There is more to decisions than meets the eye:

Cortical motor activity and previous motor responses predict sensorimotor decisions

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Synopsis

Abstract

Human behavior is largely guided by sensory information about our environment. The process of transforming sensory evidence into appropriate behavior is called sensorimotor decision making. Despite the many advances in uncovering its neural basis, it remains unclear which role cortical motor areas play in the functional architecture enabling sensorimotor decision making. Specifically, it is unknown whether cortical motor areas actually contribute to the decision making process, e.g. by casting a vote on the response alternatives, or whether they alternatively simply produce the behavior selected elsewhere. To investigate the involvement of cortical motor areas in sensorimotor decision making, we conducted two experiments in which human participants made choices about motion in visual stimuli and reported the choice with one of two manual responses, i.e. button presses with the left or the right index finger. Using magnetoencephalography to measure neural activity during decision making, in the first experiment we showed that activity in sensorimotor areas was predictive of upcoming choices several seconds before the button press and even before stimulus presentation. In part, this activity could be linked to the neural aftermath of the previous trial's choice report, which shifted a measure of cortical activity in sensorimotor areas towards the previously unchosen response alternative in the current trial. This previously unknown tendency to alternate between hands when reporting sensorimotor decisions was significant and varied in size with the size of the neural aftermath of the previous button press over sensorimotor areas across several independent statistics. The results show that beyond the current stimulus, i.e. beyond what meets the eye, other factors like the previous motor act may influence response selection in sensorimotor decision making. Additionally, the results suggest that this is driven by the neural aftermath of previous responses in cortical motor areas. More generally, this suggest that neural fluctuations in cortical motor areas can influence response selection in sensorimotor decision making. This means that cortical motor areas may be more than an output stage in sensorimotor decision making. Consistent with this interpretation, we showed that response alternation in sensorimotor decision making can be manipulated in a directed fashion through instructed and non-choice-related simple button

presses in an independent group of participants in our second study. This result establishes that previous motor acts can influence response selection in sensorimotor decision making, independent of whether they are choice-related or simply instructed. Given this generalization beyond choice-driven button presses, the results of the second experiment are consistent with the interpretation that response alternation is at least partly driven by neural correlates of previous motor acts. In summary, our results suggest that neural fluctuations in cortical motor areas can influence response selection in sensorimotor decision making, in turn suggesting that motor areas may be more than an output stage of the brain during sensorimotor decision making.

General introduction

Imagine driving a car across Tübingen's Neckar Bridge. Just as you approach the intersection to Gartenstraße, the traffic light turns orange. How do you respond?

Each day, we make hundreds of decisions like that, where we have to select between different actions guided by sensory evidence – a process we call sensorimotor decision making.

Often, there is more than one correct response: as you approach the traffic light, your reaction to hit the breaks or accelerate may depend on whether there's a car behind you or a pedestrian on the sidewalk eager to cross the street. In other words, decision making is context-dependent and must be highly flexible. Therefore, the general consensus is that even simple decisions like that are made in higher brain areas (Heekeren et al., 2004, 2006; Noppeney et al., 2010; Merten and Nieder, 2012; Mante et al., 2013; Filimon et al., 2013; Siegel et al., 2015) thought to orchestrate behavioral flexibility (Miller and Cohen, 2001; Buschman et al., 2012; Crowe et al., 2013). Nevertheless, putting the decision into effect to reach a certain goal can only be achieved by carrying out one movement alternative - moving the foot to the left or keeping it on the right pedal. In other words, the sensorimotor decision process comes down to selecting a movement plan and carrying it out, which sometimes has to happen extremely fast.

Given that sensorimotor decision making culminates in selection between competing actions, one interesting question to ask is how this is reflected in the functional architecture underlying sensorimotor decision making. Are motor areas just an output stage of the brain, merely producing the behavior decided upon elsewhere, or are they rather an active part of the architecture underlying sensorimotor decision making?

Traditional view

Had you asked the early cognitive scientists, they surely would have replied that motor areas are only an output stage of the brain because they viewed cognition and action as separate modules of the mind's functional architecture (Fodor, 1983). According to a modular view, each module - and associated brain area - has its own specialized function, each of which is completed before its result is transferred to the next module: Sensory areas analyze sensory input with respect to their preferred features, upon which a central executive builds mental representations of the world. Then, the central executive decides on how to behave and sends appropriate commands to motor areas which carry out movement. Therefore, in a modular mind, motor areas are merely the output stage that controls execution of movement.

Choice variables throughout the brain

Instead, the rich body of current evidence supports a less rigid, more fluid view of the brain: recent neural and behavioral evidence suggest that decisions arise in networks and information is shared continuously between brain areas in the network to support time- and context-flexible behavior such as decision making (Lafuente and Romo, 2006; Heekeren et al., 2008; Pesaran et al., 2008; Siegel et al., 2011; Filimon et al., 2013; Crowe et al., 2013; Siegel et al., 2015). For instance, such a network with continuous information flow is demonstrated by a study in monkeys making perceptual decisions about the color and direction of randomly moving dots which they had to report with saccades (Siegel et al., 2015). While the monkeys were making decisions, the activity in six cortical areas (V4, MT (middle temporal), IT (inferior temporal), LIP (lateral intraparietal), lPFC (lateral prefrontal cortex), FEF (frontal eye field)) was measured simultaneously. The authors analyzed the temporal dynamics of information about decision-related variables (the choice itself, the task-rule, the identity of the rule-cue and sensory information about the motion and about the color) flowing through the network. The choice itself first arose in two areas simultaneously, i.e. in LIP, an area in posterior parietal cortex (PPC) which is concerned with transforming sensory information into saccadic motor plans, and PFC. Slightly later, it was also represented in downstream motor areas (FEF) and upstream higher sensory areas (V4,MT,IT).

Fronto-parietal areas as the locus of choice commitment

Given that choice-predictive activity first arose in fronto-parietal areas, Siegel's data support the current predominant view that sensorimotor decisions are computed and committed to in fronto-parietal areas (Huk and Shadlen, 2005; Freedman and Assad, 2006; Heekeren et al., 2006; Hanks et al., 2006; Merten and Nieder, 2012; Filimon et al., 2013; Mante et al., 2013; Latimer et al., 2015; Li Hegner et al., 2015) [but see also recent literature challenging the causal involvement of parietal areas (Erlich et al., 2015; Katz et al., 2016)]. Beyond that, several other properties qualify the fronto-parietal activity as the decision variable proposed in influential models of decision making (Smith and Ratcliff, 2004; Gold and Shadlen, 2007): First of all, during presentation of sensory evidence, neurons in prefrontal and posterior parietal regions often show very similar response profiles (Pesaran et al., 2008; Siegel et al., 2015) [but see also (Hanks et al., 2015)]. Second, the activity profile looks like a temporal integral of neural activity from sensory areas, i.e. fronto-parietal activity ramps up as sensory evidence is accumulated during stimulus presentation. Also, after the stimulus is over, the activity level is maintained until a choice-contingent motor response can be made as if to keep the choice available before it is translated into motor output (Shadlen and Newsome, 1996; Kim and Shadlen, 1999; Shadlen and Newsome, 2001; Roitman and Shadlen, 2002; Mazurek et al., 2003; Huk and Shadlen, 2005; Kiani et al., 2008; Bennur and Gold, 2011; Merten and Nieder, 2012). Third, when aligned to the time of saccade onset, neural activity in the fronto-parietal network always reaches a certain activity level (Kim and Shadlen, 1999; Shadlen and Newsome, 2001; Roitman and Shadlen, 2002; Kiani et al., 2008) which can be interpreted as evidence for a threshold that activity needs to pass for the monkey to be able to commit to a choice. Together, these properties qualify the fronto-parietal network as a good candidate where choices are made.

Choice variables modulate motor activity

To put a sensorimotor decision into effect and reap its benefits, a movement such as a saccade or a hand movement has to be made. Therefore, choice-predictive activity can also be decoded from motor areas (Kim and Shadlen, 1999; Thompson and Schall, 1999; Gold and Shadlen, 2000, 2003; Song and Nakayama, 2009). The activity predictive of the upcoming choice in motor areas is not all-or-none, as would suffice for preparation of a motor response. Instead,

it is graded: like the integrated decision variable, it builds up during the accumulation process with just a few milliseconds delay with respect to the fronto-parietal choice network and is graded depending on the available perceptual evidence. That means for difficult decisions with less sensory evidence choice-predictive motor activity ramps more slowly than for easy decisions with a lot of sensory evidence. This was shown for example for FEF neurons in monkeys reporting choices about the random dot motion stimulus with saccades (Kim and Shadlen, 1999). It was also demonstrated by an elegant study where neurons in FEF were stimulated to evoke a saccade into a direction orthogonal to two saccadic choice targets (Gold and Shadlen, 2000). Gold and Shadlen measured the deviation of the evoked saccade away from the orthogonal axis towards one of two choice targets and found that it deviated as a function of motion strength and stimulus duration. That means, neural motor plans not only reflect the upcoming choice but even the available perceptual evidence for the different choice alternatives.

Similar results have been reported for premotor and primary motor areas in tasks where choices guided selection between different upper limp movements in monkeys (Lafuente and Romo, 2006; Pastor-Bernier and Cisek, 2011; Thura and Cisek, 2014) and in humans (Donner et al., 2009; Wyart et al., 2012; Kubanek et al., 2013; Lange et al., 2013), showing that modulation with decisional variables is a general principle that holds across different effector systems.

Furthermore, Pastor-Bernier and Cisek showed that premotor neurons encoded the reward value of the target in their response field dependent on which value the other target had. That is, reward value was encoded in a relative manner in premotor neurons showing that modulation of neural motor plans is not exclusive to perceptually-guided choices but extends to reward-related information, too (Roesch and Olson, 2003; Pastor-Bernier and Cisek, 2011).

Also, modulation with the decisional variables does not stop at the cortex. Even beyond cortex, decision formation can modulate neural activity (Michelet et al., 2010; Selen et al., 2012). For example, Selen and colleagues have recently demonstrated that reflex gains (derived from the electromyogram (EMG) in response to perturbations of a joystick) changed with the strength of sensory evidence and viewing duration in a perceptual decision task. Similar results have been found for value-based decisions: cortico-spinal excitability measured

with transcranial magnetic stimulation (TMS) over primary motor cortex reflected the dynamics of choice-to-action transformation (Klein-Flügge et al., 2013).

Prestimulus motor activity predicts sensorimotor decisions

The available evidence on motor activity during the decision making process reviewed above points towards an architecture where detailed choice-related information continuously flows all the way into cortical motor areas and beyond. Such an architecture allows for motor activity to reflect decisional variables during decision making, leading to the fact that decisional variables and choice-contingent response planning often cannot be distinguished in motor area activity. Because this is the case, activity in motor areas may play an active role in sensorimotor decision making beyond reflecting decision variables routed here from elsewhere (Shadlen et al., 2008). Indeed, it has even been suggested that motor areas may be the locus of choice commitment (Cisek and Kalaska, 2005, 2010, Cisek, 2006, 2007). However, the evidence reviewed above is also fully compatible with a functional architecture where motor areas are only the output stage of the brain. In this case, their modulation with decision variables may merely be a consequence of continuous flow, e.g. reflecting probability for one movement relative to another.

Here, we investigated the role of motor areas in sensorimotor decision making. More specifically, we were interested whether choice-predictive signals in motor areas merely reflect continuous flow of decisional variables into motor areas for choice-contingent response preparation, or whether alternatively, motor areas can actually influence decision making.

This question had not been investigated so far, because the levels “choice” and “response” are often inextricably linked in experimental paradigms. In Siegel’s experiment for instance, to report having seen more green dots (choice: “green”), monkeys always made a saccade towards the left (response: “left”), whereas for choosing “red”, they always made a saccade towards the right. Such a fixed relationship between choices and responses does not allow a separate analysis of the choice content (“red” / ”green”) and the behavioral response (“left” / ”right”), nor a separation of these levels in the neural motor activity. Therefore, it is unknown how much of the choice-predictive activity in

motor areas was relayed there, i.e. reflected true information about the choice, and how much was contributed by neural fluctuations (such as pre-mature response plans or even neural noise).

To elucidate the role of motor areas for sensorimotor decision making, we had 20 right-handed human participants make decisions about the presence of coherent motion (“yes” / ”no”) in the random dot motion stimulus. They reported their choices with button presses with the left and right index finger. Importantly, we randomly assigned the mapping from choice to response anew on each trial (Bennur and Gold, 2011; Merten and Nieder, 2012) to be able to analyze the impact of neural fluctuations in motor areas for decision making. If cortical motor areas can causally contribute to response selection in sensorimotor decision making, neural motor activity should predict upcoming responses before decisions can be made.

Using beta oscillations to index motor preparation and decision making

To assess the impact of neural fluctuations in motor areas during sensorimotor decision making, we measured the participants’ magnetoencephalogram (MEG) while they were making the sensorimotor decisions. The MEG represents the summed activity of thousands of neurons, more specifically the tiny magnetic fields co-occurring with intracellular current flow in the apical dendrites of pyramidal neurons following transmembrane ion flow caused by synaptic activation (Hämäläinen et al., 1993; Baillet et al., 2001). We focused on low-frequency oscillations in the beta range, i.e. from 12 to 30 Hz that we source-reconstructed from cortical motor areas. In sensorimotor areas, these beta oscillations have mainly been associated with motor function and are known to change stereotypically during different phases of movement (Pfurtscheller, 1981; Jenkinson and Brown, 2011; Kilavik et al., 2013). Typically, before a movement, power in the beta range is reduced. This change is found in both hemispheres, but often this reduction in power is stronger over the hemisphere contralateral to the upcoming movement (Pfurtscheller, 1981; Leocani et al., 2001; Szurhaj et al., 2003; Doyle et al., 2005; Zhang et al., 2008). After movement, power in beta oscillations over sensorimotor areas rebounds, i.e. rises beyond pre-movement amplitudes, again in a lateralized fashion but with opposite polarity as the pre-movement power-drop (Salmelin and Hari, 1994; Pfurtscheller et al., 1996; Jurkiewicz et al., 2006; Parkes et al., 2006).

These stereotypic changes in lateralization of power of beta oscillations accompanying movement allow predicting the side of upcoming movement.

By monitoring it throughout the whole trial, i.e. before and after the choice has been made and the choice-response mapping is revealed, we can answer our experimental question. Of course, as soon as participants are informed about the mapping from choice to a particular motor response, beta power will strongly lateralize in preparation of a choice-contingent motor response. Conversely, any lateralization occurring before participants know the mapping from choice to response or even before they have seen the stimulus to make their choice about it, cannot be related to the choice level but to the response level only. If such lateralization existed before the choice-response mapping, it would suggest an active involvement of motor areas in response selection in sensorimotor decision making.

To assess the temporal dynamics of possible neural fluctuations in motor areas during sensorimotor decision making, we kept choice and response level distinct for as long as possible. That means we informed participants about the choice-response mapping only one second after they had formed a decision. In a control condition, we already informed the participants about the choice-response mapping before decision making, to assess the impact of choice-contingent motor planning on the role of motor areas can influence sensorimotor decision making. Results were very similar across conditions:

Results

We found that fluctuations of beta lateralization predicted the upcoming response already more than six seconds before the button press when lateralization could not yet reflect choice-contingent signals. Because these fluctuations were present already at trial onset, we wondered whether they may be related to the neural aftermath of the previous response, i.e. the beta rebound. Indeed, the beta rebound of the previous response had a strong impact on beta lateralization in the current trial and was still present at the time of button press in the current trial. As the beta rebound has the opposite polarity as the pre-movement beta lateralization and dominated lateralization in a long-lasting manner, this made a specific prediction for behavior: If fluctuations of beta lateralization in motor areas can impact response selection, participants should tend to choose the opposite of the previous response on the current trial. As expected, in the behavioral analysis, we found that the previous motor

response negatively predicted the motor response on the current trial. That is, across trials, participants had a tendency to alternate between the left and right button press. Linking behavior and beta rebound, this response alternation tendency was correlated to the size of the beta rebound on the single trial as well as the group level. Furthermore, correcting for the previous response removed part of the variance in prestimulus response predictive lateralization, showing that the previous response's beta rebound contributed to the response-predictive lateralization. However, even after removing the impact of the beta rebound, neural fluctuations still predicted the upcoming response. Following the response alternation bias should of course be disadvantageous for performance on the sensorimotor decision task. And indeed it was, as we found a correlation between the strength of alternation and accuracy.

Discussion

These results allow new insights into sensorimotor decision making on the behavioral as well as on the neural level. By demonstrating that neural fluctuations in motor areas can predict upcoming responses even before decision making, they suggest that neural fluctuations in motor areas may play a role for response selection in sensorimotor decision making. On the behavioral level, we were able to uncover the behavioral bias of response alternation which was previously unknown to exist. Several lines of evidence suggested an involvement of the neural aftermath of the preceding response in driving this effect. Together these results suggest that motor areas are more than an output stage in sensorimotor decision making because they seem to be able to tip the scales during response selection in sensorimotor decision making. If the interpretation is correct and the fluctuations in cortical motor areas are causal rather than correlational for response selection, then this would suggest an active role of motor areas in sensorimotor decision making. This interpretation goes beyond what is commonly accepted for the role of motor areas in decision making, but resonates well with theories on action-centered cognition.

Signal fluctuations in motor areas seem to impact sensorimotor decision making

Our results suggest that neural noise in motor areas can bias response selection in sensorimotor decision making. As such they accord well with reports that stimulation in motor areas can influence selection of motor responses in

cognitive tasks. For instance, Javadi et al recently tested the effects of transcranial direct current stimulation (tDCS) on primary motor cortex during a perceptual decision making task with bimanual responses (Javadi et al., 2015). They found that anodal stimulation which is known to have excitatory effects without directly eliciting responses (Nitsche and Paulus, 2000, 2001) biased response selection when reporting perceptual choices towards the hand contralateral to stimulation, whereas cathodal stimulation which is inhibitory biased participants' responses toward ipsilateral hand responses. This is an important study supporting our conclusion that motor areas may be involved in sensorimotor decision making, because it actually shows both increases and decreases of lateralized responses when reporting perceptual choices in a stimulation-polarity- and stimulation-site-selective manner. More support for this conclusion comes from studies showing reductions of motor responses contralateral to reversible inactivation of one area during cognitive but not in simple motor tasks (Schieber, 2000; McPeek and Keller, 2004), and increases of contralateral behavior after stimulation to an area (Carello and Krauzlis, 2004). It also follows from our observations, that choice-predictive activity in cortical motor areas in many decision making tasks reported in the literature may actually be a conglomerate of both choice-contingent activity routed here from upstream areas and motor-cortex intrinsic activity fluctuations related to movements.

Beta - causal or epiphenomenal?

Several independent statistics suggest that between-hand response selection in our decision making task was biased by the neural aftermath of previous responses, i.e. the beta rebound. As such the results suggest that beta oscillations may have causal relevance for motor function, a question which is still discussed (Jenkinson and Brown, 2011). A number of recent studies provide converging evidence for a causal involvement of beta oscillations in motor function. For instance, Brown and colleagues showed that upregulation of cortical beta synchrony using transcranial alternating current stimulation (tACS) during a Go/No-Go task reduced the initial rate of force development and peak force rate (Joundi et al., 2012). Similarly, entrainment of beta oscillations using tACS slowed peak acceleration and deceleration in a motor tracking task (Pogosyan et al., 2009).

Moreover, it is well-known that cortical beta oscillations are synchronous with activity in muscles (electromyographic activity, EMG) (Conway et al., 1995;

Baker et al., 1997, 2003; Kilner et al., 2000). One both elegant and insightful study makes use of this link and shows results suggesting that beta oscillations may be of causal importance: Brown and colleagues measured the peak acceleration of cued finger movement as a function of cortical beta synchrony (Gilbertson et al., 2005). Participants were cued to move based on the amplitude of cortical beta synchrony. Because this couldn't be assessed with sufficient spatial resolution in healthy participants with noninvasive methods, it was assessed indirectly by measuring microtremor in finger muscles which in turn is a product of corticospinal synchrony. The authors found that during periods of elevated beta synchrony, cued movements were slower than during periods of low beta synchrony. The study confirmed their results in two patients where cortical beta synchrony was measured with intracranial measurements (electrocorticography, ECoG). Similarly, participants upregulate corticospinal beta synchrony when they are warned of an upcoming finger stretch and in turn downregulate beta when they are warned of the necessity to make a speeded finger movement (Androulidakis et al., 2007).

This body of evidence suggesting causal relevance of beta oscillations for motor control is also in line with extensive research on Parkinson's disease patients who in parallel display chronically increased levels of beta oscillations and characteristic behavioral symptoms such as bradykinesia, i.e. slowed movement (Brown, 2007).

Bi-hemispheric and cortico-spinal mechanisms contributing to response selection and competition

The aforementioned evidence suggests a causal, anti-kinetic role of beta oscillations measured in motor areas. Nevertheless, its exact function during different phases of movement, e.g. how suppression of beta power contributes to movement selection, or what the function of the beta rebound is, remains unclear (Kilavik et al., 2013).

Partly, this uncertainty may prevail because also more generally it is still unclear how motor control is organized at a network level (Graziano, 2011; Churchland et al., 2012; Shenoy et al., 2013), giving rise to signals that we can measure with MEG. Also, the lateralization of motor control may be less clear-cut than previously thought: there is increasing evidence that bilateral networks contribute to unilateral movement control (Horenstein et al., 2009; McCambridge et al., 2011; Montgomery et al., 2013; Buetefisch et al., 2014;

Uehara et al., 2015; Li et al., 2016). Ipsilateral control may be exerted via co-activation of homologue areas via interhemispheric connections via the corpus callosum or by subcortical connections and/or via uncrossed fibers from ipsilateral primary motor cortex (Uehara and Funase, 2014).

Because of this data situation, we can merely speculate about the exact mechanisms driving our effects, i.e. whether, how, when and where the beta oscillations that we observed as predictive of upcoming responses may have contributed to between-hand response selection. Several mechanisms are conceivable based on the literature: For instance, the lateralized beta rebound may simply have reduced the capacity for movement planning in one hemisphere and in turn passively increased the probability for motor output from the opposite hemisphere, in line with the suggested (Salmelin et al., 1995; Solis-Escalante et al., 2012), yet debated idea (Kilavik et al., 2013), that beta rebound's function is active inhibition. Alternatively, there may be some form of active interhemispheric competition (Ferbert et al., 1992; Vidal et al., 2003; Carbonnell et al., 2004; Praamstra and Seiss, 2005; Müller-Dahlhaus et al., 2008). While these ideas are intriguingly simple, they likely do not capture the whole picture.

One noteworthy study directly set out to investigate the question how beta oscillations contribute to between-hand response selection by analyzing bi-hemispheric, simultaneous up- and down-regulation of cortical beta synchrony, corticospinal coherence and corticospinal phase synchronization (Wijk et al., 2009). After a response-instructive cue, they found contralateral beta power to be down- and ipsilateral corticospinal coherence and phase synchronization to be up-regulated simultaneously. Later, that is beginning 1s before the response, all three measures showed less synchrony in the contra- than in the ipsilateral hemisphere. Because neither measure individually showed both up- and down-regulation in opposite hemispheres simultaneously, their results suggest that all investigated mechanisms may contribute to between-hand selection in concerto, acting together to select and suppress competing responses. These results are in line with literature suggesting involvement of corticospinal coherence in motor control by demonstrating relationships between motor outputs and levels of coherence between cortex and EMG (Conway et al., 1995; Baker et al., 1997; Schoffelen et al., 2008; Muthuraman et al., 2012; Mehrkanoon et al., 2014).

This relationship between behavior, cortical beta and corticospinal coherence demonstrated by Wijk's results can potentially also offer an explanation for an open question concerning our data: It may explain why we observed the response-predictive lateralization only before the stimulus interval (Fig. 2d in (Pape and Siegel, 2016)) despite the fact that the beta rebound of the previous response continued throughout the next stimulus presentation until the next response (Fig. 3c in (Pape and Siegel, 2016)). As shown by Wijk and colleagues, several measures of beta oscillations seem to change simultaneously during between-hand response competition. It is therefore possible that during stimulus presentation the response bias was present in a measure other than the lateralization of beta power, e.g. in corticospinal phase synchronization. Independent from such a possible mechanism, it is important to note that observing the response-predictive lateralization only before the stimulus does not allow us to draw any inferences about the point in time when motor fluctuations may have contributed to response selection in sensorimotor decision making.

Subcortical contributions to motor control and decision making

Beyond the cortex and the cortical mechanisms underlying motor control discussed here, there are of course many subcortical structures and mechanisms contributing both to motor control and to decision making. One subcortical structure that seems to combine both functions are the basal ganglia, a fact which makes them worth mentioning here. The basal ganglia have long been known to be involved in motor control because of their heavy bi-directional connections to cortical motor areas (Mink, 1996; Shadmehr and Krakauer, 2008; Redgrave et al., 2010; Brittain and Brown, 2014), and recently have also been implicated in perceptual decision making (Ding and Gold, 2010, 2013; Green et al., 2013; Wei et al., 2015; Herz et al., 2016; Perugini et al., 2016; Seymour et al., 2016; Zénon et al., 2016). In line with that, patients with Parkinson's disease, who lack dopaminergic input to the basal ganglia, suffer not only from severe impairments in motor control, but also from cognitive deficits, e.g. in decision making (Perugini et al., 2016). Moreover, areas in basal ganglia display beta oscillations synchronous to cortical motor areas (Hirschmann et al., 2011; Litvak et al., 2012). It may therefore be interesting to look for changes in oscillations measured in basal ganglia nuclei in a task like ours and see whether the basal ganglia may also be involved in response alternation.

Gamma and oscillations more generally

Oscillations such as beta oscillations reflect synchronized activity within or between populations of neurons. Our findings suggesting the involvement of beta oscillations in response selection in sensorimotor decision making fit well into the existing literature delineating the view that oscillations have functional significance for computations within local and long-range networks (Engel et al., 2001; Buzsáki and Draguhn, 2004; Fries, 2005; Womelsdorf and Fries, 2006; Donner and Siegel, 2011; Hipp et al., 2011; Supp et al., 2011; Siegel et al., 2011; Singer, 2011; Joundi et al., 2012; Siegel et al., 2012; Picazio et al., 2014; Fries, 2015; Bastos et al., 2015). Beyond oscillations in the beta range also changes in gamma band frequencies are frequently observed in motor areas at the time of movement execution (Crone et al., 1998; Pfurtscheller et al., 2003; Cheyne et al., 2008; Donner et al., 2009; Muthukumaraswamy, 2010). Likewise, stimulation studies suggest their functional importance for motor control (Joundi et al., 2012), more specifically, a pro-kinetic function. Investigating the involvement of gamma oscillations may be an interesting endeavor to fill in the blanks in the picture how oscillations contribute to motor control and decision making.

Preceding motor responses predict sensorimotor decisions

As discussed above, the strength of lateralized pre-stimulus beta power indexed the probability for response alternation according to several independent statistics. Therefore, several lines of evidence provide reason to assume that the previous response's neural aftermath in motor areas drove the response alternation effect across consecutive trials of our sensorimotor decision task. This interpretation makes a testable prediction: If it is correct, any movement sharing the motor cortical circuitry with an upcoming choice-contingent response should be able to impact response selection in the upcoming sensorimotor decision.

Alternatively, response alternation may be an effect that is specific to decision making, which would suggest that it may have a "cognitive" origin. An example for such a mechanism is the "Gambler's fallacy", which is the false belief that random events are not independent, i.e. that high incidence of one event category will be followed by low incidence (Jarvik, 1951; Senders and Sowards, 1952). Along these lines, the response alternation effect may for instance reflect a "strategy" to sample different motor outputs when the participant faces low sensory evidence. Importantly, suggesting that the response alternation effect be of cognitive origin demotes motor signals such as the beta rebound as a mere *correlate* instead of a driving force of response alternation. Therefore, according to this hypothesis, response alternation as we have observed it should occur preferably between choice-contingent motor responses, but not subsequent to a choice-unrelated movement.

We can distinguish between these two alternative hypotheses, i.e. the motor origin from the cognitive origin, by introducing instructed, choice-unrelated button presses between choices. If response alternation is of cognitive origin, it should persist under these circumstances because response alternation should occur systematically only between choice-related motor responses. If alternatively response alternation really is driven by motor signals as we suspect, then we should be able to manipulate it by instructing extra button

presses. This is because movements are stereotypically followed by the beta rebound (Salmelin et al., 1995; Pfurtscheller et al., 1996, 1998) and effects should therefore play out on selection of the following choice-contingent motor response.

Results

First, to see whether we can replicate the response alternation effect, we measured the level of response alternation without any intermediate instructed button press. The analysis confirmed our previous finding that participants tend to alternate between response options on consecutive choice-related button presses. This condition also serves as a baseline to which we compared the manipulation conditions: To tackle the question whether this response alternation effect is of a cognitive or motor origin, we randomly instructed participants to press a button in between choice-related button presses. As expected if response alternation has a motor origin, we found that the instructed button presses did have an effect on the response alternation effect. Importantly, and in line with the motor origin, we found different effects depending on whether the instructed button press is the same or different than the previous trial's choice-related button press. In "different" trials, where the instructed button press and previous choice-related button press were different, there was no correlation between two choice-related button presses, suggesting that the intermediate button press disturbed the response alternation effect. A direct test between the response relationship in the baseline and on "different" trials confirmed that the instructed different button press significantly reduced the tendency to alternate between choice-related button presses. In contrast, we found a correlation between two choice-related button presses on "same" trials where instructed and previous choice-related button press were the same. Importantly, the correlation between choice-contingent button presses on "same" trials was increased, as was revealed by the direct comparison between baseline and "same" trials. Indeed, the motor hypothesis predicts that the response alternation effect may be slightly stronger when the instructed button press is a repetition of the choice-related button press, because the neural aftermath of two same responses may add up and push the motor system towards selecting the opposite motor response. Two further findings support the motor hypothesis. First, response alternation also exists between the instructed and the following choice-related response. Interestingly, the strength of this

effect was dependent on the laterality of the previous choice-contingent response, suggesting a shared neural substrate. Second, another sequential effect, namely the choice-repetition effect (Fründ et al., 2014; John-Saaltink et al., 2016) was left intact by our manipulations, suggesting that our manipulation specifically affected response selection mechanisms in the motor system.

Discussion

Our results confirm the idea that preceding movement has an impact on sensorimotor decisions. The pattern of results that we observed matched exactly what was predicted if neural correlates of preceding movement in cortical motor areas can bias response selection. That means our results suggest that response alternation is driven at least in part by the neural aftermath of preceding movement in motor areas.

Decisions are more than meets the eye

Our results establish the preceding movement as a factor that needs to be accounted for in the analysis and design of experiments. Beyond the current stimulus, a long list of factors is known to influence sensorimotor decision making: noise in sensory areas (Britten et al., 1992; Faisal et al., 2008; Wimmer et al., 2015), expectations about stimuli (Kok et al., 2013), rewards (Rorie et al., 2010) or motor costs (Cos et al., 2011; Moher and Song, 2014; Gross et al., 2015; Marcos et al., 2015), the recent history of previous choices such as the choice repetition effect (Gao et al., 2009; Lange et al., 2013; Fründ et al., 2014; John-Saaltink et al., 2016) and the “Gambler’s fallacy”, i.e. the erroneous belief that the probability of random events is somehow linked.

Response alternation – a feature or a bug?

It is not straight-forward what the function of the response alternation effect may be, i.e. why repeating the previous motor response should be avoided in a sequence of decisions. Beyond the response alternation effect, we found another sequential behavior in our data: the tendency to repeat the previous choice (Fründ et al., 2014). Unlike the response alternation effect this tendency operates on the level of the percept and a functional explanation is available: it is thought to reflect the visual constancy prior (Burr and Cicchini, 2014; Fischer and Whitney, 2014; Liberman et al., 2014). The visual constancy prior denotes a shifted prior probability towards a previous percept, reflecting the statistics of natural environments, where the visual scene is constant in time and space over short timescales. Thereby, it improves perception under natural conditions. In

laboratory settings, however, where stimuli are presented randomly, the prior is known as the choice repetition bias and impairs performance in perceptual decision making (Lange et al., 2013; Fründ et al., 2014; John-Saaltink et al., 2016). Interestingly, our results suggest that the visual constancy prior playing out as the choice-repetition bias may be much stronger than thought because it is counter-acted by the response alternation bias when the mapping between choices and motor responses is fixed.

In analogy to the relationship between choice repetition bias and visual constancy prior, may response alternation also reflect a prior or more generally be beneficial under natural conditions? To answer this, we look at behaviors involving alternation behavior. However, for many sensorimotor behaviors, the levels of analysis choice and motor response are typically not dissociable under natural conditions. That means, it is often not clear whether alternation happens between movements or between the effects these movements bring about. Therefore, we have to turn to studies inspired by such naturally-occurring alternation behavior which experimentally separate these levels: One such study is based on the observation that humans have a preferred hand for carrying out speed- or accuracy-demanding movements like hand-writing or throwing a ball, but also use the non-preferred hand for less demanding movements like reaching. This is also true for monkeys (Gardinier et al., 2005). To investigate what drives between-hand choice in reaching, Lee and Schieber examined the interaction of effector and target laterality in three monkeys reaching for one of two identical food morsels (Lee and Schieber, 2006). When the monkeys had a choice about both the target and the hand to reach with, they tended to alternate between targets as well as the hand with which to reach for it. Because the tendency to alternate between hands was stronger than the tendency to alternate between targets, and because monkeys had a preference to reach ipsilaterally, the authors concluded that the alternation between targets was driven at least partly by the tendency to alternate between hands. This suggests that response alternation as we observed it in our sensorimotor decision task in an artificial setting where motor responses were guided by arbitrary visual choices may be an instance of naturally occurring behavior investigated by Lee and Schieber. The authors of this study offered various explanations why such behavior may be optimal in natural conditions: for instance to avoid motor fatigue or when competing for resources with intelligent individuals where non-stereotypical, non-predictable behavior such as switching

between hands may provide advantages. These explanations may also apply to response alternation.

Exploration / Exploitation

Alternatively, Lee and Schieber suggested, that response alternation in reach choices between food morsels may be an instance of exploration in the tradeoff between exploration and exploitation. This trade-off guides spontaneous behavior in natural environments, meaning that organisms are thought to behave based on the answer to the question “Given the outcome of my current behavior, should I keep doing what I do or should I change my behavior?” (Cohen et al., 2007). For instance during foraging, animals have to make decisions about whether to exploit one resource or move on and search for a better source. This conceptual framing of exploration/exploitation being based on reward and outcomes suggests that it operates on the level of choices and that there is a cognitive component to it, in line with the fact that exploration/exploitation behavior is known to correlate with frontal dopamine levels (Beharelle et al., 2015; Kayser et al., 2015). This explanation based on choices and choice outcomes of exploration/exploitation behaviors makes it unlikely that response alternation is an instance of exploration. Nevertheless, it may be interesting to test it because when facing low levels of sensory evidence, concomitant high perceptual uncertainty and lowered chances of reward, it may be beneficial to favor variability in responses to increase the chances of finding a profitable input-response relationship. Also, both data and models suggest that dopaminergic inputs to the basal ganglia which are heavily connected to cortical motor areas and involved in both decision making as well as action selection are also involved in the trade-off between exploration/exploitation (McClure et al., 2005; Humphries et al., 2012). The idea that response alternation in our paradigm may be an instance of exploration can be tested by analyzing the pupil diameter of observers during decision making: Exploration and exploitation is known to be controlled by the locus coeruleus-norepinephrine-system (Aston-Jones and Cohen, 2005). In turn, levels of norepinephrine can be tracked by measuring the pupil size in monkeys and humans (Rajkowski et al., 1994; Aston-Jones and Cohen, 2005) and tonic increases in pupil dilation are known to mark exploration periods (Jepma and Nieuwenhuis, 2010; Hayes and Petrov, 2016). Therefore, to see whether response alternation may be an instance of exploration, it would be interesting to investigate the link between response alternation and pupil size.

Our results are consistent with the idea that response alternation is driven by the neural aftermath of preceding movement in motor areas. This suggests that response alternation arises from the motor neural circuitry involved. It remains unclear whether beyond avoiding motor fatigue it has other behavioral benefits under natural conditions. As we cannot entirely exclude that beyond the suggested motor circuitry other circuitry is involved, this raises the possibility that response alternation in sensorimotor decision making may be an expression of another behaviorally relevant function (c.f. choice repetition). This possibility may be investigated in future work.

General Discussion

Summary

To investigate the involvement of motor areas in sensorimotor decision making, we conducted two experiments. In both, participants had to make choices about motion in a visual stimulus and report the choice with one of two manual responses, i.e. button presses with the left or the right index finger. Using MEG, in the first study, we showed that activity in sensorimotor areas was predictive of upcoming responses several seconds before the button press and even before stimulus presentation. In part, this activity represented the neural aftermath of the previous trial's response, and biased neural motor activity towards the previously unchosen response alternative. Several independent statistics suggested a link between this motor activity and the previously unknown tendency to alternate motor responses in sensorimotor decision tasks. These results suggest that neural fluctuations in motor areas can impact response selection in sensorimotor decision making. This means that motor areas may be more than an output stage of decision-related activity routed here from upstream areas. Instead neural signals pertaining to motor responses may be able to actively impact response selection in sensorimotor decision making. In the second study, we were able to confirm the influence of the previous motor act for sensorimotor decision making and the tendency to alternate between response alternatives. We showed that upcoming sensorimotor decisions can be manipulated in a directed fashion through instructed, simple button presses made before decision making. This result established that previous motor acts can influence response selection in sensorimotor decision making, independent of whether they are choice-related or simply instructed. Given this generalization to different button presses, these results support the idea that neural correlates of previous motor acts bias response selection and drive response alternation. In turn, these results are consistent with the idea that motor areas are more than an output stage of the brain. Beyond the current stimulus, i.e. beyond what meets the eye, other factors, e.g. like the previous motor act can influence sensorimotor decision making.

Locus of choice commitment in motor areas?

Our results resonate well with the suggestion that sensorimotor decision making and motor control may share a common neural circuitry (Cisek, 2007; Cisek and Kalaska, 2010; Shadmehr et al., 2016) because both decision making and motor control seem to be governed by the principle to minimize effort and maximize gains. For instance when humans make free choices between reach targets, the different biomechanics of the reach paths can predict both choice and movement speed towards reach targets even if the reach paths are identical from a visual perspective (Cos et al., 2011; Shadmehr et al., 2016). The less effortful trajectory is chosen more often and participants move faster towards it. Similar results have been shown for production of grip force and choice between walking or flying in starlings (Shadmehr et al., 2016). Interestingly, such effects cannot only be observed in free choices, where it's intuitive that decision making should be sensitive to motor cost and reward, but also in tasks where animals should choose between options along a different dimension such as motion in a random dot motion stimulus. For instance, geometrical properties of the competing motor acts can alter both behavior (e.g. reaction time and number of changes of mind) (Moher and Song, 2014; Marcos et al., 2015; Lepora and Pezzulo, 2015; McPeek et al., 2003) as well as neural motor activity before choice commitment (McPeek et al., 2003; Pastor-Bernier and Cisek, 2011). These effects can be accounted for by assuming that decision making and motor control share a common utility function, and that instead of choosing a target and movement kinematics independently from each other, the two are chosen together based on a utility assigned to a target/movement pair (Shadmehr et al., 2016). In turn, this idea suggests a strong coupling between neural circuits involved in generating movement and the circuits involved in sensorimotor decision making as proposed e.g. by the action affordance hypothesis (Cisek and Kalaska, 2005; Cisek, 2006, 2007; Cisek and Kalaska, 2010). The action affordance hypothesis suggests that different choice alternatives compete with each other as alternative movement plans coded in sensorimotor space, while upstream areas such as the fronto-parietal network merely modulate motor activity for choice commitment (Cisek, 2006). While our results do show that motor areas are involved in sensorimotor decisions making, they do not tell us anything about the locus of choice commitment. They are compatible with evidence suggesting that decisions are made in a fronto-

parietal network as well as with the suggestion of the action affordance hypothesis that choices may be committed to in sensorimotor space.

Network architecture underlying decision making

Our results suggest that neural fluctuations in motor areas may influence decision making. In turn this suggests that decision making may be a network achievement where information about decision-related variables flows between areas, such that activity in all of the areas may be able to impact the decision process. An abundance of experimental data suggests that information may flow bi-directionally through such networks, i.e. that beyond the bottom-up flow from sensory cortices to motor cortices via the fronto-parietal choice network, there is some form of top-down flow originating in prefrontal or fronto-parietal areas (Lamme and Roelfsema, 2000; Nienborg and Cumming, 2009; Nienborg et al., 2012; Siegel et al., 2015). For instance, Siegel and colleagues showed that information about the choice flows not only in the forward direction, i.e. from the fronto-parietal network to motor areas, but also in the top-down direction i.e. back to sensory cortices, possibly through recurrent interactions (Lamme and Roelfsema, 2000; Wang, 2008) made possible by abundant cortico-cortical connections (Felleman and Van Essen, 1991; Johnson et al., 1996). Beyond decision making, this bi-directional flow of information seems to be a prevalent organizational principle underlying many computations with important functions attributed to top-down e.g. goal-directed attentional selection (Corbetta and Shulman, 2002; Buschman and Miller, 2007; Gregoriou et al., 2014).

If we accept the idea of top-down flow as a prevalent principle and combine it with our results suggesting that motor areas seem to contribute to sensorimotor decision making, the question arises whether there may not also be feedback connections from motor areas back to other nodes of the network, e.g. to fronto-parietal areas. Our results cannot answer this question, because they are in principle consistent with an architecture with and without recurrent connections, just as there exist both purely feedforward (Usher and McClelland, 2001; Mazurek et al., 2003; Smith and Ratcliff, 2004) as well as recurrent network models (Wong and Wang, 2006) which both can successfully explain many aspects of the decision making process. Yet, it would be very interesting to explore it. Exploring this question and thinking about possible functions of top-down connections from motor areas could be inspired by and may reveal further support for action-centered approaches to cognition (Engel et al., 2013; König

et al., 2013). Ideas of embodied / enactive cognition and related theories (Varela et al., 1992; Clark, 1999; O'Regan and Noë, 2001; Engel et al., 2013) propose that cognition is dependent on aspects of the body or that action possibilities shape cognition (Engel et al., 2013; König et al., 2013). Put differently, these theories state that we perceive the world in terms of actions, i.e. that the role of cognition is not to create veridical representations of the world but to build action-relevant perception. Top-down feedback about activated action plans could then help to guide perception along action-relevant dimensions.

Testing for top-down flow

How can we test for such top-down flow? One possibility is to ask "Do neural fluctuations in motor areas impact sensorimotor decision making only through the lever on response selection or do they actually alter the choice itself?" This question may be answered by asking how confident subjects feel about their choice in response alternation trials, i.e. how much they actually believe in what they respond when they follow the neural bias in motor areas. If beyond acting as a lever on response selection, motor fluctuations can truly impact decision making on the choice level through top-down feedback from motor areas to e.g. fronto-parietal areas, participants should feel more confident when following the motor bias than when acting against it. Preliminary evidence for an involvement of motor areas in computation of perceptual confidence comes from a brain stimulation study (Fleming et al., 2015).

Another way to tackle the question of top-down information flow from motor areas is to directly investigate the network architecture and information flow between brain areas. The latter one could be done using directed connectivity measures like Granger causality (Bernasconi and König, 1999; Bernasconi et al., 2000) and possibly cross-correlation of power correlations between brain regions (c.f. Womelsdorf et al., 2007). The former one, i.e. non-directed connectivity analysis through power correlations (Bruns et al., 2000) has been done as part of my doctoral work (Siems et al., 2016). In the respective paper we show that simultaneously acquired 64-channel electroencephalography (EEG) and MEG data reveal similar network structures in frequency-resolved power correlations during resting state. The results corroborate power correlation analyses as a connectivity measure that can capture connectivity in large scale networks during perceptual tasks (Hipp et al., 2011) and at rest (Siems et al., 2016).

How well do these results generalize?

We have investigated the importance of motor areas for decision making using a visual stimulus and bimanual responses, an instance of sensorimotor decision making. An important question that remains is whether our results generalize to decisions that have to be reported using other effector systems and more generally to decision making that does not result in overt behavior?

Sensorimotor decisions in different effector systems

To act upon the world, we can use different effectors, some of which are under cranial nerve control such as the eyes and the mouth, and some of which are under control of skeletal muscles such as the hands and the feet. Given that cortical control of skeletal muscles is organized in a macroscopically contralateral pattern (Penfield and Boldrey, 1937), one can assume that all sensorimotor decision processes cumulating in skeletal muscle movement be sensitive to motor fluctuations and also display response alternation. Whether and how motor fluctuations should play out for sensorimotor decisions culminating in eye movements is less clear, as the two eyeballs move in synchrony to the same target in space, which means there is no motor competition between them. Instead, motor fluctuations may play out as competition between targets. Indeed, evidence shows that stimulating the superior colliculus, a subcortical structure involved in the control of eye movements, during a target-distractor task can lead to biases in target selection (Carello and Krauzlis, 2004). Similarly, stimulating one hemisphere's FEF, an important cortical area underlying voluntary control of eye movements, leads to saccades to contralateral space only (Sherrington and Grünbaum, 1901; Bruce et al., 1985). Thus, motor fluctuations which were implicated to give rise to response alternation in between hand choice in our task, might play out as alternation between targets in space for eye movements. Indeed, such behavior is known to exist under the name of "inhibition of return" (Posner and Cohen, 1984), i.e. delayed or even reduced responding to previously visually attended targets in space and is known to be at least partly driven by motor processes (Briand et al., 2000; Pastötter et al., 2007). Together, these considerations outline how our results may generalize to sensorimotor decisions culminating in selection between other effectors than the hands.

Abstract, non-motor decision making

In our task, participants knew throughout the whole trial that they would eventually have to make one of two button presses to report a choice,

suggesting that motor areas may have prepared neural motor plans even when choice-response mappings were revealed only after the stimulus. This has been shown to happen for instructed motor actions (Cisek and Kalaska, 2005; Tzagarakis et al., 2010, 2015; Klaes et al., 2011; Grent-'t-Jong et al., 2014). However, when decision making does not culminate in an action at all, motor areas are most likely not part of the decision making network. For instance, for many value-based decisions but also simple perceptual decisions that do not immediately ensue a motor act, neural correlates of the decision process do not seem to involve motor areas (Filimon et al., 2013). That means when it is not clear with which effector system the decision will have to be reported during choice formation, motor areas do not seem part of the decision making network. In turn, it should of course be impossible for motor fluctuations to impact response selection under these circumstances.

However, our effects may also apply to more "complicated" decisions than perceptual decisions: also in preference- or reward-based decisions choice variables can be decoded from motor areas (Wunderlich et al., 2010; Pastor-Bernier and Cisek, 2011; Hunt et al., 2013). Such observations suggest that as soon as motor areas *can* be recruited to the decision making network, because either the response modality or even the choice-response relationships are known to the decision maker, these areas also *will* be recruited. This suggests that our results may generalize to all kinds of decisions, i.e. also value-based decisions may be subject to motor fluctuations as soon as the decisions guide selection between different actions.

It is worth considering that sensorimotor decision making is most likely the phylogenetically oldest form of decision making. Very likely, evolution's forces selected our ancestors concerning their brains' ability to control sensorimotor behavior. Because brain evolution has been relatively conservative concerning structural changes (Butler and Hodos, 1996; Katz and Harris-Warrick, 1999), we may therefore assume that much of the neural architecture underlying sensorimotor decision making is also part of more complex, abstract forms of decision making and cognition more generally.

Conclusion

Our results provide new insights into sensorimotor decision making on both behavioral and neural levels, suggesting that there is more to sensorimotor decisions than the visual stimulus about which observers are asked to decide.

First of all, we uncovered that a preceding motor response can influence upcoming sensorimotor decisions, leading to a tendency to alternate between response options on subsequent decisions. This establishes preceding movements as a so far unknown factor that needs to be considered in the design and analysis of sensorimotor decision making paradigms. It would be worthwhile to investigate whether and how this alternation tendency relates to other alternation behaviors. More generally, it would be interesting to establish whether alternation between movements may serve a function or simply is a product of neural circuitry.

Our neural results suggest that response alternation is at least in part brought about by the neural aftermath of preceding movements in cortical motor areas. Together with the finding that neural fluctuations in motor cortical areas predict upcoming choice reports long before decision making about the stimulus has begun, this suggests that fluctuations in motor areas can actually cast a vote in response selection during sensorimotor decision making. Our results suggest that the role of cortical motor areas may exceed the previously assumed function of merely planning and executing choice-contingent actions. This interpretation is consistent with hypotheses assuming large overlaps of crucial neural circuitry between decision making and motor planning and more generally action-centered accounts of cognition.

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Papers

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Motor cortex activity predicts response alternation during sensorimotor decisions

Anna-Antonia Pape[1,2] & Markus Siegel[1]

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.

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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[1–4]. 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[4]. Before choice commitment, motor cortex activity already reflects competing response options[5–8], and if choices are inextricably linked to a specific response during decision formation, activity in motor areas[6,9–12] 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 s, 10–44 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 to 1.1 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).

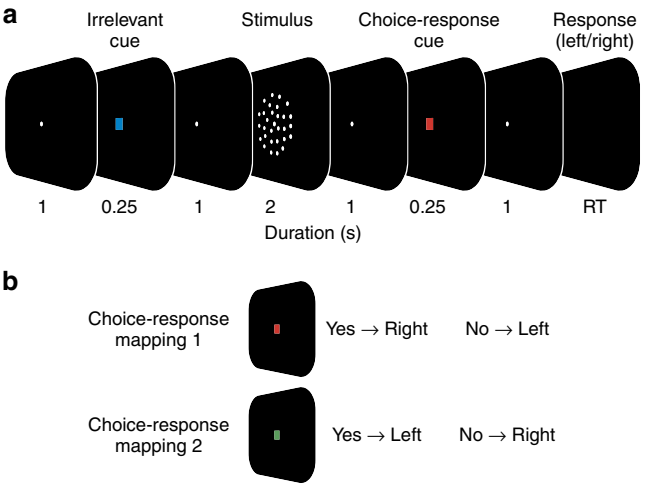

Figure 1 | Visuomotor decision task. (**a**) Participants reported the presence of coherent motion in a display of randomly moving dots with a left- or right-hand button-press. In each trial, the mapping from choice to response hand was newly assigned with a colour cue after the stimulus (choice-response cue). Successive trials were separated by a variable length ITI (median ITI: 1,290 ms). (**b**) For a red cue (choice-response mapping 1), participants reported the presence and absence of coherent motion with a right and left hand button-press, respectively. The mapping from choice to response was reversed for the green cue (choice-response mapping 2).

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

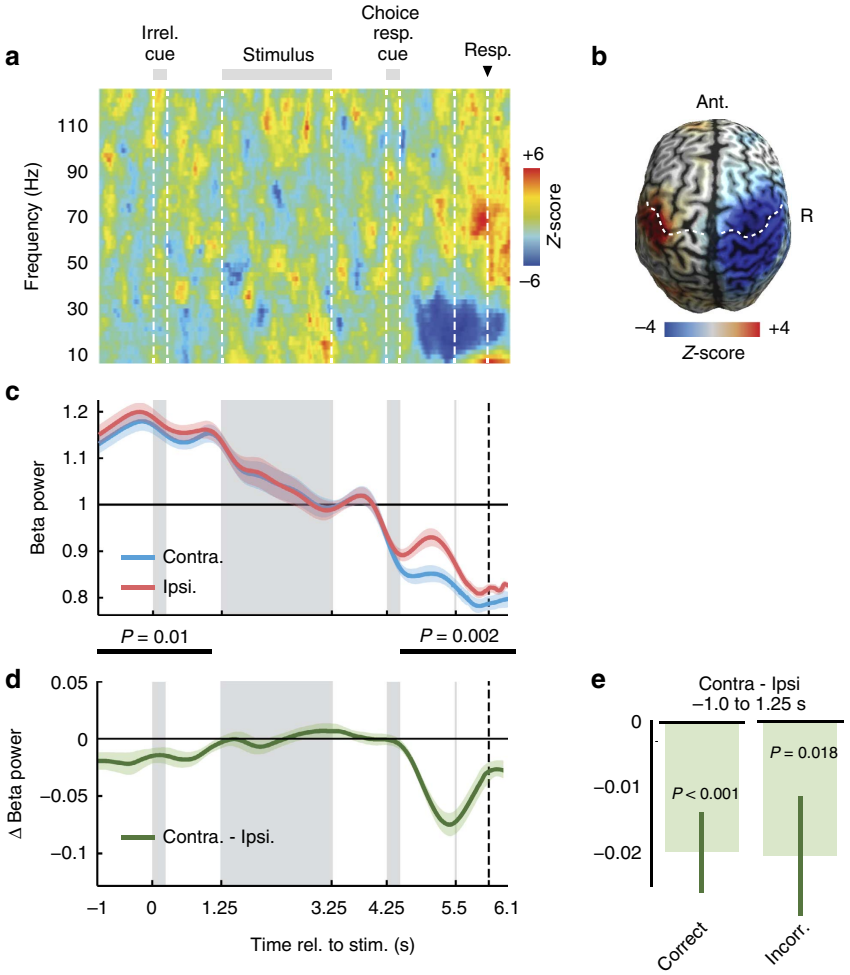

Figure 2 | Motor cortex activity predicts upcoming responses. (**a**) Time-frequency analysis of the difference of source-reconstructed motor cortex activity contralateral minus ipsilateral to each button-press. Beta power (12–30 Hz) shows a characteristic contralateral suppression before the button-press. *Z*-scores across subjects ($n=20$ subjects). (**b**) Beta power (12–30 Hz) immediately before left minus right button-presses (4.5–5.5 s). Beta power suppression is focused on motor cortex. White dashed lines mark the central sulcus. (**c**) Time-course of beta power (12–30 Hz) in motor cortex contra- and ipsilateral to the button-press. Activity is normalized by the mean across trials. Shaded areas indicate SEM across participants. Black bars mark significant differences, that is, response-predictive activity (-1.0 to 1.1 s, $P=0.01$; 4.5–6.6 s, $P=0.002$; two-tailed one-sample cluster permutation tests, $n=20$). (**d**) Time-course of response-predictive beta activity, that is, of the difference in beta power between hemispheres contra- and ipsilateral to the button-press. (**e**) Difference between contra- and ipsilateral beta power averaged across the prestimulus period (-1 to 1.25 s) is significantly different from 0 in both correct ($P<0.001$) and incorrect trials ($P=0.018$, two-tailed one-sample permutation tests, $n=20$).

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 tendency 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 $r=0.13$, $P=5.366\times10^{-4}$; 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

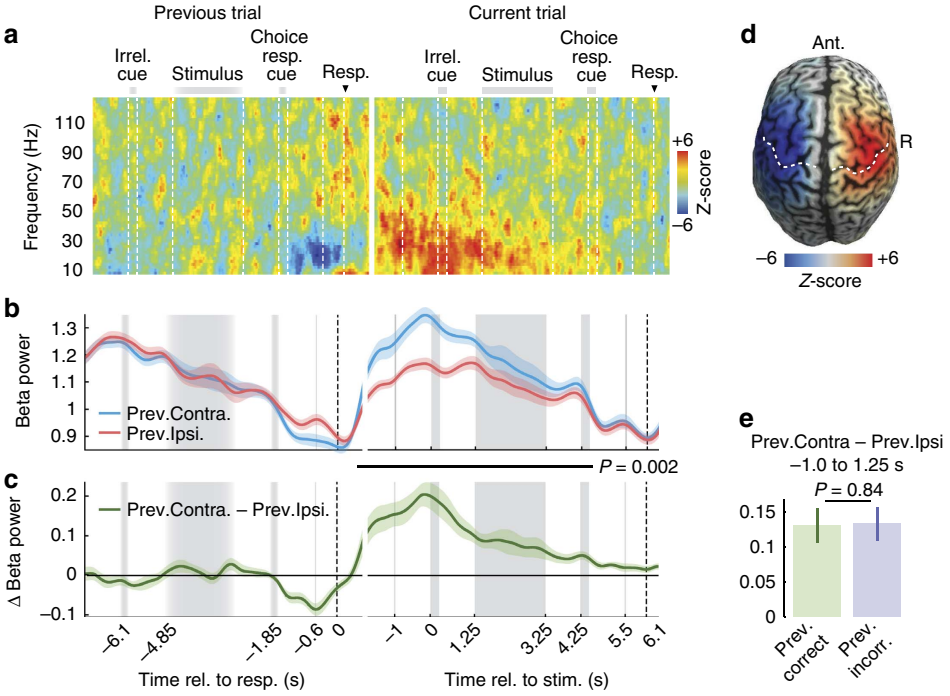

Figure 3 | Motor signals from previous trial affect current trial. (**a**) Time-frequency analysis of motor cortex activity contralateral minus ipsilateral to previous trial's button-press in pairs of consecutive trials. Data from previous trial is aligned to button-press. Data from the current trial are aligned to stimulus onset. Data from consecutive trials are concatenated according to median ITI. *Z*-scores across subjects ($n=20$). (**b**) Time-course of beta power (12–30 Hz) in motor cortex contra- and ipsilateral to previous trial's button-press. Activity is normalized by the mean across trials. Shaded areas indicate SEM across participants. Black bar marks a significant difference from 0.7 s after the previous button-press to 4.6 s in the current trial ($P=0.002$, two-tailed one-sample cluster permutation tests, $n=20$). (**c**) Time-course of the difference in beta power contra- and ipsilateral to the previous trial's button-press. (**d**) Beta power after left minus right button-presses (−1 to 1.25 s). Dashed lines indicate the hand representation of primary motor cortex and the central sulcus, respectively. (**e**) Beta rebound averaged across the prestimulus period is not significantly different after correct and incorrect choices ($P=0.84$, two-tailed paired permutation test, $n=20$).

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 were participants 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-

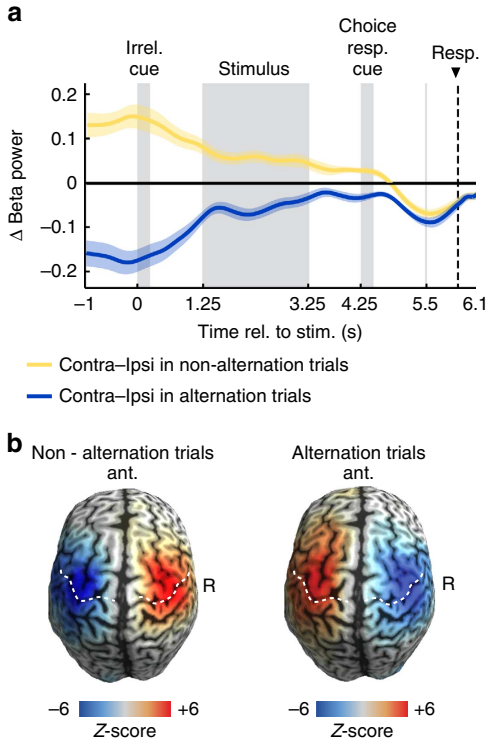

Figure 4 | Response-predictive activity in trials with and without alternation. (**a**) Beta lateralization (12–30 Hz; contralateral-ipsilateral to the response) for non-alternation trials (that is, trials where the same button is pressed in the current trial as in the previous trial) is lateralized with a positive sign throughout most of the trial, that is, opposite to the lateralization immediately preceding the button-press at 6.1 s. For alternation trials, the lateralization is negative throughout the entire trial. (**b**) Beta power (12–30 Hz) across the whole cortex during the prestimulus interval (−1 to 1.25 s) for left minus right button-presses (in the current trial) plotted separately for whether the button-press in the current trial is a non-alternation (that is, repetition of the previous button-press) or an alternation with respect to the previous button-press.

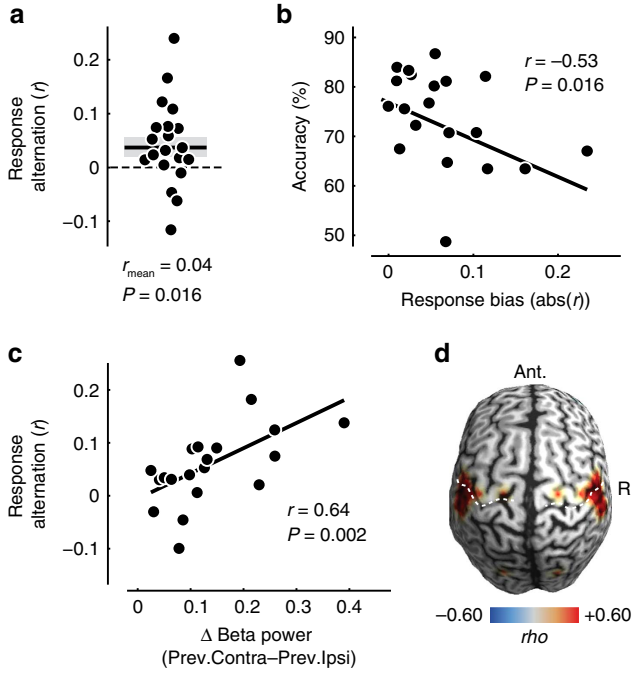

Figure 5 | Response alternation and its relation to the beta rebound. (**a**) Each participant's tendency to alternate response hands expressed as Pearson's *r* between opposite hands for consecutive trials. Note that dots are scattered horizontally to avoid overlap. Shaded area denotes SEM. (**b**) Relationship between response bias and choice accuracy across participants. (**c**) Relationship between beta rebound (lateralization with respect to previous button-press from −1 to 1.25 s of the current trial) and response alternation across participants. (**d**) Cortical distribution of the relationship between beta rebound and response alternation across participants. Correlations are masked at $P<0.05$. Note that the image is symmetric across the midline, because the beta rebound was calculated as the lateralization between corresponding points in both hemispheres.

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 held 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

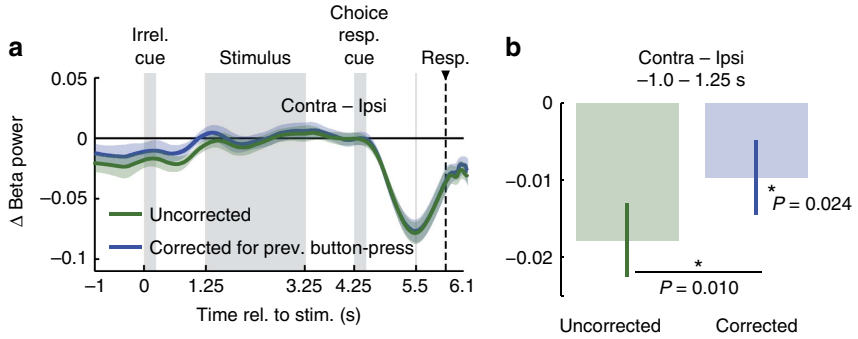

Figure 6 | Correcting response-predictive activity for previous responses. (**a**) Time-course of response-predictive beta-lateralization, corrected and un-corrected for previous responses. (**b**) Response-predictive beta-lateralization, corrected and un-corrected for previous responses averaged across the prestimulus window (−1 s to 1.25 s). Error bars show SEM. Correcting for the previous response reduces prestimulus lateralization significantly ($P = 0.010$, one-tailed paired permutation test, $n = 20$). After correcting for the previous button-press, prestimulus lateralization still predicts the upcoming button-press ($P = 0.024$, one-tailed one-sample permutation test, $n = 20$).

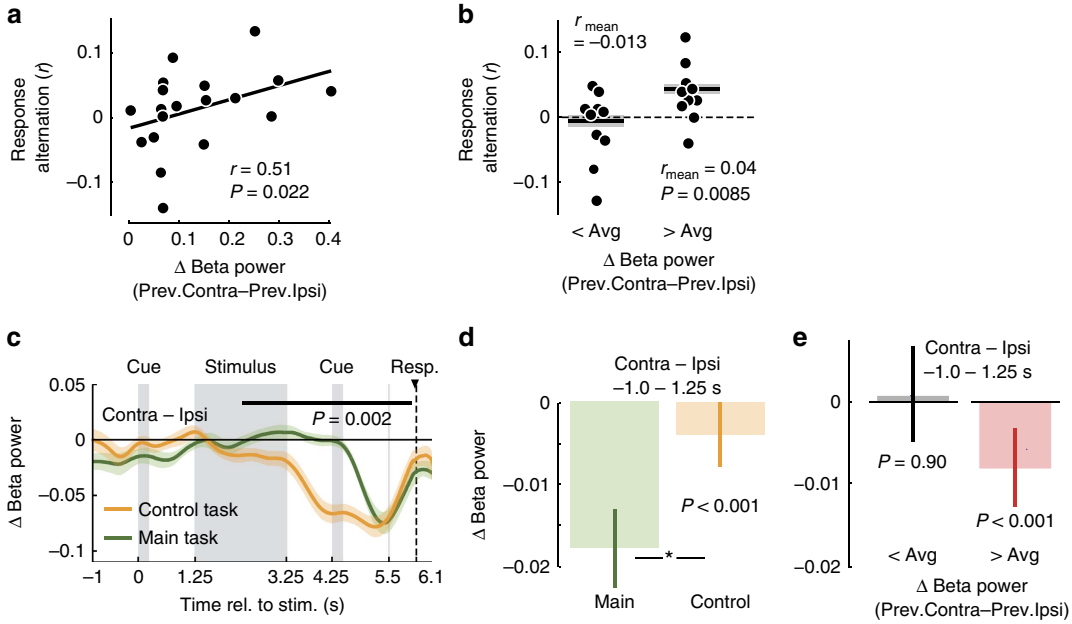

Figure 7 | Decision making with known choice-response mapping. (**a**) Relationship between beta rebound (lateralization with respect to previous button-press from −1 to 1.25 s of the current trial) and response alternation across participants. (**b**) Participants' response alternation. Participants are grouped according to above or below median beta rebound. (**c**) Time-course of response-predictive beta activity, that is, of the difference in beta power between hemispheres contra- and ipsilateral to the button-press. The black bar marks significant response-predictive activity in control trials (2.3–6.1 s, $P = 0.002$, two-tailed one-sample cluster permutation test, $n = 20$). Data from main task re-plotted from Fig. 2c for comparison. (**d**) Lateralization with respect to the upcoming button-press in the prestimulus window of main and control trials. (**e**) For subjects with above median beta rebound, prestimulus lateralization predicts the upcoming response in the control task ($P < 0.001$, time window from −1 to 1.25 s, one-tailed one-sample permutation test, $n = 10$), whereas this is not possible in subjects with below median beta rebound ($P = 0.90$, one-tailed one-sample permutation test, $n = 10$).

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[28,29], top-down factors such as stimulus[10] and reward[30] expectations, motor costs associated with response options[31–33] or sequence effects such as the 'Gambler's fallacy',

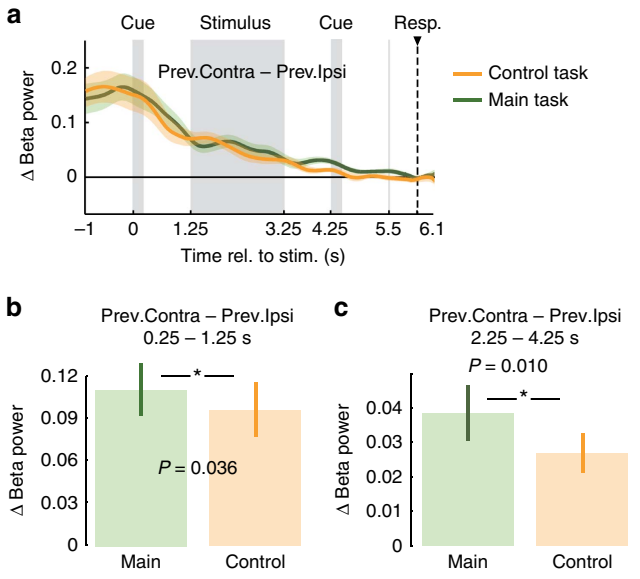

Figure 8 | Differences in beta rebound between main and control task. (**a**) Lateralization of beta power with respect to the previous button-press (beta rebound) for main and control tasks across the trial. (**b**) Beta rebound for the main and control task averaged between the end of the first cue (0.25 s) and stimulus onset (1.25 s) are significantly different ($P = 0.036$, one-tailed paired permutation test, $n = 20$). (**c**) Beta rebound for the main and control task averaged across the second half of the stimulus and the delay period before the second cue (2.25 s–4.25 s) are significantly different ($P = 0.010$, one-tailed paired permutation test, $n = 20$).

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 correlative[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[9,10,16,45]. 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[18,46], but that motor cortex activity itself can act on the resolution of response competition in a distributed network of decision making[12]. 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[4–6,8,11,15,47]. 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-press 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).

For the main and control task, this choic–response cue was presented after or before the stimulus, respectively. For temporal symmetry, an irrelevant cue (blue) was presented before or after the stimulus for the main and control task, respectively. Each trial started with a 1.5 s fixation period, followed by the first 0.25 s cue period, a blank 1 s delay, 2 s of stimulus presentation, another 1 s delay, the second 0.25 s cue period, another 1s delay and a brief (33 ms) dimming of the fixation spot, which served as the go-cue to respond. The mean (across subjects) median +/− 5/95 percentile (within subject) response times were 0.63 +/− 0.37/1.42 s and 0.62 +/− 0.35/1.38 s for the main and control experiments, respectively. There was no significant difference of response times between right- and left-hand responses for the main or control experiment (both $P>0.05$; permutation test, $n=20$). 250 ms after the response, a brief (100 ms) visual feedback was presented centrally (red or green circle; 2.1 degree diameter; green: correct, red: incorrect). The following ITI was controlled by the participants through their fixation behaviour. The experiment was paused as long as participants did not fixate the central fixation spot or closed their eyes. The pause was indicated by presentation of thin red lines at the edges of the screen. This resulted in variable inter trial intervals with a median duration of 1,290 ms. Participants were instructed to blink only during the ITI. Subjects completed 240 trials of the main task and 240 trials of the control task in two consecutive recording sessions. In addition, participants performed 240 trials (cued task), for which participants did not have to make a decision about the stimulus but received explicit instructions which button to press on each trial. Furthermore, for another 80 trials (passive task) participants had to press no button at all, but were instructed to passively view the stimulus. Cued and passive task trials were not analysed for the present study. All tasks were randomly interleaved. Before the recording, participants practiced the task for at least 45 min.

Stimuli. Dynamic random dot patterns were presented for 2 s and consisted of 1,500 white dots (dot diameter: 0.12 deg) on a black background, moving at 10 deg s^{-1} according to the 'random direction, different rule'[51] in a circular aperture of 8.5 deg diameter. For each participant, there were exactly two stimuli, both presented half of the trials: in the noise-only stimulus, there was no coherent motion, whereas in the target stimulus, a fraction of dots moved coherently downwards. Motion coherence of target stimuli was titrated to each participant's perceptual threshold employing a staircase procedure with 280 trials and a Weibull function fit (average target motion coherence: 9%). All colour cues had the same luminance (14 cd m^{-2}) and size (0.85 deg diameter). Choice-response mapping was assigned as follows: red: Yes → right, No → left, green: Yes → left, No → right, blue: uninformative.

Setup and neurophysiological recordings. We recorded the MEG (Omega 2000, CTF Systems, Inc., Port Coquitlam, Canada) with 275 channels at a sampling rate of 2,343.75 Hz in a magnetically shielded chamber. Participants were comfortably seated upright in a dark room. Stimuli were projected onto a screen at a viewing distance of 55 cm using a hue and luminance calibrated liquid crystal display projector (Sanyo PLC-XP41, Moriguchi, Japan) at 60 Hz refresh rate. Stimuli were constructed offline and presented using the Presentation software (NeuroBehavioral Systems, Albany, CA, USA). In addition to the MEG, we recorded the electrooculogram and electrocardigram for offline artefact rejection.

Eye movement recordings. Throughout the experiment, we recorded the participants' eye movements with a video-based eye-tracker (EyeLink 1000, SR Research, Ottawa, Canada). This ensured continuous fixation and allowed participants to conveniently control the length of the ITI.

Structural MRI. For source reconstruction based on each participant's individual anatomy, we recorded structural T1-weighted MRIs of each participant (echo time (TE) = 2.18 ms, repetition time (TR) = 2.3 ms, longitudinal relaxation time (T1) = 1.1 ms, flip angle = 9°, 192 slices, voxel size $1\times1\times1$ mm^3) with a Siemens 3T Tim Trio scanner and a 32 channel Head Coil.

MEG preprocessing. MEG data were downsampled to 1,000 Hz and high-pass filtered at 4 Hz (two-pass Butterworth filter, filter order 6). Line noise and its harmonics were notched out (49.5–50.5 Hz, 99.5–100.5 Hz, 149.5–150.5 Hz ; 199.5–200.5 Hz, 249.5–250.5 Hz, 299.5–300.5 Hz, 349.5–350.5 Hz two-pass Butterworth filter, filter order 4), and after careful visual inspection of the respective signals, trials with eye blinks, saccades, strong muscle artifacts, or signal jumps were excluded from further analyses (on average 20% and 18% of all trials for the main and control task, respectively).

Source analysis. We used adaptive linear spatial filtering (beamforming)[52,53] to estimate neural population signals at the source level. We used frequency-domain beamforming dynamical imaging of coherent sources (DICS)[52] to investigate the cortex-wide distribution of response-predictive beta-band activity before the button-press. We used time-domain beamforming linearly constrained minimum variance (LCMV)[53] to analyse the dynamics of frequency-specific neural activity in motor cortex.

The implementation details of the beamformer were as follows: for each time t, frequency f (for frequency-domain beamforming) and source location r, three orthogonal filters ($\hat{\mathbf{A}}=[A_x, A_y, A_z]$; one for each spatial dimension) were computed that pass activity from location $\mathbf{r}$ with unit gain, while maximally suppressing activity from all other sources:

$$\hat{\mathbf{A}}(\mathbf{r},t,f)=[\mathbf{L}^{\mathrm{T}}(\mathbf{r})\mathbf{C}_{\mathrm{real}}(t,f)^{-1}\mathbf{L}(\mathbf{r})]^{-1}\mathbf{L}^{\mathrm{T}}(\mathbf{r})\mathbf{C}_{\mathrm{real}}(t,f)^{-1} \quad (1)$$

Here, $\mathbf{L}(\mathbf{r})$ is a matrix whose columns are the leadfields of three orthogonal dipoles at source location $\mathbf{r}$, and $\mathbf{C_{real}}$ denotes the real part of the complex cross-spectral-density matrix for the data at frequency f and time t, and $^{\mathrm{T}}$ indicates the matrix transpose. For time-domain beamforming, filters are not frequency dependent and $\mathbf{C}_{\mathrm{real}}$ denotes the 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 $\mathbf{r}$):

$$\mathbf{w}=[w_1,w_2,w_3]=Eig_1(\hat{\mathbf{A}}(\mathbf{r},t,f)C_{\mathrm{real}}(t,f)\hat{\mathbf{A}}(\mathbf{r},\mathrm{t},\mathrm{f})^{\mathrm{T}}) \quad (2)$$

$$\mathbf{A}(\mathbf{r},t,f)=w_1\cdot A_1(\mathbf{r},t,f)+w_2\cdot A_2(\mathbf{r},t,f)+w_3\cdot A_3(\mathbf{r},t,f) \quad (3)$$

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

$$\mathbf{X}_{\mathrm{source}}(\mathbf{r},t,f)=\mathbf{A}(\mathbf{r},t,f)\cdot\mathbf{X}_{\mathrm{sensor}}(t,f) \quad (4)$$

where $\mathbf{X}_{\mathrm{sensor}}(t,f)$ is the frequency-domain representation at time t and frequency f at the sensor level and $\mathbf{X}_{\mathrm{source}}(\mathbf{r},t,f)$ is the corresponding source signal at location $\mathbf{r}$. For time-domain beamforming, $\mathbf{X}_{\mathrm{sensor}}$ and $\mathbf{X}_{\mathrm{source}}$ denote the sensor-level and source-level timecourses, 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 source 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 'handknob' 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 (DPSS) 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, 17 discrete prolate spheroidal sequences tapers).

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[9,21,22]. This contrast can be formalized as:

$$(\text{contra}-\text{ipsi})_{\text{current}}=\frac{(L_{xr}-R_{xr})+(R_{xl}-L_{xl})}{2} \quad (5)$$

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, l, and × denote right, left and either response hand, respectively. Thus, L_{xr} denotes the left hemispheric activity measured for trials with left- or right-hand button-press on the previous trial and right-hand button-press on the current trial. 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):

$$(\text{contra}-\text{ipsi})_{\text{previous}}=\frac{(L_{rx}-R_{rx})+(R_{lx}-L_{lx})}{2} \quad (6)$$

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 3b,c and 7c), we calculated cluster permutation statistics that account for multiple comparisons with a first-level threshold of $P=0.05$ (two-tailed) and 1,000 subject-level permutations[55,56]. For all contrasts tested on specific time windows (Figs 2e, 3e, 6b, 7d,e and 8b,c), we employed permutation statistics on un-smoothed data with 1,000 subject-level permutations. One often employed time-window was from −1 to 1.25 s (Figs 2e, 3e, 6b, 7d,e). We used this window, because this period includes the entire prestimulus interval that well matches the extent of the early response-predictive beta lateralization (Fig. 2d). All statistics were computed across subjects (random effects) with two-tailed tests unless noted otherwise.

Correction for previous responses. To investigate the beta rebound's contribution to the early response-predictive activity, we computed the lateralization relative to the current trial's response corrected for the previous response (Fig. 6):

$$(\text{contra}-\text{ipsi})_{\text{current corr.}}=\frac{(L_{lr}+L_{rr})-(R_{lr}+R_{rr})+(R_{ll}+R_{rl})-(L_{ll}+L_{rl})}{4} \quad (7)$$

The effect of previous responses is corrected for by computing the responses contralateral and ipsilateral to the current response averaged across trials with equal weighting across both possible previous responses (the four bracketed terms in equation (7)). In other words, we replace the four numerator terms in equation (5) with the same terms balanced for the previous response. By re-ordering equation (7) it becomes evident that this balancing removes the previous trial's effect:

$$(\text{contra}-\text{ipsi})_{\text{current corr.}}=\frac{(L_{lr}-L_{ll})+(L_{rr}-L_{rl})+(R_{ll}-R_{lr})+(R_{rl}-R_{rr})}{4} \quad (8)$$

Each of the four bracketed numerator terms in equation (8) isolates the effect of the current response (contralateral–ipsilateral) and subtracts out the effect of a specific previous response for a specific hemisphere. By removing the effect of previous responses, this correction removes the neuronal variability specific to the previous response, that is, the beta rebound. We employed this correction not only to test if the beta rebound contributed to the early response-predictive activity, but also to test if spontaneous, that is, beta-rebound independent, fluctuations of motor cortex lateralization predict responses.

We applied the same correction also when comparing the size of the beta rebound between main and control tasks (Fig. 8a–c). This allowed us to rule out potential confounding by different alternation behaviour across tasks (for example, less alternation trials for the control task) because correcting for the previous response is equivalent to correcting for alternation behaviour. Again, this becomes evident by re-ordering equation (7) accordingly:

$$(\text{contra}-\text{ipsi})_{\text{current corr.}}=\frac{(L_{lr}-L_{rl})+(L_{rr}-L_{ll})+(R_{rl}-R_{lr})+(R_{ll}-R_{rr})}{4} \quad (9)$$

Now, each of the four bracketed terms in equation (9) isolates the effect of the current response (contralateral–ipsilateral) and subtracts out the effect of the previous response being the same or different from the previous response.

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' *r*-values and applied 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 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[57].

Data availability. The data that support the findings of this study are available from the corresponding authors upon request.

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Author contributions

A.-A.P. and M.S. designed the research and wrote the manuscript, A.-A.P. recorded and analysed the data.

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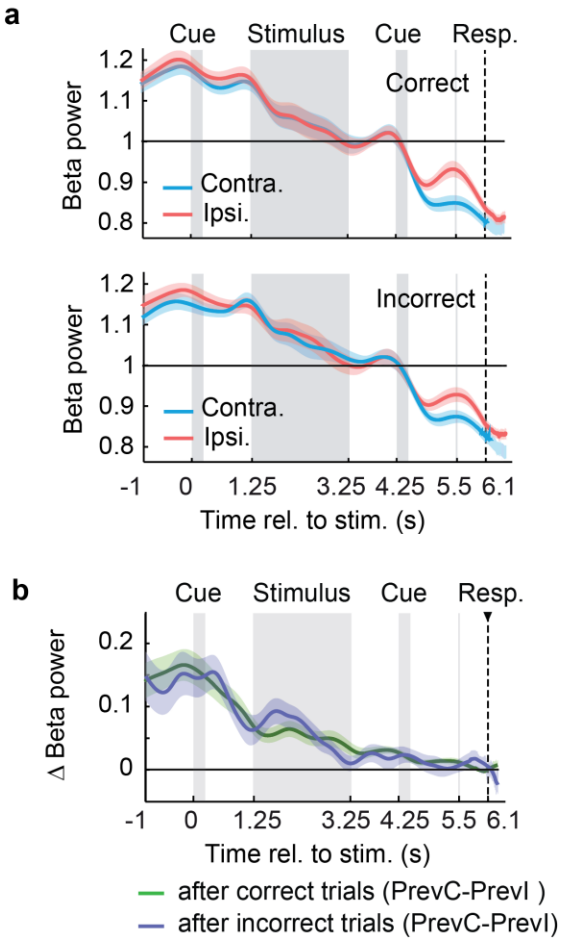
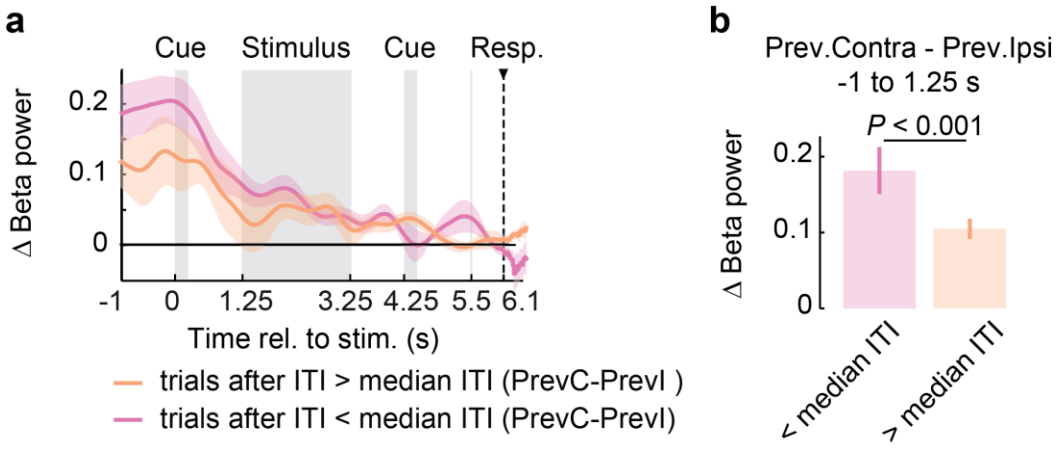
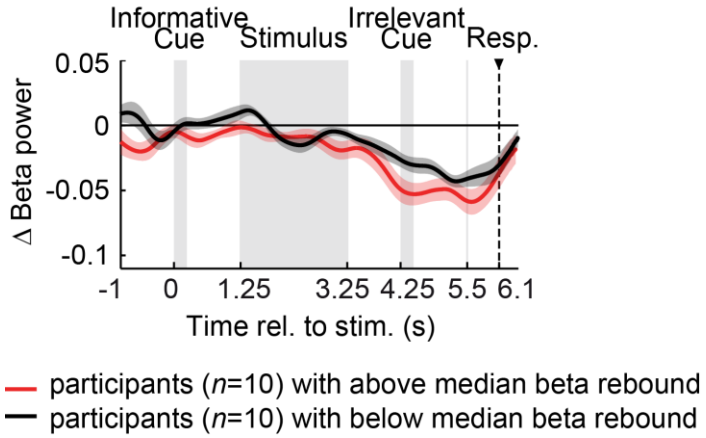


Supplementary Figure 1. Accuracy has no effect on neural variables (**a**) Beta power contra- and ipsilateral to the buttonpress plotted separately for correct (upper) and incorrect (lower) choices on the current trial. Contralateral beta power is lower than ipsilateral beta power in the prestimulus period in both trial categories, i.e. independent from accuracy. (**b**) Beta rebound (i.e. beta power lateralization calculated with respect to the previous button-press) following correct and incorrect choices.



Supplementary Figure 2. Size of beta rebound after long and short intertrial intervals

Participants controlled the length of intertrial intervals (ITI) with their fixation behavior. As the beta rebound occurs locked to the previous button-press, it should decay over time, i.e. it should be smaller for longer ITIs. (**a**) Time-course of the beta rebound for trials following short (< median) and long (> median) ITIs (median ITI = 1290ms). (**b**) As hypothesized, the beta-rebound in the prestimulus interval was significantly smaller following long as compared to short ITIs ($P < 0.001$, two-tailed paired permutation test, $n = 20$). This may lead to weaker alternation behavior for long ITIs. Indeed, while for short ITIs participants showed significant response alternation (mean $r = 0.052$, $P = 0.046$, one-tailed one-sample T-test, $n = 20$), for long ITIs, response alternation was weaker and not significant (mean $r = 0.019$, $P = 0.45$, one-tailed one-sample T-test, $n = 20$). However, a direct comparison between short and long ITIs did not reach statistical significance ($P = 0.21$, one-tailed paired T-test, $n = 20$).



Supplementary Figure 3. Response-predictive activity in control trials

To investigate if the same trend of response-predictive prestimulus lateralization holds for the control task, we split the participants according to the size of their beta rebound. Here, we show the time-course of lateralization for subjects with above and below median beta-rebound separately.

Factor	Mean*(r)*-value	*P*-value	*n*
Prev. response hand (left or right hand)	0.067	0.24	20
Prev. choice (yes or no)	0.012	0.65	20
Prev. stimulus (coherent motion present or absent)	0.006	0.78	20
Prev. accuracy (correct or incorrect choice)	0.001	0.97	20
Prev. RT (time from Go cue to button-press)	-0.010	0.66	20
ITI duration	-0.162	< 0.0001	20

Supplementary Table 1. Impact of prev trial parameters on size of the beta rebound

Results of a partial multivariate correlation between the size of the beta rebound (beta-power contralateral minus ipsilateral to the previous button-press in the time window -1 to 1.25s of the current trial) and 5 parameters of the previous trial, calculated in each participant and tested for significance across participants. Only the ITI duration was significantly correlated with the strength of the beta rebound. This effect is further quantified in Supplementary Figure 2.

Motor actions influence subsequent sensorimotor decisions

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Sensorimotor decisions are influenced by factors beyond the current sensory input, but little is known about the effect of preceding motor actions. Here, we show that choice-unrelated motor actions influence subsequent sensorimotor decisions. By instructing participants to perform choice-unrelated motor responses before visuomotor decisions, we were able to manipulate upcoming choices in a directed fashion. Our results suggest that the neural aftermath of simple movements can influence sensorimotor decision making.

Choice formation in sensorimotor decision making is influenced by many factors beyond the current stimulus such as reward expectations[1], neural noise at sensory stages[2,3], or previous decisions[4–6]. Recent evidence suggests that also previous choice-related motor responses[7] can influence sensorimotor decision making. However, it is unclear if this influence is restricted to previous motor responses related to decision making, or if it reflects a general influence of motor actions on decision making[8]. To address this, we dissociated choices and responses in a sensorimotor decision-making task, and tested if decision making can be manipulated by simple choice-unrelated motor actions before decisions.

Participants made decisions about the global motion direction (up/down) in dynamic random dot patterns and reported their decision with a left or right hand button-press (Fig. 1a). The motion strength was adjusted for each subject to perform near the individual discrimination

threshold (mean correct performance: 68.2 % +/- 0.72 % sem). The mapping from choice (motion: up/down) to response (button: left/right) was random on each trial and indicated by a choice-response cue before the stimulus (Fig. 1c). This allowed us to dissociate choices and responses[7,9]. To test if the decision process could be manipulated by choice-unrelated motor actions, before each perceptual visual-motion decision, a response cue instructed participants to press either the left, right or no button (Fig. 1b).

First, we determined the dependency of successive choice-related responses when participants did not press a button in between two successive perceptual decisions (Baseline). In accordance with previous results[7], subjects showed a significant tendency to alternate between left and right button-presses for consecutive visual-motion decisions (Fig. 2a, P = 0.029, mean $r_{Baseline}$ = - 0.038 +/- 0.06, $t(17)$ = -2.38, n = 18, two-tailed one-sample t-test on Fisher-z-transformed correlation coefficients describing the relationship between choice-related responses in each participant). This result confirms that motor responses made to report preceding choices influence consecutive choices on the same perceptual choice task[7].

If this response alternation is driven by any previous motor act independent of a preceding choice, introducing instructed button-presses between perceptual decisions should interfere with the response alternation observed in the Baseline condition. Specifically, we hypothesized that instructing a button-press different from the previous choice-related button-press (Different-condition) should counteract, i.e. reduce, the response alternation between successive perceptual choices. Indeed, we found that an intermittent “different” button-press strongly reduced response alternation ($r_{Baseline}$-$r_{Different}$ = -0.050 +/- 0.10, P = 0.024, $t(17)$ = -2.12, left-tailed paired t-test; $r_{Different}$ = 0.012 +/- 0.01, P = 0.47, $t(17)$ = 0.74, two-tailed one-sample t-test, Fig. 2a).

This pattern of results supports the hypothesis that even choice-independent previous motor actions push responses on subsequent decision making towards response alternations. However, alternatively, the intermittent instructed button-press may simply have disturbed the response alternation between choice-related button-presses. Introducing an instructed button-

press repeating the previous choice-related button-press (Same-condition) can dissociate these two alternatives. If response alternation is motor-driven, the “same” button-press should leave response alternation untouched, or even strengthen it because the neural bias in favor of an alternation[7] may build up over two button-presses. Otherwise, if response alternation is merely disturbed by introducing an intermittent button-press, a “same” intermittent button-press should induce the same reduction of response alternation as a “different” intermittent button-press.

As predicted by the motor-driven hypothesis, response alternation between choice-related responses with an intermittent instructed “same” button-press was significantly stronger than without an intermittent button-press ($r_{Baseline}$-r_{Same} = 0.038 +/- 0.08, P = 0.039, t(17)=1.87, right-tailed paired t-test; r_{same} = -0.076 +/- 0.07 , P = 0.0002, t(17)=-4.6, two-tailed one-sample t-test, Fig. 2a).

In summary, introducing a simple instructed button-press alters response alternation between choice-contingent button-presses in a directed fashion. This shows that response alternation is not limited to choice-related motor responses, but that previous motor acts with the same effector can generally impact sensorimotor decisions.

To further test this effect, we next directly analyzed the relationship between the cued intermittent response and the following choice-related response. Indeed, we found that participants also alternated between the cued button-press and the following choice-related button-press (Fig. 2b, r = -0.044 +/- 0.011, P = 0.0009, t(17) = -3.99, two-tailed one-sample t-test). Furthermore, we found an interaction between the effect of the previous choice-related response and the intermittent cued response. There were more response alternations from the cued response to the next choice-response if the cued response was a repetition of the previous choice-related button-press than when it was an alternation itself ($r_{Different}$-r_{Same} = -0.064 +/- 0.023, P = 0.012, t(17) =2.79, two-tailed paired t-test). This suggests a common neuronal substrate underlying the effect of the cued response and of the choice-related response, e.g. the post-movement rebound of neural activity in sensorimotor areas[7,10].

The fact that also choice-unrelated responses influence subsequent choices suggests that this response sequence effect acts on the response-selection rather than on the choice-selection or perceptual stage. This is in contrast to the known choice-repetition effect[5], which is thought to act on the perceptual stage[4,6] and may reflect a constancy bias of the visual system. Thus, we hypothesized that a choice-repetition effect is not affected by intermittent cued responses. Indeed, we found that subjects showed a significant tendency to repeat choices across subsequent visual-motion decisions (Fig 2c, all conditions: $r > 0.1$, $P < 0.001$, $t(17)>4.0$, two-tailed, one-sample t-tests, n=18), but, as expected, this choice-repetition effect did not differ between Baseline, Same or Different conditions ($r_{Baseline}$-r_{Same}: 0.0069 +/- 0.09, $P = 0.739$, $t(17)=0.338$, two-tailed paired t-test; $r_{Baseline}$-$r_{Different}$ = 0.0014, +/- 0.06, $P = 0.918$, $t(17) = 0.10$, two-tailed paired t-test).

In summary, we show a response-alternation bias that is independent of the preceding percept or choice, because simple, instructed motor actions can manipulate subsequent sensorimotor decisions in the same effector system in a directed fashion. Together with previous findings[7], the pattern of results observed here suggests that the impact of previous motor acts on subsequent choices is driven by a rebound of motor-cortical activity following motor actions[10].

Figures

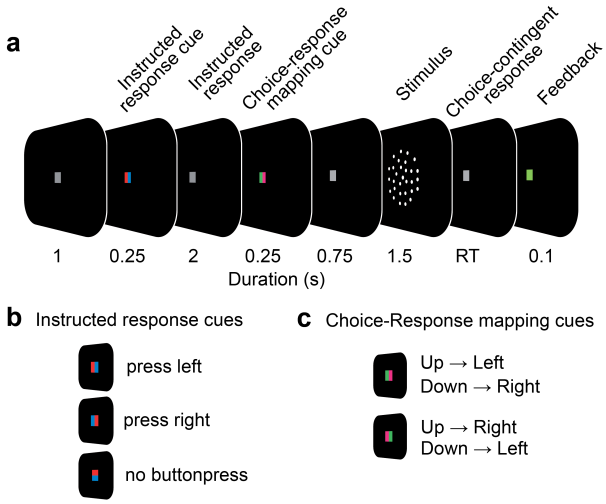

Figure 1 Visual-motion decision task and experimental manipulation (**a**) Participants reported the direction (up/down) of coherent motion in a display of randomly moving dots with a left- or right-hand button-press. For each trial, the mapping from choice to response was newly assigned with a cue before the stimulus (choice-response mapping cue). Furthermore, at the beginning of each trial, an instructed-response cue indicated subject to either press the left button, the right button or no button. (**b**) Instructed-response cues. (**c**) Choice-response mapping cues.

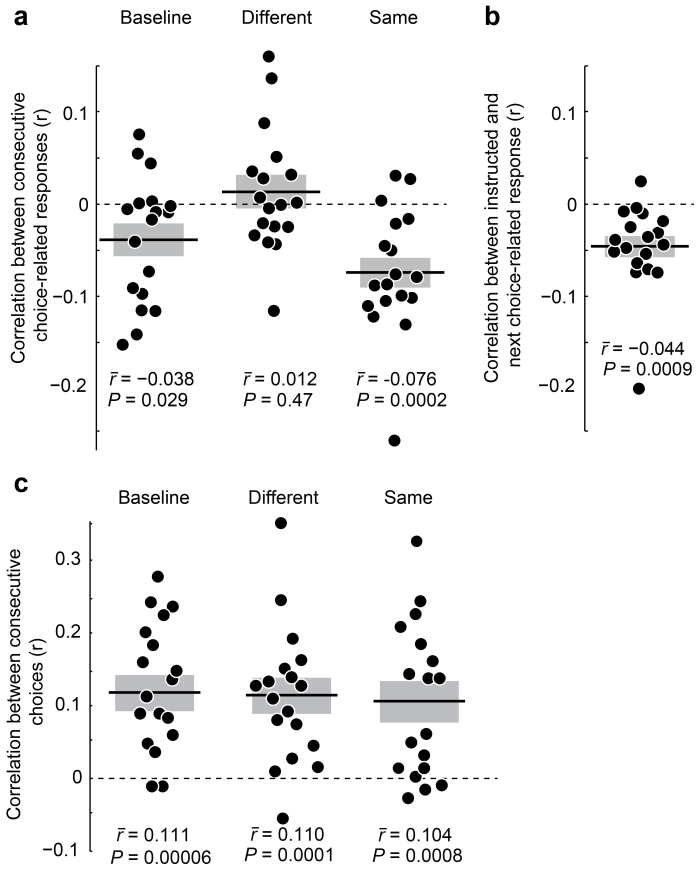

Figure 2 Effect of intermittent instructed button presses. Each dot denotes one participant's correlation-coefficient. Black lines and gray bars denote the mean and SEM of correlation coefficients across participants, respectively. For each panel, the group mean of correlation coefficients and the P-value of a t-test of Fisher-Z-transformed correlation coefficients against 0 are stated. (**a**) Correlation coefficients between consecutive choice-contingent button-presses. 'Baseline' condition: no intermittent button-press; 'Different' condition: instructed button-press different from preceding choice-response; 'same' condition: instructed button-press the same as preceding choice-response. (**b**) Correlation coefficients between instructed responses and the following choice-contingent response. (**c**) Correlation coefficients between consecutive visual-motion decisions.

Data availability

The data that support the findings of this study are available from the corresponding authors upon request.

Acknowledgements

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Author Contributions

A-AP and MS designed the research and wrote the manuscript, A-AP and NN set up the experiment, A-AP recorded and analyzed the data.

Competing Financial Interests

The authors have no competing financial interests.

Methods

Participants

18 healthy volunteers (11 female, mean age 28 years) participated in this study. All participants 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 Tübingen. All participants gave written informed consent before participating.

Behavioral Task

On each trial, participants decided if they saw coherent motion going upward or downward in a centrally presented dynamic random dot pattern and reported their percept (up/down) by button-press 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 color cue (Fig. 1c) whose orientation was informative of how to map the choice (up/down) to a response (left/right). In addition, and before decision making, participants were randomly instructed by an instructed-response cue to press either the left, the right or no button (Fig. 1b). Each instructed button-press was later considered as “same” or “different” according to the last choice related button-press.

Each trial started with the instructed-response cue presented centrally for 0.25 s, followed by a fixation period of 2 s, during which participants had to respond to the instructed-response cue. Next, the choice-response mapping cue was shown centrally for 0.25 s, followed by a fixation period of 0.75 s. Then, the motion stimulus was presented for 1.5 s. The offset of the stimulus served as the go-cue to report the choice (upward/downward motion) with a button-press (left/right). The mean (across subjects) median +/- 5/95 percentile (within subject) response time was 0.63 +/- 0.37/1.42 s. Visual feedback was provided 0.1 s after the button-press by turning the fixation spot green (correct) or red (incorrect choice) for 0.1 s. During the inter-trial interval (1 s) the fixation spot was shown.

Stimuli

Dynamic random dot patterns were presented for 1.5 s and consisted of 1500 white dots (dot diameter: 0.11 deg) on a black background, moving at 10 deg/s according to the "random direction, different rule"[11] in a circular aperture of 7.70 deg diameter. In the upward-moving stimulus, a fraction of dots moved coherently upwards, whereas in the downward-moving stimulus, a fraction of dots moved coherently downward. In both stimuli, noise dots had a life-time of exactly 1 frame. All instructed-response cues and stimulus-response mapping cues had the same luminance (38 cd/m²) and size (0.31 deg diameter) as the fixation spot.

Setup

Participants were seated in a dimly lit room in front of a screen at a distance of 58 cm. They were observing stimuli presented on a CRT monitor (Eizo Flexscan F931). The screen was controlled by a NVIDIA GeForceGTX 460 graphics controller with a refresh rate of 60 Hz at a spatial resolution of 1024x768 pixels. The experiment was controlled using PsychToolbox[12] for MATLAB.

Procedure

Before the recording, participants practiced the task for at least 10 minutes. Then, they completed a staircase procedure with 250 trials to determine a level of coherence near the individual perceptual threshold (staircase target: 66 % correct choices; average motion coherence: 3.8 %). The staircase was followed by two experimental sessions with 456 trials each. During the experimental sessions, the coherence level of the stimuli was further adjusted every 50 trials, to target 66 % correct performance.

Eye movement recordings

Throughout the experiment, we recorded the participants' eye movements with an infrared-video based eye-tracker (Arrington Research Inc., USB-220). This ensured continuous fixation.

Correlation analyses and statistics

To quantify relations between nominal behavioral variables (responses "left" or "right" and choices "up" or "down") we used Pearson's correlation coefficient for binary variables (Phi coefficient). To assess statistical significance of correlation coefficients at the group level, we

Fisher-z-transformed subjects' correlation coefficients and applied two-tailed t-statistics across subjects unless noted otherwise.

All analyses were performed in MATLAB (MathWorks Inc., Natick, USA) using custom software.

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Measuring the cortical correlation structure of spontaneous oscillatory activity with EEG and MEG

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ABSTRACT

Power correlations of orthogonalized signals have recently been introduced for MEG as a powerful tool to non-invasively investigate functional connectivity in the human brain. Little is known about the applicability of this approach to EEG, and how compatible the results are between EEG and MEG. To address this, we systematically compared power correlations of simultaneously recorded and source co-registered 64-channel EEG and 275-channel MEG in resting human subjects. For both modalities, connectivity peaked at around 16 Hz. For this frequency range, seed-based correlation maps showed comparable patterns across modalities, with generally more distinct patterns for MEG. A brain-wide pattern correlation analysis also revealed maximum similarity around 16 Hz. Correcting for different signal-to-noise ratio (SNR) across frequencies and modalities revealed pattern correlation between modalities close to one across a broad frequency range from 1 to 32 Hz and only slightly smaller for higher frequencies. The decrease above 32 Hz likely reflected higher susceptibility to muscle artifacts for EEG than for MEG. Our results show that power correlation of orthogonalized signals is feasible for studying functional connectivity with 64-channel EEG. Furthermore, besides differences in SNR, for frequencies from about 8 to 32 Hz, EEG and MEG measure the same correlation patterns across the entire brain.

Introduction

Electroencephalography (EEG) and magnetoencephalography (MEG) measure neuronal population activity in the human brain with millisecond temporal resolution. This temporal resolution allows for assessing the spectral features of neural activity and connectivity. More specifically, spectrally resolved analyses allow investigating the covariance of narrow band oscillations between brain regions as a window into large-scale neuronal interactions in the healthy and diseased human brain (Brookes et al., 2012; Hipp et al., 2012, 2011; Nolte et al., 2004; Siegel et al., 2012; Stam, 2014). Recently, this approach has been fostered by methodological advances that overcome key limitations for studying co-fluctuations of neuronal oscillations with M/EEG (Brookes et al., 2012; Hipp et al., 2012). Nearby sensors or source estimates share artifactual variance due to electromagnetic field spread and the limited spatial resolution of source-projection methods (Palva and Palva, 2012; Schoffelen and Gross, 2009). By removing signals with zero phase lag through orthogonalization, this artifactual correlation can be discounted (Brookes et al., 2012; Hipp et al., 2012). Recent studies have established this approach as a promising tool for investigating large-scale neuronal interactions with MEG: The correlation structure of spontaneous cortical oscillations is well structured, stable over subjects, and frequency dependent (Brookes et al., 2012; Hipp et al., 2012; Wens et al., 2014). It reflects known functional relationships between brain regions (Hipp et al., 2012; O'Neill et al., 2015), predicts BOLD fMRI correlations (Cabral et al., 2014; Hipp and Siegel, 2015), and is altered in brain pathologies (Hawellek et al., 2013; Kitzbichler et al., 2015).

So far, no study has systematically investigated the feasibility of power correlations from orthogonalized signals with EEG. Thus, although EEG is more readily available than MEG, in particular for clinical contexts, it remains unclear how these results transfer to EEG. Inhomogeneous tissue distribution, the high resistance of the skull and the anisotropy of current flow have stronger effects on EEG than on MEG. As a result, EEG forward models are typically more complex and MEG yields better source estimation accuracy (Babiloni et al., 2004; Brookes et al., 2011b; Fuchs et al., 1998; Klamer et al., 2015; Malmivuo, 2012; Molins et al., 2008; Perdue and Diamond, 2013). However, dipole sensitivity decreases faster with depth in MEG than in EEG (Malmivuo, 2012; Molins et al., 2008): At around 70% of the head's radius, MEG source localization accuracy drops below EEG (Fuchs et al., 1998). Additionally, differences in sensor sensitivity to dipole orientations further imply that the information collected with both measures are at least partly

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independent (Baillet et al., 2001; Cho et al., 2015; Hari and Salmelin, 2012; Malmivuo, 2012). It is unclear how these differences between modalities influence measures of functional connectivity in the resting state. In sum, it is unknown if EEG allows for assessing large-scale neuronal interactions from power correlations of orthogonalized signals and, if so, how the correlation structure relates to that of MEG. Here we address these questions.

We simultaneously recorded standard EEG (64 channels) and MEG (275 channels) from eleven healthy subjects. We applied the same processing pipeline – except for physical forward models, which differ by construction – to both measures and co-registered both measures at the source-level. We then quantified and compared functional connectivity by the correlation of power envelopes of orthogonalized signals at the source-level.

Methods

Participants

We collected simultaneous EEG and MEG resting-state measurements from eleven healthy subjects (mean age 26.6 years ± 4.6 years std., 7 female). The subjects were instructed to let their minds wander and think about nothing in particular with their eyes closed (resting state). The study was approved by the local ethics committee and conducted in accordance to the Declaration of Helsinki. All participants gave written informed consent before participating.

Electrophysiological recordings

We collected 10 min of simultaneous MEG and EEG data per subject. The MEG was continuously recorded with a 275-channel whole-head system (Omega 2000, CTF Systems Inc., Port Coquitlam, Canada) in a magnetically shielded room. The head position relative to the MEG sensors was controlled using three head localization coils (nasion, left/right preauricular points). MEG signals were recorded at a sampling rate of 2343.75 Hz (anti-aliasing filter at 1/2 Nyquist-frequency).

Simultaneously, we recorded the continuous 64-channel EEG with Ag–AgCl ring electrodes mounted on an elastic cap (antNeuro waveguard, Enschede, Netherlands), referenced against the mastoid. We kept electrode impedances below 20 kΩ. EEG signals were recorded at 2000 Hz sampling rate (anti-aliasing filter at 1/2 Nyquist-frequency). Additionally, a 2-channel (horizontal and vertical) bipolar electro-oculogram (EOG) and a 1-channel bipolar electro-cardiogram (ECG) were measured to control for cardiovascular and eye-movement artifacts. Offline, synchronization between EEG and MEG was achieved in two steps: First, we estimated and corrected for differences in system clocks and temporal offsets between the MEG and EEG systems based on triggers that were simultaneously sent to both systems every second during the entire recording. Second, we low-pass filtered and interpolated the data of both modalities to a common sampling rate of 1000 Hz (300 Hz anti-aliasing low-pass filter, 4th order zero-phase Butterworth).

Artifact rejection

Artifact rejection was carried out in a two-step procedure: First, we filtered line noise with a notch filter at 50 Hz and at the first six harmonics (stop-band width: 1 Hz). We visually inspected the data for muscle-, eyeblink-, and technical artifacts (i.e. SQUID-jumps) and rejected corresponding time intervals and malfunctioning or noisy channels (mean: 1 MEG-channel; 1.6 EEG-channel; range: 0 to 3 channels). Second, we high-pass filtered the data at 0.5 Hz with a 4th order Butterworth filter and split the data into two frequency bands: A low frequency band from 0.5–20 Hz (EEG) and 0.5–30 Hz (MEG) and a high frequency band with frequencies above 20 or 30 Hz, respectively. Independent component analysis (ICA) (Hyvärinen and Oja, 2000) was performed for each frequency range and modality. This approach takes advantage of the different nature of physiological artifacts: eyeblink, eye-movement and cardiovascular artifacts show prominent low frequency features whereas muscle activity comprises higher frequencies. We chose different cutoff frequencies between modalities to optimally separate between low and high frequency artifacts in each modality. In principle, this may have introduced differences between modalities that affected our results in this frequency range. Repeating our analysis on uncut, non ICA-cleaned data provided a control that this was not the case. We did not find differences in the 16–32 Hz frequency range between the ICA-cleaned data and the uncut, non ICA-cleaned data, suggesting that the different cutoff frequencies between EEG and MEG had little effects on our main analyses and results (see Fig. 6a).

Independent components were visually inspected and artifactual components rejected according to their topology, time-courses and spectra (Chaumon et al., 2015; Hipp and Siegel, 2013). Subsequently, both frequency bands were recombined.

MRI data acquisition

The subjects were scanned in a Siemens MAGNETOM Trio 3T scanner (Erlangen, Germany) with a 32-channel head coil. A T1-weighted sagittal MP-RAGE (TE = 2.18 ms, TR = 2300 ms, TI = 1100 ms, flip angle = 9°, 192 slices, voxel size = $1 \times 1 \times 1$ mm^3) and a sagittal T2-weighted fat suppressed scan (TE = 185 ms, TR = 3200 ms, 192 slices, voxel size = $1 \times 1 \times 1$ mm^3) were obtained from each participant to construct individual high-resolution head models.

Spectral analysis

Time-frequency representations $TFR(t,f)$ of time-domain MEG and EEG data $D(t)$ were generated using Morlet's wavelets (Goupillaud et al., 1984):

$$w(t,f) = \left(\sigma_t\sqrt{\pi}\right)^{-\frac{1}{2}} e^{-\frac{t^2}{2\sigma_t{}^2}} e^{-i2\pi ft} \quad (1)$$

$$TFR(t,f) = (D*w)(t,f) \quad (2)$$

where TFR is the number-of-sensors by samples matrix. The number of samples varies with frequency. The bandwidth of wavelets was 0.5 octaves ($f/\sigma_t = 5.83$, kernel size was 6 σ_t). We derived spectral estimates for frequencies f from 0.8 to 128 Hz ($2^{-1/2}$ to 2^7) in quarter octave steps. The temporal step-size for frequency f was $\sigma_t/2$. Time points, for which the kernel overlapped with periods marked as artifacts, were discarded.

Physical forward model

Source-reconstruction of EEG and MEG was based on individual head models generated from each subject's MRI data. MRI scans were segmented using a customized SimNIBS pipeline (Windhoff et al., 2013), which combines the segmentation algorithms of FreeSurfer (Fischl, 2012) and FSL (Smith et al., 2004) to produce optimized head models. We conducted the segmentation based on regular T1 images and the summation of the T1 and T2 MRI-contrasts to optimize the separation of skull and skin. Head models consisted of five tissue classes: gray (GM) and white matter (WM), cerebrospinal fluid (CSF), skull and skin. Based on this segmentation we generated a single shell head model for MEG source-reconstruction (Nolte, 2003) and a five-compartment Finite Element Method (FEM) model for EEG source-reconstruction (Windhoff et al., 2013). FEM conductivities were set to: GM 0.276, WM 0.126, CSF 1.654, skull 0.010, and skin 0.465 (Thielscher et al., 2011).

Forward models (leadfields) were computed for sources on a regularly spaced grid with 1 cm distance covering the entire brain (3294 sources) in MNI-space. The leadfield describes how a dipole with fixed current pointing to each of the 3 principal axes (x-, y-, z-axis) projects to the sensors. We non-linearly transformed the source locations to individual headspace using each participant's T1-weighted MRI scan. The coordinates for the seed-based connectivity analyses were adopted from Hipp et al. (2012). For every seed, the source location with minimum Euclidean distance from the seed coordinates was chosen. Source coordinates, head model, EEG and MEG channels were co-registered on the basis of the three head localization coils.

Source projection

Sensor-level EEG and MEG data were projected to source-space using adaptive linear spatial filtering (DICS, beamforming; Gross et al., 2001; Van Veen et al., 1997). For each source i, the filter A is derived from the MEG/EEG cross-spectral density matrix CSD specific for frequency f and the physical forward model L (leadfield), which is the number of sensors-by-3 matrix corresponding to three orthogonal dipoles. The CSD is the outer product over time points of the frequency-domain sensor level data $TFR(f)$ with its complex conjugate transpose (Hermitian transpose) normalized by the number of time points n_t:

$$CSD(f) = TFR(f)TFR^{'}(f)/n_t \tag{3}$$

where $TFR^{'}(f)$ denotes the complex conjugate transpose of $TFR(f)$. For each frequency band f and source position i, we derived the 3-dimensional filter-matrix A (Van Veen et al., 1997):

$$A(i,f) = \left[L(i,f)^T CSD_{real}(f)^{-1} L(i,f)\right]^{-1} L(i,f)^T CSD_{real}(f)^{-1} \tag{4}$$

where $CSD_{real}(f)$ is the real part of the sensor-level cross-spectral density matrix. Beamforming filters transmit activity from sources of interest with unit gain, while maximally suppressing contribution from all other sources. While theoretically beamforming assumes uncorrelated sources, the method is robust to moderate, i.e. biologically plausible, levels of correlation (Van Veen et al., 1997). The regularization parameter was set to 5% of the frequency specific signal power. Beamforming was applied separately for EEG and MEG. For each source, we projected the sensor-level CSD through the filter, which yielded the 3-by-3 source-level CSD ($CSD_i(f)$), where it's real part describes the covariance between the 3 orthogonal dipole orientations. We performed principal component analysis (singular value decomposition) on the source-level CSD to estimate the source-level data along the dominant dipole orientation:

$$CSD_i(f) = real\left(A(i,f)CSD(f)A(i,f)^T\right) \tag{5}$$

$$U(i,f)S(i,f)V(i,f)^T = svd(CSD_i(f)) \tag{6}$$

The first Eigenvector of the principle component analysis equals the dominant dipole orientation at the given source. Hence, we projected the filter onto the first Eigenvector U_1.

$$A_{pri}(i,f) = U_1(i,f)^T A(i,f) \tag{7}$$

Finally, for every source position i, we derived the source-level data x_i by multiplying the complex sensor-level data TFR with the real-valued filter A_{pri}.

$$x_i(t,f) = A_{pri}(i,f)TFR(t,f) \tag{8}$$

Functional connectivity analysis

As a measure of functional connectivity, we computed brain-wide pairwise correlations (Pearson's r) between log-transformed power at all source locations i and j. In the following sections i always denotes the row index and j the column index. Without further processing, these correlations suffer from source leakage: Source estimates are not independent due to field spread and limited filter resolution. This leads to an overestimation of short-distance correlations (Brookes et al., 2012; Hipp et al., 2012). Source leakage has an effect only on instantaneous relations, since electromagnetic fields propagate at speed of light. To eliminate these spurious correlations Hipp et al. (2012) proposed to orthogonalize any two signals before correlating (Pearson's r) their envelopes (see also Brookes et al., 2012).

$$x_{i\ orth}(t,f) = imag\left(x_i(t,f)\frac{x_j'(t,f)}{|x_j(t,f)|}\right) \tag{9}$$

By projecting the original complex signal x_i as a function of frequency f and time t onto the vector orthogonal to the complex signal $x_j(t,f)$, we derived the orthogonalized signal $x_{i\ orth}(t,f)$. This approach suppresses instantaneous contribution of signal x_j on x_i. As was shown by Brookes et al. (2014) removal of volume conduction by orthogonalization is optimal for data with Gaussian distributions (Brookes et al., 2014). Furthermore it should be noted, that the employed orthogonalization does only discount mutual field spread between the two sources of interest, but it does not affect any field-spread of other sources on these two sources (Palva and Palva, 2012). For all pairs of source locations i and j, we computed log-power envelopes $p(t,f)$ applying the orthogonalization in both directions:

x_i on x_j;

$$p_{i\ orth}(t,f) = \log\left(x_{i\ orth}(t,f)x_{i\ orth}'(t,f)\right) \tag{10}$$

$$p_j(t,f) = \log\left(x_j(t,f)x_j'(t,f)\right) \tag{11}$$

x_j on x_i;

$$p_i(t,f) = \log\left(x_i(t,f)x_i'(t,f)\right) \tag{12}$$

$$p_{j\ orth}(t,f) = \log\left(x_{j\ orth}(t,f)x_{j\ orth}'(t,f)\right) \tag{13}$$

We computed correlations for the complete adjacency matrix $C(f)$ as the averaged correlations across both orthogonalization directions C_{dir}(x_i on x_j; and x_j on x_i):

$$c_{i,j\ dir}(f) = \frac{\underset{1\le t\le T}{cov}\left(p_{i\ orth}(t,f), p_j(t,f)\right)}{\sqrt{\underset{1\le t\le T}{var}\left(p_{i\ orth}(t,f)\right)\underset{1\le t\le T}{var}\left(p_j(t,f)\right)}} \tag{14}$$

$$c_{j,i\ dir}(f) = \frac{\underset{1\le t\le T}{cov}\left(p_i(t,f), p_{j\ orth}(t,f)\right)}{\sqrt{\underset{1\le t\le T}{var}\left(p_i(t,f)\right)\underset{1\le t\le T}{var}\left(p_{j\ orth}(t,f)\right)}} \tag{15}$$

$$c_{i,j}(f) = c_{j,i}(f) = \tanh\left(atanh\left(c_{i,j\ dir}(f)\right)/2\right) + \operatorname{atanh}\left(c_{j,i\ dir}(f)\right)/2) \tag{16}$$

where $c_{i,j}$ denotes the i-th row and j-th column element of matrix C. Discarding the relation between sources with zero phase lag causes underestimating correlations by a factor of approximately 0.577 (Hipp et al., 2012). All correlations were corrected accordingly.

Seed-based analysis

We computed log-power correlations of orthogonalized signals between homologous early sensory areas and whole brain seed correlations. Seed correlations were further computed for selected higher order cortices. We adapted seed locations from Hipp et al. (2012): left auditory cortex (lAC) [−54, −22, 10]; left somatosensory cortex (lSSC) [42, −26, 54]; left visual cortex (lVC) [−20, −86, 18]; right dorsal prefrontal cortex (rDPFC) [54, −63, −8]; and right lateral parietal cortex (rLPC) [46, −45, 39] (all MNI coordinates).

For our further analyses we focused on the spatial structure of amplitude correlations. To pinpoint the spatial structure while discounting for general offsets in correlation strength between subjects, we standardized (z-scored) the square adjacency matrices $C(f)$ within each column j and participant:

$$c_{i,j\ zsc}(f) = \left(c_{i,j}(f) - \underset{1\le i\le N_{source}}{mean}\ c_{i,j}(f)\right) / \underset{1\le i\le N_{source}}{std}\ c_{i,j}(f) \tag{17}$$

Each element of the standardized adjacency matrix $C_{i,j\ zsc}$ corresponds to the row-wise standardized connection strength within a given subject. Each row describes the standardized correlation pattern of a source j. For the seed-based analysis we tested across all S subjects, which connections showed a standardized correlation $c_{i,j\ zsc}$ larger than zero, i.e. which connections showed correlations significantly stronger than the average correlation across all sources (Student's t-test). The resulting p-values were corrected for false discovery rate (FDR; Benjamini and Hochberg, 1995) within each column of $C_{zsc}(p_{\mathrm{corr}} < 0.05)$.

Intermodal correlation and attenuation correction

For the whole-brain analysis of the similarity of EEG and MEG, we correlated the standardized adjacency matrices (correlation structures) – for different subjects $s1 \neq s2$, at each source j (column of c_{zsc}), and frequency f – between modalities $r_{between}$:

$$r_{j,s1,s2\ \mathrm{between}}(f) = \frac{\underset{1\le i\le N_{source}}{cov}\left(c_{i,j,s1\ \mathrm{zsc\ EEG}}(f), c_{i,j,s2\ \mathrm{zsc\ MEG}}(f)\right)}{\sqrt{\underset{1\le i\le N_{source}}{var}\left(c_{i,j,s1\ \mathrm{zsc\ EEG}}(f)\right)\ \underset{1\le i\le N_{source}}{var}\left(c_{i,j,s2\ \mathrm{zsc\ MEG}}(f)\right)}} \tag{18}$$

Correlation was computed between different subjects to match the computation of within-modality reliabilities (see below). We averaged the overall possible $S(S-1)$ combinations of subjects, which yields the average brain-wide between-modality similarity of correlation patterns ($r_{between}$).

The spatial structure of intermodal correlation patterns does not only depend on the true correlations of the underlying correlation patterns but also on the fidelity (signal-to-noise ratio, SNR), with which each modality measures these patterns. Signal power is neither equally distributed over space nor over frequencies. Furthermore, EEG and MEG are sensitive to different proportions of artifacts and environmental noise. To control for different levels of SNR, we first estimated the SNR for each modality and frequency as the between subject reliability of correlation structure within each modality (r_{EEG}, r_{MEG}).

$$r_{j,s1,s2\ \mathrm{EEG}}(f) = \frac{\underset{1\le i\le N_{source}}{cov}\left(c_{i,j,s1\ \mathrm{zsc\ EEG}}(f), c_{i,j,s2\ \mathrm{zsc\ EEG}}(f)\right)}{\sqrt{\underset{1\le i\le N_{source}}{var}\left(c_{i,j,s1\ \mathrm{zsc\ EEG}}(f)\right)\ \underset{1\le i\le N_{source}}{var}\left(c_{i,j,s2\ \mathrm{zsc\ EEG}}(f)\right)}} \tag{19}$$

We correlated the standardized adjacency matrices for each column j between subjects $s1 \neq s2$, which yielded a single reliability value for each source pattern and comparison. We computed the reliability maps of MEG accordingly and derived the average within-modality reliabilities r_{EEG} and r_{MEG} by averaging over all $S(S-1)/2$ between-subject comparisons.

To compute the SNR corrected between-modality correlations r_{corr}, we employed Spearman's correction of attenuated correlation coefficients (Bergholm et al., 2010a; Hipp and Siegel, 2015; Spearman, 1904):

$$r_{i,j\ corr}(f) = r_{i,j\ between}(f) / \sqrt{r_{i,j\ MEG}(f) r_{i,j\ EEG}(f)} \tag{20}$$

The between-modality correlation $r_{between}$ was normalized by the pooled within-modality reliabilities r_{EEG} and r_{MEG}. Correlations can only be corrected for reliable estimates. Thus, only sources i with correlation-pattern reliabilities consistently larger than zero for both modalities were included (Student's t-test at $p_{\mathrm{uncorr}} < 0.05$).

$$t_{i\ EEG}(f) = \frac{r_{i\ EEG}(f)}{\underset{1\le s\le S}{std}\ r_{i,s\ EEG}(f) / \sqrt{S(S-1)/2 - 1}} \tag{21}$$

The index s denotes the pairwise comparisons for which $s1 \neq s2$ holds. Sources with a non-significant reliability in either of the two modalities were excluded from correction. Attenuation correction was independently applied in each frequency.

Degree distribution of functional networks

To assess the strength of connectivity of specific sources within the network structure we computed the network degree of each source for each frequency band and modality. Adjacency matrices C of each subject s were binarized to values larger than the 90th percentile r_{p90}.

$$C_{bin}(s) = C(s) > r_{p90} \tag{22}$$

We calculated the degree Deg as the marginal of the binarized matrices and averaged across subjects s for each frequency f.

$$\mathrm{Deg}_j(f) = \frac{1}{S} \sum_{s=1}^{S} \sum_{i=1}^{N_{source}} c_{i,j\ bin}(s,f) \tag{23}$$

For each source, this yields the average number of connections with correlation above the 90th percentile. This approach partly circumvents the problem of different average degrees with fixed thresholds in binary graphs (Van Wijk et al., 2010).

All analyses were performed in Matlab (Mathworks, Massachusetts, USA) using custom scripts and the FieldTrip (Oostenveld et al., 2010), and SPM toolboxes (Friston et al., 2011).

Results

Interhemispheric correlations of homologous sensory areas

Previous studies have demonstrated prominent anatomical (Shen et al., 2015) and functional (Biswal et al., 1995; Brookes et al., 2011a, 2011b; Cordes et al., 2001; Hipp et al., 2012; Nir et al., 2008) connectivity between homologous sensory cortices in both hemispheres. Thus, we first compared power envelope correlations of orthogonalized left and right sided auditory, somatosensory and visual seeds between both modalities (Fig. 1a,b).

Our MEG and EEG findings confirmed the results of Hipp et al. (2012). Overall, the raw correlations were highly similar and positive in both modalities. Correlations peaked in the frequency range from 8 to 32 Hz with correlations significantly above average in both modalities ($p < 0.05$). The visual areas showed a pronounced peak in the alpha range (8 to 11 Hz) and another peak around 19 Hz for MEG. The auditory areas showed a distribution similar to the average connectivity strength. The somatosensory areas showed a broader pattern with peak connectivity between 8 and 22 Hz.

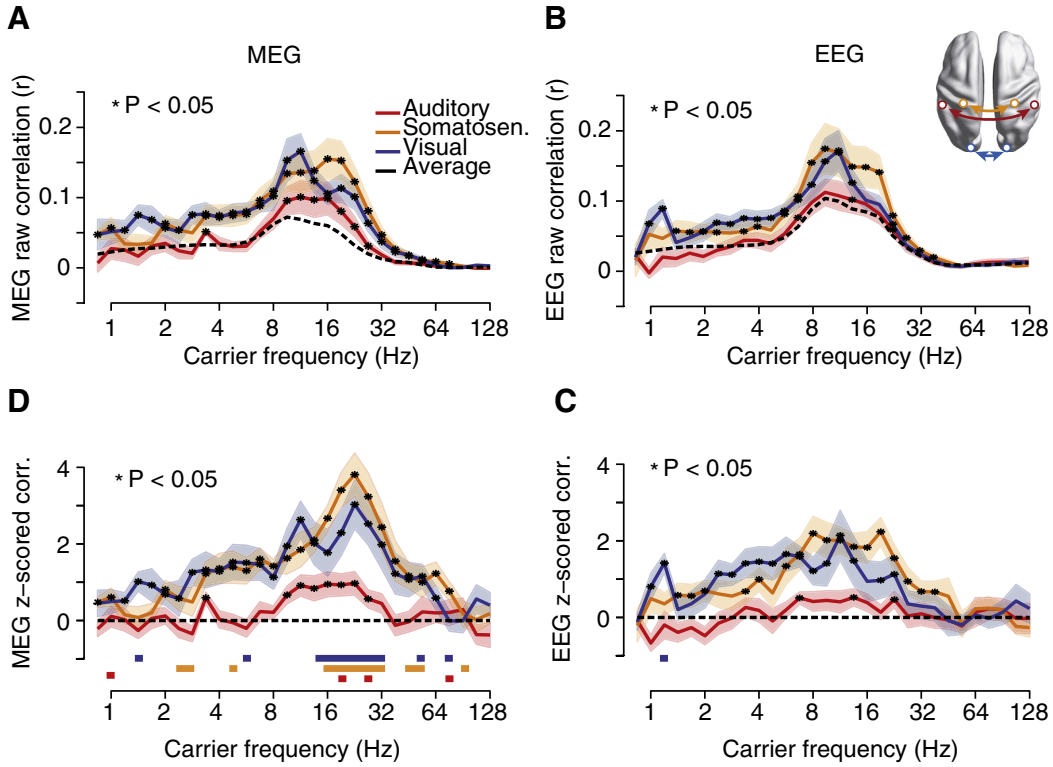

Fig. 1. Interhemispheric correlations between homologous early auditory (red), somatosensory (yellow) and visual (blue) areas for A) MEG and B) EEG. C) Standardized interhemispheric correlations for MEG and D) EEG. Asterisks indicate correlations that are significantly ($p < 0.05$) stronger than the average across the brain (dashed line). Colored bars in C) and D) indicate significantly larger standardized correlations in the respective modality than in the other modality ($p < 0.05$). A schematic of the investigated connections is shown as inset in B). Shaded areas indicate standard errors.

Next, we assessed the spatial structure of correlations between sensory areas as compared to the rest of the brain. To this end, we standardized (z-scored) the correlations for each participant by subtracting the mean and dividing by the standard deviation of correlations across all connections (Fig. 1c,d). This revealed generally higher standardized correlations, i.e. spatial structure of correlations in early sensory areas, for MEG (Student's t-test; $p < 0.05$), in particular, in the frequency range with strongest correlations (8 to 32 Hz).

We next investigated if power correlations in MEG and EEG were mutually predictive. In other words, we asked if subjects with strong interhemispheric correlation in MEG also showed strong interhemispheric correlation in EEG. Indeed, we found that in particular in the alpha and beta frequency range (8–32 Hz), the strength of MEG and EEG power correlations were mutually predictive for all sensory seeds as well as for the grand average across connections (Fig. 2a).

The comparison of standardized correlations for sensory regions suggested a stronger spatial correlation structure for MEG than for EEG. To investigate the correlation structure across the entire brain, we compared the distribution of power envelope correlations across all connections between modalities. More specifically, we quantified the skewness of MEG and EEG correlation distributions across connections (Fig. 2b). We hypothesized that above average correlations, i.e. spatial structure of connectivity, result in a right-tailed distribution with positive skewness. Indeed, for both modalities, we found positive skewness over the entire spectrum. For MEG, the skewness spectrum well resembled the average correlation spectrum (Fig. 1a) with highest skewness around 16 Hz. For frequencies below 32 Hz, MEG and EEG behaved similarly. MEG showed higher skewness corresponding to more distinct connectivity peaks than EEG ($p < 0.05$). For frequencies above 32 Hz, we found a qualitative difference between EEG and MEG:

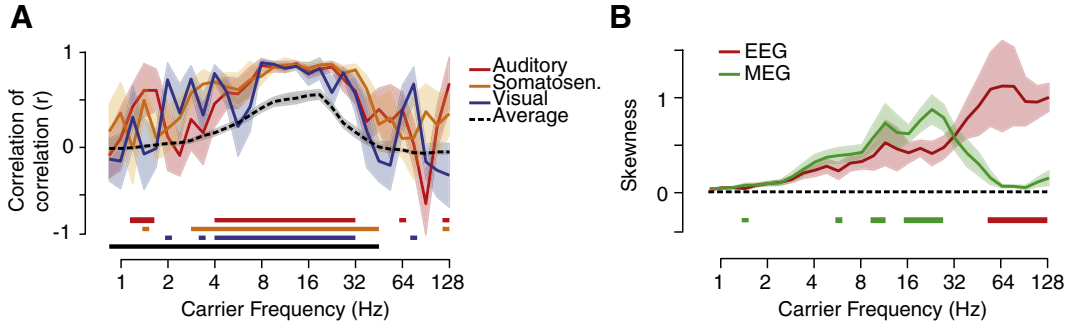

Fig. 2. Relation between EEG and MEG connectivity. A) Predictive power (Pearson correlation) between EEG and MEG interhemispheric correlation for early auditory (red), somatosensory (yellow) and visual (blue) areas as well as for all connections (dashed line) across participants. Shaded areas indicate standard errors. Colored bars indicate significant correlation between modalities ($p < 0.05$). B) Skewness of standardized correlation distributions for EEG (red) and MEG (green) averaged across participants. Shaded areas show standard deviation across participants. Colored bars indicate significant differences of skewness between modalities (green MEG > EEG, red EEG > MEG; $p < 0.05$).

While MEG skewness dropped similar to the average correlation spectrum (Fig. 1a), EEG skewness strongly increased, yet above the skewness for lower frequencies and the maximum skewness in MEG (Fig. 2b).

Correlation structure of early sensory and higher order cortices

To further investigate the spatial structure of power envelope correlations assessed with both modalities, we spatially resolved the connectivity of specific cortical seeds (Fig. 3). First, we imaged the brain-wide correlation strength for the three sensory seeds investigated above. We focused this analysis on 16 Hz carrier frequency because it showed the strongest raw correlations over the entire brain in both modalities (Fig. 1), and to foster the comparison with previous MEG studies (Hipp et al., 2012). In accordance with these previous results (Hipp et al., 2012), for both MEG and EEG, all three sensory seeds showed the strongest correlation to neighboring cortical regions and homologous regions in the other hemisphere. In general, EEG and MEG correlation patterns were well compatible (lAC: $r = 0.83$; lSSC: $r = 0.89$; lVC: $r = 0.87$). However, as indicated above (Figs. 1 and 2), standardized correlations were generally higher for MEG than for EEG with stronger inter-hemispheric correlations. This was in particular the case for the auditory cortex (compare also Fig. 1c,d). Furthermore, correlation patterns generally appeared spatially smoother in MEG than in EEG.

Next, we investigated the brain-wide correlation structure of higher order cortices (Fig. 4). We focused on the right dorsal prefrontal cortex (rDPFC) and the right lateral parietal cortex (rLPC). These brain regions have previously been shown to exhibit a complex power correlation structure in MEG (Hipp et al., 2012) and are thus well suited to test for complex correlation structure in EEG. Again, our MEG results confirmed previous findings. For rLPC the patterns overlapped well between MEG and EEG ($r = 0.89$) with ipsilateral and contralateral

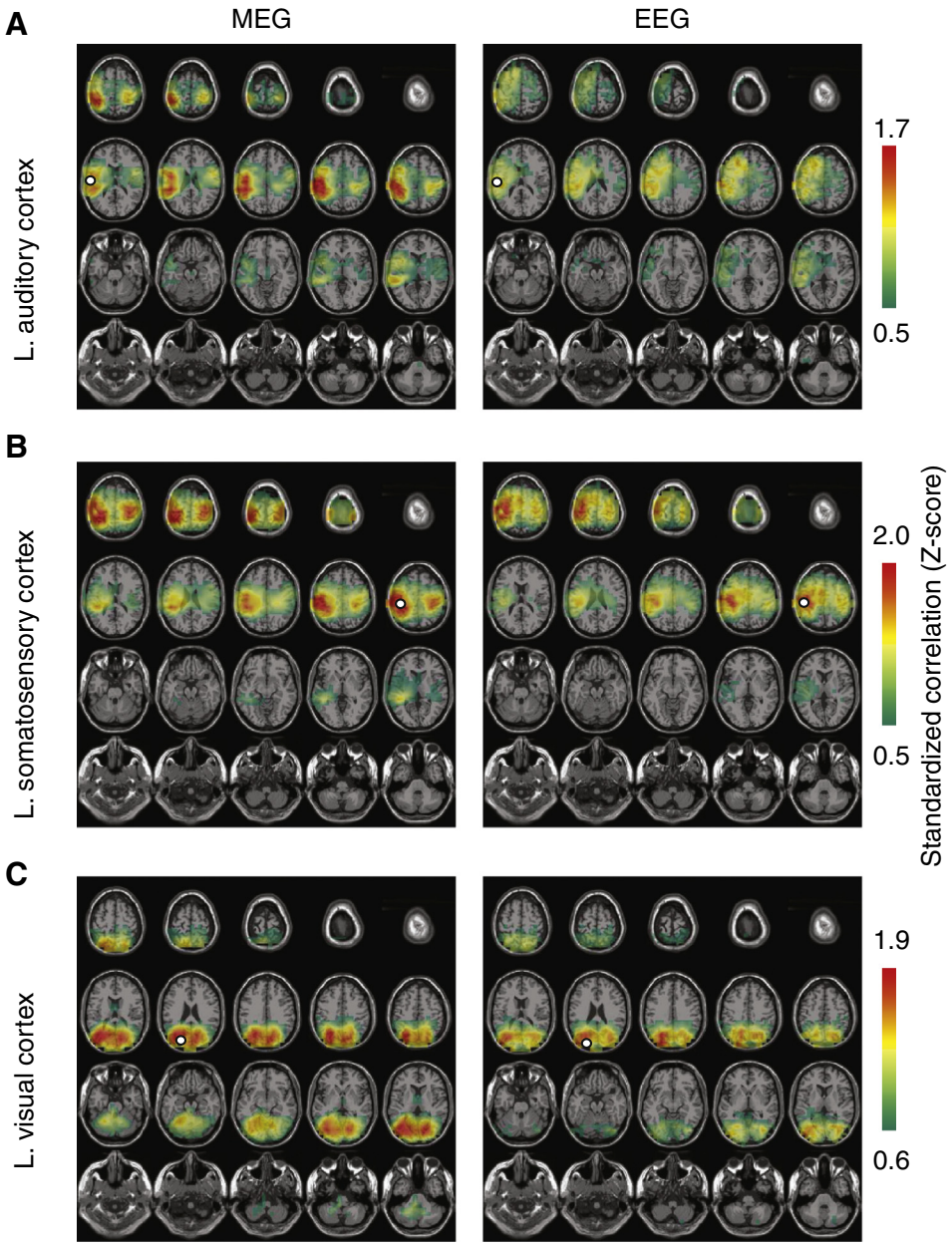

Fig. 3. Seed-based correlation structure for the left auditory (top), left somatosensory (middle) and left visual cortex (bottom) for MEG (top row) and EEG (bottom row). Blue dots mark seed locations. Correlation structure is masked for correlation significantly larger than the average correlation ($p < 0.05$, FDR corrected). The color scale is adapted to the minimum 5% and maximum 95% percentile for each seed.

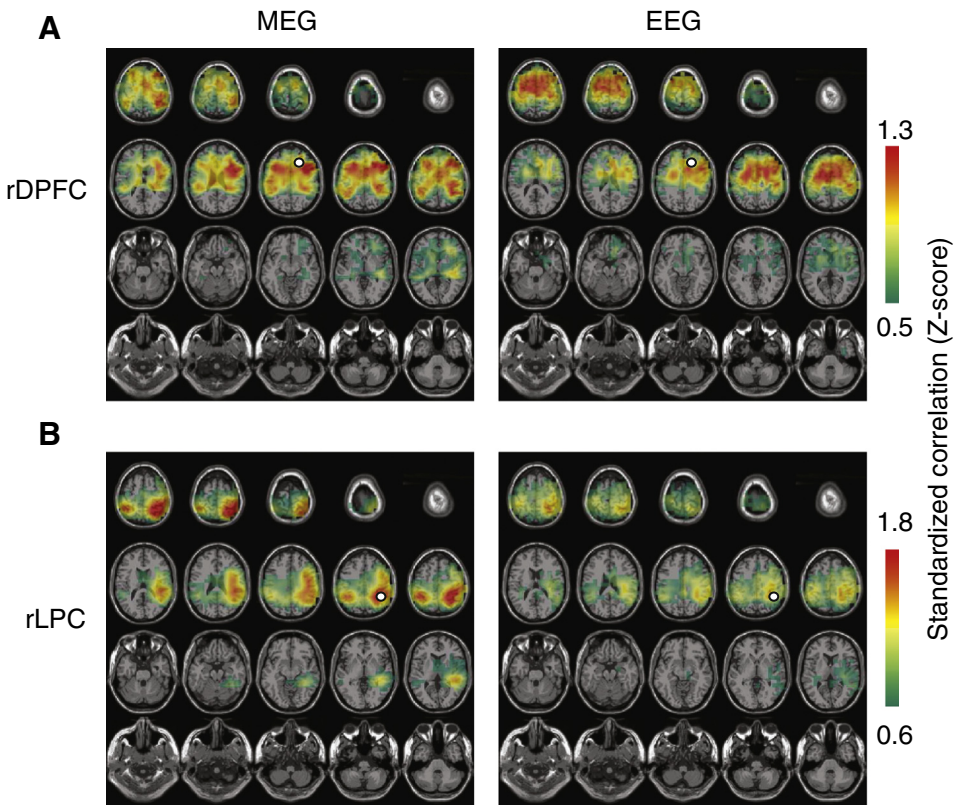

Fig. 4. Seed-based correlation structure for A) right dorsal prefrontal cortex (rDPFC) and B) the right lateral parietal cortex (rLPC) for MEG (left) and EEG (right). Blue dots mark seed locations. The correlation structure is masked for connections significantly larger than the average ($p < 0.05$, FDR corrected). The color scale is adapted to the minimum 5% and maximum 95% percentile for each seed.

peaks in LPC. For the rDPFC, MEG correlations peaked ipsi- and contralaterally around the DPFC as well as bilaterally in the postcentral gyrus. In contrast, EEG did not resolve a distinct correlation peak in the contralateral postcentral gyrus. However, pattern correlation still indicated a strong overlap between modalities ($r = 0.80$). Furthermore, standardized correlation values were again generally lower for EEG than for MEG (Student's t-test, $p < 0.01$).

Conclusively, seed correlation patterns of 275-channel MEG and 64-channel EEG were generally very well compatible. In accordance with the much larger number of MEG sensors, distinct correlation patterns were generally better resolved with MEG.

Brain-wide intermodal relation of correlation structure

So far, we investigated the similarity of EEG and MEG correlation structure for 16 Hz carrier frequency and selected seeds in sensory and higher order cortices. To systematically analyze the pattern similarity across the entire brain and spectrum, we correlated the seed correlation maps of MEG and EEG for every source and frequency (Fig. 5). Averaged across all seeds, we found positive correlations for all frequencies with highest correlations in the frequency range from 8 to 32 Hz (Fig. 5a). For lower frequencies (1 to 8 Hz), similarities were highest for occipital regions (Fig. 5b). Around 16 Hz, peak similarity shifted toward parietal and frontal regions. In the low gamma range (32 Hz), similarity peaked again in occipital and parietal areas. From 64 to 128 Hz similarity patterns became very sparse, again with consistent peaks in occipital and parietal regions.

Signal-to-noise corrected brain-wide intermodal correlation of patterns

The observed spectral and spatial distributions of pattern similarity between MEG and EEG resemble the known signal-to-noise (SNR) characteristics of cortical population signals as measured with these techniques (Niedermeyer and da Silva, 2005). For example, in the alpha frequency range intermodal patterns overlap best in occipital regions, whereas in the beta frequency range motor areas show peak similarity, which matches the strong alpha and beta rhythms found in occipital and motor regions, respectively. Also the peak of average pattern similarity around 16 Hz matches prominent cortical population signals and strongest correlations structure in that frequency range (Fig. 1). Thus, the observed spectral and spatial distribution of intermodal similarity may not reflect the genuine relation between MEG and EEG, but the fidelity i.e. the SNR of the compared correlation patterns within each modality. E.g. low intermodal similarity at frequencies below 16 Hz may rather reflect the low SNR of correlation patterns within EEG and MEG, than a substantial difference in the patterns measured by both modalities.

To investigate the influence of SNR, we computed the attenuation corrected correlation between MEG and EEG (Bergholm et al., 2010b; Hipp and Siegel, 2015; Spearman, 1904) (Fig. 6). Attenuation corrected correlation can be interpreted as the correlations that would have been measured for perfect reliability within each modality (i.e. no measurement error). In this procedure, the within-modality reliability of patterns across subjects is taken as the measure for the SNR. Raw intermodal correlations are then divided by the pooled within-modality reliabilities (see "Methods" section). Attenuation correction was only performed if both EEG and MEG showed significant within-modality reliability (Fig. 6b). If both modalities measure the same underlying networks, but are prone to noise processes, the attenuation correction converges to one. In contrast, if both modalities are sensitive to different aspects of the signal, independent of the influence of noise, attenuation corrected values are smaller than one.

Attenuation corrected intermodal correlations approached 1 for almost all frequencies (Fig. 6a, solid blue line). However, above 32 Hz,

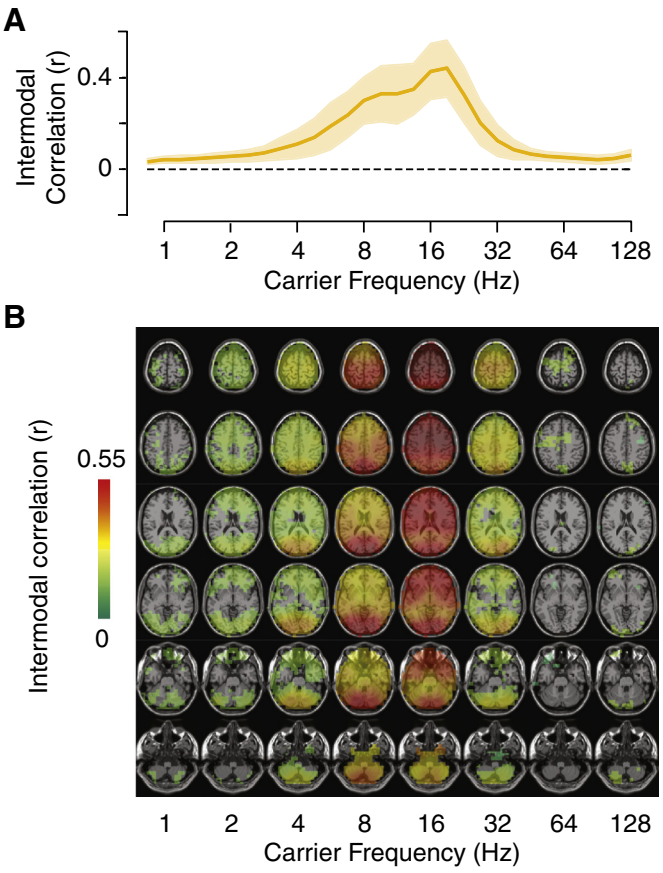

Fig. 5. A) Spectrum of intermodal correlation of seed-correlation patterns averaged across all seeds. The shaded band indicates standard deviation across seeds. B) Cortical maps of intermodal correlations of seed-correlation patterns masked for significant correlations ($p < 0.05$). Color scale was adapted to 0 and the maximum of the 95% percentile across all frequencies. We did not find any significant negative correlations.

attenuation corrected correlations slightly decreased to around 0.8. From about 4 to 32 Hz, many sources that were broadly distributed across the brain showed significant reliabilities to estimate attenuation corrected intermodal correlation (Fig. 6b and c). For very high and low frequencies, only few sources showed significant reliabilities to estimate attenuation corrected intermodal correlation. But the remaining sources still had mean attenuation corrected intermodal correlations around 0.98 (range: 0.95–0.99) and 0.8 (range: 0.70–0.98) for low and high frequencies, respectively.

The decrease of attenuation corrected intermodal correlations for high frequencies suggests that MEG and EEG do not reflect identical patterns of power correlations for these frequencies. We hypothesized that this may reflect the stronger sensitivity to muscle artifacts for EEG. To test this, we repeated the analysis on the raw data without removing independent components reflecting muscle activity (Fig 6a, dashed purple line). If the decrease of attenuation corrected correlation reflected the effect of muscle activity we expected this decrease to be more pronounced for the raw data. Consistent with this hypothesis, the attenuation corrected correlation in the not-cleaned data decreased from 32 to 128 Hz to around 0.6 (range: 0.55–0.64).

Degree distribution of functional networks

In a final set of analyses, we applied simple graph-theoretical measures (degree) to identify cortical hubs, i.e. brain regions with strongest connectivity to other regions. This approach may identify differences in spatial sensitivity between modalities that are not picked up by the correlation of correlation structures. For each participant and frequency

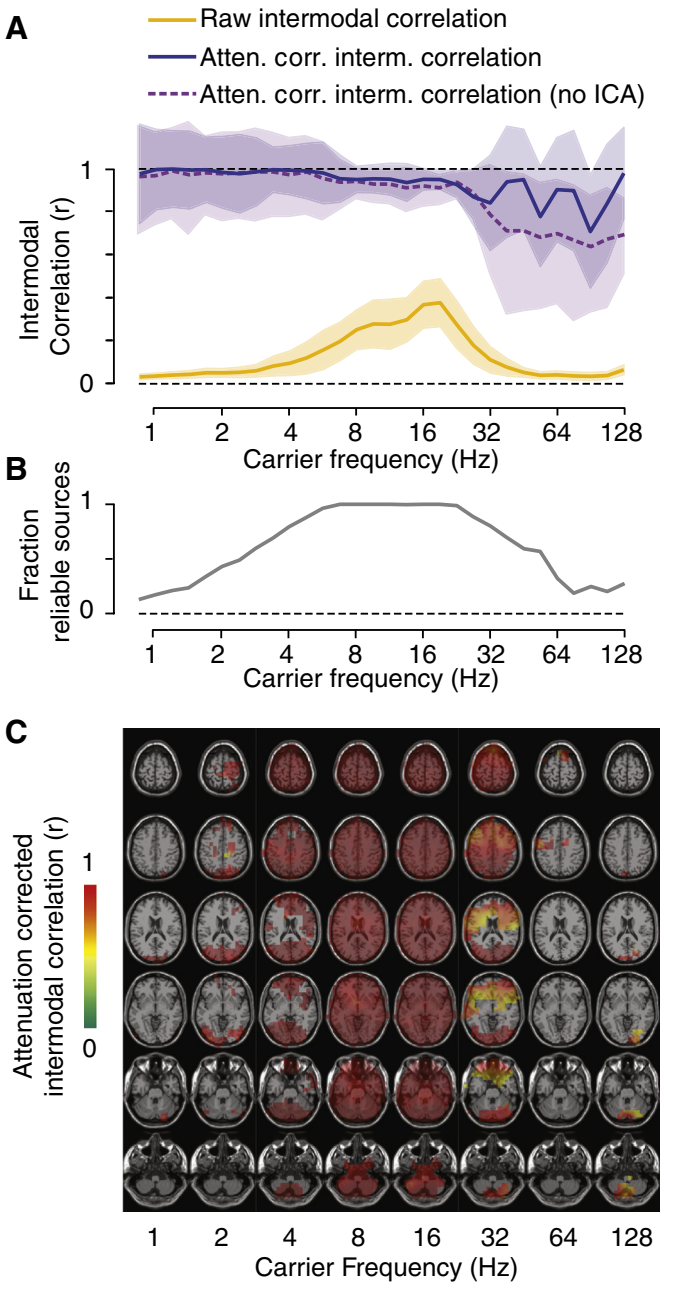

Fig. 6. Signal-to-noise corrected correlation coefficients for intermodal correlations. A) Spectrum of attenuation corrected intermodal correlations (solid blue) in comparison to the distribution of raw intermodal correlations (solid yellow) and attenuation corrected intermodal correlations for non-ICA cleaned data (dashed purple). Shaded bands indicate standard deviation across sources. B) Fraction of source with reliable correlation patterns in both modalities for the ICA cleaned data ($p < 0.05$ for both modalities). The intermodal correlation can be corrected only for sources with reliable patterns in both modalities. C) Spatially resolved spectrum of attenuation corrected intermodal correlations. Sources without significant reliability in both modalities ($p > 0.05$ for at least one modality) are masked. The color scale ranges from 0 to 1. There were no negative SNR-corrected intermodal correlations.

band, we defined a connection if the correlation between the orthogonalized signals of two sources was higher than the 90th percentile. We computed the marginal of the resulting binary connectivity matrices and averaged over participants. The resulting vector yielded the brain-wide degree distribution (Fig. 7).

For frequencies below 32 Hz, there was good correspondence of the cortical degree distributions between EEG and MEG. From 1 to 4 Hz, highest degrees were broadly distributed across occipital, parietal, and frontal areas. From 8 to 16 Hz, the patterns became spatially very

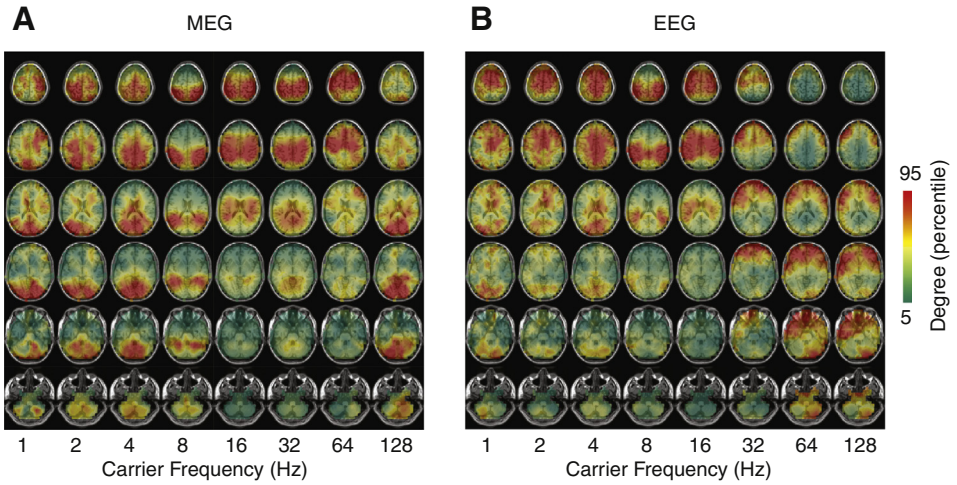

Fig. 7. Spatially resolved degree spectrum of power correlations for A) MEG and B) EEG. For each frequency and participant, a binary connection was assigned if the raw correlation between two sources was above the 90th percentile across all pair-wise correlations. The color scale is adapted to the 5% and 95% percentile for each modality and frequency.

distinct: The degree prominently peaked in bilateral posterior parietal cortex spreading also further anterior at 16 Hz. From 32 Hz on, the patterns diverged between modalities. For MEG, at 32 Hz and 64 Hz the degree distribution peaked in central regions. At 128 Hz, occipital cortex showed maximum degree. In contrast for EEG, from 32 to 128 Hz highest degrees were consistently located in anterior frontal and temporal regions.

Discussion

Here, we systematically compared the power correlations of simultaneously recorded, source-level co-registered, and orthogonalized MEG and EEG. Our results demonstrate the feasibility of this approach for EEG, and reveal several commonalities as well as differences between 275-channel MEG and 64-channel EEG.

Seed-based connectivity analyses

Our seed-based analysis showed that the spectral pattern of inter-hemispheric correlations is very similar in both modalities and that the spatial structure of seed-correlations is well compatible between modalities. Inter-hemispheric correlation as well as correlation structure peaked around 16 Hz for MEG and EEG. Furthermore, inter-hemispheric connectivity correlated well between modalities in particular in this middle frequency range. For both modalities, seed-correlation maps at 16 Hz indicated connectivity of homologous inter-hemispheric regions. For higher order cortices both modalities revealed complex patterns of bilateral connectivity that accorded well with the previous MEG results (Hipp et al., 2012). In sum, our seed-based results showed a strong overlap between MEG and EEG and were well compatible with recent MEG studies (Brookes et al., 2011b, 2012; Hipp et al., 2012). It should be noted though, that the identified networks had simple spatial structure as compared to resting-state networks identified with functional connectivity magnetic resonance imaging (fcMRI) (Fox and Raichle, 2007). This is largely due to the limited spatial resolution of non-invasive electrophysiological techniques. However, the application of more complex analytical approaches, such as graph theory (Hipp et al., 2012) or ICA (Brookes et al., 2011a) to MEG, allows for revealing more subtle spatial patterns with similarity to fcMRI networks. Nevertheless, the relation between electrophysiological and BOLD-related functional networks is complex and power correlations of a single frequency band are not sufficient to describe fcMRI networks (Hipp and Siegel, 2015).

Besides general similarity, the seed-based analysis also revealed differences between MEG and EEG. Three lines of evidence indicate that for 275-channel MEG correlation patterns were generally more distinct than for 64-channel EEG. First, standardized correlations, which measure the spatial structure of correlations, were consistently larger for MEG than for EEG (Fig. 1c,d). Second, some spatial features of correlation structure that were well resolved by MEG could not be clearly measured by EEG (Fig. 3 auditory seed and Fig. 4a rDPFC seed). Third, for frequencies up to 32 Hz, MEG showed a higher skewness of the correlation distribution than EEG, i.e. more positive outliers relative to a normal distribution. Interestingly, above 32 Hz, correlation skewness was larger for EEG than for MEG. As we will further discuss below, we hypothesize that this effect reflects the modality specific influence of muscle artifacts.

The observed advantage of MEG for resolving distinct correlation patterns is likely at least partly due to the much larger number of MEG sensors (275) as compared to EEG electrodes (64) employed. Thus, the observed difference should not be interpreted as a general advantage of MEG over EEG. In fact, given the difference in the number of sensors, it seems remarkable how similar correlation patterns were between modalities and how well the 64-channel EEG could resolve the patterns obtained with 275-channel MEG. On the one hand, this suggests that a standard 64-channel EEG, which is readily available in clinical settings, may suffice for resolving a substantial fraction of the correlation structure detectable with 275-channel MEG. On the other hand, high-density EEG, e.g. with 256 channels, may allow for resolving further spatial structure that was not available with the present 64-channel EEG system (Malmivuo, 2012).

Another difference between MEG and EEG was that MEG correlation patterns were generally smoother than EEG patterns. This may be surprising at first, because the number of channels, and thus the implicit degrees of freedom or smoothness of the source-reconstruction was lower for EEG as compared to MEG. This suggests that the more noisy appearance of EEG correlation patterns may reflect the more complex head model physics (FEM vs. single-shell) and field distributions in EEG as compared to MEG (Cho et al., 2015; Malmivuo, 2012).

Brain-wide intermodal correlation

The systematic correlation of seed-correlation maps between modalities revealed strongest intermodal similarity around 16 Hz in occipital and central regions. We adapted an approach that was recently introduced for the comparison between MEG and fMRI (Hipp and Siegel, 2015) to test if this spectral and spatial structure reflected

genuine difference between modalities or rather their SNR. For frequencies below 32 Hz, accounting for the SNR yielded almost perfect pattern similarity for regions with reliable correlation patterns in both modalities. Furthermore, from about 8 to 32 Hz reliable patterns were identified for more than 95% of the brain. Thus, from about 8 to 32 Hz, MEG and EEG power correlations display the same functional networks.

This finding sheds new light on the nature of the neuronal activity driving the power correlations measured with EEG or MEG. Both modalities have different sensitivities for specific dipole orientations and locations (Cho et al., 2015; Malmivuo, 2012). In particular, MEG is blind to a substantial number of sources, i.e. radially oriented dipoles on the top of gyri and at the bottom of sulci. Thus, our results suggest that the spatial extend of cortical sources that drive the measured correlation patterns is broader than the resolution of gyri and sulci, which differs between EEG and MEG. We conclude, that although on the level of raw dipole measurements EEG and MEG have different sensitivities, on the level of power correlations, especially from 8 to 32 Hz, both measures reflect the same underlying network activity. These results are promising for potential clinical applications (Stam, 2014), for which EEG is more readily available than MEG. Our results suggest that recent MEG findings (Brookes et al., 2012; Hawellek et al., 2013; Hipp et al., 2012; Kitzbichler et al., 2015; O'Neill et al., 2015) may be well extrapolated to potential EEG applications. However, it remains to be determined to what extent EEG network analyses could provide a diagnostic and predictive tool in the clinical routine, particularly on the single-subject level.

Furthermore, it should be noted that attenuation correction is only applicable for sources with reliable correlation patterns within both modalities. For frequencies below 8 Hz or above 32 Hz, we did not find reliable correlation patterns for the entire brain in both modalities. Thus, in principle for these frequencies ranges, there may be differences in correlation patterns between modalities in areas we could not correct.

Muscle artifacts

Our results reveal a small but substantial difference between MEG and EEG for frequencies above 32 Hz. Specifically, our results suggest that for this frequency range, i.e. the gamma band, electromyogenic artifacts substantially confound EEG power correlations, which is much less the case for MEG. Several findings support this reasoning. First, for this frequency range attenuation corrected intermodal correlations did not approach one, but values around 0.8 (range: 0.70–0.98). Intermodal pattern similarity further decreased, if independent components that reflected muscle activity were not removed from the data (range: 0.55–0.64). Second, above 32 Hz, the skewness of EEG power correlation progressively increased, indicating a substantial amount of strong correlations beyond a normal distribution. This was not the case for MEG. Third, for EEG above 32 Hz, the degree distribution, which pinpoints to those regions with strongest correlations, peaked in superficial anterior frontal and temporal regions well compatible with leaked electromyogenic activity from forehead, eye, and temporal muscles. This pattern was not found for MEG.

Notably, we observed the effects of muscle activity on EEG power correlations although we applied several complementary approaches to discount electromyogenic artifacts for MEG and EEG. We excluded epochs with obvious muscle activity, we rejected ICA components reflecting muscle activity, and we employed adaptive spatial filtering (beamforming) for source-reconstruction. This suggests that EEG power correlations are particularly sensitive to confounding by electromyogenic artifacts and more so than MEG. This accords well with recent reports of the substantial influence of electromyogenic artifacts on EEG gamma-band activity (Goncharova et al., 2003; Hipp and Siegel, 2013; Whitham et al., 2008; Yuval-Greenberg et al., 2008) and highlights the importance of careful artifact rejection in network analyses of electrophysiological data.

Conclusions

In summary, our results show that power correlation of orthogonalized signals is feasible for studying functional connectivity with 64-channel EEG. Furthermore, besides the differences in SNR, for frequencies from about 8 to 32 Hz, EEG and MEG reflect identical underlying correlation patterns across the brain. Above 32 Hz, power correlation patterns differ between modalities, which likely reflects the higher susceptibility to muscle artifacts for EEG than for MEG.

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