwidth to use in calculating the aperiodic signal is relatively unclear; however, it should be noted that use of different bandwidths may have different implications for which neuronal processes are captured. Seminal work linking aperiodic activity with E/I balance (Gao et al., 2017) did so using a bandwidth of 30–45 Hz, and this methodological detail is sometimes overlooked, with related work using a bandwidth of 1–30 Hz. In general terms, a 1–30 Hz bandwidth is more likely to involve an influence of the low-frequency oscillatory component of the EEG, whereas a 30–45 Hz bandwidth is more likely to reflect excitation in aperiodic activity with reduced influence of low frequency oscillatory activity. Further research is required to understand the influence of different researcher choices such as these in what is measured by the aperiodic component, and individual researchers will have to be careful that their choices are reasonable and justified, based on an understanding of the mechanisms and physiology involved. Here, we have argued that sleep need may result from changes in the E/I balance across hours of wakefulness. As such, a narrowband estimate (30–45 Hz) may be more appropriate to use for the detection of sleep need. A broadband (1–30 Hz) range may also show effects, but these may be due to the influence of low-frequency oscillatory activity in the delta and theta bands. This highlights an important methodological consideration in this area, which should be directly examined in future work.

Taken together, there is a solid body of evidence that suggests that aperiodic metrics of brain function in the EEG may be useful in understanding sleep as a biological phenomenon, and sleepiness as an important biological signal. Here, the use of the aperiodic signal is argued for and it is noted how aperiodic neural activity may explain elements of the literature on sleepiness and the EEG — mostly around the existence of circadian, local and use-dependent effects therein. It must also be noted that physiological and computational accounts posit a tight relationship between oscillatory and aperiodic phenomenon, such that aperiodic activity influences oscillatory activity and vice versa (Donoghue et al., 2020; Gao et al., 2017; Gao et al., 2020). Thus, it is an open question as to whether classic, established findings regarding resting-state, low-frequency activity as predictors of sleepiness in humans could be reconceptualised as resulting from changes in aperiodic activity. Further, it should be noted that studies seeking EEG-derived markers of sleepiness have typically, but not always, analysed tonic/resting-state, non-task related EEG (Cajochen et al., 2002; Cajochen et al., 1995; Achermann et al., 1993). However, there is also evidence to suggest that sleep loss may influence task-related EEG activity (Drummond et al., 2000; Hoedlmoser et al., 2011). That is, whether aperiodic measures can be meaningfully applied to task-related EEG in the context of preserving cognitive functioning in conditions of sleep loss may also be of interest to researchers, particularly in terms of avoiding or in monitoring against these (Balkin, 2011). Similarly, use-dependency and circadian effects on the aperiodic signal as it relates to homeostatic sleep pressure would seem to be an important future direction for research. Such research is not only important in increasing knowledge of sleep and the homeostatic sleep drive in the active human but is also a deeply practical affair – objective measurement of sleepiness is, ironically, onerous. Obtaining it has the potential to improve both scientific rigour, medical care, and industry safety.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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