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1 Title: Human sensorimotor beta event characteristics and aperiodic signal are highly herita-
2 ble

3

4 Short title: Sensorimotor beta & aperiodic signal heritability

5

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36

37 **Abstract**

38 Individuals' phenotypes, including the brain's structure and function, are largely determined
39 by genes and their interplay. The resting brain generates salient rhythmic patterns that can be
40 characterized non-invasively using functional neuroimaging such as magnetoencephalog-
41 raphy (MEG). One of these rhythms, the somatomotor ('rolandic') beta rhythm, shows inter-
42 mittent high amplitude 'events' that predict behavior across tasks and species. Beta rhythm is
43 altered in neurological disease. The aperiodic ('1/f') signal present in electrophysiological re-
44 cordings is also modulated by some neurological conditions and aging. Both sensorimotor
45 beta and aperiodic signal could thus serve as biomarkers of sensorimotor function. Knowledge
46 about the extent to which these brain functional measures are heritable could shed light on
47 the mechanisms underlying their generation. We investigated the heritability and variability of
48 human spontaneous sensorimotor beta rhythm events and aperiodic activity in 210 healthy
49 male and female adult siblings' spontaneous MEG activity. The most heritable trait was the
50 aperiodic 1/f signal, with a heritability of 0.87 in the right hemisphere. Time-resolved beta event
51 amplitude parameters were also highly heritable, whereas the heritabilities for overall beta
52 power, peak frequency and measures of event duration remained nonsignificant. Human sen-
53 sorimotor neural activity can thus be dissected into different components with variable herita-
54 bility. We postulate that these differences partially reflect different underlying signal generating
55 mechanisms. The 1/f signal and beta event amplitude measures may depend more on fixed,
56 anatomical parameters, whereas beta event duration and its modulation reflect dynamic char-
57 acteristics, guiding their use as potential disease biomarkers.

58

59 **Significance statement**

60 The resting brain shows a prominent, highly modulated beta-range rhythm closely linked to
61 sensorimotor function in health and disease. We investigated the heritability of human spon-
62 taneous sensorimotor beta rhythm and its different components in a large cohort of 210 sib-
63 lings' MEG data. We find that particularly beta event amplitude and its variation as well as
64 aperiodic signal characteristics are highly heritable. The study demonstrates that time-re-
65 solved electrophysiological measures of spontaneous human sensorimotor brain activity are
66 determined to a significant degree by genes. We discuss the findings in the context of known

67 and postulated structural underpinnings of MEG signal generation, to highlight their transla-
68 tional relevance. The findings have clinical implications, e.g., when considering sensorimotor
69 beta alterations as biomarkers of neurological disease.

70

71 **Introduction**

72 Individuals' phenotypes are largely determined by their genetic blueprint that regulates prop-
73 erties ranging from cell products (Barroso and McCarthy, 2019) to system-level brain macro-
74 structure (Geschwind et al., 2002; Peper et al., 2007). Genetic influences also underlie func-
75 tional brain measures which are constant within, but highly variable between individuals. Elec-
76 troencephalography (EEG) and magnetoencephalography (MEG) have been successfully ap-
77 plied to quantify the heritability and identify genetic determinants of functional brain measures
78 (Van Beijsterveldt et al., 1996; Smit et al., 2006; Koten et al., 2009; Renvall et al., 2012; van
79 Pelt et al., 2012).

80

81 The brain generates 'background' electrical activity with salient rhythmic, but also arrhythmic
82 patterns during wakeful resting. One of the prominent spontaneous rhythms is the somatomo-
83 tor (rolandic) beta rhythm (Hari and Salmelin, 1997) that is observed across several mamma-
84 lian species (Haegens et al., 2011; Feingold et al., 2015; Sherman et al., 2016). It is modulated
85 by perceptual and cognitive functions, including tactile processing (Pfurtscheller et al., 2001;
86 Haegens et al., 2011), motor function (Salmelin and Hari, 1994; Feingold et al., 2015), action
87 perception (Hari et al., 1998; Babiloni et al., 2002) and attention (Van Ede et al., 2011; Sacchet
88 et al., 2015). Beta band activity is modulated over time, manifesting in intermittent high ampli-
89 tude 'events' (Feingold et al., 2015; Jones, 2016) relevant for behavior: In the sensorimotor
90 cortex, beta event rate predicts behavior across tasks and species (Shin et al., 2017). Both
91 beta power and beta events are altered in neurological conditions affecting motor function,
92 such as genetically determined Unverricht-Lundborg disease (Silén et al., 2000), stroke (Laak-
93 sonen et al., 2012) and Parkinson's disease (Vinding et al., 2020; Pauls et al., 2022).

94

95 Besides rhythmic, or periodic, components, MEG power spectra also contain aperiodic ('1/f')
96 components (He, 2014). These two are important to disentangle as they are probably gener-
97 ated by different neural mechanisms. Aperiodic signal is believed to represent excitation-inhi-
98 bition balance (Gao et al., 2017), and it is modulated, e.g., by brain maturation (McSweeney
99 et al. 2021; Hill et al. 2022), aging (Voytek et al., 2015; Wilson et al., 2022) and several neu-
100 rological and psychiatric conditions (Molina et al., 2020; Ostlund et al., 2021; Semenova et al.,

101 2021). Cortical beta rhythm (Laaksonen et al., 2012; Pauls et al., 2022) and aperiodic activity
102 (Helson et al., 2023) both relate to clinical symptoms, show good or excellent test-retest reli-
103 ability (Pauls et al., 2023, bioRxiv), and thus have potential as diagnostic or prognostic bi-
104 omarkers.

105

106 Interpretability of rhythmic and aperiodic neural signals is important for both research and
107 clinical diagnostic applications. MEG signal arises from spatial and temporal summation of
108 underlying neuronal activity (Buzsáki et al., 2012). Structure and function are closely related:
109 e.g., peak oscillation frequency decreases with increasing cortical thickness and processing
110 hierarchy (Mahjoory et al., 2020). Decoding the structure-function-genetics relationship of
111 M/EEG signal generation could help understand signals' individuality and their degradation in
112 neurological diseases, raising their value as diagnostic tools: M/EEG may detect pathology
113 before observable structural changes in neurological disorders (Terry et al., 1991). Heritability
114 reflects the contribution of genetic vs. environmental factors to the differences observed be-
115 tween individuals, and the quantification of the heritability of neural signals can thus lead to
116 insights of the biology behind the measurable phenotypes (Visscher et al., 2008). Beta and
117 other frequency bands' global spectral power is heritable (Van Baal et al., 1996; Van Beijster-
118 veldt et al., 1996; Smit et al., 2005; Salmela et al., 2016); the beta power variability has been
119 linked to a GABA_A receptor locus (Porjesz et al., 2002). Heritability of time-resolved beta
120 events, however, has not been investigated.

121

122 We investigated the heritability and variability of time-resolved human cortical sensorimotor
123 beta rhythm and aperiodic activity using healthy adult siblings' spontaneous MEG data. We
124 propose that knowledge about the relative heritability of different neural components of sen-
125 sorimotor activity can shed light on the underlying generating mechanisms and help interpret
126 changes observed in, e.g., patient populations with sensorimotor dysfunction.

127

128 **Materials and methods**

129 Subjects

130 210 Finnish-speaking siblings from 100 families participated in the study (8 families with three
131 siblings, 1 family with four; 148 females [mean \pm SD age 29 \pm 10 years, range 18-60 years],
132 62 males [30 \pm 9 years, range 19-52 years]; 206 right-handed, three ambidextrous, one left-
133 handed). None of the participants had a history of neurological or psychiatric disorders. The

134 study was approved by the Hospital District of Helsinki and Uusimaa ethics committee, and all
135 participants gave their written informed consent to participate.

136 MEG recordings

137 Spontaneous cortical activity was recorded in a magnetically shielded room with a 306-chan-
138 nel Vectorview neuromagnetometer (Elekta Oy, Helsinki, Finland) that contains 204 planar
139 gradiometers and 102 magnetometers. Head positioning was measured at the beginning of
140 the measurement. Three minutes of data was collected while participants were resting with
141 their eyes open (REST), as well as while they clenched both hands alternatingly about once
142 per second, self-paced, keeping the eyes open (MOT). The MEG signals were band-pass
143 filtered at 0.03–200 Hz and sampled at 600 Hz.

144 MEG signal processing and beta event extraction

145 For suppressing external artifacts, MEG data were preprocessed using the signal space sep-
146 aration method (SSS, (Taulu and Simola, 2006)) implemented in MaxFilter software (MEGIN
147 Oy, Helsinki, Finland). Individual MEG recordings were transferred to one subject's head
148 space using a signal space separation based head transformation algorithm (Taulu et al.,
149 2004), implemented in MaxFilter. Further signal processing was done using MNE-python ver-
150 sion 0.22 (Gramfort et al., 2013). After band-pass filtering the data to 2-48 Hz with a one-pass,
151 zero-phase, non-causal FIR filter (MNE firwin filter design using a Hamming window), power
152 spectral density (PSD) was calculated using Welch's method (MNE's psd_welch function) with
153 a non-overlapping Hamming window and 1024-point Fast Fourier Transformation (FFT).

154 The subsequent analysis steps are illustrated in **Figure 1**. The data analysis was performed
155 on the 204 gradiometer signals. First, a channel pair with the highest spectral peak in the beta
156 range ('the peak channel pair') was selected from the region of interest (ROI) of 15 gradiom-
157 eter channel pairs per hemisphere centered over the sensorimotor cortices, and the frequency
158 at the power peak noted ('peak beta frequency') (see **Figure 1A**). In order to quantify PSD at
159 each recording site, we computed the vector sum of the two orthogonally oriented planar gra-
160 diometers at each sensor location ('vector PSD'):

$$161 \text{ PSD}_{\text{vector}} = \sqrt{(\text{PSD}_{\text{ch1}}^2 + \text{PSD}_{\text{ch2}}^2)}$$

162 The resulting 15 vector-sum PSDs per hemisphere were then decomposed into a periodic and
163 aperiodic component using FOOOF (Donoghue et al., 2020). FOOOF models the power spec-
164 trum as a combination of two distinct functional processes: an aperiodic component, reflecting
165 1/f like characteristics (exponential decay with an offset and an exponent), and a variable
166 number of periodic components (putative oscillations), as peaks rising above the aperiodic

167 component. After subtraction of the aperiodic component, the remaining periodic component
168 was plotted for all 15 vector-sum PSDs for both REST and MOT conditions in the frequency
169 range of 14-30 Hz. The resulting plots were visually inspected by two observers (AP and OK)
170 to manually select the beta signal frequency modulated most by MOT compared to REST.

171 As the manual channel selection may be prone to human observer bias, we compared the
172 inter-rater agreement between two slightly different approaches, conducted independently
173 years apart on the same data. The peak beta band frequencies had previously been extracted
174 by one of the authors (HR) without separating the $1/f$ aperiodic signal part and by using
175 Welch's method with 4096-point FFT, eight data segments overlapping by 50% and Hamming
176 windowing. When allowing deviation of ± 3 Hz in the extracted peaks (taken the different FFT
177 sizes and different handling of the aperiodic $1/f$ component), the two approaches resulted in
178 85% agreement, which is considered good.

179 Using the manually selected peak frequencies, the periodic components of the 15 vector-sum
180 PSDs were searched automatically to determine the recording channel with the highest peak
181 and its frequency (± 1 Hz) for both hemispheres' ROIs, and visually inspected again by AP.

182 The peak beta frequency and corresponding peak power of the chosen vector-sum PSD, the
183 total beta band power (periodic part of PSD area under curve (AUC) from 14-30 Hz, $1/f$ com-
184 ponent subtracted), as well as the aperiodic component information obtained via FOOOF (off-
185 set and exponent χ), were further used in the heritability analysis. All electrophysiological
186 parameters included in the heritability analysis are illustrated in **Figure 1C**.

187 The channel pair and peak beta frequency corresponding to the chosen vector-sum PSD were
188 used for beta burst analysis (see **Figure 1B**). Beta event extraction was carried out similarly
189 to the method described in Pauls et al. (2022): the channel pair's raw unfiltered time series
190 data were downsampled to 200 Hz, high-pass filtered at 2 Hz and decomposed by convolving
191 the signal with a set of complex Morlet wavelets over the frequency range of 7-47 Hz with 1
192 Hz resolution and $n_cycles=frequency/2$. The signal was then averaged within the individual
193 narrow-band beta frequency range, *i.e.*, ± 1.5 Hz around the individual peak beta frequency,
194 discarding the other frequencies. The vector sum over the two channels' beta band time series
195 was calculated as described above, and the resulting signal was rectified to obtain one beta
196 band amplitude envelope for the channel pair. The envelope was smoothed with a 100-ms
197 FWHM kernel and thresholded at the 75th percentile value. Periods exceeding this threshold
198 for 50 ms or longer were defined as beta events. For event amplitude and event duration, the
199 mean, median, robust maximum (defined as mean of the top 5% values) and standard devia-
200 tion values were calculated. Furthermore, events per second (event rate) and event dispersion
201 were calculated similarly to Pauls et al. (2022). Times between beta events were defined as

202 waiting times. To estimate the variation of waiting times ('event dispersion'), we calculated the
203 coefficient C_V proposed by Shinomoto et al. (Shinomoto et al., 2005), defined as the waiting
204 times' standard deviation σ divided by their mean μ :

$$205 \quad C_V = \frac{\sigma}{\mu}$$

206

207 All values were calculated for both hemispheres in all subjects (see also **Figure 1B**).

208 Effect sizes for MEG features were based on Cohen's d values for single group designs:

$$209 \quad D = M/S$$

210 where M and S are the mean and the standard deviation of the feature values across subjects
211 (Goulet-Pelletier and Cousineau, 2018).

212 Heritability analysis

213 Heritability is defined as the proportion of (additive) genetic variance of the total phenotypic
214 variance of a population.

215

$$216 \quad h^2 = V_{\text{genetic}} / V_{\text{phenotypic}}$$

217

218 Phenotype heritabilities were calculated using the software program Merlin version 1.1.2 (Abe-
219 casis et al., 2002), which employs a variance component approach as detailed by Amos
220 (Amos, 1994). Heritability estimates are calculated based on variance components. The coef-
221 ficient estimating genetic variance is adjusted by the degree of relationship, which is 0.5 (50%
222 shared genes) in full siblings. The full sibling status of our study individuals has been confirmed
223 by [an earlier] DNA analysis (Renvall et al. 2012).

224

225 Merlin requires non-negative values for correct interpretation so phenotypes with negative val-
226 ues were multiplied by -1. Such a transformation is standard for the Merlin analysis tool.
227 Correctness of the input data format was checked by the Pedstats program (Wigginton and
228 Abecasis, 2005). As the analysis assumes the studied phenotypes to be normally distributed
229 while many of them were not, we also re-ran the analyses after first correcting the phenotype
230 values' distributions with the inverse normal correction internal to Merlin. As both analyses

231 produced highly concordant results, we report here the results based on the non-corrected
232 values.

233

234 The probability of the observed heritability values being different from zero was assessed by
235 permuting the family labels of the study subjects 6000 times and calculating the heritability for
236 each of the permuted datasets. For each phenotype, the number of permutations k where the
237 permuted heritability was higher than the heritability observed in the real data was recorded
238 and used to calculate the one-tailed probability of the observed heritability exceeding zero as
239 $k/6000$. This permutation scheme may slightly inflate the permuted heritabilities, as it does not
240 explicitly ensure that the permutation does not reproduce any of the original sibships. This
241 may lead to conservative significance estimates. Likewise, to correct for the multiple tests
242 performed ($n = 30$), we performed a Bonferroni correction, which may be overly conservative
243 considering that some of the phenotypes were correlated.

244 Code and data accessibility

245 These data cannot be made publicly available due to Finnish data protection law. Data can,
246 however, be shared for research collaboration with an amendment to the research ethics per-
247 mit and a related data transfer agreement. All analysis code is available on GitHub
248 (<https://github.com/BioMag/Beta-sibling-study>).

249 **Results**

250 A summary of the beta band phenotypic features (both beta PSD features as well as beta
251 band burst characteristics) is given in **Table 1**. **Figure 2A** shows examples of different beta
252 power spectral phenotypes observed, and **Figure 2B** depicts beta band phenotypes for pairs
253 of siblings. Typical PSD phenotypes were *i*) ones with a narrow peak on either side of 20 Hz,
254 *ii*) a broad band activity typically spanning 15-25 Hz, and *iii*) two distinctive peaks, one typically
255 in the lower beta range (14-20 Hz) and the other in the high beta range (20-30 Hz).

256

		Left hemisphere				Right hemisphere			
PSD characteristics		mean	median	std	range	mean	median	std	range
peak beta frequency (Hz)		19.7	19.3	3.0	14.1-25.8	19.8	19.3	3.1	14.1-29.3
peak beta power ($\mu\text{T/cm}^2$)		276	144	374	15-3231	133	70	173	5-1251
total beta band power (periodic)		2979	1686	3318	164-16284	1093	619	1322	26-9652
1/f component exponent		1.03	1.01	0.19	0.34-1.76	1.07	1.05	0.18	0.67-1.66
1/f component offset		-22.95	-22.99	0.37	-23.75- -21.75	-23.28	-23.32	0.34	-24.19 -22.25
Beta event characteristics		mean	median	std	range	mean	median	std	range
duration (ms)	mean	256.9	248.0	49.4	181.7-498.2	265.2	253.7	50.0	182.0-454.0
	median	199.0	195.0	32.3	152.5-420.0	201.4	195.0	34.9	150.0-355.0
	standard deviation	198.4	182.4	63.2	99.8-487.5	213.0	198.3	67.4	67.3-531.7
	robust maximum	858.0	788.6	257.4	439.5-2145.0	912.1	852.2	263.9	382.7-2135.0
amplitude ($\mu\text{T/cm}$)	mean	325	285	165	104-994	221	188	113	73-654
	median	301	260	155	93-984	203	173	105	70-618
	standard deviation	86	71	48	21-264	62	52	34	14-202
	robust maximum	564	486	286	174-1467	390	335	196	114-1128
event rate (1/s)		1.00	1.00	0.16	0.50-1.36	0.97	0.98	0.16	0.55-1.37
dispersion		1.14	1.05	0.44	0.65-5.59	1.16	1.06	0.44	0.41-5.58

257

258 **Table 1 – PSD (beta & 1/f) and beta event descriptives**

259 Parameters used in the heritability analysis. Peak frequency – frequency between 14-30 Hz most mod-
260 ulated by hand movement; peak power – PSD amplitude at peak frequency; total beta band power
261 (periodic) – total AUC from 14-30 Hz of the periodic part of the signal (1/f signal component subtracted);
262 1/f component chi – exponential decay coefficient and offset describing 1/f (aperiodic) signal compo-
263 nent. Beta event characteristics: robust maximum – mean of top 5 % values; burst rate – number of
264 bursts/recording time; dispersion – stdev(inter-burst intervals)/mean(inter-burst intervals).

265

266 Heritability results are shown in **Table 2**. Overall, the right-hemispheric parameters were more
267 heritable than the left-hemispheric ones. The right hemisphere's 1/f aperiodic exponent and
268 offset were significantly heritable (exponent $h^2=0.87$, offset $h^2=0.69$). Measures of beta burst
269 amplitudes were also significantly heritable (range of significant heritability values h^2 of 0.28-
270 0.81). Notably, of the beta burst amplitude measures, the measures reflecting the dynamic
271 range (beta event amplitude maximum and its standard deviation) were most highly heritable.
272 Apart from the peak beta power with moderate effect size in both hemispheres (Cohen's d
273 0.74-0.77), all effect sizes were either large (Cohen's $d > 0.80$) or very large (Cohen's $d > 1.2$).

274

275

PSD characteristics		Left hemisphere			Right hemisphere		
		h^2	p	n sig. (/6000)	h^2	p	n sig. (/6000)
peak beta frequency		0.45	0.0047	28	0.41	0.0103	62
peak beta power		0.28	0.0648	389	0.58	0.0072	43
total beta band power (periodic)		0.49	0.0068	41	0.44	0.0157	94
1/f component exponent*		0.47	0.0035	21	0.87	0.0000*	0
1/f component offset*		0.35	0.0258	155	0.69	0.0000*	0
Beta event characteristics		h^2	p	n sig. (/6000)	h^2	p	n sig. (/6000)
duration	mean	0.45	0.1350	810	0.36	0.0222	133
	median	0.28	0.1338	803	0.40	0.0172	103
	standard deviation	0.49	0.2495	1497	0.32	0.0412	247
	robust maximum	0.47	0.2383	1430	0.33	0.0372	223
amplitude	mean*	0.35	0.0060	36	0.75	0.0002*	1
	median*	0.45	0.0110	66	0.72	0.0002*	1
	standard deviation*	0.28	0.0005*	3	0.81	0.0000*	0
	robust maximum*	0.49	0.0007*	4	0.79	0.0000*	0
event rate		0.47	0.0543	326	0.38	0.0137	82
dispersion		0.35	0.2850	1710	0.00	1.0000	6000

Table 2. Heritability h^2 of the oscillatory phenotypes calculated by Merlin. The nominal probability that the heritability differs from zero is calculated from an empirical distribution based on 6000 permutations of the sibship statuses/family IDs of the subjects. The variables and values that are significant after a Bonferroni correction for multiple testing are given in bold.

Discussion

To our knowledge, this is the first study investigating the heritability of spontaneous time-resolved sensorimotor beta event dynamics and aperiodic neural activity. Time-resolved beta event amplitude parameters were highly heritable, whereas the heritabilities for peak frequency and measures of event duration were not significantly different from zero. Interestingly, the most heritable trait was the aperiodic 1/f exponent, with a heritability of 0.87 in the right hemisphere. Overall, the right-hemispheric phenotypic traits were more heritable than the left-hemispheric ones.

Heritability of MEG/EEG traits including beta oscillatory activity

Heritability of electrophysiological traits has been little investigated to date. In twin studies, EEG alpha, beta, theta and delta range peak frequencies (Van Beijsterveldt et al., 1996), occipital alpha power and peak frequency at rest (Smit et al., 2006), as well as MEG visual task-related gamma peak frequency (van Pelt et al., 2012) have been found to be highly heritable. We have previously demonstrated that auditory evoked fields' amplitude (Renvall et al., 2012) as well as occipital resting-state alpha oscillatory activity (Salmela et al., 2016) are heritable in siblings, and that MEG power spectral features at rest allow identification of sibling relationship (Leppäaho et al., 2019). These MEG traits were associated with certain genetic loci /

300 genomic regions (Renvall et al., 2012; Salmela et al., 2016; Leppäaho et al., 2019) but it is
301 likely that most functional brain traits are controlled polygenetically. Furthermore, functional
302 connectivity in theta, alpha and beta bands as measured with MEG appears progressively
303 more similar as the strength of genetic relationship increases (Colclough et al., 2017).

304

305 MEG signal generative mechanisms and possible relation to heritability

306 MEG measures magnetic fields arising from the *temporal* and *spatial summation* of electric
307 currents occurring in the underlying brain tissue (Buzsáki et al., 2012). The measured raw
308 signal time series can be summarized in different ways, e.g., as power spectral density. Re-
309 duction in global beta power can result from various changes in the neuronal signalling, such
310 as smaller amplitude beta oscillation events, or fewer or shorter beta oscillation events without
311 simultaneous changes in amplitude. Thus, decomposing beta power into components gives
312 additional information about the underlying neural processing. We postulate that these MEG
313 dynamical measures reflect different aspects of MEG signal generation. The upper panel of
314 **Figure 3** schematically summarizes factors that contribute to the generation of MEG signals,
315 and the lower panel indicates how those factors may relate to the functional parameters ad-
316 dressed in this study.

317

318 What underlies the heritability of beta event amplitude?

319 We postulate that the MEG beta event amplitude reflects relatively fixed anatomical factors
320 summarized in **Figure 3** (upper panel, left). Pyramidal cells are neocortex' most abundant cell
321 type. Synaptic currents and their state-dependent modulation are the main determinants of
322 intra- and extracellular field strength, and their *spatial summation* is governed by pyramidal
323 cell morphology, cortical microstructure and layering, as well as synaptic input density
324 (Buzsáki et al., 2012). Beta event amplitudes are probably crucially dependent on these mi-
325 crostructural properties: While both temporal and spatial superposition determine event am-
326 plitude, especially the amplitude's dynamic range is limited by local cortical microstructure.
327 Interestingly, in the current study, event amplitudes' dynamic range measures (standard devi-
328 ation, maximum) were most strongly heritable.

329

330 Brain anatomical traits such as cortical thickness (Geschwind et al., 2002; Schmitt et al., 2014)
331 and cortical myelination (Schmitt et al., 2020) have previously been shown to be heritable. By
332 late adolescence, differences in cortical thickness in the sensorimotor regions are largely due

333 to heritable factors, whereas environmental factors play only a weak role (Schmitt et al., 2014).
334 Thus, throughout development, sensorimotor cortical structure appears increasingly governed
335 by the underlying genetics.

336

337 Both beta peak amplitude and the power at the beta band (which is determined by the ampli-
338 tude, number and duration of individual beta events) appeared more heritable in the right than
339 left hemisphere. Our result is in agreement with earlier studies that have found cortical mor-
340 phology/volume to be more genetically controlled in the right than left hemisphere in right-
341 handed individuals (Geschwind et al., 2002); functional studies point in the same direction
342 (Smit et al., 2006).

343

344 Why are event duration parameters not similarly heritable?

345 In the current cohort, measures of beta event duration were not significantly heritable. *Tem-*
346 *poral summation* of neural events, which determines the timing and duration of beta events,
347 arises from the interplay between several brain areas, their connections and relative input
348 timings and strength (**Figure 3**, top panel, right). Important cortical pyramidal cell afferent in-
349 puts originate from other adjacent pyramidal cells (intrinsic input) (Lorente de No, 1949), cor-
350 tico-cortical connections (Kandel et al., 2000) and thalamic connections, including connections
351 from sensory organs, and from other cortical areas ('higher-order' thalamic input) (Sherman
352 et al., 2016; Mo and Sherman, 2019). Computational models suggest that sensory induced
353 beta events are generated by synchronous bursts of excitatory synaptic drive to superficial
354 and deep cortical layers, with asymmetry in the respective input strengths (Jones et al., 2009;
355 Sherman et al., 2016; Neymotin et al., 2020): The stronger the superficial input, the more
356 prominent is the beta activity (Sherman et al., 2016). Experimental data are compatible with
357 this model (Sherman et al., 2016; Bonaiuto et al., 2021; Law et al., 2022). Thus, beta event
358 timing and duration appear to depend on the timing and strength of inputs from several differ-
359 ent cortical and subcortical input sources.

360

361 Network resonance could also play a role in beta event generation: In a dopamine-depleted
362 state, cortical beta events are associated with increased synchrony between EEG/ECOG cor-
363 tical activity and basal ganglia spiking activity (Cagnan et al., 2019). In animal models of par-
364 kinsonism, high cortical beta synchrony can be generated by changing the relative timings

365 between thalamic and cortico-cortical inputs (Reis et al., 2019). Hence, network resonant prop-
366 erties could contribute to temporal summation at least in some disease states, but possibly in
367 a dopamine-dependent fashion also in healthy brains.

368

369 Thus, compared to spatial summation, temporal summation relies on more individual factors
370 and their interplay (e.g., network structural and functional properties), making heritability more
371 multifactorial and thus less likely to show heritability in the present analysis. Methodological
372 factors could also contribute to the lack of heritability: signal-to-noise ratio of the recordings
373 affects event duration more than event amplitude measures. Finally, the resting-state beta
374 event duration could be a randomly fluctuating parameter, governed by stochastic events and
375 their timing. These explanations, however, seem less likely given the outlined experimental
376 evidence, as well as our test-retest reliability results (Pauls et al. 2023, bioRxiv).

377

378 Why is the aperiodic signal component heritable?

379 Aperiodic signal components were the most heritable of the investigated parameters in the
380 present study. The aperiodic signal is closely related to anatomical microstructure: Cortical
381 pyramidal cells and their dendritic morphology and density are believed to be the most im-
382 portant determinants of the mammalian cortical 1/f signal observed with MEG (Lindén et al.,
383 2010; Buzsáki et al., 2012). The 1/f signal is thought to stem from passive dendrite filtering
384 properties (Haines et al., 2016) but it is also modulated in an activity-dependent way (Pettersen
385 et al., 2014). It has been shown to be affected by brain maturation (McSweeney et al.
386 2021; Hill et al. 2022) and aging (Voytek et al., 2015; Wilson et al., 2022) as well as neurolog-
387 ical (Semenova et al., 2021) and psychiatric diseases (Ostlund et al., 2021). Furthermore, 1/f
388 reflects the attentional state (Waschke et al., 2021) and may contribute to integration of signals
389 over longer periods of time (Maniscalco et al., 2018). Thus, the signal's relative stability over
390 extended periods of time, and its close relationship to cortical microstructure may explain the
391 high heritability.

392

393 Stability of beta events and aperiodic activity - a prerequisite for clinical use

394 Movement-related beta suppression and rebound at the sensorimotor cortices show excellent
395 test-retest stability over weeks in EEG recordings (Espenhahn et al., 2017). Similarly, beta
396 rhythm modulation after tactile and proprioceptive stimulation was recently demonstrated to
397 be highly reproducible in healthy subjects within a year (Illman et al., 2022). In an independent

398 cohort of 50 healthy subjects measured twice during wakeful resting, both the aperiodic power
399 spectral features as well as several beta event characteristics showed good to excellent test-
400 retest stability (Pauls et al., 2023, bioRxiv). Recordings of 2-3 minutes of resting state data
401 were sufficient to obtain stable results for most parameters, speaking for their feasibility in
402 clinical settings. In the future, the heritability of dynamic oscillatory activity also outside the
403 somatosensory cortices could be addressed. This would, however, likely require automated
404 approaches which, in turn, might be more prone to signal-to-noise variations than the partly
405 manual phenotyping applied here.

406

407 Limitations

408 As the analysis assumes normal distribution of the phenotypes, the fact that many of the phe-
409 notypes were non-normally distributed may have decreased the statistical power of the study.
410 The permutation procedure adopted for testing the significance of the heritability values
411 should, however, correct for any inflation of the heritabilities caused by the non-normality. The
412 analyses were additionally conducted with the internal normality correction functionality of
413 Merlin, resulting in values qualitatively similar to (although slightly more significant than) those
414 based on the non-corrected data presented here.

415

416 Any measurement noise contributes to the phenotypic variability, thus reducing estimated her-
417 itability. The effect sizes calculated here did not suggest a systematic effect of signal-to-noise
418 ratio on the observed heritabilities: for example, the effect size for event duration was higher
419 than the effect size for event amplitude. Furthermore, in our recent study (Pauls et al., 2023,
420 bioRxiv) the test-retest reliability of somatomotor beta activity was not directly related to rela-
421 tive heritabilities observed in the current study. Thus, the observed heritability differences do
422 not solely reflect differences in the signal reliability nor the signal-to-noise ratio.

423

424 Conclusion

425 We here show that the human sensorimotor beta and aperiodic cortical activity can be dis-
426 sected into highly heritable and non-heritable components. We postulate that the different
427 heritabilities reflect, in part, different underlying signal generating mechanisms and their
428 weighting in the generation of different signal characteristics. In combination with increased
429 information resulting from the time-resolved beta signal decomposition, the results generate
430 an interesting framework to interrogate and interpret M/EEG data both in healthy subjects as

431 well as patient populations. This framework also increases the potential of whole-brain elec-
432 trophysiology measures, such as beta band activity, as disease biomarkers.

433 **References**

- 434 Abecasis GR, Cherny SS, Cookson WO, Cardon LR (2002) Merlin — Rapid analysis of
435 dense genetic maps using sparse gene flow trees. *Nat Genet* 30:97–101.
- 436 Amos CI (1994) Robust variance-components approach for assessing genetic linkage in
437 pedigrees. *Am J Hum Genet* 54:535–543.
- 438 Babiloni C, Babiloni F, Carducci F, Cincotti F, Coccozza G, Del Percio C, Moretti DV, Rossini
439 PM (2002) Human cortical electroencephalography (EEG) rhythms during the observa-
440 tion of simple aimless movements: A high-resolution EEG study. *Neuroimage* 17:559–
441 572.
- 442 Barroso I, McCarthy MI (2019) The Genetic Basis of Metabolic Disease. *Cell* 177:146–161
443 Available at: <https://pubmed.ncbi.nlm.nih.gov/30901536/>.
- 444 Bonaiuto JJ, Little S, Neymotin SA, Jones SR, Barnes GR, Bestmann S (2021) Laminar dy-
445 namics of high amplitude beta bursts in human motor cortex. *Neuroimage* 242 Available
446 at: <https://pubmed.ncbi.nlm.nih.gov/34407440/>.
- 447 Buzsáki G, Anastassiou CA, Koch C (2012) The origin of extracellular fields and currents-
448 EEG, ECoG, LFP and spikes. *Nature Reviews Neuroscience* 13:407–420 Available at:
449 <https://pubmed.ncbi.nlm.nih.gov/22595786/>.
- 450 Cagnan H, Mallet N, Moll CKE, Gulberti A, Holt AB, Westphal M, Gerloff C, Engel AK, Hamel
451 W, Magill PJ, Brown P, Sharott A (2019) Temporal evolution of beta bursts in the parkin-
452 sonian cortical and basal ganglia network. *Proc Natl Acad Sci U S A* 116:16095–16104.
- 453 Colclough GL, Smith SM, Nichols TE, Winkler AM, Sotiropoulos SN, Glasser MF, Van Essen
454 DC, Woolrich MW (2017) The heritability of multi-modal connectivity in human brain ac-
455 tivity. *Elife* 6 Available at: <https://pubmed.ncbi.nlm.nih.gov/28745584/>.
- 456 Donoghue T, Haller M, Peterson EJ, Varma P, Sebastian P, Gao R, Noto T, Lara AH, Wallis
457 JD, Knight RT, Shestyuk A, Voytek B (2020) Parameterizing neural power spectra into
458 periodic and aperiodic components. *Nat Neurosci* 23:1655–1665.
- 459 Espenhahn S, de Berker AO, van Wijk BCM, Rossiter HE, Ward NS (2017) Movement-re-
460 lated beta oscillations show high intra-individual reliability. *Neuroimage* 147:175–185.
- 461 Feingold J, Gibson DJ, Depasquale B, Graybiel AM (2015) Bursts of beta oscillation differen-
462 tiate postperformance activity in the striatum and motor cortex of monkeys performing
463 movement tasks. *Proc Natl Acad Sci U S A* 112 Available at:
464 <http://dx.doi.org/10.1073/pnas.1517629112>.
- 465 Gao R, Peterson EJ, Voytek B (2017) Inferring synaptic excitation/inhibition balance from
466 field potentials. *Neuroimage* 158:70–78.
- 467 Geschwind DH, Miller BL, DeCarli C, Carmelli D (2002) Heritability of lobar brain volumes in
468 twins supports genetic models of cerebral laterality and handedness. *Proc Natl Acad Sci*
469 *U S A* 99:3176–3181.

- 470 Goulet-Pelletier J-C, Cousineau D (2018) A review of effect sizes and their confidence inter-
471 vals, Part 1: The Cohen's d family. *Tutor Quant Methods Psychol* 14:242–265.
- 472 Gramfort A, Luessi M, Larson E, Engemann DA, Strohmeier D, Brodbeck C, Goj R, Jas M,
473 Brooks T, Parkkonen L, Hämäläinen M (2013) MEG and EEG data analysis with MNE-
474 Python. *Front Neurosci* 7 Available at: <https://pubmed.ncbi.nlm.nih.gov/24431986/>.
- 475 Haegens S, Nacher V, Hernández A, Luna R, Jensen O, Romo R (2011) Beta oscillations in
476 the monkey sensorimotor network reflect somatosensory decision making. *Proc Natl*
477 *Acad Sci U S A* 108:10708–10713.
- 478 Halmes G, Mäki-Marttunen T, Keller D, Pettersen KH, Andreassen OA, Einevoll GT (2016)
479 Effect of Ionic Diffusion on Extracellular Potentials in Neural Tissue. *PLoS Comput Biol*
480 12 Available at: <https://pubmed.ncbi.nlm.nih.gov/27820827/>.
- 481 Hari R, Forss N, Avikainen S, Kirveskari E, Salenius S, Rizzolatti G (1998) Activation of hu-
482 man primary motor cortex during action observation: A neuromagnetic study. *Proc Natl*
483 *Acad Sci U S A* 95:15061–15065.
- 484 Hari R, Salmelin R (1997) Human cortical oscillations: A neuromagnetic view through the
485 skull. *Trends in Neurosciences* 20:44–49 Available at: [https://pub-](https://pubmed.ncbi.nlm.nih.gov/9004419/)
486 [med.ncbi.nlm.nih.gov/9004419/](https://pubmed.ncbi.nlm.nih.gov/9004419/).
- 487 He BJ (2014) Scale-free brain activity: Past, present, and future. *Trends in Cognitive Sci-*
488 *ences* 18:480–487 Available at: <https://pubmed.ncbi.nlm.nih.gov/24788139/>.
- 489 Helson P, Lundqvist D, Vinding MC, Kumar A (2023) Cortex-wide topography of 1/f-expo-
490 nent in Parkinson's disease. *bioRxiv:2023.01.19.524792*.
- 491 Hill AT, Clark GM, Bigelow FJ, Lum JAG, Enticott PG (2022) Periodic and aperiodic neural
492 activity displays age-dependent changes across early-to-middle childhood. *Dev Cogn*
493 *Neurosci* 54:101076.
- 494 Illman M, Laaksonen K, Jousmäki V, Forss N, Piitulainen H (2022) Reproducibility of
495 Rolandic beta rhythm modulation in MEG and EEG. *J Neurophysiol* 127:559–570.
- 496 Jones SR (2016) When brain rhythms aren't "rhythmic": implication for their mechanisms and
497 meaning. *Current Opinion in Neurobiology* 40:72–80 Available at: [https://pub-](https://pubmed.ncbi.nlm.nih.gov/27400290/)
498 [med.ncbi.nlm.nih.gov/27400290/](https://pubmed.ncbi.nlm.nih.gov/27400290/).
- 499 Jones SR, Pritchett DL, Sikora MA, Stufflebeam SM, Hämäläinen M, Moore CI (2009) Quan-
500 titative analysis and biophysically realistic neural modeling of the MEG mu rhythm:
501 Rhythmogenesis and modulation of sensory-evoked responses. *J Neurophysiol*
502 102:3554–3572.
- 503 Kandel ER, Schwartz JH, Jessell TM (2000) The Anatomical Organization of the Central
504 Nervous System. In: *Principles of Neural Science*, pp 317–336.
- 505 Koten JW, Wood G, Hagoort P, Goebel R, Propping P, Willmes K, Boomsma DI (2009) Ge-
506 netic contribution to variation in cognitive function: An fMRI study in twins. *Science*
507 323:1737–1740.
- 508 Laaksonen K, Kirveskari E, Mäkelä JP, Kaste M, Mustanoja S, Nummenmaa L, Tatlisumak

509 T, Forss N (2012) Effect of afferent input on motor cortex excitability during stroke re-
510 recovery. *Clin Neurophysiol* 123 Available at:
511 <http://dx.doi.org/10.1016/j.clinph.2012.05.017>.

512 Law RG, Pugliese S, Shin H, Sliva DD, Lee S, Neymotin S, Moore C, Jones SR (2022)
513 Thalamocortical Mechanisms Regulating the Relationship between Transient Beta
514 Events and Human Tactile Perception. *Cereb Cortex* 32:668–688.

515 Leppäaho E, Renvall H, Salmela E, Kere J, Salmelin R, Kaski S (2019) Discovering heritable
516 modes of MEG spectral power. *Hum Brain Mapp* 40:1391–1402.

517 Lindén H, Pettersen KH, Einevoll GT (2010) Intrinsic dendritic filtering gives low-pass power
518 spectra of local field potentials. *J Comput Neurosci* 29:423–444.

519 Lorente de No R (1949) Cerebral Cortex. Architecture, intracortical connections, motor pro-
520 jections. In: *Physiology of the Nervous System*, 3rd ed. (Fulton JF, ed), pp 288–330.
521 New York: Oxford University Press.

522 Mahjoory K, Schoffelen JM, Keitel A, Gross J (2020) The frequency gradient of human rest-
523 ing-state brain oscillations follows cortical hierarchies. *Elife* 9 Available at:
524 <http://dx.doi.org/10.7554/ELIFE.53715>.

525 Maniscalco B, Lee JL, Abry P, Lin A, Holroyd T, He BJ (2018) Neural integration of stimulus
526 history underlies prediction for naturalistically evolving sequences. *Journal of Neurosci-*
527 *ence* 38:1541–1557.

528 McSweeney M, Morales S, Valadez EA, Buzzell GA, Fox NA (2021) Longitudinal age- and
529 sex-related change in background aperiodic activity during early adolescence. *Dev*
530 *Cogn Neurosci* 52:101035.

531 Mo C, Sherman SM (2019) A sensorimotor pathway via higher-order thalamus. *Journal of*
532 *Neuroscience* 39:692–704.

533 Molina JL, Voytek B, Thomas ML, Joshi YB, Bhakta SG, Talledo JA, Swerdlow NR, Light GA
534 (2020) Memantine Effects on Electroencephalographic Measures of Putative Excita-
535 tory/Inhibitory Balance in Schizophrenia. *Biological Psychiatry: Cognitive Neuroscience*
536 *and Neuroimaging* 5:562–568.

537 Neymotin SA, Daniels DS, Caldwell B, McDougal RA, Carnevale NT, Jas M, Moore CI,
538 Hines ML, Hämäläinen M, Jones SR (2020) Human neocortical neurosolver (HNN), a
539 new software tool for interpreting the cellular and network origin of human MEG/EEG
540 data. *Elife* 9 Available at: <https://pubmed.ncbi.nlm.nih.gov/31967544/>.

541 Ostlund BD, Alperin BR, Drew T, Karalunas SL (2021) Behavioral and cognitive correlates of
542 the aperiodic (1/f-like) exponent of the EEG power spectrum in adolescents with and
543 without ADHD. *Dev Cogn Neurosci* 48 Available at: [https://pub-](https://pubmed.ncbi.nlm.nih.gov/33535138/)
544 [med.ncbi.nlm.nih.gov/33535138/](https://pubmed.ncbi.nlm.nih.gov/33535138/).

545 Pauls KAM, Korsun O, Nenonen J, Nurminen J, Liljeström M, Kujala J, Pekkonen E, Renvall
546 H (2022) Cortical beta burst dynamics are altered in Parkinson's disease but normalized
547 by deep brain stimulation. *Neuroimage* 257 Available at: [https://pub-](https://pubmed.ncbi.nlm.nih.gov/35569783/)
548 [med.ncbi.nlm.nih.gov/35569783/](https://pubmed.ncbi.nlm.nih.gov/35569783/).

- 549 Pauls KAM, Nurmi P, Ala-Salomäki H, Renvall H, Kujala J, Liljeström M (2023) Human sensorimotor resting state beta events and 1/f response show good test-retest reliability. bioRxiv Available at: <http://dx.doi.org/10.1101/2023.08.16.553499>.
- 552 Peper JS, Brouwer RM, Boomsma DI, Kahn RS, Hulshoff Pol HE (2007) Genetic influences on human brain structure: A review of brain imaging studies in twins. *Human Brain Mapping* 28:464–473 Available at: <https://pubmed.ncbi.nlm.nih.gov/17415783/>.
- 555 Pettersen KH, Lindén H, Tetzlaff T, Einevoll GT (2014) Power Laws from Linear Neuronal Cable Theory: Power Spectral Densities of the Soma Potential, Soma Membrane Current and Single-Neuron Contribution to the EEG. *PLoS Comput Biol* 10 Available at: <https://pubmed.ncbi.nlm.nih.gov/25393030/>.
- 559 Pfurtscheller G, Krausz G, Neuper C (2001) Mechanical stimulation of the fingertip can induce bursts of β oscillations in sensorimotor areas. *J Clin Neurophysiol* 18:559–564.
- 561 Porjesz B et al. (2002) Linkage disequilibrium between the beta frequency of the human EEG and a GABAA receptor gene locus. *Proc Natl Acad Sci U S A* 99:3729–3733.
- 563 Reis C, Sharott A, Magill PJ, van Wijk BCM, Parr T, Zeidman P, Friston KJ, Cagnan H (2019) Thalamocortical dynamics underlying spontaneous transitions in beta power in Parkinsonism. *Neuroimage* 193:103–114.
- 566 Renvall H, Salmela E, Vihla M, Illman M, Leinonen E, Kere J, Salmelin R (2012) Genome-wide linkage analysis of human auditory cortical activation suggests distinct loci on chromosomes 2, 3, and 8. *Journal of Neuroscience* 32:14511–14518.
- 569 Sacchet MD, LaPlante RA, Wan Q, Pritchett DL, Lee AKC, Hämäläinen M, Moore CI, Kerr CE, Jones SR (2015) Attention drives synchronization of alpha and beta rhythms between right inferior frontal and primary sensory neocortex. *Journal of Neuroscience* 35:2074–2082.
- 573 Salmela E, Renvall H, Kujala J, Hakosalo O, Illman M, Vihla M, Leinonen E, Salmelin R, Kere J (2016) Evidence for genetic regulation of the human parieto-occipital 10-Hz rhythmic activity. *Eur J Neurosci* 44:1963–1971.
- 576 Salmelin R, Hari R (1994) Spatiotemporal characteristics of sensorimotor neuromagnetic rhythms related to thumb movement. *Neuroscience* 60:537–550.
- 578 Schmitt JE, Neale MC, Fassassi B, Perez J, Lenroot RK, Wells EM, Giedd JN (2014) The dynamic role of genetics on cortical patterning during childhood and adolescence. *Proc Natl Acad Sci U S A* 111:6774–6779.
- 581 Schmitt JE, Raznahan A, Liu S, Neale MC (2020) The genetics of cortical myelination in young adults and its relationships to cerebral surface area, cortical thickness, and intelligence: A magnetic resonance imaging study of twins and families: Genetics of Cortical Myelination, Area, Thickness, and Intelligence. *Neuroimage* 206 Available at: <https://pubmed.ncbi.nlm.nih.gov/31678229/>.
- 586 Seedat ZA, Quinn AJ, Vidaurre D, Liuzzi L, Gascoyne LE, Hunt BAE, O'Neill GC, Pakenham DO, Mullinger KJ, Morris PG, Woolrich MW, Brookes MJ (2020) The role of transient spectral “bursts” in functional connectivity: A magnetoencephalography study. *Neuroimage* 209 Available at: <http://dx.doi.org/10.1016/j.neuroimage.2020.116537>.

- 590 Semenova U, Popov V, Tomskiy A, Shaikh AG, Sedov A (2021) Pallidal 1/f asymmetry in pa-
591 tients with cervical dystonia. *Eur J Neurosci* 53:2214–2219.
- 592 Sherman MA, Lee S, Law R, Haegens S, Thorn CA, Hämäläinen MS, Moore CI, Jones SR
593 (2016) Neural mechanisms of transient neocortical beta rhythms: Converging evidence
594 from humans, computational modeling, monkeys, and mice. *Proc Natl Acad Sci U S A*
595 113:E4885–E4894.
- 596 Shin H, Law R, Tsutsui S, Moore CI, Jones SR (2017) The rate of transient beta frequency
597 events predicts behavior across tasks and species. *Elife* 6 Available at:
598 <http://dx.doi.org/10.7554/eLife.29086>.
- 599 Shinomoto S, Miura K, Koyama S (2005) A measure of local variation of inter-spike intervals.
600 In: *BioSystems*, pp 67–72. Biosystems.
- 601 Silén T, Forss N, Jensen O, Hari R (2000) Abnormal reactivity of the ~20-Hz motor cortex
602 rhythm in unverricht lundborg type progressive myoclonus epilepsy. *Neuroimage*
603 12:707–712.
- 604 Smit CM, Wright MJ, Hansell NK, Geffen GM, Martin NG (2006) Genetic variation of individ-
605 ual alpha frequency (IAF) and alpha power in a large adolescent twin sample. *Int J Psy-*
606 *chophysiol* 61:235–243.
- 607 Smit DJA, Posthuma D, Boomsma DI, De Geus EJC (2005) Heritability of background EEG
608 across the power spectrum. *Psychophysiology* 42:691–697.
- 609 Taulu S, Kajola M, Simola J (2004) Suppression of interference and artifacts by the signal
610 space separation method. *Brain Topogr* 16:269–275.
- 611 Taulu S, Simola J (2006) Spatiotemporal signal space separation method for rejecting
612 nearby interference in MEG measurements. *Phys Med Biol* 51:1759–1768.
- 613 Terry RD, Masliah E, Salmon DP, Butters N, DeTeresa R, Hill R, Hansen LA, Katzman R
614 (1991) Physical basis of cognitive alterations in Alzheimer's disease: synapse loss is the
615 major correlate of cognitive impairment. *Ann Neurol* 30:572–580.
- 616 Van Baal GCM, De Geus EJC, Boomsma DI (1996) Genetic architecture of EEG power
617 spectra in early life. *Electroencephalogr Clin Neurophysiol* 98:502–514.
- 618 Van Beijsterveldt CEM, Molenaar PCM, De Geus EJC, Boomsma DI (1996) Heritability of
619 human brain functioning as assessed by electroencephalography. *Am J Hum Genet*
620 58:562–573.
- 621 Van Ede F, De Lange F, Jensen O, Maris E (2011) Orienting attention to an upcoming tactile
622 event involves a spatially and temporally specific modulation of sensorimotor alpha- and
623 beta-band oscillations. *Journal of Neuroscience* 31:2016–2024.
- 624 van Pelt S, Boomsma DI, Fries P (2012) Magnetoencephalography in twins reveals a strong
625 genetic determination of the peak frequency of visually induced gamma-band synchroni-
626 zation. *Journal of Neuroscience* 32:3388–3392.
- 627 Vinding MC, Tsitsi P, Waldthaler J, Oostenveld R, Ingvar M, Svenningsson P, Lundqvist D
628 (2020) Reduction of spontaneous cortical beta bursts in Parkinson's disease is linked to

629 symptom severity. *Brain Communications* 2 Available at: [http://dx.doi.org/10.1093/brain-](http://dx.doi.org/10.1093/brain-comms/fcaa052)
630 [comms/fcaa052](http://dx.doi.org/10.1093/brain-comms/fcaa052).

631 Visscher PM, Hill WG, Wray NR (2008) Heritability in the genomics era--concepts and mis-
632 conceptions. *Nat Rev Genet* 9:255–266.

633 Voytek B, Kramer MA, Case J, Lepage KQ, Tempesta ZR, Knight RT, Gazzaley A (2015)
634 Age-related changes in 1/f neural electrophysiological noise. *Journal of Neuroscience*
635 35:13257–13265.

636 Waschke L, Donoghue T, Fiedler L, Smith S, Garrett DD, Voytek B, Obleser J (2021) Modal-
637 ity-specific tracking of attention and sensory statistics in the human electrophysiological
638 spectral exponent. *Elife* 10 Available at: <https://pubmed.ncbi.nlm.nih.gov/34672259/>.

639 Wigginton JE, Abecasis GR (2005) PEDSTATS: Descriptive statistics, graphics and quality
640 assessment for gene mapping data. *Bioinformatics* 21:3445–3447.

641 Wilson LE, Castanheira J da S, Baillet S (2022) Time-resolved parameterization of aperiodic
642 and periodic brain activity. *Elife* 11 Available at: [https://pub-](https://pubmed.ncbi.nlm.nih.gov/36094163/)
643 [med.ncbi.nlm.nih.gov/36094163/](https://pubmed.ncbi.nlm.nih.gov/36094163/).

644 **Figure Captions**

645 **Figure 1: Extraction of sensorimotor beta phenotype characteristics.**

646 **A.** Channel selection. A region of interest (ROI) was defined for both hemispheres. The 15 selected
647 gradiometer-channel pairs were combined into 15 vector-sum PSDs (one per channel pair). The peri-
648 odic spectral component of the vector-sum PSD was obtained using FOOOF. From these, a peak beta
649 frequency and peak channel pair were selected. **B.** Beta event extraction. The peak channel pair and
650 peak frequency selected in A were used to calculate the channel pair's amplitude envelope. From the
651 raw data, narrow-band filtered data were obtained using wavelet decomposition, and the individual
652 channels' band-filtered signals were combined to one amplitude envelope using vector sum calculation.
653 **C.** Parameters for heritability analysis. Both PSD characteristics (beta peak power and frequency, total
654 beta power at 14-30 Hz (periodic part), 1/f exponent; upper panel) and time-resolved beta oscillatory
655 characteristics (beta events; lower panel) were used in the heritability analysis.

656

657 **Figure 2: Beta phenotypes**

658 **A. Phenotypic spectrum of beta activity.** Examples of typical beta range PSD patterns: (a) narrow
659 beta peak, (b) broad range, 'beta brush' like activity, (c) double peaks of comparable strength, one in
660 the lower, one in the higher beta range. **B. Beta PSD patterns in siblings.** Examples of siblings' beta
661 PSD patterns (two families with two siblings, one family with three siblings, one family with four sib-
662 lings).

663

664 **Figure 3. Sources of MEG signals and their putative relationship to the MEG parameters exam-** 665 **ined in the present study.**

666 The schematic figure's upper panel summarizes the main anatomical and morphological factors, as well
667 as factors determining timing of events, that contribute to the generation of MEG signals. The lower part
668 indicates the putative relationship of those factors to the MEG parameters examined here. We postulate
669 that the 1/f signal and beta event amplitude parameters are more heavily dependent on fixed, anatom-
670 ical parameters, whereas beta event duration and its modulation are more dynamic characteristics, yet
671 keeping in mind that timing is very much constrained by network anatomy. Brain slice modified from

672 [https://commons.wikimedia.org/wiki/File:Human_basal_ganglia_nuclei_as_shown_in_two_coro-](https://commons.wikimedia.org/wiki/File:Human_basal_ganglia_nuclei_as_shown_in_two_coronal_slices_and_with_reference_to_an_illustration_in_the_sagittal_plane.svg)
 673 [nal_slices_and_with_reference_to_an_illustration_in_the_sagittal_plane.svg](https://commons.wikimedia.org/wiki/File:Human_basal_ganglia_nuclei_as_shown_in_two_coronal_slices_and_with_reference_to_an_illustration_in_the_sagittal_plane.svg)

674

PSD characteristics		Left hemisphere				Right hemisphere			
		mean	median	std	range	mean	median	std	range
peak beta frequency (Hz)		19.7	19.3	3.0	14.1-25.8	19.8	19.3	3.1	14.1-29.3
peak beta power (fT/cm) ²		276	144	374	15-3231	133	70	173	5-1251
total beta band power (periodic)		297 9	1686	331 8	164- 16284	109 3	619	132 2	26-9652
1/f component exponent		1.03	1.01	0.1 9	0.34-1.76	1.07	1.05	0.1 8	0.67-1.66
1/f component offset		- 22.9 5	- 22.99	0.3 7	-23.75- 21.75	- 23.2 8	- 23.32	0.3 4	-24.19- 22.25
Beta event characteristics		mean	median	std	range	mean	median	std	range
duration (ms)	mean	256. 9	248.0	49. 4	181.7- 498.2	265. 2	253.7	50. 0	182.0- 454.0
	median	199. 0	195.0	32. 3	152.5- 420.0	201. 4	195.0	34. 9	150.0- 355.0
	standard deviation	198. 4	182.4	63. 2	99.8- 487.5	213. 0	198.3	67. 4	67.3- 531.7
	robust maximum	858. 0	788.6	257. 4	439.5- 2145.0	912. 1	852.2	263. 9	382.7- 2135.0
amplitude (fT/cm)	mean	325	285	165	104-994	221	188	113	73-654
	median	301	260	155	93-984	203	173	105	70-618
	standard deviation	86	71	48	21-264	62	52	34	14-202
	robust maximum	564	486	286	174-1467	390	335	196	114-1128
event rate (1/s)		1.00	1.00	0.1 6	0.50-1.36	0.97	0.98	0.1 6	0.55-1.37
dispersion		1.14	1.05	0.4 4	0.65-5.59	1.16	1.06	0.4 4	0.41-5.58

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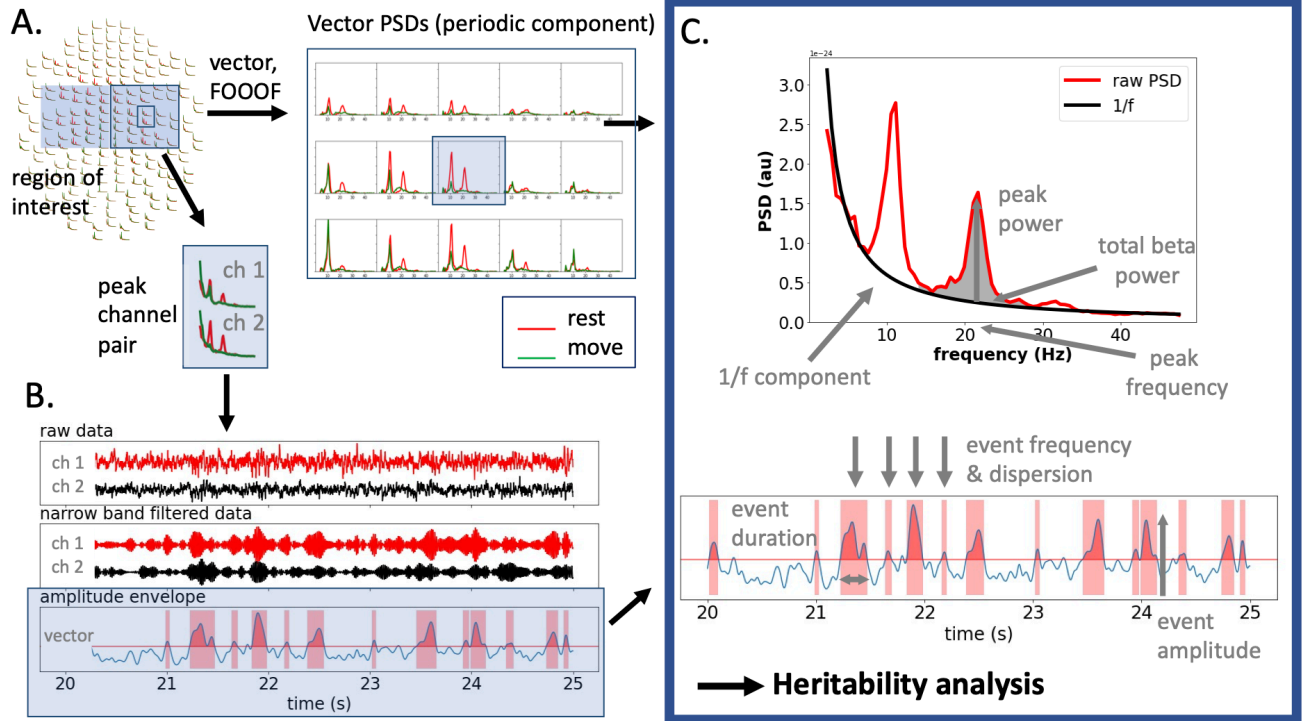
		Left hemi- sphere			Right hemi- sphere		
PSD characteristics		h ²	p	n sig. (/6000)	h ²	p	n sig. (/6000)
peak beta frequency		0.45	0.0047	28	0.41	0.0103	62
peak beta power		0.28	0.0648	389	0.58	0.0072	43
total beta band power (peri- odic)		0.49	0.0068	41	0.44	0.0157	94
1/f component exponent *		0.47	0.0035	21	0.87	0.0000	0
1/f component offset *		0.35	0.0258	155	0.69	0.0000	0
Beta event characteristics		h ²	p	n sig. (/6000)	h ²	p	n sig. (/6000)
duration	mean	0.45	0.1350	810	0.36	0.0222	133
	median	0.28	0.1338	803	0.40	0.0172	103

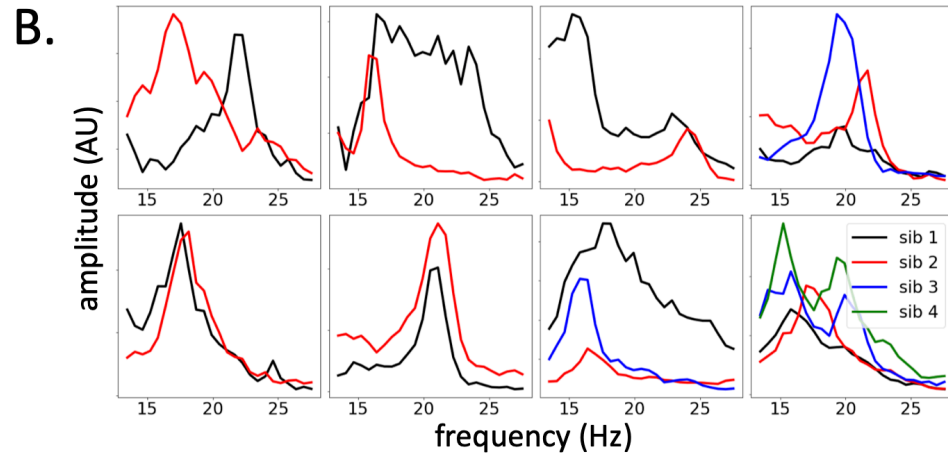
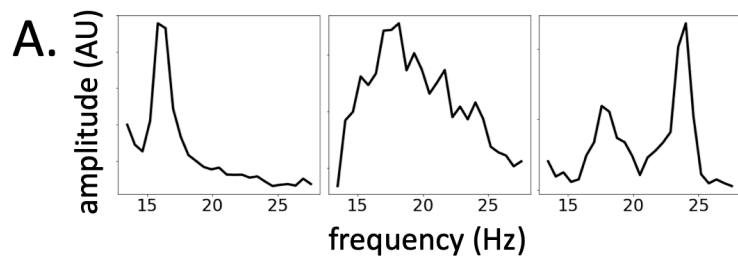
	standard deviation	0.49	0.2495	1497	0.32	0.0412	247
	robust maximum	0.47	0.2383	1430	0.33	0.0372	223
amplitude	mean *	0.35	0.0060	36	0.75	0.0002	1
	median *	0.45	0.0110	66	0.72	0.0002	1
	standard deviation *	0.28	0.0005	3	0.81	0.0000	0
	robust maximum *	0.49	0.0007	4	0.79	0.0000	0
	event rate	0.47	0.0543	326	0.38	0.0137	82
	dispersion	0.35	0.2850	1710	0.00	1.0000	6000

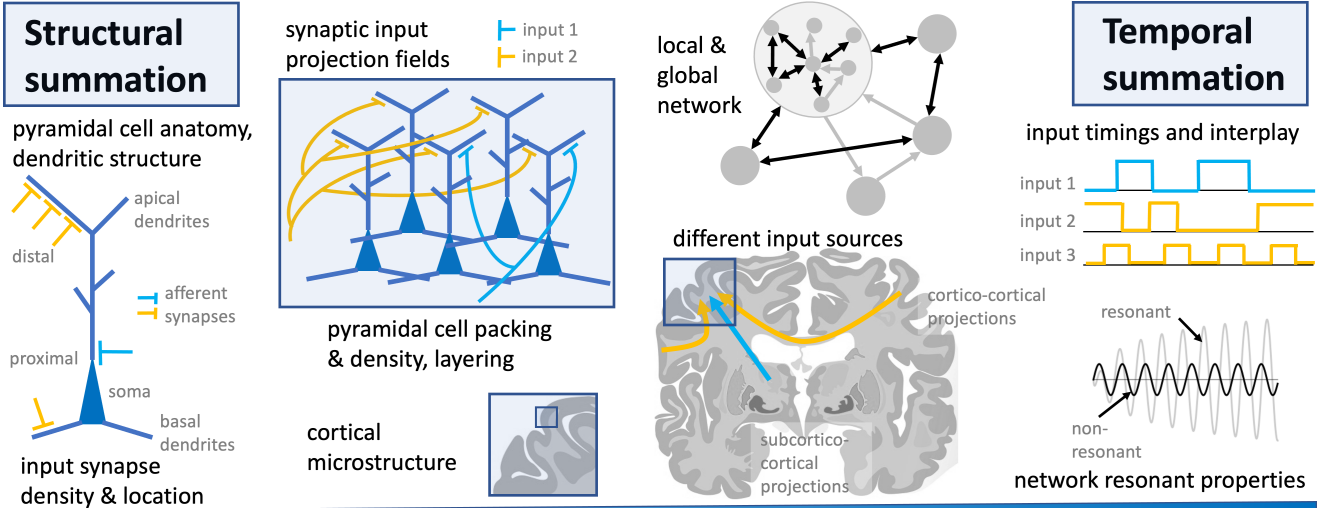
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TIMING	
ANATOMY	DYNAMIC
FIXED	DYNAMIC

1/f signal component
 tissue/apical dendrite filtering properties
 → highly heritable

beta event amplitude & its dynamic range
 size, morphology and biophysical properties of cortical cells, cortical microstructure
 → heritable

beta event duration & dynamics
 interplay of inputs from different sources and their interaction, network resonance properties → not significantly heritable