Our actions are constantly guided by decisions based on sensory information. The motor cortex is traditionally viewed as the final output stage in this process, merely executing motor responses based on these decisions. However, it is not clear if, beyond this role, the motor cortex itself impacts response selection. Here, we report activity fluctuations over motor cortex measured using MEG, which are unrelated to choice content and predict responses to a visuomotor task seconds before decisions are made. These fluctuations are strongly influenced by the previous trial’s response and predict a tendency to switch between response alternatives for consecutive decisions. This alternation behaviour depends on the size of neural signals still present from the previous response. Our results uncover a response-alternation bias in sensorimotor decision making. Furthermore, they suggest that motor cortex is more than an output stage and instead shapes response selection during sensorimotor decision making.
We constantly use sensory information to choose between alternative motor actions. The neural processes underlying such sensorimotor choices include the representation of sensory evidence, possibly weighing in top-down factors, deciding between choice alternatives and finally executing the appropriate motor response\(^1\). Traditionally, these processes were viewed as sequential stages, in which the motor cortex acts as the final output stage that merely executes responses (for example, a specific button press) corresponding to the choices made in other brain regions (for example, ‘yes—I saw the target’).

In contrast to this sequential view, recent evidence suggests a more continuous flow of information and that the motor cortex, that is, primary and pre-motor cortex, is more directly involved in the decision-making process itself\(^1\). Before choice commitment, motor cortex activity already reflects competing response options\(^2\)–\(^8\), and if choices are inextricably linked to a specific response during decision formation, activity in motor areas\(^6\)–\(^9\) as well as corticospinal excitability\(^13\),\(^14\) and motor reflexes\(^15\) track the evolution of upcoming choices.

However, if choice–response contingencies are specified before decision making, choices and associated responses cannot be dissociated, neither behaviourally nor neurally. Therefore, it is unclear if intrinsic fluctuations of motor cortex activity have a direct impact on the decision-making process beyond representing upcoming choice-contingent responses. Here, we overcome this limitation by dissociating choices and responses, and investigate with magnetoencephalography (MEG) the motor cortex’ role in human sensorimotor decision making.

We show that fluctuations over motor cortex before decision making are predictive of upcoming responses. These signal fluctuations are partly carried over from the previous response and predict a tendency to alternate between response alternatives for consecutive choices. Our results reveal a tendency to alternate responses in perceptual decision making. Furthermore, they suggest that motor cortex can impact response selection during decision making.

**Results**

**Dissociating choices from responses.** We recorded MEG from 20 human participants while they judged the presence of weakly coherent motion in a display of randomly moving dots (Fig. 1a; see ‘Methods’ section). For each participant, stimuli were adjusted for near-threshold performance (average correct performance: 73.9 % \(+/-9.4\)%). Subjects reported their choice (‘yes’/’no’) with a left or right hand button-press. Two design features dissociated choices from motor responses during the decision-phase\(^16\)–\(^18\). First, the mapping between choice and response hand was randomly re-assigned on each trial. Second, for each trial, the choice-response mapping was indicated with a colour cue only after the stimulus presentation was completed (Fig. 1b). Thus, subjects had to form their decision during stimulus presentation, but could only later map their choice onto a response.

**Early response-predictive motor cortex activity.** We reconstructed neuronal activity in the left and right motor cortices as a function of time and frequency (Fig. 2a). After the choice–response cue and directly preceding the button-press, we observed the typical reduction of beta-band power (12–30 Hz) in the hemisphere contralateral to the button-press (Fig. 2a, \(P = 0.012\), two-tailed one-sample cluster permutation test; \(n = 20\), \(4.7–6.6 \text{ s, } 10–44 \text{ Hz}\))\(^9\),\(^10\),\(^19\)–\(^22\). Because the cortical distribution of this lateralized pre-response activity peaked pre- and post-centrally (Fig. 2b; 4.5–5.5 s; 12–30 Hz), we refer to it as sensorimotor cortex activity in the following. To test if sensorimotor cortex activity also predicted responses earlier, that is, before the choice-response cue allowed for choice-contingent response selection, we compared beta-band activity (12–30 Hz) contra- and ipsilateral to the response throughout the trial (Fig. 2c,d). This revealed significant response-predictive lateralization not only after the choice–response cue (Fig. 2c,d; 4.6–6.1 s; \(P = 0.002\), two-tailed one-sample cluster permutation test; \(n = 20\)) but also at the beginning of the trial (\(-1.0 \text{ to } 1.1 \text{ s}; \ P = 0.01\), two-tailed one-sample cluster permutation test; \(n = 20\)). Beta-band activity contralateral to the button-press was significantly lower than ipsilateral. This early response-predictive activity was independent of accuracy. It was present for both, correct and error trials (Fig. 2e, and Supplementary Fig. 1).

In sum, neuronal activity in sensorimotor cortex predicted which button participants eventually pressed not only after, but even before the choice-response cue, before the stimulus and more than 6 s before the final motor response. Importantly, because choices and responses were dissociated at this point in time, this response-predictive lateralization reflects neuronal encoding of the upcoming response, but not of the reported choice content.

**Long-lasting effect of beta rebound.** Because response-predictive activity appeared already at trial onset, we hypothesized that it was related to the previous trial’s response. The contralateral beta power decrease in motor cortex before a response is typically followed by a characteristic increase of beta power, the ‘beta rebound’\(^20\),\(^23\),\(^24\). To investigate if this affected the early response-predictive activity, we analysed the evolution of the beta rebound that followed the previous trial’s button-press (Fig. 3). Indeed, we found a prominent increase of beta power contralateral to, and following the previous button-press that lasted for several seconds into the current trial until presentation of the next choice-response cue (Fig. 3a–c, 0.7 s after the previous trial’s button-press to 4.6 s of the current trial, \(P = 0.002\), two-tailed one-sample...
correct (following the previous response in combination with the early
Fig. 4a,b). We hypothesized that this reversed lateralization
separately for response alternation and non-alternation trials,
lateralization with respect to current button-press plotted
to the lateralization before the previous button-press (but see
pushes the sensorimotor cortices into a lateralized state opposite
the beginning of the current trial, the sensorimotor cortex was not
the lateralization right before the previous button-press. Thus, at
the beta-rebound lateralization was about three times as strong as
Fig. 1). At its maximum before the current trial’s stimulus onset,
response accuracy. Furthermore, the beta-rebound did not differ
following correct and error trials (Fig. 3e and Supplementary
irrelevant to the button-press). Thus, at
cluster permutation test; \( n = 20 \). The cortical distribution of this
beta-rebound peaked over sensorimotor cortices (Fig. 3d), and
similar to the response-predictive activity, was independent of
response accuracy. Furthermore, the beta-rebound did not differ
following correct and error trials (Fig. 3e and Supplementary
Fig. 1). At its maximum before the current trial’s stimulus onset,
the beta-rebound lateralization was about three times as strong as
the lateralization right before the previous button-press. Thus, at
the beginning of the current trial, the sensorimotor cortex was not
in a neutral state, but even stronger and reversely lateralized than
preceding the previous response.

**Beta rebound predicts response alternation.** The beta rebound
pushes the sensorimotor cortices into a lateralized state opposite
to the lateralization before the previous button-press (but see
lateralization with respect to current button-press plotted
separately for response alternation and non-alternation trials,
Fig. 4a,b). We hypothesized that this reversed lateralization
following the previous response in combination with the early
response-predictive lateralization for the current response may
induce a behavioural bias towards response alternations across
successive trials. Indeed, participants showed a significant ten-
dency to alternate the response hand from one trial to the next
(Fig. 5a, mean \( r = 0.04 \), \( P = 0.016 \), one-tailed one-sample \( t \)-test;
\( n = 20 \)). Because our design enabled us to dissociate responses
from choices, we could unequivocally dissociate this response
alternation bias from the well-known preference to repeat the
previous choice \( 10,25-27 \), which was also present in our data (mean
\( r = 0.13 \), \( P = 5.366 \times 10^{-4} \); two-tailed one-sample \( t \)-test; \( n = 20 \)).
The response bias also affected overall performance: The stronger
the participants’ response bias, the worse they performed in the
actual motion detection task (Fig. 5b, \( r = -0.53 \), \( P = 0.016 \),
Spearman correlation; \( n = 20 \)).

While the above findings of a long-lasting beta rebound and
response alternation suggest a mechanistic link between these two
phenomena, they might also merely coexist. Therefore, we sought
more direct evidence for a link between these two phenomena.
If they were mechanistically related, variance in one variable
should explain variance in the other. First, we tested if, across

\[ Z = \]
participants, the strength of the beta rebound predicted the tendency to alternate responses. This is what we found (Fig. 5c, \( r = 0.64, P = 0.002 \), Spearman correlation; \( n = 20 \)): the stronger a participant’s beta rebound, the more likely the participant was to alternate responses. We repeated this analysis across the entire cortex (Fig. 5d). This revealed that the beta rebound predicted response alternation specifically in regions compatible with sensorimotor cortex and similar to those regions showing maximum pre-response lateralization (Fig. 2b). Second, we tested if the relationship between beta rebound and response alternation also held on the single-trial level. Indeed, we found that the stronger the beta rebound at the beginning of a trial, the more likely participants were to alternate responses on this trial (random effects: \( P = 0.021 \); fixed effects: \( P = 0.005 \); two-tailed one-sample permutation tests on beta rebound averaged in the window \(-1 \) to \(-1.25 \) s; \( n = 20 \)). Another third line of evidence suggested a close relation between beta rebound and alternation behaviour: If the response-predictive activity at trial onset (Fig. 2d) reflects the effect of the beta rebound on response behaviour, then removing neural variability due to the beta rebound should reduce the response-predictive effect. To test this, we removed neural variability due to the beta rebound by correcting for the effect of previous responses (see ‘Methods’ section). Indeed, we found that this correction significantly reduced the response-predictive effect (Fig. 6a,b, \( P = 0.010 \), one-tailed paired permutation test; \( n = 20 \)). This finding provides additional evidence for a mechanistic link between beta rebound and response alternation behaviour.

We next tested if the strength of the beta rebound was modulated by different aspects of the previous trial. We found that only the duration of the preceding inter-trial interval (ITI; \( P < 0.001 \); two-tailed one-sample \( t \)-test; \( n = 20 \)), but not the previous choice, response hand, target presence, accuracy, or reaction time (all \( P > 0.05 \); two-tailed one-sample \( t \)-tests, all \( n = 20 \)) predicted the strength of the following beta-rebound (Supplementary Table 1). Corresponding to this decay of the beta-rebound, also the alternation bias was descriptively weaker and not significant for trials following long (mean \( r = 0.019, P = 0.45 \), one-tailed one-sample \( t \)-test; \( n = 20 \)) as compared with short (mean \( r = 0.052, P = 0.046 \), one-tailed one-sample \( t \)-test; \( n = 20 \)) inter-trial intervals (direct comparison \( P = 0.21 \), one-tailed paired \( t \)-test; \( n = 20 \), Supplementary Fig. 2).

In sum, our findings suggest that the beta rebound drives response-predictive fluctuations of sensorimotor cortex activity at trial onset.

**Spontaneous fluctuations of beta lateralization predict responses.**
Do also spontaneous fluctuations of motor cortical activity beyond the beta rebound predict responses? In other words, can response variability be explained by prestimulus neural variability—over and above the fact that responses depended on previous responses, and the fact that each response produces a beta rebound? Removing the neural variability due to the beta-rebound allows for also addressing this question. Indeed, we found that even after removing neural variability due to the beta-rebound, motor cortex lateralization at trial onset predicted upcoming responses (\( P = 0.024, \ -1 \) to \( 1.25 \) s, one-tailed one-sample permutation test; \( n = 20 \), Fig. 6a,b). Thus, the response-
predictive sensorimotor activity was not limited to the neural aftermath of the previous trial, that is, the beta rebound, but also spontaneous fluctuations unrelated to the previous button-press predicted which button would be pressed 6 s later.

The effect of choice-contingent response planning. All of the above results hold in a situation where choices could be translated into motor responses only after choice formation. Do motor fluctuations also predict responses when choices can be directly mapped onto motor responses? To test this, we recorded MEG during a second decision task in which the choice-response mapping was already cued before the stimulus by swapping the order of the irrelevant and the choice–response cues (choice–response cue for control task: 0–0.25 s).

Motor activity also predicted motor responses in this control task, but weaker. We first focused on the beta rebound as the major source of motor fluctuations. Again, we found evidence for a mechanistic link between beta rebound and response alternation: Across participants, stronger beta rebound significantly predicted stronger response alternation (Fig. 7a, \( r = 0.51 \), \( P = 0.022 \), Spearman correlation; \( n = 20 \)), but descriptively the relationship was weaker than for the original task. Correspondingly, participants showed a weaker tendency to alternate responses in the control task, which was only significant in participants with above average beta rebound (Fig. 7b, mean \( r = 0.04 \), \( P = 0.0085 \), one-tailed one-sample \( t \)-test; \( n = 10 \)), but not across the entire sample (all participants: mean \( r = 0.015 \), \( P = 0.132 \), one-tailed one-sample \( t \)-test; \( n = 20 \)). Also the response-predictive effect of early motor lateralization was significantly weaker in the control task than in the original task (Fig. 7d, \( P < 0.001 \), one-tailed permutation test, \(-1 \) to \( 1.25 \) s; \( n = 20 \)), and reached significance only in participants with above-average beta rebound, not in all participants (Fig. 7 e, and Supplementary Fig. 3, \(-1 \) to \( 1.25 \) s, all participants: \( P = 0.18 \), \( n = 20 \), one-tailed one-sample permutation test; participants with above average beta rebound: \( P < 0.001 \), \( n = 10 \), one-tailed one-sample permutation test). The preference for repeating the same choice as in the previous trial was present in the control task as in original task (mean \( r = 0.055 \), \( P = 0.0075 \), two-tailed one-sample \( t \)-test).

Why was the effect of motor fluctuations on response selection weaker when the choice-response mapping was cued before the stimulus? We hypothesized that this may reflect interference of early response planning with the prestimulus motor lateralization. Indeed, in accordance with previous reports\(^9,10\), for the control task, response-predictive lateralization started already during the stimulus interval (Fig. 7c, \( 2.3 \)–\( 6.1 \) s, \( P = 0.002 \), two-tailed one-sample cluster permutation test, \( n = 20 \)). Thus, in the control task, subjects already mapped choices onto response plans during decision formation, that is, earlier than in the main task. The possibility to plan responses early on may have decreased the
preexistent motor lateralization. To test this hypothesis, we compared the beta rebound between the original and the control task while ruling out confounds due to the different alternation behaviour (Fig. 8a, see ‘Methods’ section). As hypothesized, the beta rebound was significantly decreased for the control task in the late stimulus interval and delay before the second cue, that is, during response planning in the control task (Fig. 8c, $P = 0.010$, one-tailed paired permutation test, $n = 20$). Notably, the beta rebound was also already reduced in the delay interval directly following the early choice-response cue in the control task (Fig. 8b, $P = 0.036$, one-tailed paired permutation test, $n = 20$), which may reflect the suppression of the beta rebound in preparation of the upcoming response planning or processing of the choice–response cue. Together, these results suggest a reduced response-alternation bias in the control task because upcoming or evolving response planning reduces motor fluctuations caused by previous responses.

**Discussion**

Our results provide new insights into sensorimotor decision making on both behavioural and neural levels. We uncovered that a previous motor response can influence sensorimotor decision making. Several factors beyond the present stimulus are known to influence sensorimotor decisions. These factors include neural noise at sensory stages, top-down factors such as stimulus and reward expectations, motor costs associated with response options, and sequences such as the ‘Gambler’s fallacy’, and reward expectations, motor costs associated with response options, and sequences such as the ‘Gambler’s fallacy’,
that is, the mistaken belief that high event incidence is followed by low incidence and vice versa\(^{27}\), or the preference to repeat the previous perceptual choice\(^{10,25–27}\) that we also observed in the present experiment. The Gambler’s fallacy and choice repetition effect are conceptualized on the choice-level, that is, the content of decisions (for example, ‘yes—I saw the target’). In contrast, our results indicate that also previous responses at the level of the motor act (for example, a specific button-press) and independent of previous choices influence which decisions are eventually reported. This unravels a previously unknown decision factor that needs to be accounted for in models of decision making as well as in the analysis and design of decision-making experiments. In fact, our results suggest that, for perceptual decision-making tasks with fixed choice–response mapping, the well-known choice-repetition bias is counteracted by an independent response-alternation bias.

While the demonstrated response-alternation bias is behaviourally detrimental for perceptual decision-making tasks, such as the one at hand, it may be beneficial in specific behavioural contexts. For instance, response alternation may improve sampling of different motor acts to succeed in a task, favoring exploration over exploitation, or it may help prevent motor fatigue.

We identified the post-movement beta-rebound as a strong source of sensorimotor cortex fluctuations that may drive the response-alternation bias. Three lines of evidence support this conclusion. First, subjects with stronger beta-rebound showed stronger response alternation. Second, the strength of beta-rebound predicted the likelihood of response alternation on the single-trial level. Third, removing neuronal variability related to the previous response’s beta-rebound reduced the early response-predictive beta lateralization.

Our results accord well with other recent studies that provide converging correlational\(^ {34–37}\) and manipulative\(^ {38,39}\) evidence for a causal role of beta-oscillations in motor control. Nevertheless, it remains difficult to pinpoint the exact neural source of the demonstrated alternation behaviour based on the present data alone. First, although we found strongest effects in regions consistent with primary motor cortex and applied source-reconstruction to extract primary motor cortex activity, the spatial resolution of MEG is limited. Thus, other regions such as for example, premotor cortex or somatosensory cortex\(^ {40}\) may well contribute to the observed effects. Second, only regions with a prominent macroscopic contralateral motor organization were apt to reflect upcoming or past responses in the present experiment. This organization decreases upstream from primary motor cortex, which reduces response-predictive lateralization. Thus, the effects that we observed over motor cortex may in principle be caused by other upstream cortical or subcortical\(^ {41,42}\) regions that encode response specific information without a somatotopic organization. In addition, post-central somatosensory areas might contribute to the observed beta oscillations. Previous research has demonstrated monosynaptic projections from S1 onto motoneurons\(^ {43}\) and beta coherence between S1 and muscle activity\(^ {40}\). Yet, S1 stimulation does not elicit or facilitate muscle activity\(^ {44}\). Thus, the role of S1 in motor control remains unclear. In sum, while our results suggest an intimate relationship of the motor cortical beta rebound and response alternation, the exact cortical mechanisms that drive response alternation remain to be determined. Ultimately, invasive and manipulative approaches will be required to unequivocally show that motor cortex activity itself causes the response-alternation bias. Independent from the exact cortical stage, our results show that a post-response rebound of neural representations of motor responses predicts response alternation in human decision making.

Furthermore, our results show that even beyond the response-related beta-rebound the state of the sensorimotor cortex before decision formation and unrelated to choice content predicts the final decision-making outcome. Previous studies showed that neuronal activity in motor areas reflects upcoming choices during evidence accumulation if choices and responses are inextricably linked\(^ {10,16,45}\). Our finding of response-predictive, but choice-unrelated activity suggests that sensorimotor cortex activity during decision making does not merely reflect the routing of decision-related activity from higher cognitive areas\(^ {1,12,18,46,48–50}\). As such our results accord well with a growing body of evidence suggesting that motor regions are directly involved in the process of decision making\(^ {1,6,8,11,15,47}\). That said, our results are also well compatible with converging data that suggest a prominent role of frontoparietal association cortices in decision making\(^ {1,12,18,46,48–50}\).

In summary, our results show that not only choice-related neuronal fluctuations but also fluctuations related to the associated motor responses predict sensorimotor decisions.

**Methods**

**Participants.** Twenty healthy, right-handed volunteers (11 female, mean age 29 years) participated in this study. All had normal or corrected-to-normal vision and received monetary reward for their participation. The study was conducted in accordance with the Declaration of Helsinki, and was approved by the ethics committee of the University of Tuebingen. All participants gave written informed consent before participating.

**Behavioural task.** On each trial, participants had to decide whether coherent motion was present in centrally presented dynamic random dot pattern (random dot kinematogram, RDK) and to report their percept (yes/no) by button-presses with the left or the right index finger (Fig. 1a, 2-alternative forced choice). The choice-response mapping was newly assigned on each trial by a colour cue (red or green).
The implementation details of the beamformer were as follows: for every time $t$, frequency $f$ (for frequency-domain beamforming) and source location $r$, three orthogonal filters ($\mathbf{\Lambda} = \{A_x, A_y, A_z\}$; for one spatial dimension) were computed that pass activity from location $r$ with unit gain, while maximally suppressing activity from all other sources:

$$A(r, t, f) = L^1(r)\mathbf{C}_\text{real}(t, f) - L^2(r)\mathbf{C}_\text{real}(t, f)^{-1} \mathbf{C}_0 (r, t, f)$$

Here, $L(r)$ is a matrix whose columns are the leadfields of three orthogonal dipoles at source location $r$, and $\mathbf{C}_\text{real}$ denotes the real part of the complex cross-spectral-density matrix for the data at frequency $f$ and time $t$, and $\mathbf{C}_0$ indicates the matrix transpose. For time-domain beamforming, filters are not frequency dependent and $\mathbf{C}_\text{real}$ denotes a covariance matrix of the sensor-level signals. We derived a joint filter for all contrasted conditions.

We linearly combined the three filters to a single filter pointing in the direction of maximal variance, that is, the dominant dipole orientation. To this end, the filters were weighted with the first eigenvectors’ elements (the eigenvector with the largest eigenvalue of the real part of the cross-spectral-density or covariance matrix at the source location $r$):

$$w = [w_1, w_2, w_3] = \text{Eig}(\mathbf{\Lambda}(r, t, f)\mathbf{C}_\text{real}(t, f)\mathbf{\Lambda}(r, t, f)^T)$$

$$A(r, t, f) = w_1 \cdot A_x(r, t, f) + w_2 \cdot A_y(r, t, f) + w_3 \cdot A_z(r, t, f)$$

To derive the complex source estimates (frequency-domain beamforming), the complex frequency-domain data was multiplied with the real-valued filter:

$$\mathbf{X}_{\text{source}}(t, f) = A(r, t, f) \cdot \mathbf{X}_{\text{sensor}}(t, f)$$

where $\mathbf{X}_{\text{source}}(t, f)$ is the frequency-domain representation at time $t$ and frequency $f$ at the sensor level and $\mathbf{X}_{\text{sensor}}(t, f)$ is the corresponding source signal at location $r$. For time-domain beamforming, $\mathbf{X}_{\text{source}}$ and $\mathbf{X}_{\text{sensor}}$ denote the sensor-level and source-level time courses, respectively.

**Source locations.** To investigate the cortical distribution of choice predictive neuronal activity before the button-press (Fig. 2b), we estimated neuronal activity at 457 locations that homogeneously covered the space at ~0.7 cm beneath the skull with a spacing of ~1.25 cm. This coverage is well adapted to the spatial resolution of MEG, samples sources with high signal-to-noise ratio (SNR) close to the sensors, and covers a large part of the cortex.

Furthermore, we reconstructed neuronal activity specific to the button-press near the hand representation of left and right primary motor cortex. We visually inspected each participant’s cortical map of the contrast between contra- and ipsilateral button-presses in main, control and cued tasks in the time-window from 4.5 to 5.5 s and the frequency range from 12 to 30 Hz. For each participant, we selected the local spatial maximum of this functional contrast closest to the anatomical hand representation, that is, the ‘hand knob’ of the precentral gyrus.

**Physical forward model for source analysis.** For all source analyses, we computed individual physical forward models (leadfields). To match participants, we nonlinearly transformed source locations defined in standard Montreal Neurological Institute (MNI) space into individual head space using the participants’ individual structural magnetic resonance image (MRI). We aligned the MEG sensors to the head geometry based on three fiducial points (nasion, left and right ear, registered during the MEG acquisition by three head localization coils). For each participant, we derived the physical relation between sources and sensors using a single shell model$^{54}$ that was computed based on the segmentation of each participants structural MRI.

**Spectral analysis.** For time-frequency analyses of neuronal activity (Figs 2a and 3a), we source-reconstructed broad-band neuronal activity using time-domain beamforming and employed a sliding window multi-taper Fourier analysis (window size: 250 ms, step size: 20 ms, 8 Hz smoothing, 3 discrete prolate spheroidal sequences tapers). To account for variable response times, we computed two time-frequency transforms: first, with data aligned to the stimulus, and second, with data aligned to the button-press. These time-frequency transforms were concatenated according to the average response time. Power was quantified as the per cent change of power relative to the average pre-cue baseline.

To image the cortical distribution of response-predictive beta-band activity directly preceding the response, we derived the sensor-level cross-spectral-density matrix for frequency-domain beamforming using multi-taper Fourier analysis (4.5–5.5 s, 12–30 Hz) down to 80 ms before the anticipated button press. To investigate the time-course of source-reconstructed beta-band activity, we band-pass-filtered the sensor-level MEG data in the time-domain (12–30 Hz; two-pass Butterworth filter, filter order 4), applied time-domain beamforming, applied the Hilbert transform, and smoothed power time-courses with a 500 ms (full-width at half-maximum) Hanning window. Finally, all time-courses were normalized by the average across time and trials.

**Response-predictive activity.** To isolate neuronal activity that predicted the specific upcoming response (left or right hand), we contrasted power in motor cortex contra- and ipsilateral to the response hand (Figs 2d, 6, 7c,d). This contrast...
isolates effector-specific signals and discards other unrelated neuronal variance providing a specific proxy on neuronal activity involved in decision formation and motor execution. This contrast can be formalized as:

\[
\text{(contra - ipsi)}_{\text{current corr}} = \left( \frac{(L_m - R_m) + (R_\mu - L_\mu)}{2} \right)
\]

where \( L \) and \( R \) stand for the neuronal activity measured in the left and right hemisphere, respectively, and the first and second subscripts denote the previous and current response hand, respectively. \( r \) and \( l \) denote right and left, respectively, \( \mu \) stand for both hand responses, respectively. This contrast can be formalized as:

\[
\text{(contra - ipsi)}_{\text{previous}} = \left( \frac{(L_m - R_m) + (R_\mu - L_\mu)}{2} \right)
\]

The left and right bracketed terms in equation (5) correspond to neural activity contralateral–ipsilateral to current right and left-hand button-presses, respectively.

**Beta rebound.** To estimate the response-specific effect of the previous button-press on the current trial, that is, the beta rebound, we contrasted power in motor cortex contra- and ipsilateral to the previous trial’s button-press (Figs 3c, 8a–c):

\[
\text{(contra - ipsi)}_{\text{previous}} = \left( \frac{(L_m - R_m) + (R_\mu - L_\mu)}{2} \right)
\]

The left and right bracketed terms in equation (6) correspond to neural activity contralateral–ipsilateral to previous right and left-hand button-presses, respectively.

To quantify the response-specific beta rebound for each subject, we averaged lateralization relative to the previous response from 1 to 1.25 s of the current trial.

**Statistical assessment of lateralization.** To assess statistical significance of response-specific lateralization across time and frequency (Fig. 2a) or across time (Figs 2c,d and 3c) and 7c), we calculated cluster permutation statistics that account for multiple comparisons with a false discovery rate of \( \alpha = 0.05 \) (two-tailed) and 1,000 subject-level permutations. For all contrasts tested on responses on the single-trial level we either tested for a difference of the beta-rebound between alternation and non-alternation trials across subjects (random effects), or we tested for a difference of the beta-rebound between alternation and non-alternation trials pooling all trials across subjects (fixed effects). For both approaches, we employed permutations statistics and we z-scored each subject’s single-trial beta rebound data. Thus, both single-trial correlation analyses (random and fixed effects) were orthogonal to the subject-level correlation analysis.

To test if the tendency to alternate responses and accuracy were related, we calculated Pearson’s correlation across participants.

All analyses were performed in MATLaB (MathWorks Inc., Natick, USA) using custom software and the Fieldtrip toolbox.

**Correlation analyses.** To quantify relations between nominal behavioural variables (responses ‘left’ or ‘right’ on current and previous trials) we used Pearson’s correlation coefficient for binary variables (Phi coefficient). To assess statistical significance of correlations, we Fisher-z-transformed subjects’ two-tailed t-statistics across subjects unless noted otherwise.

To test if different aspects of the previous trial modulated the strength of the beta rebound we performed a multivariate partial correlation analysis, with the predictors previous choice, previous response hand, previous target presence, previous accuracy, previous reaction time, and ITI duration following the previous response. For each subject, partial correlation was performed across trials and the significance of predictors was assessed using a two-tailed t-statistics of the Fisher-z transformed r-values across subjects.

To quantify the relation between each participant’s beta rebound and tendency to alternate responses on the subject level, we computed Spearman’s rank correlation across subjects (Fig. 5c). We used the same approach to test for each cortical region, how its beta rebound predicted response alternation (Fig. 5d). To test if the strength of the beta rebound also predicted the tendency to alternate responses on the single-trial level we either tested for a difference of the beta-rebound between alternation and non-alternation trials pooling all trials across subjects (fixed effects). For both approaches, we employed permutations statistics and we z-scored each subject’s single-trial beta rebound data. Thus, both single-trrial correlation analyses (random and fixed effects) were orthogonal to the subject-level correlation analysis.

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