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REVIEW

The anterior midcingulate cortex might be a neuronal substrate for the ideomotor mechanism

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Abstract

The way the brain controls voluntary movements for normal and pathological subject remains puzzling. In this selective review, we provide unreported harmonies between the anterior midcingulate cortex (aMCC) activities and the ideomotor mechanism postulating that voluntary movements are controlled by the anticipation of the expected perceptual consequences of an action, critically involving bidirectional interplay of a given motor activity and corresponding sensory feedback. Among other evidence, we found that the required asymmetry in the bidirectional interplay between a given motor command and its expected sensory effect could rely on the specific activity of aMCC neurons when observing errors and successes. We confirm this hypothesis by presenting a pathological perspective, studying obsessive–compulsive and other related disorders in which hyperactivated and uniform aMCC activities should lead to a circular-reflex process that results in persistent ideas and repeated actions. By evaluating normal and pathological data, we propose considering the aMCC at a central position within the cerebral network involved in the ideomotor mechanism.

Keywords Circular reflex · Compulsion · Ideomotor theory · Midcingulate cortex · Voluntary movement

Introduction

The way the brain controls voluntary movements for pathological or normal subjects is a puzzling issue, and there is no doubt that an interdisciplinary approach is needed to tackle this fundamental question. However, unifying the findings arising from various disciplines is always difficult because comparable questions provide answers that often remain confined to a particular field. Even in a single domain such as neuroscience, studies interested in the neurobiological basis of voluntary movement focused on distinct aspects depending on author interests or laboratory traditions, which is not surprising if we consider that the theoretical accounts of voluntary movement are often conceived separately from perception, cognition, execution and evaluation.

Ideomotor theory tries to avoid these caveats and attempts to capture the global complexity of voluntary movement by gaining a full understanding of the intermingled relationship between perception and action. According to the ideomotor mechanism, a movement is planned by anticipating the cognitive representation of the expected perceptual effect (for example, from vision, audition, touch or proprioceptive information), resulting in a stable movement-outcome association (see Shin et al. 2010 for reviews; Badets et al. 2016; Pfister 2019). Critically, this association is also activated in the opposite direction since any mentally anticipated effect could directly prime a behavioral response. We propose that this bidirectional link is in part supported by the anterior midcingulate cortex (aMCC), whose deregulation could be associated with compulsions observed in obsessive–compulsive disorder (OCD) or related disorders. These pathologies are considered in this article to provide good support in favor of our hypothesis when considered, at least to some extent, a consequence of a pathological "circular reflex", which can be defined as an endless repetition of the same action.

In this article, we develop this hypothesis throughout five sections with the goal of questioning voluntary movements from normal to pathological accounts. The five sections are as follows: an account of ideomotor theory (1), detailing its neuronal correspondence with aMCC activity during either action (2), evaluation (3) or their interplay (4) and

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finally deriving its implication for pathological behaviors (5) in which aMCC dysfunction should lead to a circular-reflex process that results in action perturbations classically found in OCD, Tourette syndrome or other tic disorders. To anticipate our conclusion, we speculate that based on this selective review, that aMCC, already known as a key player in voluntary action (Hoffstaedter et al. 2014), could be a central part of the ideomotor mechanism and may advance the comprehension of voluntary movement control by opening new lines of research specifically questioning the role of the aMCC in the ideomotor framework. Two figures presenting the location of neuronal correlates or lesion sites discussed in this paper (Fig. 1) as well as a schematic representation of the ideomotor mechanism and its pathophysiological implications (Fig. 2) illustrate the theory.

Ideomotor theory

Ideomotor theory proposes that an action is controlled by the anticipation of the expected perceptual consequences of the action's impact on the environment. The main role of this perceptual expectation is to predict and efficiently select the appropriate action (Hommel et al. 2001) and to detect possible movement errors during and/or after the action's execution. Crucially, merely thinking about the sensory consequences of our movements can lead to the selection of related actions. Accordingly, actions are planned in terms of their anticipated perceptual consequences so that the representations of both actions and expected feedback share a "perceptual" framework. Functionally, this shared format, or "common coding" of perception and action (Prinz 1997), implies a bidirectional linkage between a motor command and its perceptual effect; once such an action-outcome association has been learned and stored, any cognitive representation (i.e., idea) of the perceptual effects will automatically activate the associated motor command (Badets et al. 2016), which is of obvious interest when considering the relationship between obsession and compulsion, as discussed later.

This theoretical interpretation has been confirmed numerous times in different experimental domains, including motor control (Elsner and Hommel 2001; Kunde 2001), sequence learning (Koch and Hoffmann 2000), social interaction (Flach et al. 2010), consciousness (Kunde 2004), numerical cognition (Badets et al. 2013), and word processing (Koch and Kunde 2002); critically, a recent study also demonstrated that nonwilled motor behavior is induced by the sole activation of its perceptual consequences (Colton et al. 2018). However, it could be argued that the forward models are sufficient to theorize human anticipation for action (Wolpert et al. 2001; see Dogge et al. 2019 for a recent review). Indeed, such models can predict action outcomes throughout the simulation of a perceptual copy of the motor command to predict sensory consequences. We acknowledge that all these movement systems are obviously implicated during enactment, but the present theoretical account, without denying such forward models, assumes a central role of the activation of an expected perceptual effect, just before the action itself (see Badets and Osiurak 2015 for a similar suggestion). According to Kunde, Koch, and Hoffmann (Kunde et al. 2004), a movement is performed when expected perceptual outcomes exceed a threshold; in other words, "the occurrence of an external (or internal) response cue induces a gradual growth of activation of the various multimodally distributed codes of response effects over time" (p. 101). This growing activation is not emphasized by forward models but resonates strongly with the perceptual superiority effect suggested by Badets and Osiurak (2015). From this perspective, the intention to perform an action in the future can increase a kind of "task tension", especially on the perceptual mechanism devoted to the expected effects in the environment. After the completion of the intended action, an inhibitory mechanism could disrupt the perceptual tension to avoid irrelevant motor repetition (see Moschl et al. 2020 for a detailed review on such deactivation that operates on a continuum from full intention retrieval to a complete deactivation or even inhibition). In this respect, we can consider the functional role of such perceptual tension in memory as a process that allows maintaining strategic control of our intended behaviors.

Another strong assumption is that an effect representation throughout an ideomotor mechanism automatically primes a motor response; this assumption is also supported by numerous experiments indicating that the observation of another's actions may also influence the observer's own actions (see Knoblich and Prinz 2005 for an in-depth review). This is in line with goal-directed action imitation, which is particularly expressed by primates and is thought to be constitutive of human culture. Indeed, unlike most species, humans are able to learn by imitation (Heyes 2001; Rizzolatti and Craighero 2004; Knoblich and Prinz 2005) (but see below for an incidental pathological effect of this property). A clear experimental demonstration that either the mere observation (i.e., not goal-directed behavior) of an action or its outcome also automatically primes a motor response (action induction) has been derived from the finding that a posterror slowing is initiated in subjects observing errors committed by another actor. Indeed, posterror slowing (a prolongation of reaction time (RT) after an erroneous response), which is often considered a typical behavioral adjustment allowing the success rate to be maximized (Rabbitt 1966), also received alternative interpretations (Rabbitt and Rodgers 1977) and has been found to be triggered in observers monitoring another's behavior (Schuch and Tipper 2007). Regardless of its functional interpretation, observation of the outcome, without any direct observation of the action itself, is sufficient to

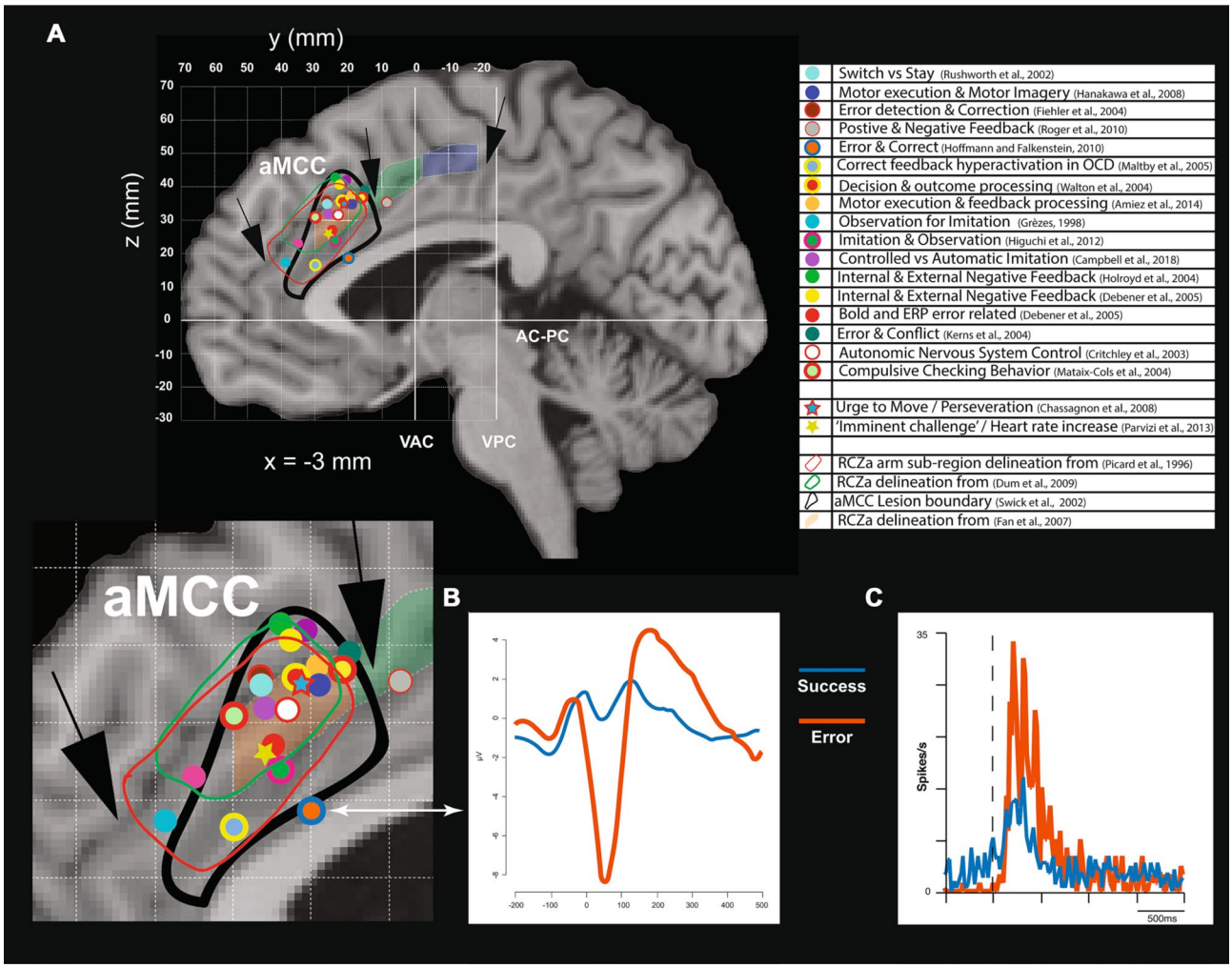

Fig. 1 Clustering of putative ideomotor-related functions within the aMCC: **A** Coordinates of activation and stimulation sites plotted on a medial-sagittal view of the brain (adapted from (Fan et al. 2007)). Coordinates from Talairach space were converted to MNI space (Lancaster et al. 2007). There is a consistent localization of neural activity (fMRI brain activation and dipole solutions; colored circles), stimulation loci (stars) and brain lesions related to ideomotor mechanisms and OCD pathophysiology in the anterior midcingulate cortex (aMCC). *VAC* vertical plane passing through the anterior commissure, *VPC* vertical plane passing through the anterior commissure. *AC–PC* line crossing the anterior commissure-posterior commissure. **B** Response-locked ($t = 0$ ms) grand averages (functionally connected zone (FCz)) showing an error-related negative deflection (vermilion) of greater amplitude than the correct-related negativity (blue) for error and correct trials (adapted from (Hoffmann and Falkenstein 2010)). The corresponding dipole solution is graphically represented in panel 1A (vermilion and blue circle). **C** Neurons that respond to both positive (blue) and negative (vermilion) feedback demonstrate greater activation after negative feedback than after positive feedback in the monkey rostral cingulate motor area, a region homologous to the human aMCC (adapted from Michelet et al. 2007). The list in panel A (upper right) presents articles reporting behavioral, anatomical or functional studies related to upper limb movements (as an example) and involving the aMCC region (see color/symbol coding and corresponding localization in the brain sections shown at left). It is important to note that the aMCC as a constituent of a four-region model (see Vogt (2009) for a comprehensive review) is a concept now widely used in an effort to harmonize nomenclature for cingulate cortex organization. However, several studies cited in the present review refer to older (and sometimes misnamed) nomenclature, making it difficult for comparisons between studies to be made (Vogt 2016). We have systematically used the term aMCC for each location corresponding to the borders defined by Vogt and collaborators (Vogt 2009, 2016). Therefore, in panel 1A, we plotted the peak voxels associated with functional activation, stimulation site or the electrophysiological source localized in these studies, coregistered with the borders (indicated by lines) of the most consistent alternative nomenclature (e.g., the anterior rostral cingulate zone: RCZa) (Picard and Strick 1996; Fan et al. 2007; Dum et al. 2009) and of the aMCC

produce this posterror slowing, further indicating that performance monitoring is able to prime or trigger an action selection (Weller et al. 2018). This agrees with previous proposals postulating that the ideomotor mechanism is dependent on the evaluative function and implies that the actual outcome of a given behavior is compared with the intended effect (Nattkemper and Ziessler 2004; Band et al. 2009).

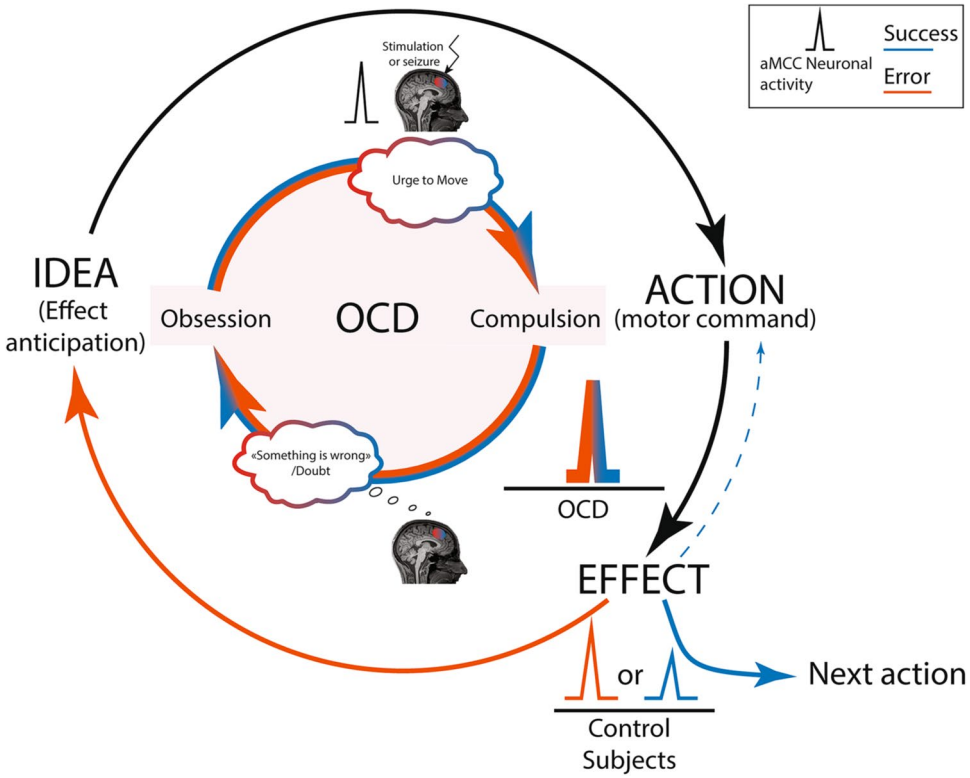

Fig. 2 Schematic representation of the ideomotor mechanism and its pathophysiological implications: According to ideomotor theory, an action is controlled by the anticipation of the expected perceptual consequences of the execution of that action in the environment. The ideomotor mechanism is thus based on a bidirectional link between the underlying motor command and its anticipated outcome. In normal control subjects, the monitoring of positive (blue) or negative (vermilion) outcomes is encoded by differential activity, which allows the bidirectional coupling to be biased toward the next action in cases of success (residual link from effect to action: dashed blue line) or to initiate a remedial action after an error (plain vermilion line). In OCD, the hyperactive and uniform (virtually identical for success and error) monitoring signals are no longer capable of biasing this bidirectional link, resulting in an "endless" circular-reflex process (merged vermilion and blue lines). The metacognitive component of the pathological circular reflex is represented in the comic strip bubbles

The neural basis of the ideomotor mechanism

At the neural level, neuroimaging studies have revealed that the ideomotor mechanism is largely distributed among the premotor cortex, inferior-parietal cortex, parahippocampal gyrus, caudate nucleus, angular gyrus, and supplementary motor area (Elsner et al. 2002; Melcher et al. 2008, 2013; Pfister et al. 2014; Badets and Osiurak 2017). Depending on the few studies investigating the neuronal bases of ideomotor mechanisms, different brain regions were alternatively proposed. For example, in a study aimed at finding brain regions involved in action–effect associative learning, the caudal supplementary motor area and the right hippocampus are found to play a role in linking auditory consequences of an action and the action itself (Elsner et al. 2002). In another ideomotor paradigm, based on the response-effect compatibility effect, the inferior parietal cortex and the parahippocampal gyrus were found to be key regions for this type of action control (Pfister et al. 2014). Hence, it seems that brain regions associated in the ideomotor mechanism are not systematically identified in imaging experiments, likely because the analysis attempted to find a contrast between different conditions that could nevertheless engage similar activity. Critically, and likely because of the choice of the experimental protocol, previous studies investigating ideomotor action control did not consider a potential engagement of the aMCC. In the present article, however, we propose experimental data that we believe are in favor of a major and, until now, unreported role of the aMCC in such a mechanism. More precisely, we will present examples of neuronal involvement of aMCC in four selected topics (action, evaluation, action-evaluation interplay and echophenomena/imitation) along with an examination of their pathological implications that directly provide a firm experimental basis for our hypothesis. We consequently propose that the properties of neurons in the aMCC allow this brain region to play a key role in linking actions and their

perceptual consequences. Incidentally, we hypothesized that hyperactivity of the aMCC leads to a reverberant pathological interaction between idea and action, as observed in OCD and other related disorders.

Action

The aMCC (Vogt 2009, 2016) is primarily considered to be a premotor area (Picard and Strick 1996; Dum et al. 2009) because of its direct anatomical connections with the primary motor cortex and the spinal cord and on the basis of stimulation and lesion studies that have confirmed its motor function; that is, electrical stimulation of the aMCC evokes simple and complex movements (Chassagnon et al. 2008), mimicking natural behavioral actions (Bancaud et al. 1976). In a recent study, Parvizi and colleagues showed that electrical stimulation of the aMCC can induce the expectation of an "imminent challenge" coupled with a determined attitude to overcome it (Parvizi et al. 2013) (Fig. 1), while earlier data reported an "urge-to-move" feeling and compulsive goal-directed behavior (Kremer et al. 2001) mimicking OCD symptomatology (Chassagnon et al. 2008) (Fig. 1). On the other hand, numerous observations from accidental stroke or surgical ablation have indicated that a lesion of the aMCC results in a reduction in spontaneous voluntary movements (akinesia) (Devinsky et al. 1995) or in behavioral error correction (Swick and Turken 2002) (see Fig. 1), whereas the motor capability per se is not impaired. These specific impairments of volitional movements thus provide a clear argument for an important role played by the aMCC in the transition between an idea (intention) and movement, that is, the ideomotor mechanism. Neuroimaging studies and neuronal recordings in humans and monkeys have also provided direct substantiating evidence that the aMCC is involved in motor execution and plays a major role in voluntary initiated action (Deiber et al. 1999; Williams et al. 2004; Hoshi et al. 2005) or intentional adaptive behavior such as task switching (Rushworth et al. 2002) (Fig. 1).

Critically, for the present article, motor neuronal responses occurring in the aMCC during complex tasks in animals are not finely tuned with detailed movement parameters (Hoshi et al. 2005; Michelet et al. 2016), indicating that such responses are associated more with an abstract intention to act or produce a preparatory signal than to define the specific parameters that are encoded in this region. This idea is the core interpretation of ideomotor theories about the idea of expected perceptual effects (Shin et al. 2010) and was confirmed by a functional magnetic resonance imaging (fMRI) study in which both execution and motor imagery were found to activate the aMCC (Hanakawa et al. 2008) (Fig. 1). In summary, the findings of these studies of aMCC neuronal activities concur with the ideomotor mechanism, suggesting that voluntary planning and initiation of an action rely more on the anticipation of the outcome of the action within the environment, rather than on the parametric specification of the actual motor action (e.g., see (Osiurak and Badets 2016) for this dissociation in tool use behavior), as could be the case in the motor and premotor cortices.

Evaluation

In a recent theoretical formulation of the ideomotor mechanism, Kunde et al. (2017) emphasized the role played by performance monitoring and proposed that an intended action could be coded in terms of the transition between two perceptual expectations: an ongoing and an intended one. This proposal explains how the "circular reflex" (which would result in an endless repetition of the same action; but see below) is circumvented because in an absence of discrepancy between the two expectations, there is no reason to repeatedly retrieve that action. While often considered a premotor area, the aMCC has most frequently been proposed to serve as a neural system for performance monitoring, a function required for evaluating the need for cognitive control in "nonhabitual" situations (Ullsperger et al. 2014). The principal neural signature of performance monitoring is the so-called error-related negativity (ERN), a sharp deflection in the event-related brain potential (ERP) that is considered mainly to occur in humans just after a person has committed an error or after negative feedback perception (feedback ERN or feedback-related negativity (FRN)) (Gehring et al. 1990; Falkenstein et al. 1995; Miltner et al. 1997). Convergent data from dipole analysis of human brain ERP signals, electrophysiological data from nonhuman primates, and human neuroimaging studies have confirmed that the aMCC is the primary generator of the ERN (Dehaene et al. 1994; Carter et al. 1998) and is also involved in subsequent behavioral error correction (Fiehler et al. 2004). Moreover, it has consistently been proposed that this brain region (Fig. 1) is also activated during correct response evaluation (Oliveira et al. 2007; Hoffmann and Falkenstein 2010; Roger et al. 2010). ERN has been recorded in both actors and observers during correct and erroneous performances (Van Schie et al. 2004; Weller et al. 2018). These data have thus strongly suggested that similar neural mechanisms are involved in monitoring one's own actions and the actions of others (Van Schie et al. 2004). Along with recordings of observational ERN, the findings of these studies further comply with previous behavioral results (see above), in which posterror slowing was also found in observers (Schuch and Tipper 2007). Hence, as postulated by ideomotor theories (Shin et al. 2010), a specific motor behavior can be triggered by the mere observation of a performance outcome, suggesting that any neuronal activity associated with a representation of an outcome will favor or prime the production of a motor command.

In almost all studies, monitoring-related aMCC activity has been found to be greater during erroneous trials than during correct trials, indicating a differential coding between success and error. This is supported by the observation in both humans (Roger et al. 2010) (Fig. 1C) and nonhuman primates (Michelet et al. 2007) (Fig. 1B) that negative neuronal-related feedback activities often have greater amplitudes than positive ones, even when encoded by the same neurons. Furthermore, it has been shown that these negative neuronal-related feedback activities are involved in triggering adaptive remedial behavior (Michelet et al. 2009). It is therefore likely that in normal subjects, the differential encoding of positive and negative feedback by the same aMCC neurons contributes to this terminating process and allows for the planning of another action in cases of success or allows for behavioral adaptation in cases of error (Fig. 2). A deregulation of this monitoring function is consistently found in OCD patients, associated with an overactivation of the aMCC during errors but also, interestingly, when trials were correctly completed (false error signals) (Maltby et al. 2005) (see Fig. 1). This undifferentiated and augmented neuronal activity is likely to cause a misinterpretation of the monitoring signals and prime a remedial adaptive behavior, whether the latter is pertinent or not. The initiation of checking behavior is no longer modulated by previous trial performance in OCD patients (Rotgé et al. 2015), indicating that this highly adaptive behavior in normal subjects (both humans (Rotgé et al. 2015) and monkeys (Bosc et al. 2017)) is likely to be altered by a dysfunction of the performance monitoring system. Consistent with this hypothesis, it has been shown that aMCC lesions result in a loss or decrease in error-corrective behavior (associated with a decrease in the ERN amplitude), confirming a monitoring impairment since the closed loop relationship between error detection and correction is abolished after a lesion develops (Swick and Turken 2002) (see Fig. 1).

Action evaluation

One of the most recent experimental findings that could be linked to the ideomotor mechanism is derived from a study by Procyk et al. (2014), who demonstrated that feedback-related activity (i.e., the expected perceptual effect) is generated by precisely the same aMCC area involved in motor command. A clear relationship was found between the modality of feedback processing and the specificity of the motor command; in their experiments on both nonhuman and human primates, the orofacial field involved in the motor control of facial expression was also activated by juice rewards representing the feedback information (i.e., the expected perceptual effect) relevant to the behavioral task. For example, the authors demonstrated a clear overlap of activation peaks in voluntary movements of the tongue and outcome-related activity (in this case, licking a liquid). They proposed that this finding could be extended to other types of feedback modalities; for instance, "tactile feedback on the hand or feedback-related to arm movement itself might be expected to involve the forelimb representation of the same brain region". This interpretation is consistent with the view that the ideomotor mechanism could also be strongly in charge of body-related action effects (Pfister 2019). This is also consistent with previous findings indicating that in a manual task, neuronal activation in the aMCC is driven by the combination of deciding on a choice and assessing the consequence of that choice (Walton et al. 2004) (but see Fig. 1 for the peak activation location that is very close to those reported in Amiez et al. 2013 and Amiez and Petrides 2014). This suggests that the aMCC involvement in motor execution and feedback processing is organized in a modality-specific manner. This is a critical condition for directly linking motor intention (or volition) with performance monitoring and consequently agrees well with the ideomotor principle. Indeed, as stated by Greenwald, "the notion of an idea or image of a response is conceived as a central-nervous-system representation of various modalities of sensory feedback from the response" (Greenwald 1970).

Echophenomena and imitation

Given that a bidirectional link between an expected perceptual effect and action is at the core of the ideomotor mechanism, it is probable that a "vicious cycle" could exist, which theorists have termed the "circular reflex" (Greenwald 1970) (see Fig. 2 and text above), a term that emphasizes the involuntary counterpart of a mechanism normally dedicated to voluntary movement control. Consequently, the establishment of an "endless" reverberant loop between the idea of an expected effect and action is highly probable and must be controlled. Though the circular reflex has sometimes been considered a limitation to ideomotor theory (Kunde et al. 2017), several diseases share many phenomenological traits that could be directly associated with the circular reflex hypothesis of the ideomotor mechanism. Up to 30% of individuals with OCD also have a lifetime tic disorder (American-Psychiatric-Association 2013) that can be echophenomena or a tic-like imitation of someone else's action (echopraxia) or sounds (echolalia), respectively, triggered by the mere perception of visual or auditory stimuli. This also includes palilalia since patients can repeat their own sounds or words. Here, again, the critical role played by the aMCC has been evaluated by electrical stimulation studies revealing the preserved behavior of repeating the same syllable after electrical stimulation (Chassagnon et al. 2008) (see Fig. 1). This is in line with a few studies reporting activity within the aMCC during observation and imitation (but see Rizzolatti et al. (2009) for a proposed role of the cingulate cortex in

the mirror mechanism). Twenty years ago, Grezes detected activity in the aMCC during the observation of hand movements in video-film scenes that later must be imitated but did not detect activity when observation was performed without any purpose (Grèzes 1998). In this respect, it is noticeable that OCD patients seem to be impaired in meaningless gesture imitation tasks (Rounis et al. 2016). In a recent study investigating imitation and observational learning of hand actions, Higuchi (Higuchi et al. 2012) found a very specific activation of the aMCC during the observation of a set of stimuli previously used during an observational practice session (i.e., learning by the observation of an action in the absence of motor execution) and during action imitation. A very recent report shed some light on the role of this brain region, emphasizing its importance for the control process over automatic imitation following action observation (Campbell et al. 2018) (Fig. 1). The cortical activation that was found in these studies was anatomically localized to the aMCC; in addition, other activations involved in internal and external error feedback processing (Holroyd et al. 2004; Debener et al. 2005), action-dependent feedback-related activations described earlier (Amiez and Petrides 2014) (Fig. 1), and both motor execution and motor imagery (Hanakawa et al. 2008) were noted. The aMCC is therefore a well-documented brain region in which a bidirectional interaction between action and expected perceptual effect occurs. From this perspective, a recent experiment (Brandt et al. 2015) clearly indicated that the visual observation of one's own tics increases tic frequency in patients with Tourette syndrome, a pathology that frequently occurs in comorbid expression with OCD. In favor of an ideomotor mechanism for such phenomena is the finding that imitations are mainly related to the goal of the action and not the specific movements actually realized by the observed subject (Flanagan and Johansson 2003).

Discussion and conclusion

The present paper analyses and merges various and, until now, unrelated experimental findings with the ideomotor mechanism to address, at least to some extent, how the aMCC is involved in the control of voluntary movements from normal to pathological circumstances. Our partial review reveals that the aMCC is a source of neuronal activities that are consistent with an ideomotor explanation and whose dysfunction could underlie some pathogenic processes, for example, when considering the cyclic pattern of obsession and compulsion at the core of OCD (Fig. 2) but also other repetitive behaviors. Indeed, strong evidence exists that the need to perform repetitive behaviors in both Tourette syndrome and OCD are associated with visual, tactile, and auditory stimuli (Prado et al. 2008), and some authors emphasized the idea of a phenomenological continuum between tics and OCD (Ferrão et al. 2013). However, it seems that along this tic-compulsion spectrum, OCD is more concerned with stimuli that evokes potential threats and are known to consistently activate the aMCC (Shackman et al. 2011). One of the most striking examples derives from symptom provocation experiments in which obsessional stimuli known to generate OCD symptoms are presented to patients. Consistent with ideomotor theory postulating that any processed expected feedback resulting from an action can be formulated as an anticipatory image for re-initiating this action (Greenwald 1970; Shin et al. 2010), the behavioral response in this type of paradigm is thus triggered by the sole perception of sensory stimuli. In a recent report, Banca and colleagues (Banca et al. 2015) evaluated the response rate of a rejection behavior when patients were exposed to individual provocation stimuli either with their hands (for example, a patient with biological contamination obsessions and washing compulsions would touch different stimuli, such as clean, potentially contaminated or believed to be contaminated gloves) or via online videos in real time from the patient's home. Either distant or proximate stimulation generates a rejection behavior. This is also consistent with self-reported accounts by OCD patients of some urges to change, rearrange, or move something in response to the experimental stimuli (Coles et al. 2005).

Obviously, other structures in the brain are involved in this complex pathology, and we do not advocate that the whole complexity of OCD is completely accounted for by aMCC dysfunction. Rather, our proposal could bridge existing gaps in the main classical hypotheses. For example, according to Graybiel, habits are acquired via experience-dependent plasticity, and she proposes that the cortico-basal ganglia circuits that normally mediate habits and automated behaviors are dysfunctional in OCD and become hyperactive or inaccessible to a stop signal (Graybiel and Rauch 2000). Our proposal is that the ideomotor circular reflex can induce repetitive behavior that could feed of and in turn be reinforced by this plasticity-dependent habit-forming process. Hence, it can become virtually uncontrollable and escalates into obsessive–compulsive behavior.

Because the ideomotor model emphasizes the role of the aMCC in an ultrashort and simple closed-feedback loop (needed for rapid adaptive behavior initiation that is not exclusive to top-down attentional and cognitive control after error or conflict (Kerns et al. 2004)), it helps to disentangle the role of awareness in aMCC-dependent behavioral adaptation. For example, the role of consciousness in error (Nieuwenhuis et al. 2001) or conflict detection (Dehaene et al. 2003) and the dependence of aMCC-related activity and their correlated remedial behaviors (Mayr 2004) have often been questioned. The ideomotor circular reflex hypothesis helps one to avoid making a causal distinction

and is consistent with earlier reports suggesting that the cingulate motor functions may be largely under the direction of "unconscious" mental processes (Talairach et al. 1973; Devinsky and Luciano 1993). Indeed, any activation of the aMCC (during sensory processing of a response effect, for example) is likely associated with changes in bodily states that can be subjectively and subsequently perceived as a conscious representation of the error/conflict situation. This hypothesis is in line with the role of the aMCC in the control of autonomic arousal states because of its anatomical connections (Critchley et al. 2003). Indeed, it projects to regions involved in autonomic control, and the aMCC has been clearly shown to be involved in autonomic control (Fig. 1) during both cognitive processing and motor behavior (Critchley et al. 2003). Thus, awareness of changes in bodily states following aMCC activation might explain the urges (and subsequent compulsions) some patients associate with the feeling of "something not being just right" or to the sensations of "incompleteness" (Coles et al. 2005). This hypothesis is also supported by the observation of heart rate acceleration following aMCC stimulation (Parvizi et al. 2013) (Fig. 1) and relates to the idea that (post hoc) rationalization of an action often follows the arrival of somatic and visceral information (Damasio 1996). Here, conscious control should be considered a consequence rather than a primary instigator (Gillan and Robbins 2014). The fact that OCD patients often acknowledge the senselessness of obsessions or describe these ideas as unwanted and extraneous intrusions into consciousness (Schwartz 1999) confirms the "automaticity" of this representation that directly emerges from the bidirectional link between an action and the associated expected perceptual effect. The conscious experience of intending to act (or the urge to move) could in principle originate from any neuronal activity involved in movement preparation, such as that expressed by aMCC neurons (Hoshi et al. 2005). This concept is highly consistent with the previously described role of the aMCC in the awareness of self and intention (Devinsky et al. 1995; Vogt and Devinsky 2000), the impairment of which is then likely to be associated with the presence of obsession, compulsion or both. We could thereby consider the feeling that "something is wrong" and the "urge to move" as the metacognitive representations of the ideomotor mechanism, with the former being related to the hypersignal from the monitoring function, whereas the latter corresponds to the automatic activation of the motor command (Fig. 2).

Our proposal is also notably based on the fact that aMCC neurons (see Fig. 1) are similarly involved in hand-related motor behaviors and in hand-related compulsive checking behavior (Mataix-Cols et al. 2004) (e.g., washing). This supports the idea that this very common symptom could originate in part from actions "too easily" triggered by cognitive representations of their expected perceptual effects. We believe that the aMCC dysfunction found in these pathological conditions provides good support in favor of this proposal since we predict that an alteration of the ideomotor mechanism could provide a probable starting point for the transition from a normal condition to a pathological condition.

To conclude, our analysis reveals that the aMCC is a constant and reliable locus for neuronal activities that can be interpreted as providing an important nervous substrate for the ideomotor mechanism. Thus, we propose that the aMCC is a key brain region for ideomotor-related processing, the dysfunction of which results in a pathological reverberant interaction between idea and action, as observed in OCD and other related disorders.

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References

American-Psychiatric-Association (2013) Diagnostic and statistical manual of mental disorders, 5th edn. American Psychiatric Association, Arlington

Amiez C, Petrides M (2014) Neuroimaging evidence of the anatomo-functional organization of the human cingulate motor areas. Cereb Cortex 24:563–578

Amiez C, Neveu R, Warrot D, Petrides M, Knoblauch K, Procyk E (2013) The location of feedback-related activity in the midcingulate cortex is predicted by local morphology. J Neurosci 33:2217–2228

Badets A, Osiurak F (2015) A goal-based mechanism for delayed motor intention: considerations from motor skills, tool use and action memory. Psychol Res 79:345–360. https://doi.org/10.1007/s00426-014-0581-5

Badets A, Osiurak F (2017) The ideomotor recycling theory for tool use, language, and foresight. Exp Brain Res 235:365–377. https://doi.org/10.1007/s00221-016-4812-4

Badets A, Koch I, Toussaint L (2013) Role of an ideomotor mechanism in number processing. Exp Psychol 60:34–43. https://doi.org/10.1027/1618-3169/a000171

Badets A, Koch I, Philipp AM (2016) A review of ideomotor approaches to perception, cognition, action, and language: advancing a cultural recycling hypothesis. Psychol Res 80:1–15. https://doi.org/10.1007/s00426-014-0643-8

Banca P, Voon V, Vestergaard MD et al (2015) Imbalance in habitual versus goal directed neural systems during symptom provocation in obsessive-compulsive disorder. Brain 138:798–811. https://doi.org/10.1093/brain/awu379

Bancaud J, Talairach J, Geier S, Bonis A, Trottier S, Manrique M (1976) Behavioral manifestations induced by electric stimulation of the anterior cingulate gyrus in man. Rev Neurol 132:705–724

Band GPH, Van Steenbergen H, Ridderinkhof KR, Falkenstein M, Hommel B (2009) Action-effect negativity: irrelevant action effects are monitored like relevant feedback. Biol Psychol 82:211–218. https://doi.org/10.1016/j.biopsycho.2009.06.011

Bosc M, Bioulac B, Langbour N et al (2017) Checking behavior in rhesus monkeys is related to anxiety and frontal activity. Sci Rep 7:45267. https://doi.org/10.1038/srep45267

Brandt VC, Lynn MT, Obst M, Brass M, Münchau A (2015) Visual feedback of own tics increases tic frequency in patients with Tourette's syndrome. Cogn Neurosci 6:1–7. https://doi.org/10.1080/17588928.2014.954990

Campbell MEJ, Mehrkanoon S, Cunnington R (2018) Intentionally not imitating: Insula cortex engaged for top-down control of action mirroring. Neuropsychologia 111:241–251. https://doi.org/10.1016/j.neuropsychologia.2018.01.037

Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD (1998) Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280:747–749

Chassagnon S, Minotti L, Kremer S, Hoffmann D, Kahane P (2008) Somatosensory, motor, and reaching/grasping responses to direct electrical stimulation of the human cingulate motor areas. J Neurosurg 109:593–604. https://doi.org/10.3171/JNS/2008/109/10/0593

Coles ME, Heimberg RG, Frost RO, Steketee G (2005) Not just right experiences and obsessive-compulsive features: experimental and self-monitoring perspectives. Behav Res Ther 43:153–167. https://doi.org/10.1016/j.brat.2004.01.002

Colton J, Bach P, Whalley B, Mitchell C (2018) Intention insertion: activating an action's perceptual consequences is sufficient to induce non-willed motor behavior. J Exp Psychol Gen 147:1256–1263. https://doi.org/10.1037/xge0000435

Critchley HD, Mathias CJ, Josephs O et al (2003) Human cingulate cortex and autonomic control: converging neuroimaging and clinical evidence. Brain 126:2139–2152. https://doi.org/10.1093/brain/awg216

Damasio AR (1996) The somatic marker hypothesis and the possible functions of the prefrontal cortex. Philos Trans R Soc Lond B 351:1413–1420. https://doi.org/10.1098/rstb.1996.0125

Debener S, Ullsperger M, Siegel M, Fiehler K, von Cramon DY, Engel AK (2005) Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. J Neurosci 25:11730–11737

Dehaene S, Posner MI, Tucker DM (1994) Localization of a neural system for error detection and compensation. Psychol Sci 5:303–305

Dehaene S, Artiges E, Naccache L et al (2003) Conscious and subliminal conflicts in normal subjects and patients with schizophrenia: the role of the anterior cingulate. Proc Natl Acad Sci 100:13722–13727. https://doi.org/10.1073/pnas.2235214100

Deiber MP, Honda M, Ibañez V, Sadato N, Hallett M (1999) Mesial motor areas in self-initiated versus externally triggered movements examined with fMRI: effect of movement type and rate. J Neurophysiol 81:3065–3077. https://doi.org/10.1152/jn.1999.81.6.3065

Devinsky O, Luciano D (1993) The contributions of cingulate cortex to human behavior. In: Vogt BABA, Gabriel M (eds) Neurobiology of cingulate cortex and limbic thalamus: a comprehensive handbook. Birkhäuser, Boston, pp 527–556

Devinsky O, Morrell MJ, Vogt BA (1995) Contributions of anterior cingulate cortex to behaviour. Brain 118:279–306

Dogge M, Custers R, Aarts H (2019) Moving forward: on the limits of motor-based forward models. Trends Cogn Sci 23:743–753. https://doi.org/10.1016/j.tics.2019.06.008

Dum RP, Levinthal DJ, Strick PL (2009) The spinothalamic system targets motor and sensory areas in the cerebral cortex of monkeys. J Neurosci 29:14223–14235. https://doi.org/10.1523/JNEUROSCI.3398-09.2009

Elsner B, Hommel B (2001) Effect anticipation and action control. J Exp Psychol Hum Percept Perform 27:229–240

Elsner B, Hommel B, Mentschel C, Drzezga A, Prinz W, Conrad B, Siebner H (2002) Linking actions and their perceivable consequences in the human brain. Neuroimage 17:364–372. https://doi.org/10.1006/nimg.2002.1162

Falkenstein M, Hohnsbein J, Hoormann J (1995) Event-related potential correlates of errors in reaction tasks. Electroencephalogr Clin Neurophysiol Suppl 44:287–296

Fan J, Hof P, Guise K, Fossella J, Posner M (2007) The functional integration of the anterior cingulate cortex during conflict processing. Cereb Cortex. https://doi.org/10.1093/cercor/bhm125

Ferrão Y, Pedro A, Hounie A, Mathis M, RosáRio M, Miguel E (2013) The phenomenology of obsessive-compulsive symptoms in Tourette syndrome. In: Martino D, Leckman JF (eds) Tourette syndrome, vol 692. Oxford University Press, New York, pp 50–73

Fiehler K, Ullsperger M, von Cramon DY (2004) Neural correlates of error detection and error correction: is there a common neuroanatomical substrate? Eur J Neurosci 19:3081–3087

Flach R, Press C, Badets A, Heyes C (2010) Shaking hands: priming by social action effects. Br J Psychol 101:739–749. https://doi.org/10.1348/000712609X484595

Flanagan JR, Johansson RS (2003) Action plans used in action observation. Nature 424:769–771. https://doi.org/10.1038/nature01861

Gehring WJ, Coles MG, Meyer DE, Donchin E (1990) The error-related negativity: An event related potential accompanying errors. Psychophysiology 27:S34

Gillan CM, Robbins TW (2014) Goal-directed learning and obsessive-compulsive disorder. Philos Trans R Soc Lond B. https://doi.org/10.1098/rstb.2013.0475

Graybiel AM, Rauch SL (2000) Toward a neurobiology of obsessive-compulsive disorder. Neuron 28:343–347

Greenwald AG (1970) Sensory feedback mechanisms in performance control: with special reference to the ideo-motor mechanism. Psychol Rev 77:73–99

Grèzes J (1998) Top down effect of strategy on the perception of human biological motion: a pet investigation. Cogn Neuropsychol 15:553–582. https://doi.org/10.1080/026432998381023

Hanakawa T, Dimyan MA, Hallett M (2008) Motor planning, imagery, and execution in the distributed motor network: a time-course study with functional MRI. Cereb Cortex. https://doi.org/10.1093/cercor/bhn036

Heyes C (2001) Causes and consequences of imitation. Trends Cogn Sci 5:253–261

Higuchi S, Holle H, Roberts N, Eickhoff SB, Vogt S (2012) Imitation and observational learning of hand actions: prefrontal involvement and connectivity. Neuroimage 59:1668–1683. https://doi.org/10.1016/j.neuroimage.2011.09.021

Hoffmann S, Falkenstein M (2010) Independent component analysis of erroneous and correct responses suggests online response control. Hum Brain Mapp 31:1305–1315. https://doi.org/10.1002/hbm.20937

Hoffstaedter F, Grefkes C, Caspers S et al (2014) The role of anterior midcingulate cortex in cognitive motor control: evidence from functional connectivity analyses. Hum Brain Mapp 35:2741–2753. https://doi.org/10.1002/hbm.22363

Holroyd CB, Nieuwenhuis S, Yeung N, Nystrom L, Mars RB, Coles MG, Cohen JD (2004) Dorsal anterior cingulate cortex shows fMRI response to internal and external error signals. Nat Neurosci 7:497–498

Hommel B, Müsseler J, Aschersleben G, Prinz W (2001) The theory of event coding (TEC): a framework for perception and action planning. Behav Brain Sci 24:849–878

Hoshi E, Sawamura H, Tanji J (2005) Neurons in the rostral cingulate motor area monitor multiple phases of visuomotor behavior with modest parametric selectivity. J Neurophysiol 94:640–656

Kerns JG, Cohen JD, MacDonald AW 3rd, Cho RY, Stenger VA, Carter CS (2004) Anterior cingulate conflict monitoring and adjustments in control. Science 303:1023–1026

Knoblich G, Prinz W (2005) Linking perception and action: an ideomotor approach. In: Freund H, Jeannerod M, Hallett M, Leiguarda R (eds) Higher-order motor disorders: from neuroanatomy and

neurobiology to clinical neurology. Oxford University Press, Oxford, pp 79–104
Koch I, Hoffmann J (2000) The role of stimulus-based and response-based spatial information in sequence learning. J Exp Psychol Learn Mem Cogn 26:863–882. https://doi.org/10.1037//0278-7393.26.4.863
Koch I, Kunde W (2002) Verbal response-effect compatibility. Memory Cognition 30:1297–1303
Kremer S, Chassagnon S, Hoffmann D, Benabid AL, Kahane P (2001) The cingulate hidden hand. J Neurol Neurosurg Psychiatry 70:264–265. https://doi.org/10.1136/jnnp.70.2.264
Kunde W (2001) Response-effect compatibility in manual choice reaction tasks. J Exp Psychol Hum Percept Perform 27:387–394. https://doi.org/10.1037//OO96-1523.27.2.387
Kunde W (2004) Response priming by supraliminal and subliminal action effects. Psychol Res 68:91–96. https://doi.org/10.1007/s00426-003-0147-4
Kunde W, Koch I, Hoffmann J (2004) Anticipated action effects affect the selection, initiation, and execution of actions. Q J Exp Psychol Sec A 57:87–106. https://doi.org/10.1080/02724980343000143
Kunde W, Schmidts C, Wirth R, Herbort O (2017) Action effects are coded as transitions from current to future stimulation: Evidence from compatibility effects in tracking. J Exp Psychol Hum Percept Perform 43:477–486. https://doi.org/10.1037/xhp0000311
Lancaster JL, Tordesillas-Gutiérrez D, Martinez M et al (2007) Bias between MNI and Talairach coordinates analyzed using the ICBM-152 brain template. Hum Brain Mapp 28:1194–1205. https://doi.org/10.1002/hbm.20345
Maltby N, Tolin DF, Worhunsky P, O'Keefe TM, Kiehl KA (2005) Dysfunctional action monitoring hyperactivates frontal-striatal circuits in obsessive-compulsive disorder: an event-related fMRI study. Neuroimage 24:495–503
Mataix-Cols D, Wooderson S, Lawrence N, Brammer MJ, Speckens A, Phillips ML (2004) Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive-compulsive disorder. Arch Gen Psychiatry 61:564–576
Mayr U (2004) Conflict, consciousness, and control. Trends Cogn Sci 8:145–148. https://doi.org/10.1016/j.tics.2004.02.006
Melcher T, Weidema M, Eenshuistra RM, Hommel B, Gruber O (2008) The neural substrate of the ideomotor principle: an event-related fMRI analysis. Neuroimage 39:1274–1288. https://doi.org/10.1016/j.neuroimage.2007.09.049
Melcher T, Winter D, Hommel B, Pfister R, Dechent P, Gruber O (2013) The neural substrate of the ideomotor principle revisited: evidence for asymmetries in action-effect learning. Neuroscience 231:13–27. https://doi.org/10.1016/j.neuroscience.2012.11.035
Michelet T, Bioulac B, Guehl D, Escola L, Burbaud P (2007) Impact of commitment on performance evaluation in the rostral cingulate motor area. J Neurosci 27:7482–7489
Michelet T, Bioulac B, Guehl D, Goillandeau M, Burbaud P (2009) Single medial prefrontal neurons cope with error. PLoS ONE 4:e6240. https://doi.org/10.1371/journal.pone.0006240
Michelet T, Bioulac B, Langbour N, Goillandeau M, Guehl D, Burbaud P (2016) Electrophysiological correlates of a versatile executive control system in the monkey anterior cingulate cortex. Cereb Cortex 26:1684–1697. https://doi.org/10.1093/cercor/bhv004
Miltner W, Braun C, Coles MG (1997) Event-related brain potentials following incorrect feedback in a time-estimation task: evidence for a "generic" neural system for error detection. J Cogn Neurosci 9:788–798
Moschl M, Fischer R, Bugg JM, Scullin MK, Goschke T, Walser M (2020) Aftereffects and deactivation of completed prospective memory intentions: a systematic review. Psychol Bull 146:245–278. https://doi.org/10.1037/bul0000221
Nattkemper D, Ziessler M (2004) Cognitive control of action: the role of action effects. Psychol Res 68:71–73. https://doi.org/10.1007/s00426-003-0145-6
Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GP, Kok A (2001) Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. Psychophysiology 38:752–760
Oliveira FTP, McDonald JJ, Goodman D (2007) Performance monitoring in the anterior cingulate is not all error related: expectancy deviation and the representation of action-outcome associations. J Cogn Neurosci 19:1994–2004. https://doi.org/10.1162/jocn.2007.19.12.1994
Osiurak F, Badets A (2016) Tool use and affordance: manipulation-based versus reasoning-based approaches. Psychol Rev 123:534–568. https://doi.org/10.1037/rev0000027
Parvizi J, Rangarajan V, Shirer WR, Desai N, Greicius MD (2013) The will to persevere induced by electrical stimulation of the human cingulate gyrus. Neuron 80:1359–1367. https://doi.org/10.1016/j.neuron.2013.10.057
Pfister R (2019) Effect-based action control with body-related effects: implications for empirical approaches to ideomotor action control. Psychol Rev 126:153–161. https://doi.org/10.1037/rev0000140
Pfister R, Melcher T, Kiesel A, Dechent P, Gruber O (2014) Neural correlates of ideomotor effect anticipations. Neuroscience 259:164–171. https://doi.org/10.1016/j.neuroscience.2013.11.061
Picard N, Strick PL (1996) Motor areas of the medial wall: a review of their location and functional activation. Cereb Cortex 6:342–353
Prado HS, Rosário MC, Lee J, Hounie AG, Shavitt RG, Miguel EC (2008) Sensory phenomena in obsessive-compulsive disorder and tic disorders: a review of the literature. CNS Spectr 13:425–432. https://doi.org/10.1017/s1092852900016606
Prinz W (1997) Perception and action planning. Eur J Cogn Psychol 2:1–26
Procyk E, Wilson CRE, Stoll FM, Faraut MCM, Petrides M, Amiez C (2014) Midcingulate motor map and feedback detection: converging data from humans and monkeys. Cereb Cortex. https://doi.org/10.1093/cercor/bhu213
Rabbitt PM (1966) Errors and error correction in choice-response tasks. J Exp Psychol 71:264–272
Rabbitt P, Rodgers B (1977) What does a man do after he makes an error? An analysis of response programming. Q J Exp Psychol 29:727–743. https://doi.org/10.1080/14640747708400645
Rizzolatti G, Craighero L (2004) The mirror-neuron system. Annu Rev Neurosci 27:169–192. https://doi.org/10.1146/annurev.neuro.27.070203.144230
Rizzolatti G, Fabbri-Destro M, Cattaneo L (2009) Mirror neurons and their clinical relevance. Nat Clin Pract Neurol 5:24–34. https://doi.org/10.1038/ncpneuro0990
Roger C, Bénar CG, Vidal F, Hasbroucq T, Burle B (2010) Rostral cingulate zone and correct response monitoring: ICA and source localization evidences for the unicity of correct- and error-negativities. Neuroimage 51:391–403. https://doi.org/10.1016/j.neuroimage.2010.02.005
Rotgé J-Y, Langbour N, Dilharreguy B et al (2015) Contextual and behavioral influences on uncertainty in obsessive-compulsive disorder. Cortex 62:1–10. https://doi.org/10.1016/j.cortex.2012.12.010
Rounis E, Banca P, Voon V (2016) Deficits in limb praxis in patients with obsessive-compulsive disorder. J Neuropsychiatry Clin Neurosci 28:232–235. https://doi.org/10.1176/appi.neuropsych.15090233
Rushworth MF, Hadland KA, Paus T, Sipila PK (2002) Role of the human medial frontal cortex in task switching: a combined fMRI and TMS study. J Neurophysiol 87:2577–2592

Schuch S, Tipper SP (2007) On observing another person's actions: influences of observed inhibition and errors. Percept Psychophys 69:828–837

Schwartz JM (1999) A role for volition and attention in the generation of new brain circuitry. Toward a nerobiology of mental force. J Conscious Stud 6:115–142

Shackman AJ, Salomons TV, Slagter HA, Fox AS, Winter JJ, Davidson RJ (2011) The integration of negative affect, pain and cognitive control in the cingulate cortex. Nat Rev Neurosci 12:154–167. https://doi.org/10.1038/nrn2994

Shin YK, Proctor RW, Capaldi EJ (2010) A review of contemporary ideomotor theory. Psychol Bull 136:943–974. https://doi.org/10.1037/a0020541

Swick D, Turken AU (2002) Dissociation between conflict detection and error monitoring in the human anterior cingulate cortex. Proc Natl Acad Sci 99:16354–16359

Talairach J, Bancaud J, Geier S, Bordas-Ferrer M, Bonis A, Szikla G, Rusu M (1973) The cingulate gyrus and human behaviour. Electroencephalogr Clin Neurophysiol 34:45–52

Ullsperger M, Danielmeier C, Jocham G (2014) Neurophysiology of performance monitoring and adaptive behavior. Physiol Rev 94:35–79

Van Schie HT, Mars RB, Coles MG, Bekkering H (2004) Modulation of activity in medial frontal and motor cortices during error observation. Nat Neurosci 7:549–554

Vogt BA (2009) Regions and subregions of the cingulate cortex. In: Vogt BA (ed) Cingulate neurobiology and disease. Oxford University Press, London, pp 3–30

Vogt BA (2016) Midcingulate cortex: Structure, connections, homologies, functions and diseases. J Chem Neuroanat 74:28–46. https://doi.org/10.1016/j.jchemneu.2016.01.010

Vogt BA, Devinsky O (2000) Topography and relationships of mind and brain. Prog Brain Res 122:11–22. https://doi.org/10.1016/S0079-6123(08)62127-5

Walton ME, Devlin JT, Rushworth MFS (2004) Interactions between decision making and performance monitoring within prefrontal cortex. Nat Neurosci 7:1259–1265. https://doi.org/10.1038/nn1339

Weller L, Schwarz KA, Kunde W, Pfister R (2018) My mistake? Enhanced error processing for commanded compared to passively observed actions. Psychophysiology. https://doi.org/10.1111/psyp.13057

Williams ZM, Bush G, Rauch SL, Cosgrove GR, Eskandar EN (2004) Human anterior cingulate neurons and the integration of monetary reward with motor responses. Nat Neurosci 7:1370–1375

Wolpert DM, Ghahramani Z, Flanagan JR (2001) Perspectives and problems in motor learning. Trends Cogn Sci 5:487–494