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RESEARCH ARTICLE

Control of Movement

Cortical beta modulation during active movement is highly reproducible in healthy adults

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Abstract

The rolandic beta (13–30 Hz) rhythm recorded over the sensorimotor cortices is known to be modified by movement execution and observation. Beta modulation has been considered as a biomarker of motor function in various neurological diseases, and active natural-like movements might offer a clinically feasible method to assess them. Although the stability of movement-related beta modulation has been addressed during passive and highly controlled active movements, the test-retest reliability of natural-like movements has not been established. We used magnetoencephalography (MEG) to evaluate the reproducibility of movement-related sensorimotor beta modulation longitudinally over 3 mo in a group of healthy adults ($n = 22$). We focused on the changes in beta activity both during active grasping movement (beta suppression) and after movement termination (beta rebound). The strengths of beta suppression and rebound were similar between the baseline and follow-up measurements; intraclass correlation coefficient values (0.76–0.96) demonstrated high reproducibility. Our results indicate that the beta modulation in response to an active hand-squeezing task has excellent test-retest reliability: the natural-like active movement paradigm is suitable for evaluating the functional state of the sensorimotor cortex and can be used as a biomarker in clinical follow-up studies.

NEW & NOTEWORTHY This research demonstrates that the beta rhythm modulation related to active hand-squeezing task has an excellent test-retest reproducibility in healthy adults over a three-month follow-up period. This natural-like active movement is thus suitable for evaluating beta modulation to assess the functional state of the sensorimotor cortex and can be utilized as a biomarker, for example, in clinical longitudinal follow-up studies.

active movement; event-related desynchronization; event-related synchronization; sensorimotor cortex; test-retest reliability

INTRODUCTION

The human cerebral cortex demonstrates several salient, intrinsic rhythms that can be characterized noninvasively with magnetoencephalography (MEG) and electroencephalography (EEG). The rolandic sensorimotor "mu" rhythm at around 13–30 Hz (beta band) is a particularly prominent one and is known to react to movement execution and observation (1, 2). The rhythm appears also clinically relevant: for example, alterations in movement-related beta activity have been observed in various neurological diseases, such as spinal cord injury (3, 4), stroke (5–7), and Parkinson's disease (7–9). Beta band modulation is a robust phenomenon across individuals (10). Before and during an active movement, the beta oscillatory activity in the sensorimotor cortex rapidly decreases.

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After completion of the movement, this so-called beta suppression—or event-related desynchronization—is followed by an increase in the beta power (postmovement beta rebound or event-related synchronization; 10–12). Beta suppression typically reaches its maximum 250–400 ms after movement onset (2, 13), and it has been shown to be sustained until the end of the muscle contraction (14). The postmovement beta rebound occurs 300–1,000 ms after movement cessation (15), and the recovery of beta power is known to depend on the speed of the movement, with earlier recovery for brisk compared with slow movements (14).

Despite extensive research, the functional relevance of beta modulation remains unclear. Beta suppression has been suggested to reflect activation of the sensorimotor cortex (16), whereas the rebound is believed to represent deactivation or active inhibition of the motor cortex (11, 12). Motor learning reduces beta suppression (17) and enhances the beta rebound (18). The beta rebound strength has also been suggested to indicate confidence in the prediction of motor outcome, with larger amplitudes indicating more confidence (19) and amplitude reductions relating to error (20). The evolution of beta modulation after lesions to the sensorimotor system may provide a marker of the clinical outcome and motor recovery, for example, in spinal cord injury patients (4, 21). In addition, the strength of beta rebound correlates positively with clinical recovery in stroke patients (5). For establishing the reliability of such biomarkers in clinical practice, the test-retest stability of such measures needs to be first demonstrated. Beta modulations addressed with electroencephalography (EEG) to precisely execute and control movements are highly stable in healthy individuals between measurements conducted within a few weeks (22). Similarly, significant reproducibility has been demonstrated for passive tactile and proprioceptive stimulation (13). Active natural-like movements, such as grasping, can provide a clinically feasible way to probe motor functions. Although their test-rest reliability over several months is not known, they have already been used in assessing the changes in the sensorimotor beta oscillations after neuromodulation treatment (paired associative stimulation; PAS) in patients with spinal cord injury (21).

Here, we used a simple active hand-squeezing task to investigate the test-retest reliability of movement-related beta modulation in a group of healthy adults over months. As suppression and rebound are estimated as movement-related changes relative to premovement baseline beta-band oscillatory activity, we also evaluated the reliability of premovement beta power. The results demonstrate excellent test-retest stability of both movement-related beta suppression and rebound over a minimum of a 3-mo follow-up period. Both responses showed strong correlations between the baseline and follow-up measurements.

MATERIALS AND METHODS

Subjects

Twenty-two healthy subjects (11 females, mean ± SD age 49 ± 15 yr; range 18–78 yr; 20 right-handed, 2 ambidextrous) participated in the study. None of the participants had any self-reported neurological disease, medications affecting the central nervous system, or identifiable physical disabilities of the arms or hands. All subjects underwent two experimentally identical MEG sessions: The time interval between the two sessions was at least 3 mo (range 98–167 days, mean ± SD interval 124 ± 25 days). Subjects gave their written informed consent before the first measurement, and the study had previously received an ethical statement from the Helsinki University Hospital Committee of Medical Research Ethics.

Experimental Protocol

The modulation of movement-related beta activity was assessed during an auditory-paced active movement paradigm. Three of the subjects were initially measured in the supine position followed by a second measurement in a sitting position, as we aimed to assess the impact of different positions on the MEG signal. Despite the change in position, the results for these subjects remained consistent with those of the others. The remaining subjects were thus measured in the sitting position on both occasions. Subjects performed two-second sustained ball squeezes with one hand, followed by a five-second rest after each squeeze. The timing of the squeezing was instructed with auditory cues (high-frequency tone cued the start of the squeeze and low-frequency tone cued the end of the squeeze). The task was performed with both hands separately and their order (left-right, right-left) was counterbalanced over the two measurement times. Beta oscillations are known to be modulated also by movement observation (1). Thus, to minimize responses evoked by seeing the movement, the hand and wrist were hidden behind a visual barrier. Furthermore, the subjects were instructed to focus on a centrally placed fixation cross to minimize eye movements. Each measurement took ∼6 min, with around 50 trials (51 ± 2, mean ± SD) per hand.

MEG Acquisition and Preprocessing

The MEG measurements were conducted with a 306-channel whole scalp MEG system (TRIUX, MEGIN Oy, Helsinki, Finland) consisting of 204 planar gradiometers and 102 magnetometer sensors. Electro-oculogram (EOG) was recorded to detect eye blinks and movements. In addition, an electromyogram (EMG) of the finger flexors in the forearms was recorded to monitor muscle activation during the squeeze. Head position was continuously tracked during the measurements using five head position indicator (HPI) coils located on the surface of the head. These indicator coils, as well as three anatomical landmarks and additional points on the scalp, were digitized before recording with 3-D digitizer (Polhemus 3Space Fastrak, Colchester, VT). A bandpass of 0.03–330 Hz and a sampling frequency of 1,000 Hz were used during the data collection.

MEG data were preprocessed with the temporal extension of the Signal Space Separation method and head movement compensation (23) as implemented in MaxFilter software (Megin Oy). Both measurement sessions for each subject were transformed to the same headspace with MaxFilter software, to enable a better comparison of the two sessions in sensor space. MEG data were further analyzed with MNE Python (v. 1.3). Eye blink artifacts were removed from the sensor level signals with principal component analysis (24). In addition, for

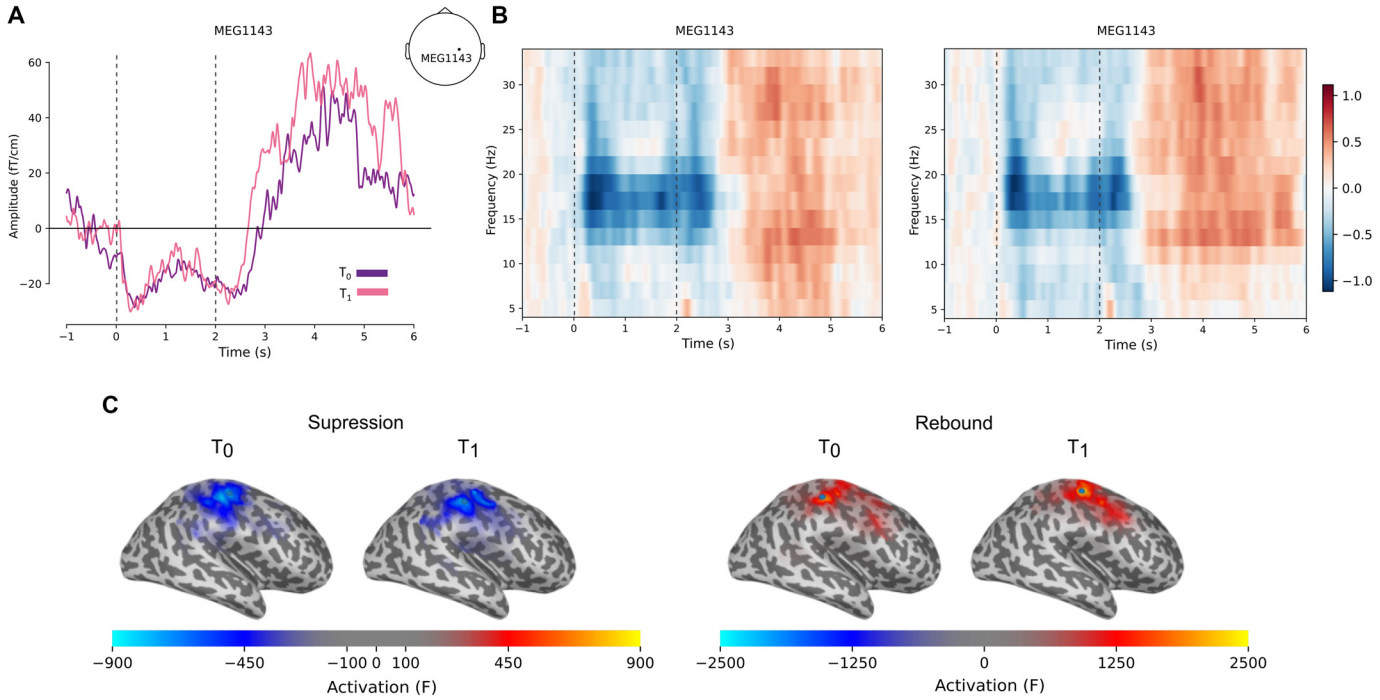

Figure 1. Modulation of the beta rhythm during active left-hand movement that began at 0 s and ended at 2 s (dashed vertical lines) in one example subject in the two MEG measurements at time points T_0 and T_1 separated by 3 mo across the two MEG measurements. *A*: temporal-spectral evolution (TSE) of the 13–25 Hz activity at the maximum channel over the right sensorimotor cortex at both measurement sessions (T_0 and T_1). *B*: time-frequency representation of the same activity. *C*: the sources of the beta suppression and rebound as estimated by DICS depicted on the subject's inflated MR images. DICS, Dynamical Imaging of Coherent Sources; MEG, magnetoencephalography.

source localization, independent component analysis (ICA), (25) implemented in MNE Python (26) was used to extract the remaining artifacts related to eye blinks and heartbeats.

Data Analysis

The modulation of movement-related beta rhythm within the sensorimotor cortex was quantified using the temporal spectral evolution method (TSE; 2). The raw data was first filtered in the beta range using a bandpass of 13–25 Hz. This range was selected based on previous data showing that individually selected beta bands coincide well with this common range (27). Somatosensory and auditory evoked responses time-locked to stimulus onset were then subtracted from each epoch in the raw data (28). The signals were smoothed using a 200-ms Gaussian kernel, and a Hilbert transform was applied to obtain the envelope signal. The data was averaged from −1,000 to 6,000 ms with respect to the auditory stimulus probing the onset of ball squeeze. The premovement baseline was calculated from −1,000 to −100 ms to the onset of the auditory stimulus.

Beta suppression lasts until the end of a muscle contraction (14), but during episodes of static postural maintenance, especially after a stable object hold, beta power can significantly increase relative to the decrement seen during the dynamic part of movement (29); this can be reflected as an initial suppression followed by a longer sustained one. To determine the strength of the beta suppression, we thus calculated both the mean signal at 100–500 ms to determine the initial phase of suppression, and the area under the curve (AUC) for a time period of 100–3,300 ms for the sustained suppression. For the beta rebound, known to last seconds after the movement discontinuation (13, 22), we used the AUC calculated at 3,500–6,000 ms.

For each subject, the gradiometer pair adjacent to the contralateral sensorimotor cortex showing the strongest beta modulation was selected for further analysis; the channel pair was kept constant between sessions. Individual magnetic resonance (MR) images were obtained from five subjects to confirm the location of the recorded activity. For source localization, we used Dynamical Imaging of Coherent Sources (DICS; 30, 31) as implemented in MNE Python (26).

Statistical Analysis

Statistical analysis was performed using Python. Shapiro–Wilk test revealed that the beta modulation strengths and baseline power follow mainly nonnormal distribution. Subsequently, a nonparametric Wilcoxon test was used to analyze the possible differences in baseline power, beta suppression and rebound strengths between the baseline and follow-up measurements.

Pearson's correlation coefficients were used to evaluate the stability of beta modulation measures between the first and the follow-up MEG sessions. In addition, the intraclass correlation coefficient (ICC) with two-way random effects and absolute agreement was calculated to evaluate the reproducibility of beta suppression and rebound between the two measurement sessions.

The *P* values resulting from statistical tests were adjusted by Bonferroni correction for multiple comparisons. A *P* value of <0.05 was considered as statistically significant.

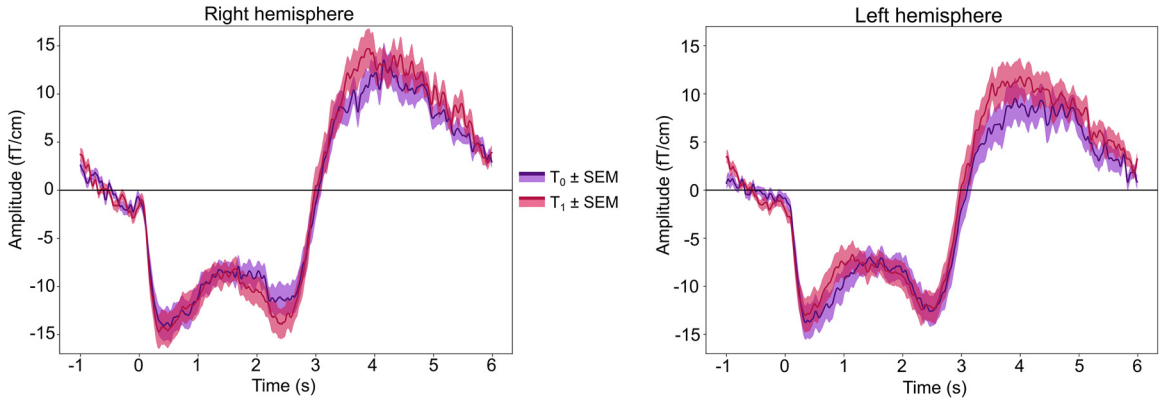

Figure 2. Grand average ($n = 22$) beta rhythm modulation (13–25 Hz) to active hand movement at the maximum channel over the contralateral sensorimotor cortices at the baseline (T_0) and follow-up (T_1) measurements.

RESULTS

Beta Modulation Strengths Correlated between the Measurements

Figure 1 shows an example of a beta modulation in one subject. After the auditory cue onset and during the left-hand motor task, a decrease in beta power (beta suppression) was observed in the right sensorimotor cortex as expected. Subsequent to the cessation of movement, a rapid increase in beta power was observed (beta rebound). Although individual electromyography (EMG) signals indicated slight variations between subjects in the timing and duration of active movements, the chosen analysis intervals adequately adapted the individual activation trends.

Figure 2 illustrates the grand average ($n = 22$) of the 13–25 Hz beta modulation TSE with respect to the auditory cue for each hand separately, and Table 1 depicts the extracted measures of beta modulation in the hemisphere contralateral to the motor movement. All the extracted values show remarkable similarity across the measurement sessions conducted at T_0 and T_1; there were no statistically significant differences ($P > 0.95$) in the strength of beta modulation between the baseline and follow-up measurements in beta suppression or rebound.

Figure 3 illustrates the scatterplots for individual suppression and rebound strengths between the baseline and 3-mo follow-up measurements, and Table 2 shows Pearson's correlation coefficients (r) and intraclass correlation coefficients (ICC) for the same values. Both beta suppression and beta rebound correlated strongly between the T_0 and T_1 measurements. For beta suppression, Pearson's correlation coefficients were $r = 0.91$–0.93 ($P < 0.001$) and $r = 0.93$–0.96 ($P < 0.001$) for the left and right-hand activation, respectively. For beta rebound, the correlation value was $r = 0.82$ for the left, and $r = 0.76$ for the right hand. For beta suppression, ICC values indicated excellent reproducibility (0.91–0.93 left hand; 0.93–0.96 right hand). Although the ICC values were slightly lower for the beta rebound (0.82 left hand; 0.76 right hand), they still indicated good reliability. To account for the possible effect of subjects' age causing the weaker reproducibility of beta rebound compared to beta suppression, we calculated the correlation between age and beta rebound AUC values. No significant correlations were observed at either timepoint T_0 and T_1 for either hand (r range between -0.11 and -0.18, n.s.).

Reproducibility of the Premovement Beta Power

Table 3 shows the mean baseline beta power from −1,000 to −100 ms before the auditory cue for the ball squeeze. The baseline beta power remained highly stable, with no significant differences between T_0 and T_1 ($P > 0.82$). Pearson's correlation coefficients were 0.93 ($P < 0.001$) for the left hand and 0.96 ($P < 0.001$) for the right hand. ICC coefficients demonstrated, again, excellent (left 0.91, right 0.96) test-retest stability between the baseline and the follow-up measurements.

DISCUSSION

We examined the test-retest reliability of movement-related and premovement sensorimotor beta oscillatory activity in an active grasping task in a group of healthy adults covering a wide age range from 18 to 78 yr of age. The results demonstrate excellent test-retest stability of both movement-related beta suppression and rebound signal over a minimum of 3 mo follow-up period. Both responses

Table 1. *Beta modulation strengths [means ± standard error of mean (SEM), AUC ± SEM] for hand activation at the contralateral sensorimotor cortex*

	Left Hand Task	Right Hand Task
	Supression	
T_0 fT/cm	11.9 ± 2.2	11.2 ± 2.0
T_1 fT/cm	12.4 ± 2.1	11.0 ± 2.2
T_0 AUC (fT/cm × ms)	33.3 ± 7.1	30.6 ± 6.5
T_1 AUC (fT/cm × ms)	34.1 ± 7.3	29.8 ± 6.4
	Rebound	
T_0 AUC (fT/cm × ms)	23.9 ± 5.1	17.9 ± 3.8
T_1 AUC (fT/cm × ms)	27.6 ± 5.9	22.5 ± 4.8

$n = 22$; AUC, area under the curve; T_0, baseline; T_1, 3-mo follow-up.

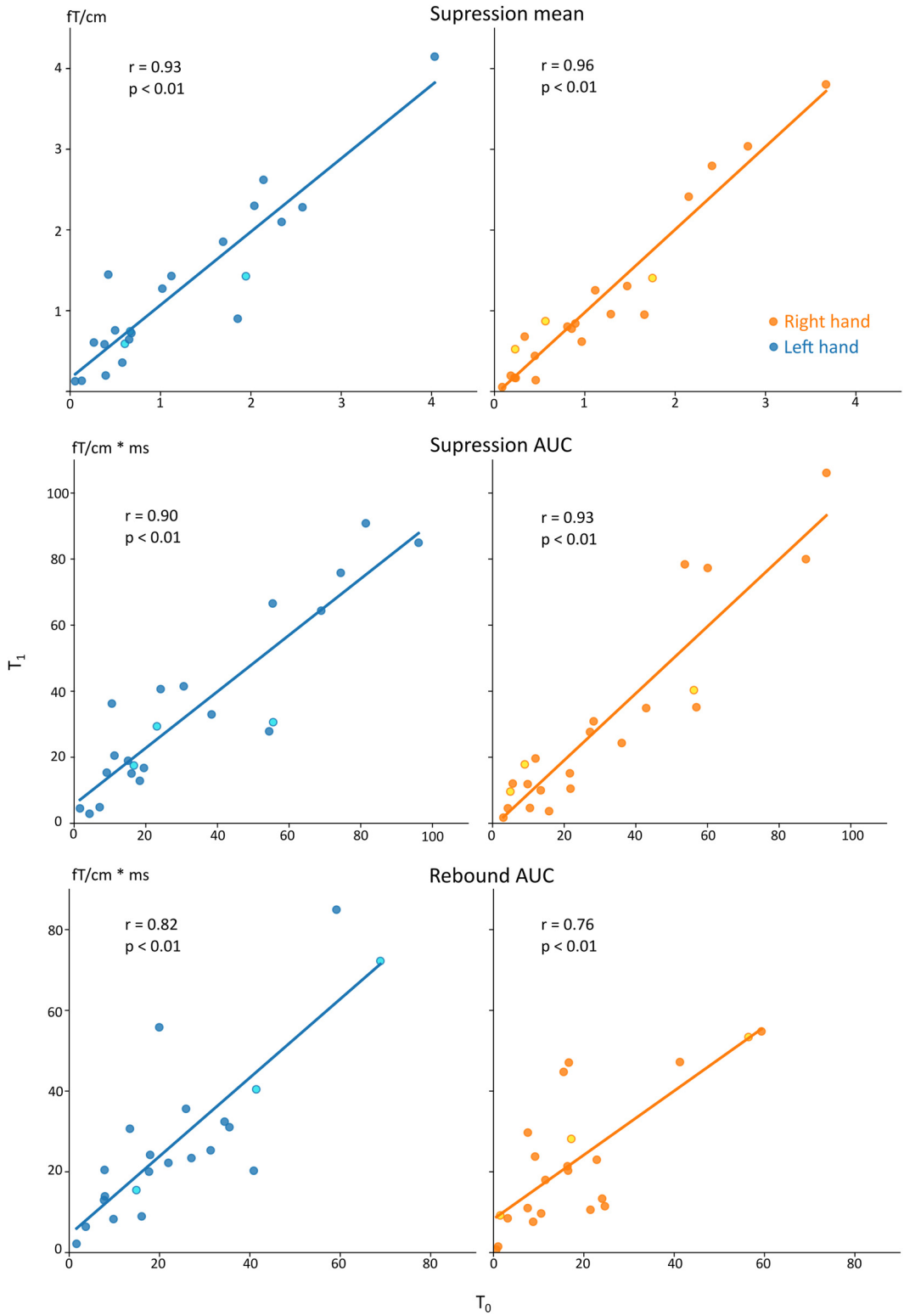

Figure 3. Correlations between the baseline and follow-up measurements for the strength of contralateral beta modulation in both hemispheres. The three subjects who were measured in the supine position during the baseline measurement and in the sitting position during the follow-up MEG have been highlighted in teal and yellow. MEG, magnetoencephalography.

showed a strong correlation between the baseline and follow-up measurements. In addition, the absolute baseline beta power remained stable across measurement sessions. The results agree with earlier studies that have addressed the stability of beta-range signals during rest (32), passive stimulation (13), and controlled movements (22). Our results further demonstrate that the reproducibility of movement-related beta signals in a natural-like, active movement

Table 2. *Pearson's correlation coefficients (r) and ICC for beta modulation strengths between sessions*

	Left Hand Task		Right Hand Task	
T_0 vs. T_1	*r*	ICC	*r*	ICC
Suppression				
Mean	0.93*	0.93	0.96*	0.96
AUC	0.90*	0.91	0.93*	0.92
Rebound				
AUC	0.82*	0.80	0.76*	0.74

$n = 22$; AUC, area under the curve; ICC, intraclass correlation coefficient; T_0, baseline; T_1, 3-mo follow-up. *$P < 0.001$.

Table 3. *Baseline (−1,000 to −100 ms) beta power values (mean ± SEM) and the corresponding Pearson's correlation coefficients (r) and ICC values*

	Baseline			
	Left Hand Task		Right Hand Task	
T_0 fT/cm	35.2 ± 7.5		37.2 ± 7.9	
T_1 fT/cm	37.5 ± 8.0		37.1 ± 7.9	
	r	ICC	*r*	ICC
T_0 vs. T_1	0.93*	0.91	0.96*	0.96

$n = 22$; AUC, area under the curve; ICC, intraclass correlation coefficient; T_0, baseline; T_1, 3-mo follow-up. *$P < 0.001$.

paradigm is excellent, and it can thus be used as a biomarker to assess the functional state of the sensorimotor cortex in longitudinal follow-up studies.

Although both beta suppression and rebound showed a strong correlation between measurements, our results were slightly more consistent for beta suppression than for rebound. This is in line with previous studies that have shown that beta rebound is more sensitive to the exact experimental condition than the suppression (14, 33). The amplitude of beta rebound, but not suppression, is modulated by the speed of voluntary movement (34). In addition, beta rebound is altered by the output force and the rate of force development (33). Indeed, beta suppression and rebound are thought to reflect different functions in sensorimotor cortical processing. The beta suppression has been proposed to be a more nongradual response, whereas the rebound would be regulated by more complex inhibitory networks and would thus be more prone to changes in the task (13).

Our study did not aim at standardizing subjects' alertness level between the baseline and follow-up sessions, or the time of day of the measurements. Circadian rhythm is known to affect both beta suppression and baseline beta power. Amplitude of beta suppression increases toward the afternoon, although this effect is not observed for beta rebound (35) Although alterations in alertness do not significantly affect beta modulation at the group level, reduced alertness at the individual level has been associated with decreased suppression and rebound strength (27). Similarly, infrequent auditory and visual distractors have been shown to affect motor cortex activity, possibly by induced startle-like responses (36). Our stimulation paradigm, with constant timing of auditory triggers to cue the onset and offset of the movement, is unlikely to produce such responses. Despite the applied auditory cues and not controlling for alertness, force, and movement speed in the ball-squeezing task, our measurements proved to be highly reproducible in our 3-mo follow-up period. The high test-retest reliability indicates that this type of active motor task can be effectively used in longitudinal assessments, even within clinical populations.

In contrast to previous studies addressing the reproducibility of beta modulation with well-controlled wrist flexion and extension, passive movements, or tactile stimuli, we used a natural ball-squeezing task. Beta suppression and rebound were observed in all but one subject, but the strengths of beta suppression and rebound showed considerable variability between individuals. This is consistent with previous findings indicating that beta rhythm modulation exhibits significant individual variability and can, in some subjects, even be indistinguishable (13). Although our sustained 2 s squeezing task involving finger flexion and postural maintenance is more challenging to standardize than passive or precisely controlled active movement (13, 22), it can be a useful monitor of sensorimotor disruption as the force maintenance requires both feedforward motor signal as well as feedback sensory control. It is likely that the stability of beta signal and its recovery as a function of time after the injury differ between patient groups with different causes of injury. The present task can, however, be a useful tool, especially for neurological patients known to have problems with deafferentation such as spinal cord injury and who can nevertheless do the active squeezing task. In this patient group, the loss of sensory feedback from movement execution has been suggested to contribute to insufficient movement-related beta suppression (3).

Conclusions

Our results demonstrate that beta suppression and rebound strengths related to the active hand-squeezing task have excellent test-retest reproducibility over a 3-mo follow-up period in a group of healthy adults. This finding suggests that the natural-like active movement paradigm is suitable for evaluating beta modulation to assess the functional state of the sensorimotor cortex and can be used as a biomarker, for example, in clinical longitudinal follow-up studies.

DATA AVAILABILITY

Data cannot be made publicly available due to Finnish data protection law. Data can, however, be shared for research collaboration with an amendment to the research ethics permit and a related data transfer agreement.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

L.N. and H.R. conceived and designed research; L.N. analyzed data; L.N., L.L., K.A.M.P., and H.R. performed experiments; L.N., K.A.M.P., and H.R. interpreted results of experiments; L.N. prepared figures; L.N. drafted manuscript; L.N., M.I., E.K., K.A.M.P, and H.R. edited and revised manuscript. L.N., L.L., M.I., E.K., M.L., K.A.M.P., and H.R. approved final version of manuscript.

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