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https://doi.org/10.1523/JNEUROSCI.1887-19.2019

Cite as: J. Neurosci 2019; 10.1523/JNEUROSCI.1887-19.2019

Received: 3 August 2019
Revised: 11 November 2019
Accepted: 12 November 2019

This Early Release article has been peer-reviewed and accepted, but has not been through the composition and copyediting processes. The final version may differ slightly in style or formatting and will contain links to any extended data.

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Behavioral / Cognitive

**β-bursts reveal the trial-to-trial dynamics of movement initiation and cancellation**

Abbreviated title: **β-bursts index movement initiation & cancellation**

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Number of pages: 33 Number of figures: 5 Number of tables: 0

Abstract word count: 250 Intro word count: 518 Discussion: 1497

**Acknowledgements**

The author would like to thank the following individuals for help with data collection: Daniel Thayer, Julian Scheffer, Cailey Parker, Hailey Billings, Carly Ryder, Megan Hynd, Carly Iacullo, Brynne Dochterman, Isabella Dutra, Alec Mather, Kylie Dolan, Nathan Chalkley, Darcy Waller, Tobin Dykstra, and Cheol Soh. Furthermore, the author would like to thank Mark Blumberg, John Freeman, and Bob McMurray for helpful discussions. Additionally, the author would like to thank Freek van Ede, Eric Maris, and Jan-Mathijs Schoffelen for help with the lagged coherence method, and Bernhard Spitzer for suggesting it. This research was supported by grants from the National Institute of Health (R01 NS102201), the National Science Foundation (CAREER 1752355), and the Roy J Carver Charitable Trust (Research Program of Excellence 17-4885).

**COI:** None reported.
Abstract

The neurophysiological basis of motor control is of substantial interest to basic researchers and clinicians alike. Motor processes are accompanied by prominent field potential changes in the $\beta$-frequency band (15-29Hz): in trial-averages, movement initiation is accompanied by $\beta$-band desynchronization over sensorimotor areas, whereas movement cancellation is accompanied by $\beta$-power increases over (pre)frontal areas. However, averaging misrepresents the true nature of the $\beta$-signal. Unaveraged $\beta$-band activity is characterized by short-lasting, burst-like events, rather than by steady modulations. Hence, averaging-based quantifications may miss important brain-behavior relationships. To investigate how $\beta$-bursts relate to movement in male and female humans (N=234), we investigated scalp-recorded $\beta$-band activity during the Stop-signal task, which operationalizes both movement initiation and cancellation. Both processes were indexed by systematic spatiotemporal changes in $\beta$-burst rates. Before movement initiation, $\beta$-bursting was prominent at bilateral sensorimotor sites. These burst-rates predicted reaction time (a relationship that was absent in trial-average data), suggesting that sensorimotor $\beta$-bursting signifies an inhibited motor system, which has to be overcome to initiate movements. Indeed, during movement initiation, sensorimotor burst-rates steadily decreased, lateralizing just prior to movement execution. In contrast, successful movement cancellation was signified by increased phasic $\beta$-bursting over fronto-central sites. Such $\beta$-bursts were followed by short-latency increases of bilateral sensorimotor $\beta$-burst rates, suggesting that motor inhibition can be rapidly re-instantiated by frontal areas when movements have to be suddenly cancelled. Together, these findings suggest that $\beta$-bursting
is a fundamental signature of the motor system, used by both sensorimotor and frontal
areas involved in the trial-by-trial control of behavior.
Significance Statement

Movement-related β-frequency (15-29Hz) changes are among the most prominent features of neural recordings across species, scales, and methods. However, standard averaging-based methods obscure the true dynamics of β-band activity, which is dominated by short-lived, burst-like events. Here, we demonstrate that both movement-initiation and cancellation in humans are characterized by unique trial-to-trial patterns of β-bursting. Movement initiation is characterized by steady reductions of β-bursting over bilateral sensorimotor sites. In contrast, during rapid movement cancellation, β-bursts first emerge over fronto-central sites typically associated with motor control, after which sensorimotor β-bursting re-initiates. These findings suggest a fundamentally novel, non-invasive measure of the neural interaction underlying movement-initiation and – cancellation, opening new avenues for the study of motor control in health and disease.
Activity in the β-frequency band (15-29Hz) is a prominent constituent of the neural local field potential. It can be observed at spatial scales ranging from extracellular to scalp recordings, in species ranging from rodents to humans, and using methods ranging from intracranial recordings to magnetoencephalography (Murthy and Fetz, 1992; Sanes and Donoghue, 1993; Engel and Fries, 2010; Shin et al., 2017). The β-frequency plays a particularly important role in the functioning of the motor system. In particular, during movement initiation, a prominent desynchronization of β-band activity is clearly observable over sensorimotor areas (McFarland et al., 2000; Pfurtscheller et al., 2003). In contrast, the rapid cancellation of movement is accompanied by β-power increases over (pre-)frontal cortical areas generally implicated in cognitive control (Swann et al., 2009; Swann et al., 2011a; Swann et al., 2012; Picazio et al., 2014; Wagner et al., 2018). Moreover, movement-related changes in β-power can also be observed in extrapyramidal parts of the motor system, including the basal ganglia (Kuhn et al., 2004; Ray et al., 2012; Brittain and Brown, 2014; Wessel et al., 2016), where abnormal β-rhythms are prominently observed in movement disorders such as Parkinson’s Disease (Hammond et al., 2007; Bronte-Stewart et al., 2009; Jenkinson and Brown, 2011; Little and Brown, 2014; Quinn et al., 2015).

Recent studies of raw, unaveraged β-band activity, however, have led to a significant reappraisal of the nature of neural activity in the β-band. While trial-averaging approaches suggest that movement-related changes in the β-band reflect steady (de)synchronizations that stretch over several hundred milliseconds, unaveraged β-band activity is primarily characterized by rapid burst-like events, which typically last less than ~150ms (Leventhal et al., 2012; Feingold et al., 2015; Sherman et al., 2016). While these burst-events appear as
slow-evolving (de)synchronizations when averaged across trials, analyses of single-trial data have found that the simple presence or absence of these β-bursts, rather than overall changes in β-power, is the most reliable predictor of trial-to-trial behavior (Shin et al., 2017).

Here, we therefore investigated the characteristics of single-trial β-bursting in humans during both the initiation and the rapid cancellation of movement. A large sample of healthy human participants (N=234) performed the stop-signal task (Logan and Cowan, 1984; Verbruggen et al., 2019), a motor task that includes both instances of movement initiation (following a Go-signal) and movement cancellation (on a subset of trials that include a subsequent Stop-signal). We used non-invasive scalp-EEG recordings to investigate how β-bursting on individual trials indexes both processes, as well as their interaction. Specifically, we investigated four questions: 1. Is human β-band activity during movement burst-like? 2. If so, do systematic spatiotemporal patterns of β-burst activity distinguish successful from unsuccessful movement cancellation, as would be expected based on the trial-average literature on β-power cited above? 3. Are there systematic relationships between initiation and cancellation-related changes in β-bursting when movements have to be rapidly stopped? 4. Do β-bursts provide a more accurate representation of brain-behavior relationships compared to changes in overall β-amplitude?

Materials and Methods

Participants

234 healthy adult humans (mean age: 22.7, SEM: .43, 137 female, 25 left-handed) from the Iowa City community participated in the study, either for course credit or for an
hourly payment. 123 of those datasets were published as part of other studies, none of which focused on β-bursting (Wessel, 2016, 2017; Dutra et al., 2018; Waller et al., 2019).

All procedures were approved by the local ethics committee at the University of Iowa (IRB #201511709).

**Task**

The task was identical to the one described in (Wessel, 2016, 2017; Dutra et al., 2018; Waller et al., 2019). In short, trials began with a fixation cross (500ms duration), followed by a white left- or rightward arrow (Go-signal). Participants were instructed to respond as fast and accurately as possible to the arrow using their left or right index finger (the respective response-buttons were q and p on a QWERTY keyboard). On one-third of trials, a Stop-signal occurred (the arrow turned from white to red) at a delay after the go-stimulus (stop-signal delay, SSD). The SSD, which was initially set to 200ms, was dynamically adjusted in 50ms increments to achieve a p(stop) of .5: after successful stops, the SSD was prolonged; after failed stops, it was shortened. This was done independently for left- and rightward go-stimuli. Trial duration was fixed at 3,000ms. Six blocks of 50 trials were performed (200 Go, 100 Stop).

**Data availability**

All data, procedures, and analysis routines can be downloaded on the Open Science Framework at [URL to be inserted after acceptance].

**EEG recording**
Scalp-EEG was recorded using two different BrainProducts (Garching, Germany) systems – one active (actiChamp) and one passive (MR plus). In both cases, 62-channel electrode caps with two additional electrodes on the left canthus (over the lateral part of the orbital bone of the left eye) and over the part of the orbital bone directly below the left eye were used. The ground was placed at electrode Fz, and the reference was placed at electrode Pz. EEG was digitized at a sampling rate of 500 Hz, with hardware filters set to 10s time-constant high-pass and 1000 Hz low-pass.

**EEG data preprocessing**

Data were preprocessed using custom routines in MATLAB, incorporating functions from the EEGLAB toolbox (Delorme and Makeig, 2004), RRID: SCR_007292. The electrode *time-series matrices for each task were imported into MATLAB and then filtered using symmetric two-way least-squares finite impulse response filters (high-pass cutoff: .3 Hz, low-pass cutoff: 30 Hz). Non-stereotyped artifacts were automatically removed from further analysis using segment statistics applied to each second-long segment of data (joint probability and joint kurtosis, with both cutoffs set to 5 SD, cf., Delorme et al., 2007). Trials that included a rejected data segment were excluded from further analysis. After removal of non-stereotypic artifacts, the data were re-referenced to common average and subjected to a temporal infomax ICA decomposition algorithm (Bell and Sejnowski, 1995), with extension to subgaussian sources (Lee et al., 1999). The resulting component matrix was screened for components representing eye-movement and electrode artifacts using outlier statistics and non-dipolar components (residual variance cutoff at 15%, Delorme et al., 2012), which were removed from the data using automated outlier-based statistics (Wessel, 2002).
The remaining components were subjected to further analyses. For all statistical analyses and all plots (except the topographical plots in Figure 1a and 2a), the data were subsequently transformed to a reference-free montage using the current-source density method (Perrin et al., 1989; Tenke and Kayser, 2005), which minimizes the effects of volume conduction on the scalp-measured activity. This was done to enable β-event detection at fronto-central electrode FCz and lateral sensorimotor electrodes C3 and C4 while reducing the change of cross-contamination by either side.

**β-burst detection**

β-burst detection was performed exactly as described in Shin and colleagues’ work (Shin et al., 2017). The description is adapted from therein. First, each electrode’s data was convolved with a complex Morlet wavelet of the form:

\[
w(t, f) = A \exp\left(\frac{t^2}{2\sigma_t^2}\right) \exp(2i\pi ft)
\]

with \(\sigma = \frac{m}{2\pi f}, A = \frac{1}{\sigma_t} \sqrt{2\pi}\), and \(m = 7\) (cycles) for each of 15 evenly spaced frequencies spanning the β-band (15-29Hz). Time-frequency power estimates were extracted by calculating the squared magnitude of the complex wavelet-convolved data. These power estimates were then epoched relative to the events in question (ranging from -500 to +1,000ms with respect to Stop- / Go-signals). Individual β-bursts were defined as local maxima in the trial-by-trial β-band time-frequency power matrix for which the power exceeded a set cutoff of 6x the median power.
of the entire time-frequency power matrix for that electrode. Local maxima were identified using the MATLAB function imregional().

**Experimental design and statistical analysis**

**Behavioral analysis**

Means were extracted for each subject for the following measures: Go-trial reaction time, failed Stop-trial reaction time, Stop-signal delay, Stopping accuracy, and Stop-signal reaction time, which was calculated via the integration method with replacement of miss-trial reaction times (Verbruggen et al., 2019). Failed Stop-trial reaction times and Go-trial reaction times were compared with a paired samples t-test.

**Topographical distribution of $\beta$-bursts (Figures 1a, 2a, 3)**

To visualize the topographical distribution of $\beta$-bursts with respect to Stop- and Go-signals, 12 windows of 25ms length, starting at 25ms after the event, were defined. For each subject, the number of $\beta$-bursts in each window at each electrode following the respective stimulus (Go/Stop) was counted. The average number of $\beta$-bursts in each time window for each of the three trial types (Correct Go, Successful Stop, Failed Stop) was then plotted in a topographical grid representing the scalp surface (Figures 1a and 2a). Figure 3 depicts the difference between the number of $\beta$-bursts on Go- and Stop-trials in each window and at each electrode, tested for significance using paired-samples t-tests. The resulting electrode * time window matrix of p-values was corrected for multiple comparisons to a significance level of $p = .0001$ using the false-discovery rate procedure (FDR, (Benjamini et al., 2006)).
Temporal development of β-bursts (Figures 1b, 2b, 2d)

To visualize the temporal development of β-bursts on Go-trials at the two electrodes of interest (C3 and C4), 11 windows of 50ms length ranging from 25ms post-event to 575ms post-event were defined. This time range spanned the entire post-Go-signal period leading up to mean reaction time. To test the linear decreasing trend observed at C3/C4 during movement initiation for significance, we submitted the means for left- and right-hand responses to the Mann-Kendall test. To test the lateralization of the linear trend towards the end of the response period (i.e., prior to mean RT), we compared the means for left- and right-hand responses at both electrodes using paired-samples t-tests, again corrected for multiple comparisons to a significance level of p = .0001 using the false-discovery rate procedure. To visualize the temporal development of β-bursts on Stop-trials at the electrode of interest (FCz), 6 windows of 50ms length ranging from 25ms post-event to 325ms post-event were defined. This time range spanned the entire post-Stop-signal period leading up to Stop-signal reaction time (SSRT).

Correlation between lateral sensorimotor β-bursts and behavior (Figure 1f)

The relative rates of β-bursts over lateral sensorimotor sites C3 and C4 for each subject and in each time-window following the Go-signal (depicted in Figure 1b,d) were then correlated with each subject’s SSRT and Go-trial reaction time using Pearson’s correlation coefficient. The resulting correlations were tested for significance and the respective p-values were corrected for multiple comparisons using the FDR procedure (12 tests each, critical p < .05).
Comparison of pre-SSRT $\beta$-events (Figure 2c)

To test the difference in the amount of $\beta$-bursts at fronto-central electrode FCz for significance, we counted the number of $\beta$-bursts in the time period ranging from the Stop-signal to each individual participants’ SSRT estimate, separately for successful and failed Stop-trials. Moreover, we counted the number of $\beta$-bursts in a time period of identical length of Go-trials. The time period of interest on Go-trials was the time period ranging from the current Stop-signal delay on that trial and the participants’ SSRT estimate. In other words, it was the Stop-signal-to-SSRT time period had there been a Stop-signal on that trial. The mean number of $\beta$-bursts for these periods were compared across subjects using a paired-samples t-tests.

Lateralized $\beta$-bursting after fronto-central $\beta$-bursts (Figure 4)

To investigate the pattern of $\beta$-bursting over bilateral electrodes C3/4 following fronto-central $\beta$-bursts in the Stop-Signal-to-SSRT period on successful Stop-trials, we identified each trial in which such a fronto-central $\beta$-burst event occurred, and counted the amount of $\beta$-bursts at electrodes C3/4 both contra- and ipsilateral to the to-be-stopped response in eight time windows of 25ms duration ranging from -100 to +100ms around the fronto-central $\beta$-burst event. In case more than one $\beta$-burst event was found, we chose the latency of the first of those events. To compare these $\beta$-burst counts to trials in which no fronto-central $\beta$-burst was found, a random time-point in the Stop-signal-to-SSRT interval was chosen from a uniform distribution and C3/4 $\beta$-bursts were quantified in an identical time window around that random time point in the pre-SSRT period. The distribution of randomly selected timepoints did not significantly deviate from the distribution of $\beta$-burst
onset times on trials with such bursts. We then compared the means burst-counts for the two conditions in the four time-windows following the event (or the 'pseudo-event' in the case of the trial without an actual fronto-central burst) using signed-rank tests (the non-parametrical equivalent of the paired-samples t-test, chosen because of the large skew of the means towards zero in these samples), corrected for multiple comparisons to a critical p-value of $p < .0001$ using the FDR-method.

**$\beta$-power amplitude analysis (Figure 5)**

To conduct the analyses presented in Figure 5, the time-frequency power estimates that were used for $\beta$-burst detection (as described above) were converted to decibel (dB) using a 250ms baseline prior to the event in question (Go-signal / Stop-signal). Activity in a specific time period was then defined not by the amount of individual $\beta$-bursts within in that period, but instead by the average amplitude in that same period. All other analyses were then performed in the same manner as the $\beta$-burst analyses described above.

**Lagged phase coherence analysis**

Lagged phase coherence was quantified as described in (Fransen et al., 2015). Computations were made in a beta version of the fieldTrip software package (Oostenveld et al., 2011), RRID: SCR_004849. Cycle number was set to 3 and frequencies between 8 and 35 Hz were chosen to cover both the $\beta$ band and the surrounding frequencies. Lagged coherence was computed on the epoched Stop-trial data for electrode FCz and on the epoched Go-trial data for electrodes C3 and C4.
Results

Both movement initiation and cancellation are accompanied by systematic spatiotemporal patterns of β-bursts

Figure 1a shows that after Go-signals (which prompt the start of movement initiation), bilateral sensorimotor sites (peaking over electrodes C3 and C4) initially showed localized β-bursting, which decreased in the time period leading up to movement execution, which resulted in a significant downward linear trend across the entire time period depicted in Figure 1b, d (linear trend for left-hand responses at C4: Z = -3.63, p = .00028, left-hand responses at C3: Z = -3.22, p = .001, right-hand responses at C4: Z = -2.81, p = .005, right-hand responses at C3: Z = -3.23, p = .0013). Furthermore, this pattern lateralized towards the end of the response period, with sites contralateral to the response hand showing a stronger sustained reduction in β-bursting (significant lateralization at p < .0001, FDR-corrected, at time five consecutive windows from 325 to 575ms for electrode C4, t(233) = -4.91, p = 1.75*10^{-6}, d = .38, t(233) = -8.25, p = 1.2*10^{-14}, d = .63, t(233) = -6.74, p = 1.24*10^{-10}, d = .52, t(233) = -6.83, p = 7.37*10^{-11}, d = .61, t(233) = -5.75, p = 2.81*10^{-08}, d = .5; and at three consecutive time windows from 325 to 475ms for electrode C3, t(233) = 5.28, p = 2.92*10^{-07}, d = .41, t(233) = 6.02, p = 6.6*10^{-09}, d = .49, t(233) = 4.81, p = 2.65*10^{-06}, d = .37). These findings parallel reports from trial-averaged power-based quantifications of sensorimotor β-band activity (e.g., McFarland et al., 2000; Pfurtscheller et al., 2003). An inspection of individual trial data showed that the single-trial β-band signal was indeed characterized by clearly visible, burst-like events, rather than by steady modulations (Figure 1c,e show data from one representative subject; plots of single trial data for each individual participant can be downloaded from the author's website [Author's note: URL to
be inserted upon acceptance, image files from N=234 were too large in size to be uploaded as supplemental material. I am also open to other options for publication of these data to accompany the manuscript}. 
a) Topographical distribution of β-bursts following GO-signals

Left-hand responses

Right-hand responses

b) Temporal distribution at RIGHT motor electrode C4

burst reduction

mean RT: 533 ms

c) C4: Individual trial data

d) Temporal distribution at LEFT motor electrode C3

burst reduction

mean RT: 533 ms

e) C3: Individual trial data

f) Relationship between Go-signal-locked contralateral C3/C4 β-bursts and GO/STOP behavior
**Figure 1.** β-burst properties during movement initiation on Go-trials. A) Topographical distribution of the average number of β-bursts on the scalp at different consecutive time-windows following the Go-signal, for both left-hand (top) and right-hand (bottom) responses. There are visible bilateral peaks over electrode sites C3 and C4 until about 200ms following the Go-signal. Channels at which the number of bursts did not exceed significant increases from zero (when corrected using FDR) were set to 0. B) Temporal development of β-burst rates following the Go-Signal at right lateral sensorimotor electrode site C4 during both left- and right hand responses. A significant linear trend is evident, such that the β-burst rates steadily decreased after the Go-signal. Moreover, a significant lateralization of this effect is evident starting at 325ms following the Go-signal, such that the β-burst rates for the contralateral hand kept diminishing, while the rates showed an early asymptote for the ipsilateral response hand. C) β-band data at right sensorimotor electrode C4 from individual Go-trials in a representative subject, clearly showing the burst-like nature of β-activity. Each plot shows frequency on the y-axis (15-29Hz) and time relative to the Go-signal on the x-axis, as demarcated on the bottom right trial (labels removed from the other trial plots for easier viewing). These same plots of single trial data for all individual participants can be downloaded from the author’s website [Author’s note: Or any other method to host these plots; the zip file is ~360MB large]. D) Same as B, but for left-lateral electrode C3. E) Same as C, but for C3. F) Inter-subject correlation between average Go-trial reaction time (green dots) / average Stop-signal reaction time (orange dots) and each subject’s C3/C4 β-burst rates contralateral to the instructed movement, separately for each of the time windows in B. Black line = least squares fit; gray lines: confidence intervals. * = p < .05 (FDR-corrected).
Increased amounts of contralateral β-bursting between 75-125ms following the Go-signal were related to longer Go-trial reaction times, as well as shorter Stop-signal reaction times.

With regards to movement cancellation, Figure 2a shows that after Stop-signals (which followed Go-signals on 1/3 of all trials at a variable delay and prompted the participants to attempt to cancel the movement instructed by the Go-signal), no coherent spatiotemporal organization of the rate of β-bursts could be observed until around 200ms after the stop-signal. At that point, a clear radial fronto-central topographical distribution emerged, centered around electrode FCz. Just like Go-signal-related activity at C3 and C4, single-trial Stop-signal-related activity at FCz clearly showed the presence of β-bursting (Figure 2d).
Figure 2. β-burst properties during movement cancellation on Stop-trials. A) Topographical distribution of the number of β-bursts on the scalp at different consecutive time-windows following the Stop-signal, separately for successful (top) and failed (bottom) Stop-trials. In the time-window towards the end of SSRT, a clear fronto-central organization of β-bursting centered around electrode FCz is evident. Channels at which the number of bursts did not exceed significant increases from zero (when corrected using FDR) were set to 0. B) Temporal development of β-burst rates following the Stop-Signal at fronto-central electrode FCz. C) Comparison of the number of β-bursts between successful and failed Stop-trials in the Stop-signal-to-SSRT period (as well as during a matched time period on Go-trials) for each subject. D) β-band data at electrode FCz from 18 individual Stop-trials in a representative subject, clearly showing the burst-like nature of β-activity following the stop-signal (vertical line). Each plot shows frequency on the y-axis (15-29Hz) and time relative to the Stop-signal on the x-axis, as demarcated on the bottom right trial (labels were removed from the remaining trials for easier viewing). The same plots of single trial data for each individual participant can be downloaded from the author’s website.

Fronto-central β-bursting is increased during successful movement cancellation

Behavior in the Stop-signal task was typical for healthy young adults (mean Go-trial reaction time: 534ms, SEM: 6.6; mean failed Stop-trial reaction time: 460ms, SEM: 5.79; stop accuracy: .52, SEM: .002, mean Stop-signal delay: 282ms, SEM: 7.91; mean Stop-Signal reaction time: 245ms, SEM: 3.62). In accordance with the race model, failed Stop-trial reaction times were consistently faster than Go-trial reaction times: t(233) = 34.7, p =
4.9 \times 10^{-94}, d = .78. On the individual-subject level, 232 out of 234 participants showed this pattern.

The observed increase of fronto-central $\beta$-bursting after Stop-signals (compared to Go-signals) starting around 200ms following the Stop-signal was highly significant. Figure 3 shows the topographical difference plots between the distribution of $\beta$-bursts following Stop- vs. Go-trials, thresholded to an effective family-wise $p = .0001$ using the FDR-correction for 62 channels and 12 time windows. Moreover, the time after the Stop-signal at which this fronto-central organization of $\beta$-bursting developed overlaps with the end of SSRT (~245ms). This time period, before the end of SSRT, is the exact time period during which neural activity reflecting movement cancellation should be maximal according to both computational models of the Stop-signal task (Boucher et al., 2007) and neural recordings from the basal ganglia and the frontal-eye fields (Hanes et al., 1998; Ogasawara et al., 2018). Moreover, a direct comparison of the pre-SSRT time period in each individual participant (i.e., the time range between the appearance of the Stop-signal and the end of each participants’ individual SSRT estimate) revealed that successful Stop-trials yielded an increased rate of $\beta$-bursts at FCz compared to failed Stop-trials ($t(233) = 3.46, p < .0007, d = .25$, Figure 2b, c) as well as compared to a matched time-period on Go-trials ($t(233) = 12.28, p = 4.8 \times 10^{-27}, d = .88$). This suggests that fronto-central $\beta$-bursts are related to movement cancellation, with successful stop-trials being accompanied by greater burst-rate increases. These findings parallel reports of increased trial-averaged $\beta$-band power found in intracranial recordings from neurological patients, where increased $\beta$-power can be found at sites in the medial wall of the frontal cortex – most notably, in the pre-supplementary motor area (Swann et al., 2012, Jha et al., 2015).
Figure 3. Statistical comparison of β-burst topographies following Stop- and Go-signals, separately for successful (top) and failed (bottom) Stop-trials. Thresholded for significance at $p < .0001$, FDR-corrected for multiple comparisons across channels and time windows. It is evident that while movement initiation (Go-trials) was accompanied by a significantly relative increase in β-burst count at bilateral sensorimotor sites until ~175ms after the Go-signal, movement cancellation was accompanied by significant relative increase of β-burst rates at fronto-central electrodes, starting at ~200ms after the Stop-signal.

Fronto-central β-bursts are followed by increased bilateral sensorimotor β-bursting

Figure 4a shows the temporal development of β-bursting at sensorimotor sites ipsi- and contralateral to the to-be-stopped movement on successful stop-trials. Importantly, rather than being time-locked to the Stop-signal (or the Go-signal), these plots are time-locked to the latency of the first fronto-central β-burst event that occurred within the pre-SSRT time period (i.e., between the Stop-signal and the participant’s SSRT estimate). These plots show a significant increase in bilateral sensorimotor β-bursting within 25ms of the first fronto-central β-burst. To evaluate significance, these values were compared to matched time-periods on successful stop-trials without fronto-central β-bursts in the pre-
SSRT period (exact values for the pairwise comparisons between trials with and without β-bursts: \( Z = 7.81, p = 5.65 \times 10^{-15} \) and \( Z = 4.36, p = 1.28 \times 10^{-05} \) for the two significant time-windows for contralateral sites and \( Z = 8.06, p = 7.7 \times 10^{-16} \) and \( Z = 4.8, p = 1.55 \times 10^{-06} \) for ipsilateral sites). Additionally, these same time windows of post-burst activity were compared to the average number of sensorimotor β-bursts that occurred on trials with fronto-central bursts, but prior to the occurrence of that bursts (i.e., the average number of bursts in the four time windows prior to the fronto-central β burst on the same trials). That comparison confirmed the significant increase in sensorimotor β bursting following the fronto-central β-burst, though in this analysis, this was limited to the first post-burst window (\( Z = 8.02, p = 1.1 \times 10^{-15} \) for contralateral, \( Z = 6.63, p = 3.35 \times 10^{-11} \) for ipsilateral).

Moreover, the same analysis performed on failed instead of successful stop-trials (which was suggested by a reviewer) yielded no significant increases in sensorimotor β following fronto-central β bursts. In essence, these findings suggest that there is a low-latency increase in lateral sensorimotor β-bursts immediately following fronto-central β-bursts that occur within the Stop-signal-to-SSRT period on successful Stop-trials. Potential effects of volume-conduction are a concern in this analysis (though these data were current-source density transformed, see Materials and Methods). However, a topographical representation of the effect reveals that the increase in β-bursting following the first fronto-central β-burst showed local maxima at bilateral sensorimotor sites (in addition to the fronto-central electrodes surrounding FCz, Figure 4b) – i.e., at remote locations that do not follow a linear or exponential signal decay from FCz. Therefore, while volume conduction effects can never be ruled out in scalp-recordings, there seems to be some specificity of this increase to bilateral sensorimotor sites.
Figure 4. Interaction between fronto-central and bilateral sensorimotor β-bursting during successful movement cancellation. A) Rates of contra- and ipsilateral β-bursts at sensorimotor electrodes C3/C4, time-locked to individual fronto-central β-bursts during the Stop-signal-to-SSRT period. 25ms following fronto-central β-bursts (green highlighting), both sensorimotor β-bursting was significantly increased at sites both ipsi- and contralateral to the to-be-stopped movement. B) Topographical representation of β-bursts in the highlighted time period (25ms following the fronto-central β-burst event) showing local maxima in increased β-burst activity at bilateral sensorimotor sites (in addition to the fronto-central sites that surround FCz).
Early β-bursting at lateral sensorimotor sites may reflect a tonic inhibitory state

The fact that lateral sensorimotor β-bursting steadily declined during movement initiation (Figure 1b, d) and restarted immediately following fronto-central β-bursts during movement cancellation (Figure 4a) suggests that this spatiotemporal signature may reflect an inhibited motor state, which has to be overcome during movement initiation and which can be rapidly reinstated following frontal control signals during movement cancellation. To further test this possibility, we correlated the amount of contralateral sensorimotor β-bursting on Go-trials with the two main behavioral indices for each subject (Go-trial reaction time and Stop-signal reaction time). This was done for the same time windows that were used to visualize these same β-burst data in Figure 1b and 1d. As can be seen in Figure 1f, the amount of lateral sensorimotor β-bursting was significantly positively related to Go-trial reaction times in all nine of the early time windows following the Go-signal (significant time-windows in which the correlation survived FDR-correction covered a time range from the Go-signal to 425ms after the signal). The strongest correlation was found during the early time windows, in particular the ones ranging from 75-175ms after the Go-signal. Specifically, subjects that showed increased rates of β-bursting during these time windows showed systematically longer Go-trial reaction times. Additionally, the rate of early sensorimotor β-bursting showed a negative relationship with SSRT: subjects with higher initial sensorimotor β-bursting rates showed faster SSRT. The latter relationship survived FDR-corrections for multiple comparisons in the 75-125ms post-Go-signal window. Taken together with the Go-reaction time correlation, this suggests that the rate of early β-bursting over lateral somatosensory areas does indeed reflect a (proactive)
inhibition of the motor system, which is detrimental to fast movement execution (as reflected in slower Go-trial reaction times), but beneficial to fast movement cancellation (as reflected in faster Stop-signal reaction times).

β-burst rates are a more accurate level of description compared to mean amplitude changes

As is evident from the individual-trial plots in Figures 1c, 1e and 2d (as well as from prior literature), individual burst-like events provide a more accurate description of the dominant feature of raw β-band activity compared to steady modulations of power. However, it is an open question whether β-bursts actually provide a more accurate description of the relationship between human brain activity and movement, especially compared to more standard mean-amplitude measurements. To investigate this question, two of the three main analyses in the current manuscript (1. The comparison of fronto-central β-activity between the different trial types, Figure 2c; 2. The correlation between sensorimotor β-activity and Go-/Stop-signal reaction time, Figure 1f) were repeated using standard mean amplitude measurements. (The third main finding in this current study – i.e., the increase in β-bursting at sensorimotor sites following individual instances of fronto-central β-burst events – cannot be reproduced using amplitude measurements.) In other words, instead of quantifying β-burst rates for the time periods of interest, overall β-amplitude activity was averaged across the same period of time, and the analyses were repeated just as presented above.

Figure 5A shows the topographical distribution of mean β-amplitude across the time periods used in the β-burst analyses in Figures 1, 2, and 3. From the statistical trial-
comparison maps (bottom two rows of Figure 5A - i.e., the analog of Figure 3), it is evident that β-band amplitude shows topographically similar features compared to β-burst-rates: there is bilateral sensorimotor desynchronization following Go-signals and a fronto-central power increase following Stop-signals. However, unlike for β-burst rates, where fronto-central electrode sites showed significant increases from zero following Stop-signals, the β-band power increase at fronto-central electrodes on those same trials was not significantly different from zero (cf., second and third row of Figure 5A).

The average time-course of the fronto-central amplitude-signal for each individual trial type can be seen in Figure 5B. An analysis of the β-amplitude data during the Stop-signal-to-SSRT time period (as well as a matched time period on Go-trials) revealed that – just like for the β-burst analyses – there was a significant increase of β-activity on successful compared to failed Stop-trials (t(233) = 2.09, p = .037, d = .12), as well as compared to Go-trials (t(233) = 7.81, p = 1.93*10^{-13}, d = .55; Figure 5C). However, the effect sizes for both comparisons were substantially reduced compared to the β-burst analysis (indeed, the 95% confidence interval for the effect size of the successful vs. failed Stop-trials contrast included an effect size of 0, which was not the case for the β-burst analysis).

The time-course of the sensorimotor signal on Go-trials can be seen in Figure 5D. As to be expected based on prior work, there was a pronounced β-desynchronization following the Go-signal, which lateralized towards the contralateral side in the lead-up to the response. While this also matches the properties of the β-burst rates reported in Figures 1b and 1d, the same correlations analyses that yielded highly significant relationships between β-burst rates and both Go- and Stop-signal reaction time (Figure 1f)
did not yield any significant relationships between mean $\beta$-power amplitude and behavior. Not only did none of the 12 time periods yield any correlations that survived corrections for multiple comparisons, but none of the windows showed even an uncorrected $p < .05$ (Figure 5e).

Taken together, these results show that while $\beta$-burst rates and amplitude changes are clearly related both theoretically (a $\beta$-burst represents a sudden, short-lived increase in $\beta$-power) and empirically (the overall topographies of both $\beta$-burst rates and $\beta$-power after Stop- and Go-signals are morphologically similar), $\beta$-burst rates not only provide a superior description of the signal on the trial-to-trial level, they also provide a much more accurate reflection of behavior – and can in fact be used to uncover behavioral associations that are entirely absent in averaged $\beta$-power data.
a) Topographical distribution of $\beta$-amplitude per trial type

Correct Go-trials

Successful Stop-trials

Failed Stop-trials

Successful Stop vs. Correct Go

Failed Stop vs. Correct Go

Time after signal (msec)

b) Mean $\beta$-amplitude at FCz

c) $\beta$-ampl. pre-SSRT

d) Mean $\beta$-amplitude at C3 / C4

e) Relationship between Go-signal-locked contralateral C3/C4 $\beta$-amplitude and GO/STOP behavior
Figure 5. β-power amplitude analyses. A) Topographical distributions of β-power amplitude following Go- and Stop-signals. These figures are analog to Figures 1a, 2a, and 3, except they do not show changes in β-burst rates, but changes in mean β-amplitude during the same time windows. Any activity that did not exceed a significant change from 0 dB (using the same significance threshold of p < .0001, FDR-corrected that was used in Figures 1a, 2a, and 3) was set to 0. B) Changes in mean β-power amplitude at fronto-central electrode FCz on the three trial types of interest in the Stop-signal task. Shaded area around each curve represents the standard error of the mean. C) Comparison of mean β-amplitude in the Stop-signal-to-SSRT period (and a matched time period on Go-trials) – analog to Figure 2c. D) Changes in mean β-power amplitude at sensorimotor electrodes C3 and C4 following Go-signals on left/right-hand Go-trials. Shaded area around each curve represents the standard error of the mean. E) Correlations between mean β-power amplitude at sensorimotor sites contralateral to the response hand on Go-trials in 12 successive time windows following the Go-signal and Go/Stop-behavior (Go-trial reaction time / Stop-signal reaction time). Analog to Figure 1F.

Is single-trial β truly burst-like?

One issue that bears discussion regarding the current paper (as well as the existing β-burst literature) is that the decision regarding whether the β-band signal is truly burst-like (or, instead, reflects a transient modulation of an ongoing oscillation) is made based on largely qualitative, subjective criteria (van Ede et al., 2018). In other words, β is deemed ‘burst-like’ because the data look ‘burst-like’ – and because there are concrete biophysical models that explain the burst-like nature of the β signal (Sherman et al., 2016), which provide a priori credence to this idea.
However, one way to actually quantify whether a neural signal is truly burst-like is lagged phase coherence (Fransen et al., 2015). Lagged phase coherence quantifies the phase consistency in successive segments of data. The assumption is that if a signal at a specific frequency is marked by an ongoing oscillation, it should be possible to predict the phase of a given data segment based on past segments. Hence, lagged phase coherence was quantified at the three main electrode sites of interest in the current study (FCz, C3, C4).

Figure 6 shows the lagged coherence spectrum. It is evident that while there is some degree of lagged coherence across all frequencies surrounding (and including) $\beta$, the $\beta$ band notably represents the trough of the spectrum and is flanked by higher relative degrees of coherence in both the alpha and gamma bands. While there is currently no way to quantify what constitutes a “significant” degree of lagged phase coherence (Fransen et al., 2016), it seems save to conclude that beta is at least less ‘oscillatory’ – and hence, more burst-like – than the surrounding frequency bands.
Figure 6. Lagged phase coherence (3 cycles) at the three electrode sites of interest at 550 frequencies including and surrounding the β-range. What can be seen is that the β-range (highlighted) contains the trough of the lagged coherence spectrum, suggesting that the β signal at those electrodes is more 'burst-like' than the signal in the surrounding frequencies.

Discussion

Human movement is accompanied by systematic spatiotemporal changes in β-bursting. Prior to movement initiation, bilateral sensorimotor sites showed localized patterns of β-bursting, potentially representing a net-inhibited state of the motor system (Engel and Fries, 2010; Picazio et al., 2014; Rossiter et al., 2014). This β-bursting steadily reduced during movement initiation, suggesting a net-disinhibition. Notably, this reduction lateralized just prior to movement execution – comparable to the movement-related lateralization of β-desynchronization in the trial-average (McFarland et al., 2000; Kaiser et al., 2001; Doyle et al., 2005). However, β-bursts contained systematic relationships to both movement execution and movement cancellation that were absent in the averaged β-power amplitudes.

In situations in which inhibition had to be rapidly reinstated – i.e., to cancel movements following Stop-signals – β-burst rates significantly increased at fronto-central sites. This increase showed its most coherent spatiotemporal organization during the time period towards the end of SSRT (Figure 1a). Such fronto-central β-bursts were followed by a rapid, low-latency (<25ms) increase of β-bursting over sensorimotor sites – both ipsi- and contralateral to the to-be-cancelled movement. We tentatively propose that this may reflect a low-latency re-instantiation of inhibition at the level of the motor system (tonic
lateral sensorimotor β-bursting), triggered by a fronto-central control signal (phasic
fronto-central β-bursts).

The β-burst patterns show intriguing conceptual and empirical overlap with the
proposed properties of the neural and behavioral processing-cascade underlying
movement cancellation. First, β-band power increases are routinely found in trial-averaged
activity during movement cancellation (Swann et al., 2009; Swann et al., 2011a; Ray et al.,
2012; Swann et al., 2012; Wagner et al., 2018), including in intracranial recordings from
sites that could conceivably underlie the fronto-centrally distributed pattern of β-bursting
observed here (e.g., the pre-supplementary motor area, (Swann et al., 2011b)). The current
results suggest that these changes may actually result from increases in β-burst rates on
individual trials. Second, the race-model of the Stop-signal task proposes that action-
stopping can be modeled by a race between two processes; a Go-process working towards
the execution of the motor response and a Stop-process working towards the cancellation
of that response (Logan and Cowan, 1984). Notably, it is still controversial whether these
two processes operate independently, or whether the Stop-process directly influences the
Go-process (Boucher et al., 2007; Verbruggen and Logan, 2008; Schmidt et al., 2013; Schall
et al., 2017). In our data, stopping-related β-bursts at fronto-central sites were immediately
followed by a re-instantiation of β-bursting over sensorimotor sites – the same signature
whose initial reduction represented the start of the Go-process. This suggests that the
initiation of the Stop-process is followed by a substantial alteration in the neural activity
underlying the Go-process – speaking in favor of an interactive race-model (Boucher et al.,
2007; Schall et al., 2017). Third, previous research has shown that humans proactively
inhibit their movement initiation when they anticipate having to potentially cancel an
action, resulting in longer Go-trial reaction times. The degree of this proactive inhibition is inversely related to the amount of reactive inhibition necessary to successfully cancel an action. In other words, subjects that exert higher degrees of proactive reaction time slowing exhibit shorter SSRTs (Chikazoe et al., 2009; Verbruggen and Logan, 2009). The degree of this proactive inhibitory control may be reflected in the level of initial lateral sensorimotor $\beta$-bursting found in individual participants: Figure 1f shows that higher initial sensorimotor $\beta$-burst rates predicted both slower Go-trial reaction times and faster SSRT. This suggests that early $\beta$-bursting may reflect the trade-off between proactive control and reactive control, with more cautious responding being reflected in increased bilateral $\beta$-bursting. Fourth, the Stop-related re-instantiation of sensorimotor $\beta$-bursting that was observed following fronto-central $\beta$-bursts was notably present at both ipsi- and contralateral sensorimotor sites (Figure 4). This parallels prior work using physiological measures of motor excitability in specific corticospinal tracts. Such studies found that rapid movement cancellation is non-selective and affects the entire motor system (Badry et al., 2009; Majid et al., 2012; Wessel et al., 2013; Duque et al., 2017). The simultaneous, bilateral re-activation of sensorimotor $\beta$-bursting after fronto-central Stop-related $\beta$-bursts observed here could be the neurophysiological expression of that same non-selective property. Beyond this theoretical and conceptual coherence with prior work, the quantification of $\beta$-bursts also substantially extends our insights into the neural dynamics of movement. $\beta$-bursts not only provide a more accurate description of the actual raw $\beta$-signal (cf., Figures 1c,e; 2b), but reveals relationships to behavior that are not evident in amplitude-averages – such as the relationship between early sensorimotor activity and Go-
Stop-signal reaction time. Moreover, trial-level comparisons of β-burst rates yielded much larger effect sizes compared to amplitude analyses. For example, the effect size for the fronto-central successful vs. failed Stop-trial comparison was twice as large for β-burst rates compared to mean amplitudes, suggesting that averaging obscures the nature of this relationship. Lastly, the β-burst-locked analysis presented in Figure 4 yielded fundamentally novel insights into the interactions between neural concomitants of the Go- and Stop-processes and their interaction during movement cancellation. Moreover, the current study could speak to a recent controversy in the field of inhibitory control. Some studies have suggested that SSRT overestimates the latency of the stopping process (e.g., Matzke et al., 2013), and that true indices of inhibition occur much earlier before SSRT as previously thought (Raud and Huster, 2017). This is a challenge for many established signatures of inhibitory control, which occur close to the end of the stopping period indicated by traditional, potentially inflated SSRT estimates (Hanes et al., 1998; Wessel and Aron, 2015; Ogasawara et al., 2018). In this respect, it is notable that while the spatiotemporal coherence in fronto-central β-bursting after stop-signals took about 200ms to develop in the current study (bringing it close to the end of average SSRT), the FCz channel data show clear separation of successful and failed stop-trials much earlier than that (cf., Figure 2). Interestingly, the data show successful/failed differences as early as the time window that is centered around 50ms after the Stop-signal. This could reflect differences in the anticipatory recruitment of reactive stopping circuitry, which has been shown to partially underlie proactive control (Swann et al., 2012; Greenhouse & Wessel, 2013; van Belle et al., 2014), though this needs further testing in future studies.
The current study has key implications for future research. First, the precise neural origin of movement-related $\beta$-bursts needs to be investigated. Biophysical modeling has suggested that these bursts may result from the integration of near-synchronous bursts of excitatory synaptic drive, targeting the dendrites of pyramidal neurons in specific cortical layers (Sherman et al., 2016). It remains to be tested whether the brain areas from which the scalp-recorded activity reported here may be generated are subject to such layer-specific drive. Second, if $\beta$-bursting over sensorimotor sites is indeed indicative of a ‘tonic’ inhibitory mode of the motor system it would make it an interesting target for more systematic investigations of proactive inhibitory control that utilize different likelihoods of Stop-signals (Jaffard et al., 2008; Verbruggen and Logan, 2009; Greenhouse et al., 2012; Stuphorn and Emeric, 2012; Vink et al., 2015; Elchlepp et al., 2016). Third, there is great interest in pathological features of the $\beta$-frequency band in movement disorders, especially Parkinson’s disease (Hammond et al., 2007; Bronte-Stewart et al., 2009; Jenkinson and Brown, 2011; Quinn et al., 2015). Recent studies have used trial-level $\beta$-burst measurements in subcortical areas (e.g., the subthalamic nucleus) to identify gait problems in PD (Anidi et al., 2018) and to develop adaptive brain stimulation approaches using closed-loop algorithms (Little et al., 2013; Tinkhauser et al., 2017). Future studies should aim to investigate whether similar relationships can be found in scalp-recorded $\beta$-bursts (Gauggel et al., 2004; van den Wildenberg et al., 2006; Obeso et al., 2011). Fourth, $\beta$-bursts may help elucidate the interactions between the subcortical aspects of the extrapyramidal motor system and the cortical areas governing higher-order motor control (Jenkinson and Brown, 2011; Leventhal et al., 2012; Bartolo and Merchant, 2015; Feingold et al., 2015). Indeed, it is tempting to hypothesize that $\beta$-bursting may be a ‘universal’ language of the
motor system, signifying distributed processing throughout the both pyramidal and extrapyramidal motor pathways.

While the current study provides fundamentally novel insights into the neural underpinnings of movement in a large dataset, it also has shortcomings. First, since the neural activity in the current study was non-invasively recorded from the scalp, only limited conclusions can be made regarding the neural origins of the observed $\beta$-bursts. Second, while movement cancellation was clearly related to increased $\beta$-burst rates at fronto-central sites, $\beta$-bursts are neither necessary nor sufficient to cancel a movement on individual trials – at least when measured at the scalp-surface. Indeed, many failed Stop-trials also included $\beta$-bursts before SSRT. Moreover, not all successful Stop-trials included a supra-threshold $\beta$-burst prior to SSRT (Figure 2c). Part of this pattern can likely be attributed to the imperfect signal-to-noise ratio of scalp-recordings. Indeed, intracranial recordings of the local field potential closer to its source will likely yield substantially higher $\beta$-burst rates. Furthermore, the search window for fronto-central $\beta$-bursts in this study was defined very conservatively. While the time period between the Stop-signal and each participants’ SSRT estimate is arguably the most theoretically motivated, straightforward window of interest, it is based on a variable, implicit measure (SSRT). Combined with the fact that frequency-resolved methods sacrifice some degree of temporal precision, some behaviorally-relevant $\beta$-bursts may have fallen just outside of the detection window. Indeed, extending the window by a single $\beta$-cycle increased the number of detected fronto-central $\beta$-burst on successful Stop-trials by 27%, while the trial-differences remained highly significant (successful Stop vs. Go: $t(233)=12.07$, $p=2.27\times10^{-26}$, $d=94$; successful vs. failed Stop: $t(233)=2.97$, $p=.003$, $d=.23$). Future investigations – ideally using
intracranial recordings – are necessary to investigate whether fronto-central \( \beta \)-bursts are truly necessary for stopping, and how the scalp-recorded \( \beta \)-burst signal relates to the original local field potential.

In summary, the current study found systematic changes in \( \beta \)-burst activity during both movement initiation and cancellation in humans. The findings suggest that \( \beta \)-bursting over lateral sensorimotor sites reflects the inhibition of the underlying areas of the motor system, which has to be overcome during movement initiation, and which can be rapidly re-instated when movement cancellation is necessary – likely signaled by phasic increases in fronto-central \( \beta \)-burst. This suggests that \( \beta \)-bursts are a fundamental signature of human motor control.


Verbruggen F et al. (2019) A consensus guide to capturing the ability to inhibit actions and impulsive behaviors in the stop-signal task. Elife 8.


