1   # A meta-analysis of periodic and aperiodic M/EEG components in Parkinson's
2   # disease
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Abstract

Parkinson's disease is characterised by a range of motor and non-motor changes that can negatively impact quality of life. Many studies have identified potential clinical electrophysiological biomarkers of Parkinson's disease with an aim of developing new methods of identifying at-risk patients, and to form the basis of therapeutic interventions. However, these studies do not present consistent results, and a formal meta-analysis is warranted to identify reliable M/EEG characteristics across datasets. In this meta-(re-)analysis of open-access M/EEG datasets (n = 6; 4 EEG and 2 MEG), we compared periodic and aperiodic characteristics of resting-state recordings in 368 patients with Parkinson's disease and 570 age-matched healthy controls. Specifically, we compared the power and peak frequency of the aperiodic-adjusted alpha and beta oscillations, and the aperiodic exponent and offset across the two groups. Using spectral parametrisation, individuals with Parkinson's disease had higher alpha-band power and a slower alpha peak frequency compared to controls, however no group differences in beta-band power and peak frequency were identified in this resting state data. Parkinson's patients were furthermore found to have consistently higher aperiodic offset and exponent, possibly indicative of increased cortical inhibition. In conclusion, this large cohort meta-analysis points to a broadly consistent pattern of both periodic and aperiodic changes in Parkinson's patients in M/EEG signal that may be used to develop diagnostics and targeted interventions in the future.

1. Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder caused by a loss of dopamine-producing neurons in the substantia nigra, which plays a critical role in motor control. PD primarily affects the execution of smooth movement, and is characterised by tremors, rigidity, bradykinesia and postural and gait impairments (Vinding et al. 2024a). Yet PD is also a multi-faceted disorder and is often accompanied by a range of non-motor symptoms, including cognitive decline, fatigue, apathy, visuospatial disturbances and sleep disruption (Kalia and Lang 2015; Vinding et al. 2024a; Yassine et al. 2023).

Electrophysiological recordings have identified specific changes in cortical oscillatory activity in patients with Parkinson's disease, particularly within the theta (4-7 Hz), beta (13-30 Hz) and gamma (40+ Hz) bands (Foffani and Alegre 2022). One of the most consistent markers of "oscillopathy" in PD is the beta activity associated with motor preparation and execution. During active movement in healthy individuals, beta power gradually desynchronises in anticipation of an action, reaches its lowest point during movement execution, and then rapidly rebounds to a level above baseline after the movement ends (Szul et al. 2023). In contrast, this pattern is disrupted in PD patients, with the beta desynchronisation before and during movement execution diminished, and the post-movement beta rebound also reduced in magnitude (Boon et al., 2019; Heinrichs-Graham, Wilson, et al., 2014; Mustile et al., 2023).

Tonically high levels of beta have also been observed at rest in Parkinson's disease and these elevated periods of beta have been directly associated with motor dysfunction in the form of bradykinesia (Zavala et al. 2015) and freezing of gait (Toledo et al. 2014). This has been corroborated by invasive electrophysiology which has identified elevated resting-state beta activity within the basal ganglia nuclei and cortex (Eisinger et al. 2020; Thomas Binns et al. 2025; Tinkhauser, Pogosyan, Little, et al. 2017a; Tinkhauser, Pogosyan, Tan, et al. 2017). However, the literature is mixed, with non-invasive resting state studies variously describing higher beta activity in PD patients relative to controls (Cao et al. 2020; McKeown, Jones, et al. 2024a), lower beta activity (Gimenez-Aparisi et al. 2023; Jaramillo-Jimenez et al. 2023a; Olde Dubbelink et al. 2013), and no difference between patients and controls (Vinding et al. 2024b; Wang et al. 2022a; Wiesman, Madge, et al. 2025a). Further, dopaminergic medication appears

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84   to effectively suppress beta activity at the cortical and subcortical levels (Binns et al. 2025).

85   Thus, although beta oscillations may show some promise as a potential biomarker to dissociate

86   PD from controls, this potential is limited due to its sensitivity to dynamic physiological and

87   pharmacological conditions.

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89   In contrast, the lower oscillatory frequencies, particularly within the alpha band (8-12 Hz) have

90   been explored less often in Parkinson's disease. In healthy adults, alpha rhythms typically reflect

91   decreased excitability of the underlying cortex and are modulated by both tonic and selective

92   attention (Jensen 2024; Thut et al. 2006). Several studies have reported an increase in alpha

93   power and decreased peak frequency in PD, which appear to be positively associated with non-

94   motor disease symptoms, specifically cognitive decline (Anjum et al. 2024; Olde Dubbelink et

95   al. 2013; Özkurt 2024; Sinanovic et al. 2005; Wiesman et al. 2023a; Wiesman, Madge, et al.

96   2025b; Yassine et al. 2023; Ye et al. 2022). However, this pattern is also inconsistent, with others

97   reporting lower alpha, or no differences between patients and healthy controls (Bosboom et

98   al. 2006; George et al. 2013; Jaramillo-Jimenez et al. 2023b; Mikkel Vinding et al. 2022).

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100   Electrophysiological signals reflect a combination of periodic (oscillatory) and aperiodic

101   components, which are postulated to arise from distinct neural mechanisms (Donoghue et al.

102   2020; Hughes et al. 2012; Wen and Liu 2016). The aperiodic component of electrophysiological

103   signals carries potential physiological significance and exhibits dynamic modulation in response

104   to factors such as age, cognitive state, and task demands (Donoghue et al. 2020). The aperiodic

105   component can be characterized by two parameters; the exponent is equivalent to the negative

106   slope of the power spectrum, and the offset reflects the uniform shift of power across

107   frequencies (Donoghue et al. 2020; Hughes et al. 2012; Wen and Liu 2016). The aperiodic

108   exponent has been linked to the balance and integration of underlying excitatory and inhibitory

109   synaptic currents (E/I ratio) (Martínez-Cañada et al. 2023; van Nifterick et al. 2023), while

110   changes in the offset reflect shifts in the broadband signal, particularly at lower frequencies

111   (Manning et al. 2009; Miller et al. 2014).

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113   Recent advancements in electrophysiological signal analysis have made it possible to detect

114   changes in the excitation-inhibition ratio, which may be detectable during (and potentially in

115   the prodrome of) disease processes. A link between the aperiodic exponent and the E/I ratio

116   in the brain is frequently reported (Martínez-Cañada et al. 2023; van Nifterick et al. 2023).

117   Ketamine, by blocking NMDA receptor-mediated input to inhibitory interneurons, induces

118   cortical disinhibition and increases glutamate release (Grunze et al. 1996; Maccaferri and

119   Dingledine 2002). In contrast, thiopental, a $GABA_A$ receptor modulator, suppresses cortical

120   excitation (Pittson et al. 2004). Both drugs have been shown to modulate the aperiodic

121   exponent in healthy individuals (Cortes-Briones et al. 2025). However, other pharmacological

122   manipulations of inhibitory neural mechanisms have failed to support the hypothesis that the

123   aperiodic exponent reliably reflects the E/I ratio in electrophysiological activity (Salvatore et al.

124   2024).

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126   Both non-invasive and invasive electrophysiology in humans and animals have identified

127   increased synchronised neural activity in PD patients (reflected by greater offset) as well as

128   elevated spontaneous neuronal activity in the basal ganglia and in the cortical level, which shifts

129   the balance of the normal E/I ratio (indicated by larger exponent, i.e. steeper slope in the power

130   spectrum) in the brain (Donoghue 2024; Filion and Tremblay 1991; Helson et al. 2023;

131   Rosenblum et al. 2023; Wang et al. 2022b; Wiest et al. 2023). Such increases in the aperiodic

132   exponent and offset in PD patients relative to controls have been documented (McKeown,

133   Jones, et al. 2024b; Vinding et al. 2024a; Wiesman et al. 2023a). However, several studies have

134   reported either a reversed pattern (Mostile et al. 2019; Peng et al. 2024; Wu et al. 2023) or no

135   significant differences in aperiodic offset and exponent between individuals with Parkinson's

136   disease and healthy controls (Bernasconi et al. 2023a; Clark et al. 2023; Martin et al. 2018;

137   Monchy et al. 2023; Pardo-Valencia et al. 2024).

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139   A recent advancement in spectral parameterisation of M/EEG data has highlighted that

140   previous studies of group differences in periodic oscillatory power may be confounded by

141   changes in aperiodic electrophysiological components (Donoghue et al. 2020; Kopčanová et al.

142   2024). By first extracting the offset and exponent separately from periodic components, we aim

143   to identify more subtle changes in cortical activity that may point to more consistent and

144   reliable biomarkers of PD that have previously been obscured.

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146 Collectively, these findings suggest the need to identify consistent patterns of altered cortical
147 electrophysiology associated with Parkinson's disease to develop novel biomarkers to diagnose
148 PD, predict its onset, and to formulate more effective treatments. In this study, we leverage
149 large-scale open-source M/EEG resting-state datasets in patients with Parkinson's disease and
150 healthy controls. We applied a single, custom processing pipeline to perform a spectral
151 parameterisation re-analysis of the raw datasets, followed by 6 meta-analyses to synthesise the
152 evidence for between-group differences in the power and peak frequency of aperiodic-
153 adjusted alpha and beta oscillations, and the aperiodic exponent and offset between PD
154 patients and healthy controls.

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2. Methods

2.1. Study Selection

158 Studies were identified through electronic databases, including bioRxiv, medRxiv, PsyArXiv,
159 PsycINFO, PubMed, ScienceDirect, Scopus, and Web of Science from inception until September
160 2024. The search terms used to identify the datasets were ("Parkinsons's disease" OR
161 "Parkinsons" OR "Parkinsonian" OR "Parkinson") AND ("resting state" OR "resting-state" OR
162 "rest state" OR "rest-state" OR "rest") AND ("EEG" OR "MEG" OR "electroencephalogram" OR
163 "magnetoencephalogram"). We included datasets where raw resting-state EEG or MEG was
164 available from both human Parkinsonian patients and healthy controls. A total of 226 studies
165 were identified (Figure 1). After removing duplicates, 159 unique records were screened and
166 123 records were excluded for the following reasons: did not include electrophysiological signal
167 recordings, described animal models, recorded deeper brain structures using ECOG, LFP, or
168 DBS, and investigated mild traumatic brain injury, multiple sclerosis, or Parkinson's disease
169 patients during sleep. Of the 36 remaining records, 13 were further excluded because they:
170 only included patients with no control group, did not include resting-state data (Agouram et al.
171 2024; Bernasconi et al. 2023b; Bringas et al. 2020; Cockburn et al. 2014; Lee et al. 2023;
172 Pellegrini et al. 2024; Rassoulou et al. 2024; Singh et al. 2018; Stuart et al. 2019; Wang et al.
173 2022c; Xefteris et al. 2020; Zhang et al. 2022a; Zimmermann et al. 2015), and 17 records were
174 not retrieved (did not allow data sharing or did not respond to our data-sharing requests).
175 Ultimately, resting-state data from six publicly available studies were included: San Diego
176 (Rockhill et al. 2020), Turku (Railo, 2021), New Mexico (Cavanagh, 2021), and Iowa (Singh et al.

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177 2023) and 2 were shared with us upon request: OMEGA (Niso et al. 2016) (the original report

178 from OMEGA dataset that we refer to is (Wiesman et al. 2023a), and NatMEG (Vinding et al.

179 2023a). Details of the searches are illustrated in Figure 1.
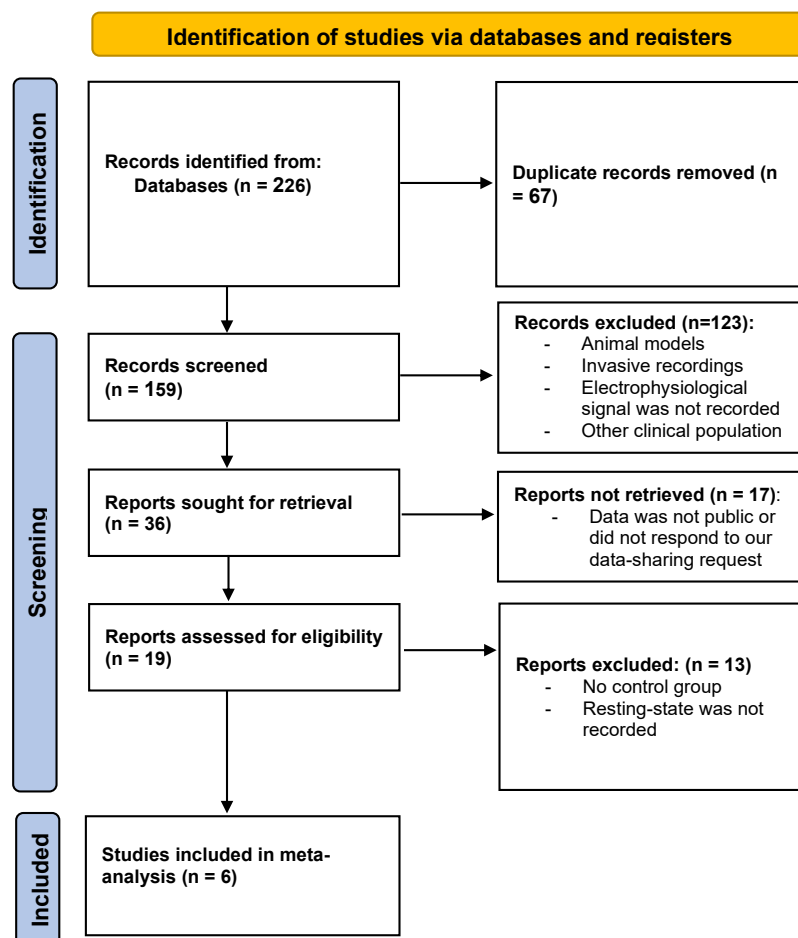

Figure 1. Flow diagram documenting the electronic database searches, abstract screening, and the reasons for excluding studies. Adapted from Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Page et al., 2021).

185 The datasets entered into the pre-processing stage involved a total of 1092 participants, of

186 which 426 participants were PD patients, and 666 were healthy aged-matched control

187 individuals. The San Diego, Turku, Iowa, and New Mexico datasets were recorded using EEG

188 with various numbers of electrodes, and the OMEGA and NatMEG datasets were recorded

189 using MEG. The San Diego, Turku, and New Mexico datasets recruited patients both in ON and

190 OFF medication states, however, we only included recordings for the ON medication state in

191 this analysis. The characteristics of each dataset are presented in Table 1 and are described

192 below.

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Dataset 1: San Diego, USA (Rockhill et al. 2020)

16 PD patients (8 women, mean age = 62.6 years, SD = 8.3) and 16 healthy controls (9 women, mean age = 63.5, SD = 9.6) were recorded. All patients had mild to moderate PD severity (Hoehn and Yahr stage II and III). PD patients were recorded both ON and OFF medication. EEG was recorded for at least 3 minutes using a 32 channel ActiveTwo (Biosemi Instrumentation system) using a sampling rate of 512Hz while participants kept their eyes open and maintained fixation.

Dataset 2: Turku, Finland (Railo, 2021)

Twenty PD patients (10 women, mean age = 69.5, SD = 7.7) and twenty healthy controls (12 women, mean age = 67, SD = 6.1). EEG was recorded for at least 2 minutes using 64 active electrodes with a sampling rate of 500Hz during eyes open and eyes closed. Twelve patients were recorded OFF medication, and five patients were recorded ON medication but only the eyes open, ON medication recordings were selected for the meta-analysis.

Dataset 3: Iowa, USA (Singh et al. 2023)

132 PD patients (43 women, mean age = 68.1, SD = 7.98) and 49 healthy controls (23 women, mean age = 70.91, SD = 7.62) were tested. 64 channel EEG was recorded with eyes open for at least 2 minutes with a sampling rate of 500Hz. All patients were tested ON medication.

Dataset 4: New Mexico, USA (Cavanagh, 2021)

25 PD patients (9 women, mean age = 69.68, SD = 8.73) and 25 age- and sex-matched controls (9 women, mean age = 69.32, SD = 9.58) were tested. EEG was recorded for around 9 minutes with 64 Ag/AgCl electrodes with a sampling rate of 500 Hz during both eyes closed and eyes open. PD patients were tested both ON and OFF medication. The eyes open, ON medication data was used in the meta-analysis.

Dataset 5: OMEGA, Canada (Niso et al. 2016)

166 PD patients (46 women, mean age = 64.95, SD = 9.12) and 488 healthy controls (307 women, mean age = 44.42, SD = 20.83) were tested. At least 5 minutes eyes open resting-state data was recorded using a 275-channel whole-head CTF MEG system at a sampling rate of 2400

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224   Hz and with an antialiasing filter with a 600 Hz cut-off. (Note: The OMEGA dataset is

225   continuously expanding, with new participants, both patients and healthy controls, are being

226   regularly added. In the original report, the dataset comprised 79 individuals with Parkinson's

227   disease and 65 healthy controls (Wiesman et al. 2023a).

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229   **Dataset 6: NATMEG, Sweden** (Vinding et al. 2023b)

230   66 PD patients (28 women, mean age = 65.6, SD = 9.5) and 68 healthy controls (27 women,

231   mean age = 63.93, SD = 8.4) were tested. At least 3 minutes of resting-state data was recorded

232   using a Neuromag TRIUX 306-channel MEG system, with 102 magnetometers, with 1000 Hz

233   sampling rate, with an online 0.1 Hz high-pass filter and 330 Hz low-pass filter while the

234   participants sat with their eyes closed. The average head movements were not significantly

235   different between groups ($t(115.8) = 0.55$, $p = 0.58$).

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248   Table *1*. Characteristics of data cohorts assessing resting-state EEG/MEG in Parkinson's patients and healthy controls.

DATASET	Total participants	Participants included in meta-analysis	M/EEG	Resting state recording duration (min)	PD Age	PD Sex	Mean PD duration (years)	Mean H&Y	Mean UPDRS score (ON medication)	HC Age	HC Sex	SpecParam analysis performed
San Diego	16 PD, 16 HC	13 PD, 10 HC	EEG	> 3	62.6 (8.3)	8F	4.9 (3.6)	-	33.7$^{⚜}$ (10.9)	63.5 (9.6)	9F	No
Turku	20 PD, 20 HC	6 PD, 20 HC	EEG	> 2	69.5 (7.7)	10F	6 (5.0)	-	23.0* (10.2)	67 (6.1)	12F	No
Iowa	132 PD, 49 HC	115 PD, 42 HC	EEG	> 2	68.1 (8.0)	43F	4.1 (3.5)	1.45	12.5* (7.2)	70.9 (7.6)	23F	No
New Mexico	27 PD, 25 HC	24 PD, 23 HC	EEG	≈ 9	69.7 (8.7)	9F	5.4 (4.1)	-	23.4* (9.9)	69.3 (9.6)	9F	Yes
OMEGA	166 PD, 488 HC	154 PD, 411 HC	MEG	≥ 5	65.0 (9.1)	42F	6.8 (4.3)	1.96 (0.7)	32.7* (14.8)	44.4 (20.8)	305F	Yes
NatMEG	66 PD, 68 HC	56 PD, 64 HC	MEG	≥ 3	64.4 (9.8)	22F	4.0 (3.4)	1.63 (0.77)	19.3$^{⚜}$ (11.1)	63.3 (8.7)	31F	Yes

249   *(⚜) UPDRS overall score, (***) UPDRS-III scores. PD = Parkinson's disease, HC = Healthy controls, F = females, UPDRS = Unified Parkinson's Disease Rating Scale,*

250   *H&Y = Hoehn and Yahr Score.*

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2.2. Preprocessing

The OMEGA, NatMEG and New Mexico datasets had previously been subjected to spectral parameterisation (Table 2; reported in the respective source publications), but for the purposes of this study we developed a single processing pipeline in order to re-analyse the 6 datasets using a single set of common parameters. Each of the datasets were first re-sampled to a uniform sampling rate of 500 Hz, then filtered between 1-100 Hz using a zero phase Infinite Impulse Response (IIR) filter and detrended to remove slow drifts. Independent component analysis (ICA) was applied to the filtered signal and non-brain components were excluded automatically using MNE-ICALabel (Li et al. 2022) for the 4 EEG datasets, and empty-room signal was used to reduce environmental noise in the 2 MEG datasets. The reference channels were available for the OMEGA dataset and were used to identify non-brain components. Both gradiometer and magnetometer channels were available for the NatMEG dataset, and only magnetometers were available in the OMEGA dataset, therefore only magnetometer channels were used for analysis of both MEG datasets. Next, the pre-processed signal was segmented into 1 second epochs. Each dataset resulted in different number of epochs due to the variable length of the recordings. Due to poor signal quality or data corruption, 9 subjects from San Diego dataset, 23 subjects from Iowa, 5 subjects from New Mexico, 45 subjects from OMEGA, and 11 subjects from NatMEG were excluded. A total of 938 subjects (368 PD patients and 570 controls) were included in the final spectral parametrisation stage.

Table 1. Comparison of SpecParam settings between previously parametrised datasets and the present meta-analysis.

Study	Frequency range (Hz)	Peak width limit (Hz)	Max number of peaks	Minimum peak height	Peak threshold	Modelling mode
OMEGA	2 - 40	0.5 – 12	3	3	2	Fixed Linear
NatMEG	0.5 - 40	0.75 – 12	8	0.05	2	Fixed Linear
New Mexico	2 - 40	1 – 8	8	0.1	2	Fixed Linear
Present meta-analysis	3 - 40	0 - 12	7	0.1	1	Fixed Linear

2.3. Spectral parameterisation

The power spectrum of each epoch was estimated using Welch's method with a segment length of 5000 samples (10 * sampling rate), 50% overlap, and a Hanning window. Power spectrum signal was then averaged over 3 different regions of interest involving the Frontal, Central, and Posterior electrodes, as well as an average across all electrodes, referred to as the AllScalp region. Next, the power spectrum of each region was parameterized using the *specparam* toolbox (Donoghue et al. 2020) in MNE python (Larson et al. 2024) using the following settings: peak width limits ([0, 12]), peak threshold (1), aperiodic mode (fixed), maximum number of peaks (7), and minimum peak height (0.1). Each of the previously parameterised datasets had examined different frequency windows (from a lower bound of 0.5, 2 or 3Hz to an upper bound of 40Hz; Table 2) and we therefore parametrized the spectra across several broad and narrow frequency ranges to identify the optimal frequency windows and spatial locations for comparison across studies (see Supplementary Data). Through this process, we selected 3-40 Hz for the common pipeline, and the data was collapsed over the full scalp before being entered into the meta-analysis.

Error metrics were used to identify and exclude samples with poor model accuracy. For each data cohort, the model fit was obtained and the z-score of the model accuracy distribution was then computed to detect and exclude subjects with poor model performance. As a result, the following participants were excluded at this stage: 1 PD patient from the San Diego dataset, 2 healthy controls from the Turku dataset, 4 PD and 2 HCs from Iowa, 1 PD and 2 HCs from New Mexico, 5 PD and 14 HCs from OMEGA, and 3 PD patients from NatMEG. The number of subjects included in the final meta-analysis from each dataset is shown in Table 1. After removing outliers, the model accuracy had a mean fit of 97.04% (range = 96.54- 97.8) for the PD group and 97.38% (range = 96.98- 97.7) for the healthy controls.

The unadjusted and adjusted spectral plots were used to select the frequencies of interest for meta-analysis (Figure 2). We observed clear peaks within the alpha (7-12 Hz) and beta (12-30 Hz) frequency windows, but no peaks were identified within the theta or gamma bands. Thus, the peak adjusted alpha and beta power, and their respective peak frequencies, were entered into the meta-analysis, together with both aperiodic components, i.e. offset and exponent.

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2.4. Meta-analysis

309 The following features extracted from each dataset and were used in the meta-analysis: alpha
310 power, alpha peak frequency, beta power, beta peak frequency, exponent, and offset. The
311 *metafor* (Viechtbauer, 2007) and *robumeta* (Fisher et al., 2017) packages for R were used to
312 perform the meta-analysis. First, the standardized mean difference and sampling variance were
313 calculated, using the *escalc* function, between Parkinson's patients and healthy controls,
314 separately for each of the 6 components. Random effects models were then run for each
315 component and mean weighted effect sizes and 95% confidence intervals were estimated using
316 restricted maximum likelihood (REML). Heterogeneity was assessed using the Q-statistic, I^2, R^2
317 and tau^2 indices. Forest plots were used to visualise the standardised mean difference across
318 datasets.

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3. Results

321 A total of 6 datasets involving 368 patients with Parkinson's disease and 570 age-matched
322 controls were included in the meta-analyses of 1) alpha peak amplitude, 2) alpha peak
323 frequency, 3) beta peak amplitude, 4) beta peak frequency, 5) exponent, 6) offset. The mean
324 unadjusted and aperiodic-adjusted signal is shown in Figure 2, separately for Parkinson's
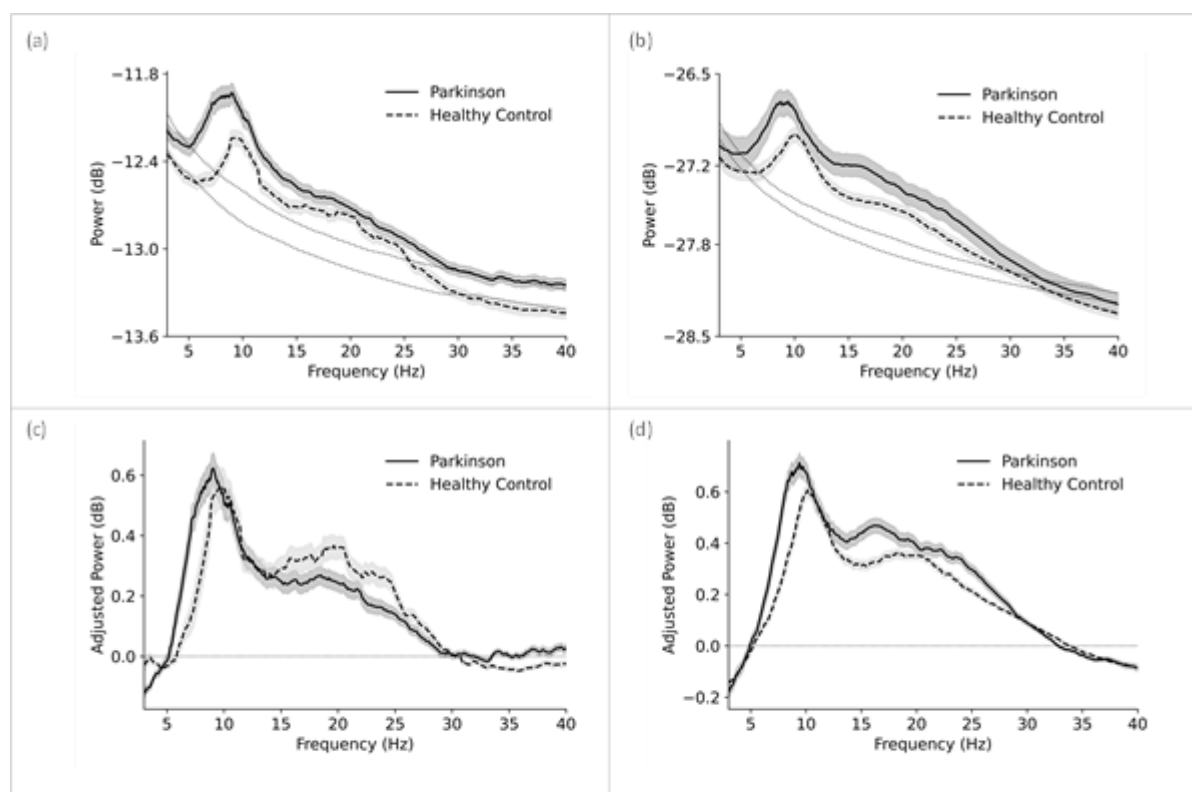325 patients and healthy controls, and for MEG and EEG datasets.

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Figure 2 (a) Mean unadjusted power in the 2 MEG datasets. (b) Mean adjusted power (aperiodic signal subtracted) in the 2 MEG datasets. (c) Mean unadjusted power in the 4 EEG datasets. (d) Mean adjusted power (aperiodic signal subtracted) in the 4 EEG datasets.

3.1. Alpha peak amplitude

A small effect of group on alpha amplitude was observed, SMD = 0.33, 95% CI = [0.15, 0.52], p = 0.00037, with a larger alpha amplitude for PD patients relative to controls (Figure 3A). There was limited evidence of heterogeneity across the 6 datasets: Q (5) = 6.05, p = 0.30, tau^2 = 0.01, I^2 = 22.50%.

3.2. Alpha peak frequency

There was a large effect of group on alpha central frequency, SMD = -0.74, 95% CI = [-0.92,-0.57], p <0.00001, with slower alpha central frequency for PD patients relative to controls (Figure 3B). There was low evidence of heterogeneity across datasets: Q(5) = 6.24, p = 0.28, tau^2 = 0.008, I^2 = 15.76%.

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3.3. Beta peak amplitude

There was no between-group difference in beta amplitude, SMD = 0.01, 95% CI = [-0.27, 0.30], p = 0.925 (Figure 3C). There was evidence of heterogeneity across datasets: Q(5) = 15.18, p = 0.009, tau^2 = 0.06, I^2 = 61.98% and visual inspection of the forest plot identified the OMEGA dataset as an outlier relative to the other 5 datasets. Since no effect of group was found when collapsing the data across all scalp electrodes, we performed separate random effects analyses for the frontal, central and posterior scalp regions. None of these models identified differences in beta amplitude between PD patients and controls (Frontal: SMD = 0.06, 95% CI = [-0.26, 0.39], p = 0.69; Central: SMD = 0.09, 95% CI = [-0.19, 0.37], p = 0.52; Posterior: SMD =-0.66, 95% CI = [-1.78, 0.45], p = 0.24).

3.4. Beta peak frequency

We identified no difference between PD patients and controls for beta central frequency, SMD =-0.05, 95% CI [-0.20, 0.08], p = 4.37e-01 (Figure 3D). There was no evidence of heterogeneity: Q(5) = 3.15, p = 0.63, tau^2 = 0.0, I^2 = 0.0%.

3.5. Exponent

PD patients had a markedly larger exponent compared to healthy controls: SMD = 0.61, 95% CI = [0.34, 0.88], p < 0.00001 (Figure 3E), although there was evidence of heterogeneity across datasets: Q(5) = 10.40, p = 0.06, tau^2 = 0.05, I^2 = 55.47%.

3.6. Offset

PD patients had a larger offset compared to controls: SMD = 0.71, 95% CI = [0.47, 0.94], p < 0.00001, (Figure 3F) and there was some heterogeneity across datasets: Q(5) = 7.91, p = 0.16, tau^2 = 0.03, I^2 = 41.87%.
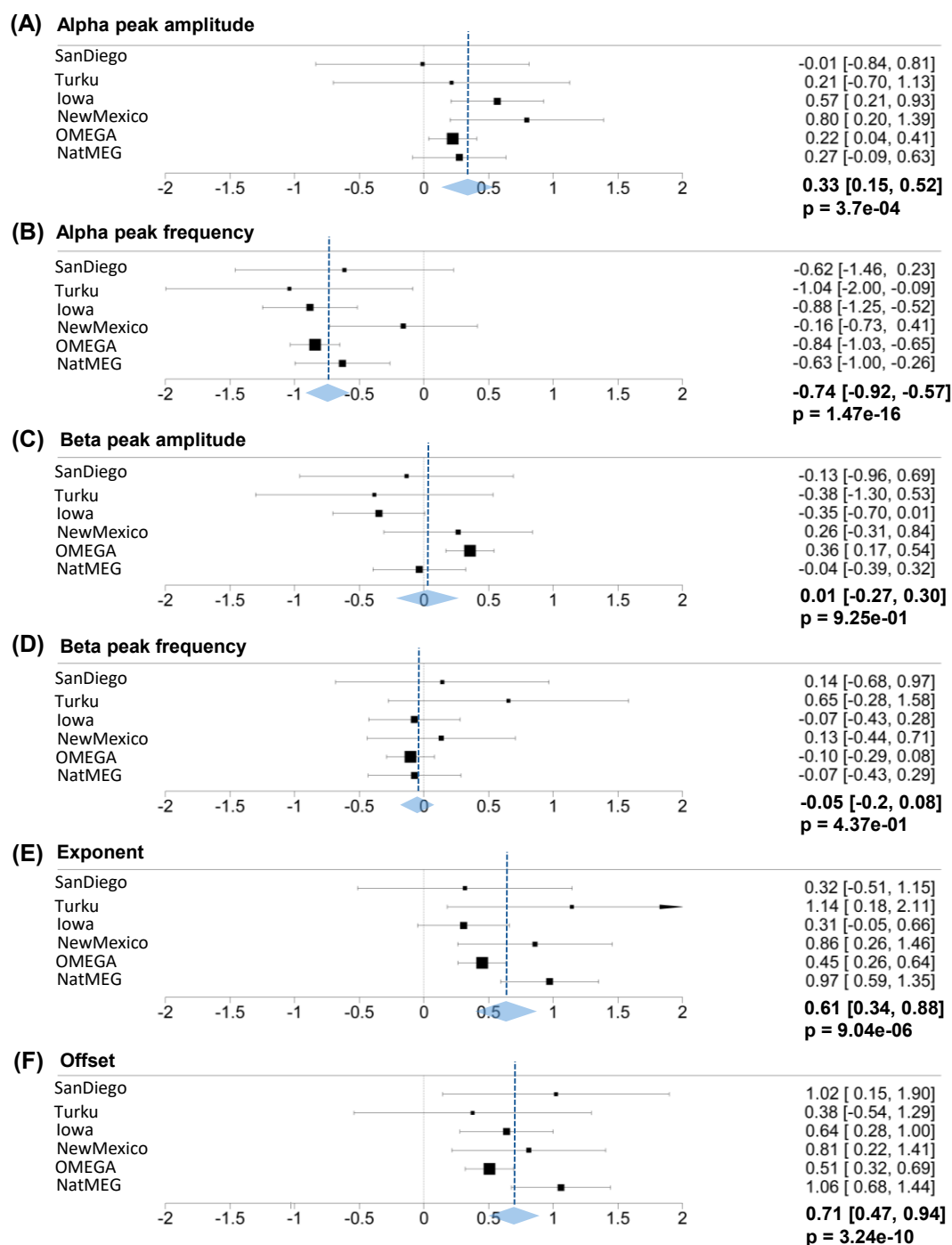
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Figure 3. Standardized mean differences between Parkinson's patients and healthy controls for A) Alpha peak amplitude, B) Alpha peak frequency, C) Beta peak amplitude, D) Beta peak frequency, E) Exponent and F) Offset. A positive number indicates values are higher in patients relative to controls, and a negative number indicates higher in controls relative to patients.

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4. Discussion

We conducted an advanced re-analysis and meta-analysis of resting-state M/EEG datasets to compare periodic and aperiodic components in patients with Parkinson's disease and age-matched healthy controls. Using a common analytical pipeline, aggregating multiple, raw, open-access M/EEG datasets, we compared the power and peak frequency of the aperiodic-adjusted alpha and beta oscillations, and the aperiodic components, i.e. exponent and offset across the two groups. A total of 6 datasets were included (4 recorded using EEG and 2 using MEG), involving 938 participants (368 with Parkinson's disease and 570 healthy controls). In summary, we identified higher alpha power and slower peak alpha frequency, in addition to higher aperiodic offset and exponent, in Parkinson's patients relative to controls, but there were no between-group differences in beta power or beta peak frequency in this resting state data.

4.1. Higher peak alpha power and slower peak alpha frequency in Parkinson's disease

The meta-analyses of alpha band measures were broadly consistent across the six included datasets, with statistical indices suggesting minimal between-study heterogeneity for both higher peak alpha power and slower peak alpha frequency in PD patients relative to controls (Sinanovic et al., 2005; Soikkeli et al., 1991; Stoffers et al., 2007; Ye et al., 2022). However, the relevance of this change in the clinical population is not immediately clear. In healthy adults, alpha rhythms are proposed to reflect decreased excitability of the underlying cortex and are modulated by both tonic and selective attention (Jensen, 2024; Thut et al. 2006). For example, increases in alpha power are observed during fatigued states (Hanzal et al. 2024; Tran et al. 2020), and linear increases in alpha activity are commonly recorded during the course of experimental sessions, as participants experience fatigue as a result of extended engagement with perceptual tasks (the "time-on-task" effect) (Benwell et al. 2019; Kasten et al. 2016).

Similarly, Parkinson's disease presents not only with motor symptoms but also with a range of non-motor symptoms, such as fatigue and apathy (Dujardin et al. 2007; Marin 1991; Pavese et al. 2010; Sáez-Francàs et al. 2013a; Siciliano et al. 2018a; Zuo et al. 2016). Around half of PD patients experience fatigue, both in terms of reduced physical and mental energy and increased effort in cognition and perception, at higher levels than healthy individuals (Siciliano et al. 2018b). Apathy is characterised by diminished motivation, goal-directed behaviour, and

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400   emotional engagement, manifesting as indifference, lack of initiative, and impaired decision-

401   making (Ineichen and Baumann-Vogel 2021; Sáez-Francàs et al. 2013b). Both apathy and

402   fatigue can appear early in PD, often preceding motor symptoms, and their prevalence often

403   increases as the disease progresses (Ineichen and Baumann-Vogel, 2021; Siciliano et al. 2020;

404   Siciliano et al. 2018b).

405

406   Although we identified a consistent increase in alpha power in patients relative to controls,

407   which may be related to transient or chronic episodes of fatigue and apathy, none of the

408   datasets included in the meta-analyses included clinical fatigue measurements to test this

409   hypothesis directly. The OMEGA dataset did include scores from the Unified Parkinson's

410   Disease Rating Scale Part I (UPDRS-I), which contains sub-item UPDRS-I.13, assessing fatigue

411   over the past few weeks. However, these scores were unavailable in other datasets, precluding

412   a formal meta-regression. Furthermore, although sub-item UPDRS-I.13 assesses fatigue, it may

413   not serve as a reliable indicator of the fatigue state experienced at the time of M/EEG recording.

414   As such, we cannot determine whether the observed alterations in alpha oscillations are driven

415   by underlying pathology, cognitive changes, elevated apathy or fatigue levels, or a combination

416   of these factors. We recommend that future research includes both physical and cognitive

417   fatigue measures taken at the time of recording, alongside longer-term assessments, to better

418   understand the relationship between Parkinson's disease and increased alpha power.

419

420   In addition to increased alpha power, we also identified a slowing of peak alpha frequency in

421   Parkinson's patients relative to controls. Although subtle, this effect was consistent across

422   datasets, with a mean slowing of 0.92 Hz (SD = 0.062). Individual alpha frequency gradually

423   changes non-linearly throughout the lifespan (Aurlien et al. 2004; Cellier et al. 2021; Chiang et

424   al. 2011; Cragg et al. n.d.; Duffy et al. 1984; Freschl et al. 2022; Grandy et al. 2013; Klimesch

425   1999; Knyazeva et al. 2018; Marshall et al. 2002; Turner et al. 2023) typically peaking around

426   10 Hz on average in young adulthood (Turner et al. 2023) followed by a gradual slowing into

427   older age. The functional significance of individual alpha frequency (IAF) is still under debate

428   (see Samaha & Romei, 2024, for a review) but has been proposed to represent the "sampling

429   rate" of the perceptual system (Samaha and Postle 2015), suggesting that slowing of IAF in

430   Parkinson's patients may reflect disrupted perceptual or cognitive processing (Chunharas et al.,

19

431   2022). Specifically, PD patients with more pronounced alpha slowing show increased disease

432   progression and cognitive decline in longitudinal studies (Olde Dubbelink et al., 2013). These

433   findings indicate that pathological changes in neural competence in Parkinson's disease may

434   directly impact neural oscillations. However, these changes could also reflect secondary effects

435   of non-motor symptoms commonly observed in PD.

436

437   Finally, we evaluated the consistency of our re-analysis results with those reported in the

438   original publications. Aperiodic-adjusted alpha power had previously been reported for three

439   of the included datasets (i.e. OMEGA, NatMEG and New Mexico). Since we re-analysed all

440   datasets using a common processing pipeline, we compared our outputs to the results from

441   the original articles. Our re-analysis was consistent across these datasets: all 3 papers had

442   previously reported an increased alpha power in PD patients and we replicated this effect. For

443   the remaining three datasets (i.e. San Diego, Turku, and Iowa), which had not previously been

444   analysed using spectral parameterisation, we replicated the absence of group-level differences

445   originally described in the San Diego dataset. However, the findings from our re-analysis

446   differed from the reported results of the Turku and Iowa studies.

447

448   Of the six datasets included in our analyses, only one (NatMEG) had previously examined peak

449   alpha frequency between groups and reported no differences. In contrast, our re-analysis of

450   the raw data identified a significant slowing of peak alpha frequency in Parkinson's disease

451   relative to controls in the NatMEG dataset (and consistently across all datasets). This

452   discrepancy is likely due to differences in the selection of electrodes analysed; the original

453   analysis focused on a fronto-central sensorimotor region of interest, whereas our approach

454   used full-scalp data, including the posterior electrodes. These findings underscore the impact

455   of analytical choices, such as electrode selection and the use of aperiodic-adjusted versus

456   unadjusted spectral analysis, on the outcomes of M/EEG data analysis. To improve

457   transparency, reproducibility, and comparability across studies, we recommend that

458   researchers report both adjusted and unadjusted power spectra, and, where possible, make

459   raw data and analysis scripts publicly available or on request.

460

461   **4.2. No group-level differences in resting-state beta activity**

462   We found no differences in resting-state beta power or beta peak frequency between

463   individuals with Parkinson's disease and healthy controls. This is perhaps unsurprising, given

464   that we analysed resting state activity, whereas beta band activity (13-30 Hz) is more strongly

465   associated with motor preparation and execution (Alayrangues et al. 2019; Rhodes et al. 2018).

466   Our re-analysis was consistent with the original findings from the NatMEG, New Mexico and

467   San Diego datasets, all of which reported no between-group differences in beta power. In

468   contrast, the original analyses of the OMEGA, Iowa, and Turku datasets reported reduced beta

469   power in Parkinson's patients. However, our re-analysis identified no group-level differences in

470   the Iowa and Turku datasets (which had not been parameterised), and higher beta power in PD

471   patients in the (parameterised) OMEGA dataset.

472

473   Our decision to analyse peak amplitudes within canonical frequency bands may have been

474   sensitive enough to capture important pathological electrophysiological changes in Parkinson's

475   disease. Beta activity often occurs in transient bursts with varying duration and amplitude (He

476   et al. 2020; Szul et al. 2023; Tinkhauser, Pogosyan, Little, et al. 2017b). Notably, beta bursts

477   occurring close in time to motor movements are associated with slower response times (Little

478   et al. 2019), and neurofeedback-driven suppression of these bursts leads to faster reaction

479   times (He et al. 2020). Thus, analysing the temporal dynamics of beta bursts may offer a more

480   sensitive and reliable method for investigating cortical motor impairments in PD.

481

482   While heterogeneity across the six datasets was low for peak beta frequency, there was high

483   heterogeneity for beta power. The OMEGA dataset emerged as an outlier, with PD patients

484   showing significantly larger beta amplitudes than controls. This difference may reflect greater

485   symptom severity of the patients in this cohort, as indicated by higher UPDRS-III and Hoehn &

486   Yahr scores, consistent with prior findings linking increased beta power to poor motor function

487   (Wiesman, Vinding, et al. 2025). Additionally, the PD patients in the OMEGA dataset were

488   substantially older than the controls (mean age PD = 65, SD = 9.1; mean age controls = 44.4, SD

489   = 20.8) and elevated beta activity is commonly observed in older adults (Park et al. 2025; Xifra-

490   Porxas et al. 2019). It is important to highlight that the version of the OMEGA dataset used in

491   the original report (Wiesman et al. 2023a) included a smaller sample of control participants,

492   with closer age matching between the patient and control groups. In contrast, in the current

21

493   study we used a larger subset of the OMEGA data, which includes a broader age range in the

494   control group. This difference in sample composition may contribute to the discrepancies in

495   beta power observed between the two studies. In short, our results suggest that *resting-state*

496   beta power and peak frequency are unlikely to serve as reliable electrophysiological biomarkers

497   of Parkinson's disease, and further synthesis of active-state M/EEG should be conducted.

498

499   ### 4.3. Higher aperiodic exponent and offset in Parkinson's disease

500   Compared to healthy controls, Parkinson's patients had a consistently larger aperiodic

501   exponent and offset across the six datasets. The functional relevance of the M/EEG exponent

502   has been variously interpreted as an index of the neural excitation-inhibition ratio (Darmani et

503   al. 2023; Helson et al. 2023; Wiest et al. 2023), oscillatory slowing (Wiesman et al. 2023b), and

504   oscillation damping (Evertz et al. 2022). We also observed a large effect of group on the

505   aperiodic offset, with PD patients showing larger offset values than healthy controls. Taken

506   together, these aperiodic changes could indicate alterations in excitation-inhibition balance and

507   increased baseline neural activity, reflecting pathological changes in network dynamics

508   associated with Parkinson's disease. Regardless of the physiological interpretation, our results

509   suggest that elevated aperiodic exponent and offset are likely to be robust and reproducible

510   characteristics across PD studies (Bush et al. 2024; Clark et al. 2023; da Silva Castanheira et al.

511   2024; Darmani et al. 2023; Liu et al. 2024; Martin et al. 2018; Monchy et al. 2023; Mostile et

512   al. 2019; Rosenblum et al. 2023; Vinding et al. 2024a; Wang et al. 2022b; Wiest et al. 2023;

513   Zhang et al. 2022b). Additionally, the aperiodic offset and exponent may also be potential

514   indicators to differentiate early- from late-onset PD (Wu et al. 2023).

515

516   Three of the included datasets (New Mexico, NatMEG, and OMEGA) had been analysed using

517   spectral parameterisation. These studies reported elevated aperiodic offset and exponent

518   values in PD patients, which our meta-analysis confirms are reliable markers consistently found

519   in Parkinson's disease across the datasets. On a methodological note, although our analysis

520   pipeline is broadly comparable, each study employed slightly different parameterisation

521   approaches, with minor variations in the parameters and frequency ranges. A systematic

522   investigation of how these methodological choices influence the spectral parameterisation

523   outputs, and the establishment of best-practice guidelines would improve consistency across

524   research groups. Reliable measures are critical for meaningful comparisons across individuals

525   and groups over time, indicating that observed changes reflect biological factors rather than

526   measurement errors (Ding et al. 2022; Ip et al. 2018). Both aperiodic offset and exponent

527   demonstrated good test-retest reliability (McKeown, Finley, et al. 2024), suggesting these

528   measures are stable over time and capable of capturing individual differences in neural activity.

529   This reliability positions them as promising markers for investigating neural mechanisms and

530   clinical conditions.

531

532   Whilst our results, and previous studies (McKeown, Jones, et al. 2024b; Vinding et al. 2024a)

533   provide evidence that the aperiodic offset can distinguish PD patients from healthy controls, its

534   functional relevance remains unclear. Although the aperiodic offset and exponent have been

535   associated with motor symptom severity in PD (Clark et al. 2023), conflicting evidence has also

536   been reported (Bush et al. 2024). Similarly, cognitive decline in PD also has been associated

537   with aperiodic activity in some studies (Rosenblum et al. 2023; Wiesman et al. 2023a), but this

538   association could not be replicated in others (Bernasconi et al. 2023a). Overall, it remains

539   uncertain whether the aperiodic offset and exponent are directly linked to cognitive and motor

540   symptoms in PD. Given the contradictory evidence, further research is needed to clarify these

541   relationships.

542

543   4.4. Limitations and future directions

544   In our re-analysis of existing raw M/EEG datasets, we developed a common processing pipeline

545   that was applied to all 6 datasets, to ensure that the aperiodic and periodic components were

546   extracted from consistently cleaned and processed signals. As shown in the Supplementary

547   data, varying the frequency ranges used for spectral parameterisation substantially affects the

548   outputs, particularly the estimate of the aperiodic exponent. For example, parameterising the

549   spectrum from 1-20 Hz results in a smaller exponent compared to using 3-40 Hz. In addition,

550   identifying peaks within the canonical frequency bands involves researcher-led definitions of

551   the upper and lower limits of those bands of interest (e.g., alpha may be variously defined as

552   8-12Hz or 8-13Hz, or further subdivided into low (8-10Hz) and high (10-12Hz) alpha). Although

553   both the clinical and control groups in this study were processed using the same pipeline,

554   allowing for a comparison of standardized mean differences between the two groups, the

23

555   impact of these experimenter-defined choices on the outputs of spectral parameterisation is

556   under-explored.

557

558   Second, to facilitate data synthesis, we chose to analyse only scalp-wide averages of the data

559   across all available electrodes, rather than conducting region of interest analysis, or an analysis

560   of individual electrodes. While this approach ensured comparability across datasets, it likely

561   obscured any subtle, region-specific changes in periodic and aperiodic activity (Donoghue

562   2024). Given that the six datasets differed in the number and placement of available electrodes,

563   it was deemed not possible to perform a direct comparison to such a degree of sensitivity.

564   However, we conducted a preliminary pilot analysis comparing aperiodic exponents across

565   frontal, central and posterior regions found no substantial regional differences between PD

566   patients and healthy controls (see Supplementary data). This absence of regional variation may

567   reflect the task-free nature of the resting-state recordings used in this study and it is possible

568   that task engagement modulates aperiodic activity differently across brain regions (Gyurkovics

569   et al., 2022).

570

571   Our decision to focus the analysis on alpha and beta frequency windows was data-driven, based

572   on the presence of observed peaks in the grand-average spectral plots (Figure 2). Consequently,

573   we did not perform a synthesis of other frequency bands, such as the theta band, which has

574   been reported to change in Parkinson's disease and may be a biomarker of PD-related cognitive

575   decline (Zawiślak-Fornagiel et al., 2023; Ye et al., 2022; Iyer et al., 2020). We also chose to only

576   include PD patients who were tested ON-medication, due to the low availability of both ON-

577   and OFF-medication datasets. It remains possible that different effects, particularly in beta

578   power and aperiodic components, would emerge when comparing patients tested OFF

579   medication states to controls. Further research examining ON versus OFF medication states, as

580   well as recording during active movement, is needed to evaluate the reliability of spectral

581   parameterisation as a biomarker for PD. Additionally, it remains unclear whether the periodic

582   and aperiodic changes observed in Parkinson's disease represent specific disease biomarkers

583   or simply correlate with disease presence.

584

585

5. Conclusion

Parkinson's patients exhibit increased alpha power and slowed peak alpha frequency, alongside elevated aperiodic offset and exponent values during resting states. This meta-analysis highlights the potential of M/EEG as a tool for identifying consistent cortical electrophysiological changes in Parkinson's disease. Further research is needed to refine these electrophysiological measures (particularly with best-practice guidelines for parameter selection during the processing stages), and to explore their potential clinical applications in the diagnosis and treatment of neurodegenerative conditions.

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