Overview

Neurocognitive mechanisms of action control: resisting the call of the Sirens

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An essential facet of adaptive and versatile behavior is the ability to prioritize actions in response to dynamically changing circumstances. The field of potential actions afforded by a situation is shaped by many factors, such as environmental demands, past experiences, and prepotent tendencies. Selection among action affordances can be driven by deliberate, intentional processes as a product of goal-directed behavior and by extraneous stimulus–action associations as established inherently or through learning. We first review the neurocognitive mechanisms putatively linked to these intention-driven and association-driven routes of action selection. Next, we review the neurocognitive mechanisms engaged to inhibit action affordances that are no longer relevant or that interfere with goal-directed action selection. Optimal action control is viewed as a dynamic interplay between selection and suppression mechanisms, which is achieved by an elaborate circuitry of interconnected cortical regions (most prominently the pre-supplementary motor area and the right inferior frontal cortex) and basal ganglia structures (most prominently the dorsal striatum and the subthalamic nucleus). © 2010 John Wiley & Sons, Ltd. *WIREs Cogn Sci*

COGNITIVE CONTROL, ONLINE ACTION CONTROL, AND ANTICIPATORY ACTION REGULATION

Cognitive control refers descriptively to the capacity to orchestrate, coordinate, and direct basic cognitive processes and their temporal structure, in accordance with internal goals and or external demands, such as to optimize behavioral outcomes. Cognitive control functions should not necessarily be considered as basic mental functions, supported by specific dedicated systems or neural circuits; they are better conceived of as emergent properties, being established by the configuration and tailoring of existing subordinate processes in such a fashion that 'new', unique functions emerge. Traditionally, the operation of cognitive control processes has been considered to require intention and awareness.[1] For instance, voluntary action-selection processes as well as response inhibition processes have typically been considered as controlled processes that require intention and awareness for their instigation; however, this view has been challenged by recent evidence suggesting that these processes can be implemented unintentionally and unconsciously.[2,3] Perhaps due to its descriptive rather than mechanistic conceptualization, cognitive control has long remained an intractable concept despite its importance in a wide variety of situations.

Action control refers to a subset of cognitive control processes involved in the requirement to coordinate one's instantaneous urges *vis-à-vis* actions that concord with our intentions or instructions. Prudence in selecting the right course of action in response to fickle circumstances is not always straightforward, as illustrated by the quick transitions in credit providers' decision policies in granting loans:

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from happy-go-lucky policies in times of prosperity (when shareholders demand boosting profits) to over-conservative risk aversion (in times of economic crisis). One's current situation presents a field of action affordances (alluring and potentiating opportunities for action in a particular situation, some more potent than others[4]). However, our responsiveness to affordances is guided by our current concerns and intentions; we are not responsive to the full constellation of affordances, but primarily to *relevant* affordances. Goals, concerns, and prior experience have shaped our sensitivity to relevant affordances, such that one is not immediately captivated by the one action affordance that presents the most potent solicitation.

In the next sections, we will derive a taxonomy and nomenclature of processes that are central to adequate action control, and review evidence that unveils the neurocognitive mechanisms underlying it. In passing, we will explore individual differences in the ability to select the appropriate action from the field of affordances and to inhibit potent impulses if they are not appropriate to the situation at hand. In certain neurological conditions, such as utilization behavior,[5] patients respond to irrelevant affordances, which results in actions that are often inappropriate given social norms.[6] Similar behavior might follow from neuropsychiatric conditions, such as attention deficit/hyperactivity disorder or Parkinson's disease, and from alcohol intoxication or addiction.[7–10]

Action Control: A Taxonomy of Processes

It is important to distinguish online action control from the anticipatory processes that regulate them.[11,a] In the existing literature, these aspects of action control are often discussed interchangeably. However, as will become evident, anticipatory and online control processes can be dissociated in terms of underlying neural networks, temporal dynamics, and sensitivity to experimental manipulations as well as individual differences. *Online action control* is exerted to suppress and overcome incorrect, inappropriate, or undesirable actions in favor of intention-driven action selection.[12] Proficient traffic navigation, for instance, requires one to arrest conversation with a passenger when approaching a complex roundabout and to overrule the habit of driving on the right side of the road when navigating traffic in England. *Anticipatory action regulation* refers to those modulatory processes that either strengthen online action control proactively or preempt the need for such online action control.[13] If a traffic accident (e.g., resulting from an experienced tendency to drive on the right side of an English road) was barely avoided, anticipatory action regulation might lead one to tighten online action control to preempt further error. Anticipatory action regulation can be instigated by several kinds of processes that monitor external and internal signals that indicate the need to adjust behavior (reviewed elsewhere[14]). Online action control operates transiently, whereas anticipatory regulation operates in a more sustained fashion.

Online action control can involve a number of component processes: (1) prompting the activation of appropriate actions based on *intention-driven action selection*, (2) resisting the activation of inappropriate actions based on extraneous stimulus–action associations that are strong enough to incur *response capture*, and (3) suppressing the activation of inappropriate actions through *active response inhibition*. In this review, we will focus on online action control, leaving the proactive or preemptive anticipatory processes that modulate them to be reviewed in future work. Yet, in order to provide a context that facilitates a richer appreciation of online action control processes, we will briefly discuss anticipatory action regulation in the section below.

Anticipatory Regulation of Online Action Control

It may not always be possible to deploy online action control processes successfully to completely cancel out the effects of response capture. However, in establishing action control it may be possible to prepare for task-inappropriate action affordances and to mitigate their undesired effects. Thus, the expression of the online action control processes may be modulated by anticipatory adjustments of action-selection priorities. In participating in traffic, for instance, one's responsiveness to action affordances is subject to fluctuations as a function of warnings, changing situations, recent experiences (good or bad), the behavior of others, and so on. Such anticipatory processes can be described in terms of two orthogonal dimensions: regulation may be prospective or reactive in nature, and it may take on proactive or preemptive forms.

In many an instance, anticipatory regulation will be *reactive* in nature; that is, adjustments of online action control will be contingent upon performance errors or internal signals of performance difficulty, such as response conflicts. In other instances, anticipatory regulation will be more *prospective* in nature; for instance, one may slow down when anticipating busy traffic or make use of explicit cues or instructions to guide adjustments of processing priorities.

Whether prospective or reactive in nature, anticipatory action regulation can be accomplished through either proactive or preemptive adjustments. One may attempt to *proactively* strengthen online action control, for instance by *a priori* amplifying those processes that help keep our horses in check when strong response capture is anticipated. Alternatively, one may attempt to *preempt* the need for online action control, for instance by increasing the focus of selective attention to filter out task-irrelevant stimuli such that these fail to elicit strong response capture in the first place.

Although an extensive review of findings regarding the mechanisms of anticipatory regulation is beyond the scope of this article (see reviews elsewhere[15,16]), it is noteworthy that several dissociations between online action control and anticipatory regulation have been reported,[17,18] and that anticipatory action regulation appears to engage brain circuits that can be dissociated from those involved in implementing online action control.[19,20] In the following sections, we will elaborate on the neural architecture and the neurocognitive mechanisms underlying online processes of action control.

ONLINE PROCESSES OF ACTION CONTROL: AN OVERVIEW

Cognitive Mechanisms and Neural Bases of Online Action Control

Especially when appropriate actions compete for activation with strong alternatives, online action control may be needed to resist interference from these alternatives and ensure the timely and uninterrupted activation of the selected response.[21] A vast literature documents a proliferation of dual-process models that distinguish between *association-driven* and *intention-driven* processes (direct vs deliberative, bottom-up vs top-down, automatic vs controlled, habitual vs goal-directed, impulsive vs deliberate, reflexive vs reflective, involuntary vs voluntary, and the like) and that seek to describe the respective contributions of those processes to behavior.[22] Such models have been used to describe and predict various aspects of human behavior, including reasoning, emotion processing, decision making, social behaviors, and addiction.[10,23–26] Although these models differ in their details and applications, their common denominator entails an understanding of behavior in terms of the interplay between relatively automatic and relatively deliberative processes.

Dual-process models have been entertained extensively in the field of action control. Although dual-route models had been formulated previously,[27] Kornblum and colleagues have set the stage with their seminal dual-route model for perception–action coupling.[28] Their rudimentary dual-route architecture has been embraced by many authors in the field.[29] Basically, upon identification, a stimulus is thought to deliberately activate the correct response via a deliberate route and to captivate activation of other (correct or incorrect) responses via a more direct processing route; the two routes converge at the level of response activation processes. If the activated responses match, the motor program already activated via the direct route can be carried out quickly; if they mismatch, this motor program must be aborted in favor of the alternative motor program, whose retrieval and execution cost extra time. Thus, on the one hand, action selection can be driven by response capture from strong extraneous stimulus–response associations, automatically activated action stereotypes or biases, response habits, urges, impulses, and so on. On the other hand, deliberate intention-driven action selection requires that the action system is shielded from such response capture, and that the activation of inappropriate actions is actively inhibited.

Timing is everything. Upon encountering stimuli that present action affordances, action control processes will zero in on selection of the intention-guided action as time progresses. The implication is that during the early stages of processing, action selection is perhaps not yet perfectly intention guided, and hence more vulnerable to potent action affordances, even if these are solicited by task-irrelevant stimulus features. As one well-known result, responses that happen to be fast are more error prone than those that happen to be slow: in conflict tasks, many fast errors are elicited when stimuli contain task-irrelevant features that afford actions incongruent with the action designated by the target feature. Note, though, that these are gradual rather than all-or-none effects: during fast responses, as compared to slower responses, action selection is driven *relatively* more by task-irrelevant affordances than by deliberate target–response translation, and on average, fast responses are *relatively* more error prone for incongruent compared to congruent stimuli. This phenomenon, referred to as response capture, is what is typically shown in *conditional accuracy functions* (CAFs, see Figure 3); we return to these in a later section. If, for some reason, effortful intention-guided action selection is less effective, then this will be expressed mostly in greater susceptibility to potent action affordances as solicited by task-irrelevant stimulus features, especially during the early stages

of processing. It is as if initially the action-selection system is 'hijacked' by the response activation as triggered by the task-irrelevant action affordances.

Resisting the Call of the Sirens

Given that we have controlled action-selection processes at our avail, why then do we allow impulses and over-learned stimulus–response associations to solicit actions that escape deliberation? We are presented with all kinds of potent and compelling but inappropriate action affordances, rendering action control far from straightforward. See Figure 1 for a famous mythical example. If heeding to the appeal of the *Sirens'* call were so universally dangerous ('a promise of mantic truths, with a false promise of living to tell them'), what adaptive value then led it to survive evolutionary selection?

Obviously, there are benefits of having intention-driven action selection complemented with (or even bypassed by) association-driven response capture. When we imagine receiving the ball in a game of table tennis, it is in fact not too difficult to see the adaptive value of having our action-selection system hijacked by association-driven action impulses: any online deliberation on the preferred course of action would cause us to be way too slow to even return the ball, let alone to take over initiative and play the ball offensively. Our reflexes serve us well. Prior experience, skill, insight, and intentional plans may serve to adjust and optimize our prepared reflexes; but certainly in some situations it is best to rely on the action affordances presented by automatic or over-learned stimulus–action associations. In driving our cars or in playing rapid sports such as table tennis, we resemble Aristotle's *phronimos*,[30] the ethical expert who (based on prior experience) does not need to deliberate prior to acting, because he is moved to act selectively to the pertinent affordances that solicit the relevant action in that specific situation.

Whatever positive role action affordances might have in some situations, relying exclusively on them could lead to dramatic consequences. Heed to the song of the Sirens, and one might not live to tell. Evidently, we can silence competing affordances, as reviewed here: action control serves not only to single out the relevant action-inviting affordance, but also insulates the attractions of competing impulses (say a Siren's call) from generating actual urges to pursue them. This begs the prominent question of how action control is implemented in the brain.

The Neural Bases of Action Selection and Suppression: A Brief Introduction

Converging evidence from studies using electrophysiology, functional magnetic resonance imaging (fMRI), or repetitive transcranial magnetic stimulation (rTMS) and from neurologically diagnosed patients and lesion studies suggests that a variety of brain regions, connected in intricate networks, are involved in action selection, response inhibition, and interference control. These brain areas include the lateral areas of prefrontal cortex (PFC), the supplementary motor complex (SMC), and the basal ganglia.[31–42]

The dorsolateral PFC appears to be involved in maintaining representations of stimulus–action associations, as used to guide action selection, and in determining which of those associations need to be accessed in a given context.[21,43,44] The dorsolateral

FIGURE 1 | *'Odysseus and the Sirens'* (1891) by J. W. Waterhouse. According to Ovid's *Metamorphoses*, Odysseus was eager to learn what the Sirens sounded like but at the same time feared giving into the appeal of their song. Odysseus therefore had his sailors tie him to the mast of his boat and ordered them to leave him there, even if he'd beg to be untied. Upon hearing the Sirens' song, he was immediately enchanted by its beauty and promise, and begged the sailors to unleash him, but they couldn't hear him because he had ordered them to plug their ears with wax (*Odyssey XII, 39*). In popular language, 'the call of the Sirens' refers to an appeal that is hard to resist but that, if heeded, will lead to a bad result.

PFC is engaged more strongly in complex tasks than in tasks with less complicated action-selection demands, suggesting that the increased working memory demands associated with complex conditions necessitate increased recruitment of dorsolateral PFC to guide action selection and suppression.[45]

The inferior frontal cortex (IFC) has been implicated in a large body of evidence as especially crucial for response inhibition.[33,46,47] Deficits of response inhibition as measured using antisaccade tasks or stop tasks have been observed after lesions to the IFC.[48,49] rTMS has been applied as a virtual lesion technique to confirm observations from patient lesion studies,[46,49] demonstrating that the right IFC is necessary for inhibiting a prepotent response in the stop-signal task.[50]

The SMC is thought to contribute to action control in a number of different ways.[37,51,52] Firing of SMC neurons precedes actual motor and oculomotor actions.[53,54] SMC neurons change their patterns of activation when individuals learn and re-learn associations between stimuli and saccadic or manual actions, respectively.[55,56] SMC function is perhaps best understood in terms of the complexity of stimulus–action associations, with a caudal-to-rostral gradient when moving from less complex to more complex stimulus–action associations.[37] Exogenously triggered actions involve stricter constraints on stimulus–action associations than do endogenously generated actions; thus, free-choice tasks preferentially activate rostral SMC [pre-supplementary motor area (pre-SMA)], whereas instructed responses involve caudal SMC [supplementary motor area (SMA)].[57–60] Stimulus–action associations are more complex when they have to be learned anew or when they have to be reversed, and hence engage more rostral SMC activation, compared to when they are overlearned.[56] Selecting the appropriate action is more complex when multiple stimulus–action association alternatives compete for activation, and hence engage more rostral SMC activation, compared to when response conflicts are absent and action selection is driven by a single stimulus–action association.[61–64]

Dorsolateral PFC, IFC, and SMC are densely connected to one another.[65] Moreover, each of these regions has direct projections to and from the striatum (the input structures of the basal ganglia).[66–69] These projections are part of somatotopically organized loops that go from frontal cortex to striatum to basal ganglia output structures (especially globus pallidus) to thalamus and then back to frontal cortex, with many of these projections being reciprocal.[70] The output of this loop serves to facilitate the selective activation of appropriate actions and the selective inhibition of inappropriate actions.[71,72]

Online action control was decomposed into a number of constituent processes that work together to select the most appropriate action. The activation of appropriate actions is based on intention-driven action selection, whereas the activation of inappropriate actions based on extraneous stimulus–action associations should be resisted to prevent response capture. As one instrument of action selection, response inhibition can be invoked to suppress the activation of competing or task-inappropriate actions. Each of these processes, and their underlying neural mechanisms, will be discussed in some detail in the subsequent sections to gain a deeper understanding of their role in establishing adequate action control.

THE ACTIVATION OF APPROPRIATE ACTIONS BASED ON INTENTION-DRIVEN ACTION SELECTION

Despite a long tradition of research within cognitive psychology on processes of stimulus–response translation,[28,29] to date little is understood about the neurocognitive mechanisms of intention-driven action selection. To further our understanding of these cognitive processes, we need to sketch a more elaborate picture of the SMC and its role in action control (see Figure 2; for a detailed but comprehensible elaboration see the review by Nachev and colleagues[37]).

The SMC consists of the SMA, the pre-SMA, and the supplementary eye field (SEF), located in the dorsomedial frontal cortex just dorsal to the anterior cingulate cortex.[73,74] The SMA, pre-SMA, and SEF constitute the medial part of Brodmann's area 6 in the superior frontal gyrus. The pre-SMA lies anterior to the SMA, with the SEF situated at the dorsal-most part of their border.[75] The SMA has reciprocal connections with the primary motor cortex and the ventral horn of the spinal cord, whereas the pre-SMA is interconnected with other prefrontal areas rather than motor areas,[76,77] such that the pre-SMA might be considered as a prefrontal rather than premotor region.[73] These patterns suggest that the pre-SMA is involved in selecting and preparing actions, whereas the SMA is related to more downstream motor activation processes. There is some discussion about whether the structural differences within SMC are better described in terms of discrete subregions or in terms of a rostrocaudal continuum, proceeding from the SMA through the SEF into the pre-SMA. Although diffusion tensor imaging

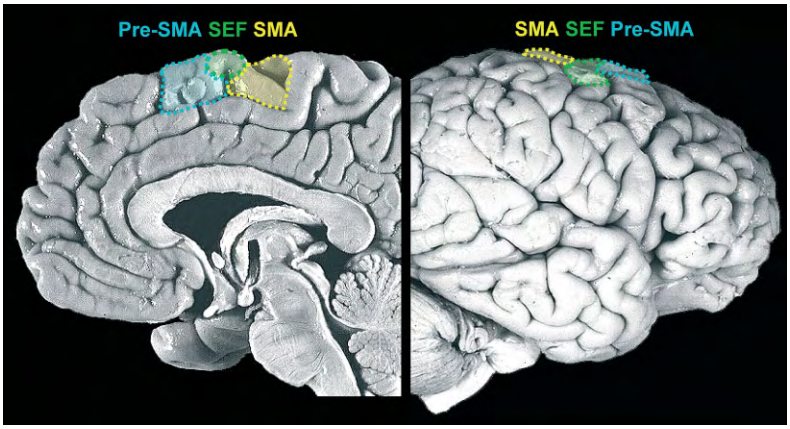

FIGURE 2 | Medial frontal cortex. Midsagittal view of the medial wall (left) and lateral prefrontal cortex surface (right), delineating the main subregions of the supplementary motor complex (supplementary motor area, supplementary eye field, and pre-supplementary motor area).

(DTI) parcellation studies in humans suggest that an important change in connectivity occurs between SMA and pre-SMA,[78] other connectivity profile studies suggest a rostrocaudal continuum rather than a discrete division, with the rostral-most portions of SMA being more similar to adjacent caudal-most pre-SMA than to caudal-most SMA.[37,77] Indeed, in moving from the rostral (pre-SMA) to the caudal (SMA) side of the SMC, the functional significance of activation appears to shift gradually from being more tightly associated with cognitive aspects to being more tightly associated with motor aspects of action control, with the SEF lying somewhere in between these extremes.[37]

Whether more gradual or discrete in nature, the differences in function between SMA and pre-SMA are prominent and of relevance to our understanding of action control. Although SMA activation is seen only during action execution, pre-SMA activation can be observed already in preparatory intervals preceding the motor command.[76,79] More than any other portion of the SMC, the pre-SMA has been considered a key node for deliberate and voluntary action selection,[51,52] especially because the representation of stimulus–response associations in the pre-SMA is flexible and adaptive. First, patterns of activation in pre-SMA neurons are altered when individuals learn and re-learn associations between stimuli and actions.[56] Second, the pre-SMA is activated in tasks that require switching between stimulus–response mapping rules.[39,80,81]

All parts of the SMC send efferents to the striatum and receive projections back from the globus pallidus pars interna via the thalamus.[69] In addition to this somatotopically organized loop, the SMA and pre-SMA have (partly overlapping) hyperdirect projections to the STN.[69,82,83] Although the role of these hyperdirect projections in action selection remains elusive, one prominent hypothesis suggests that activations along these pathways serve to keep basal ganglia output in check until voluntary action selection has completed.[84,85]

CAPTURE OF INAPPROPRIATE ACTIONS THROUGH EXTRANEOUS STIMULUS–ACTION ASSOCIATIONS

Although Odysseus was curious to hear the song of the Sirens, he had himself tied to the mast of his boat because he did not intend to actually answer their call. Likewise, in their pursuit of adequate task performance, participants in laboratory experiments generally have no explicit intention to select their actions based on extraneous stimulus–response associations, automatically activated action stereotypes or biases, response habits, urges, impulses, and so on. Yet, these extraneous action affordances are often difficult to resist; it is as if they capture the action system non-deliberately. Indeed, stimuli that present the individual with an action affordance (such as graspable objects) have been shown to activate the SMA even when there is no requirement to actually act on those stimuli.[86] These processes of response capture are considered to be rapid, immediate, and non-reflective in nature.

Often, action affordances as triggered by external stimuli are detrimental, as in the case of the Sirens' call. In laboratory tasks, stimuli are often manipulated to present task-irrelevant and inappropriate action affordances. Yet, the existence of such affordances is not necessarily maladaptive. Quite to the contrary, these affordances exist because they have proven to represent adaptive value; they stem from potent, habitual, over-learned, or even instinctive action tendencies, such as looking in the direction of potential danger or catching a ball thrown at us or imitating someone else's action. A stimulus–action association that was consistently reinforced in the past can serve as an affordance for the same action when

the same stimulus is presented again. Such affordances potentiate those actions that yield improvement in the direction of equilibrium: moving toward appetitive stimuli and away from aversive stimuli. Such action affordances hence provide a basis for association-driven action selection.

The phenomenology of action affordances is typically described in terms of attracting or repelling solicitations: intrinsic to the experience of an affordance is that stimuli incite or summon certain actions.[87] Affordances present not only motivational significance but also potentiate an actual and specific tendency to act. Such an attraction bears resemblance to emotional reactions,[88] at least according to Frijda's[89] definition of emotion in terms of a change in action readiness. Just seeing a spider can make one freeze and just hearing the Sirens' song may drive one into the water. However, the fact that stimuli may attract (or repel) certain actions does not imply that execution of these actions is inevitable.[90] Calls can be resisted, at least in principle. Moreover, the strength of the action disposition as potentiated by an affordance, such as the desire to grab a peanut, can be modulated by contextual factors or internal states (such as hunger and satiety).

Behavioral Expressions of Response Capture

Ideomotor theory, put forward by William James[91] and others before him and revived by Wolfgang Prinz and co-workers,[92] proposes that actions are represented in terms of their perceptual consequences and that stimuli and actions are therefore represented in a common coding space. Along these lines, common coding provides a possible mechanism for direct visuo-motor transformations that take precedence over more deliberate stimulus–action mappings—rapid but error prone. Such direct visuo-motor links might constitute one possible avenue for instilling response capture. On a descriptive level, the strength and the time course of response capture can be revealed by *CAFs* that plot accuracy rates as a function of reaction time (RT). The CAF presented in Figure 3(a) illustrates the well-documented finding that fast responses are relatively more prone to errors than slower responses. In conflict tasks (such as the Stroop task, the Simon task, and the Eriksen flankers tasks), for a relatively large proportion of the fast responses, action selection is captured by automatically activated extraneous stimulus–response associations to such an extent that deliberate intention-driven action selection is bypassed and an overt response error is committed. The CAF illustrates that within a clinical sample of patients diagnosed with Parkinson's disease, the amount of response capture in fact distinguishes between subgroups of patients, as the proportion of fast errors correlates with disease severity as indexed by a symptom rating scale.[57,93]

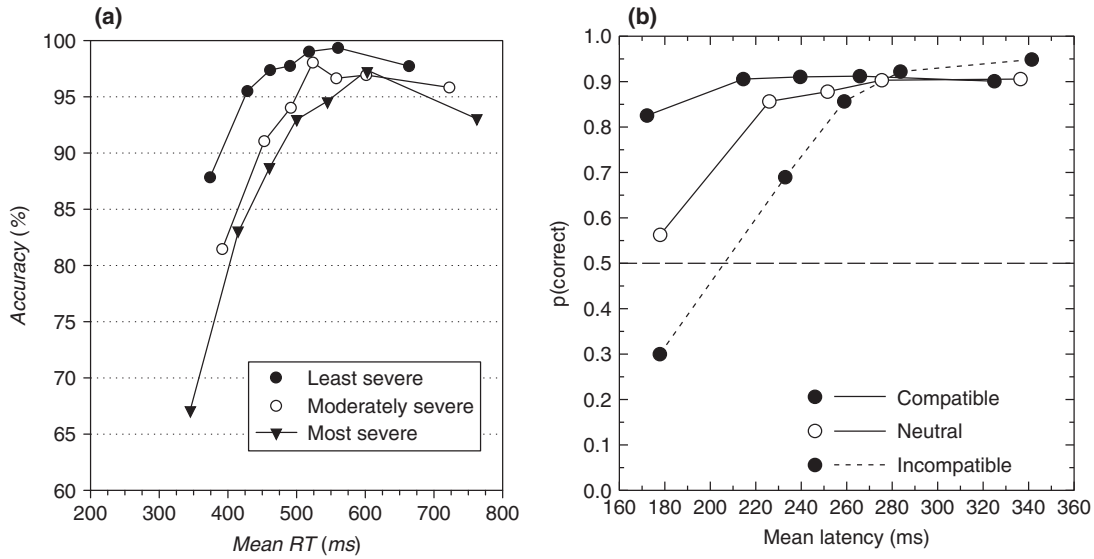

FIGURE 3 | Conditional accuracy functions. (a) Three subgroups of patients diagnosed with Parkinson's disease divided in terms of symptom severity. The subgroup with most severe symptoms displays more fast errors on incompatible trials compared to the other two subgroups (adapted with permission from Ref 93 Copyright 2010 The MIT Press). (b) Performance of healthy participants in the Simon task reveals that the probability of rapid activation of the muscle involved in the *correct* response is high for compatible trials, but significantly below chance in incompatible trials, indicating strong capture of the response solicited by the task-irrelevant stimulus position. Re-analysis of data that were originally reported in Ref 94.

Although detectable in overt behavior, response capture is essentially a covert process. Recording electromyographic (EMG) activity of the muscles involved in the responses has proved very effective in revealing such covert response capture.[94] Indeed, during behaviorally correct trials, EMG recordings sometimes reveal small, subliminal EMG bursts in the muscles involved in the *incorrect* response. Such incorrect response activations are more numerous on incompatible trials (in which the task-irrelevant affordance primes the incorrect response) than on compatible trials (with response capture of the correct response). When coupled to the CAF approach, such partial response activation reveals the strength of response capture. Figure 3(b) represents the probability of correct response activation (as measured by the EMG activity) as a function of time elapsing after stimulus presentation in a Simon task (a task in which stimulus color designates the response, but the task-irrelevant spatial position of the stimulus provides a direct action affordance that is inappropriate on a substantial proportion of the trials). In the incompatible situation, the probability of *correct* response activation drops clearly below chance level for the fastest responses, indicating a very strong capture of the response solicited by the task-irrelevant stimulus position.

The Functional Anatomy of Response Capture

Studies on the functional neuroanatomy of response capture have been scarce. When immediate action affordances are considered as exogenously rather than endogenously triggered actions, one might assume that they involve direct stimulus–action transformations, and hence engage caudal rather than rostral portions of the SMC.[57–60] A more traditional distinction that may also be relevant for present purposes holds that although endogenous action selection relies mostly on the medial structures located in the SMC, exogenously triggered actions capitalize on more laterally situated circuits in premotor cortex (PMC).[95] Canonical neurons (in monkeys' lateral premotor area F5) become active both when grasping an object and when seeing the same (graspable) object without moving. Neurons with such properties may play an important role in object-directed action and may provide a basis for understanding the neural mechanisms underlying responsiveness to affordances.[86,96,97]

The PMC receives projections mostly from posterior parietal cortex (PPC[95]). Within the visual processing system, the dorsal stream is an occipitoparietal pathway that terminates in PPC, and this is considered crucial for the visual guidance of actions toward an object.[98,99] The PPC–PMC circuit has been suggested as the neurophysiological basis for direct visuo-motor transformations (direct-route processing in the dual-process accounts reviewed above) which may provide a neurophysiological correlate of association-driven action affordances. Monkey and patient studies confirmed that activation of the right PPC is essential to visuo-motor transformations,[100,101] in particular for mapping spatial stimulus attributes onto spatial response attributes.[102,103] In a recent intervention study,[104] volleys of rTMS were administered to interrupt neural activity in PPC while participants performed a Simon task. rTMS volleys to the right PPC served to ameliorate the interference effect as elicited by the task-irrelevant stimulus position, thus confirming the role of PPC in establishing direct visuo-motor links.

Indirect evidence for the neural mechanism underlying response capture comes from a recent fMRI study from our laboratory,[105] in which the subjects' task was to respond to target probe stimuli, given that these were preceded by a specific cue. In a training phase, some specific cue stimuli became associated with face probe stimuli, whereas certain other cues became associated with house probes. In a subsequent test phase, the same cue stimuli were followed by the associated probe stimuli (target probes: face and house stimuli, respectively) most of the time, but incidentally the cues could instead be followed by non-target probe stimuli (house and face probes, respectively). Presentation of the face-associated cue stimuli triggered immediate reactivation of the fusiform face area (FFA, thought to be prominently involved in face processing), whereas presentation of the house-associated cues reactivated the parahippocampal place area (PPA, thought to be involved in processing scene stimuli such as houses and landscapes). Reactivation of FFA and PPA yielded performance benefits when these cues were followed by target probes, but performance costs when followed by (infrequent) non-target probes. These patterns can be likened to response capture: cue-elicited reactivation of specific visual processing areas leads to the immediate activation of target responses, even when the probe stimulus actually requires a non-target response.

When multiple stimulus–action association alternatives compete for activation, the demands on action control are highest, and selecting the appropriate action engages stronger activation of the pre-SMA compared to when response conflicts are absent.[41] Indeed, the strength of activation in pre-SMA covaries with the extent to which inappropriate responses are

captured by stimulus–action association as driven by task-irrelevant stimulus features,[33,106] and neurodisruption or lesions of this region compromise the efficiency of action selection in the face of response capture by competing stimulus–action association alternatives.[60,63,64] A recent voxel-based morphometry study from our laboratory[107] confirmed the role of pre-SMA in the ability to select the appropriate response in the face of competing alternatives, by demonstrating a strong negative correlation between pre-SMA gray-matter volume and the susceptibility to response conflict.

Taken together, the amassed evidence points to a role for the pre-SMA as an action-selection director, modulating the action-selection gate through which the available action affordances are translated into actual actions. One way in which such gatekeeping may be implemented is by modulating the strength of coupling between input areas (which process the stimuli that present action affordances) and output areas (action-selection circuits downstream of the pre-SMA, including the basal ganglia and the motor system). Modulation of the basal ganglia input structures (e.g., putamen[106] and STN[82]) may serve to coordinate the relative weighting of actions; the selected action is strengthened by removing tonic inhibitory signals sent via the thalamus to the corresponding motor pattern generators in PMC, whereas non-selected actions are suppressed by increasing tonic inhibition.

A further instrument of action control is active top-down response inhibition, as discussed next.

SUPPRESSING THE ACTIVATION OF INAPPROPRIATE ACTIONS THROUGH ACTIVE RESPONSE INHIBITION

Inhibition can be defined as the set of processes that results in the suppression of prepotent behavioral responses when such actions are premature or inappropriate in a given context and/or when such actions interfere with goal-directed behavior. Inhibitory control is postulated as one of the mechanisms by which action control exerts its coordinating effects on subsidiary processes implemented in other cortical and subcortical regions to optimize behavior. Like intention-driven action selection, we consider response inhibition as an active process that involves suppression of a prepotent action either in favor of engaging more appropriate action alternatives or with the aim of refraining from responding altogether (see also the recent review by Mostofsky and Simmonds[36]).

The example of Odysseus and the Sirens illustrates the use of selective suppression that works against detrimental urges and as such helps selecting more appropriate behavior: The rope tying him to the mast (i.e., selective inhibition) helps him to overcome his prepotent urge to give in to the appeal of the Sirens' song (i.e., detrimental action affordances), so that he can continue his voyage home (i.e., goal-directed behavior). The conflict situation is brought about by the simultaneous activation of two mutually exclusive action affordances, one of which is coupled with an automatically activated impulse. As such, the call for selective inhibition of inappropriate actions to resolve the conflict is generated internally.

Here we survey the literature on the neurocognitive mechanisms underlying response inhibition, reviewing correlational evidence from behavioral, electrophysiological, and neuroimaging studies, as well as more direct evidence from neurological and lesion studies. We focus our review on the two classes of experimental paradigms most prominently associated with response inhibition: the Go/NoGo and stop-signal paradigms on the one hand and the conflict paradigm on the other.

Go/NoGo tasks[108] and stop-signal tasks[109,110] require subjects to engage in intention-driven action selection by performing speeded responses on Go trials (such as pressing a button in response to a target stimulus), but to inhibit responding on incidental NoGo trials (containing non-target stimuli) or stop trials (when the target stimulus is followed by a stop signal) (Figure 4). Behavioral indices of inhibitory control in Go/NoGo and Stop tasks are (1) the percentage of commission errors (failures to refrain from responding) and (2) (in the Stop task) the duration of the stop process, mathematically approximated as the stop-signal reaction time (SSRT). The recruitment of response inhibition in Go/NoGo and stop tasks has been demonstrated at the level of corticospinal excitability using motor-TMS procedures.[111,112]

In contrast to the complete inhibition of any action affordance as measured by Go/NoGo and stop tasks, selective inhibition of competing action affordances is thought to be invoked in conflict tasks (such as the Stroop, Simon, and Eriksen tasks[113–115]). Conflict tasks, which capitalize on action affordances induced by extraneous stimulus–action associations, measure the suppression of a response whose incitement is triggered by some task-irrelevant (or extraneous) feature of the stimulus and that conflicts with a response associated with intention-driven action selection based on a relevant stimulus feature (Figure 5).

Based on classic monkey lesion work, inhibitory control has for a long time been associated with the

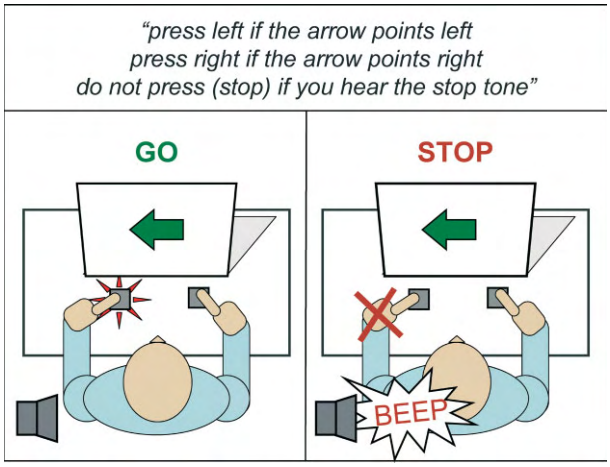

FIGURE 4 | Stop task. Participants press response buttons, either with their left or right hand, according to the direction of the green arrow (go signal), but try to stop responding upon the incidental presentation of a subsequent auditory tone (stop signal).

IFC,[116] a picture strengthened by more recent fMRI studies. However, recent reviews demonstrate that, rather than a single structure, a network of areas is important in implementing inhibition, depending in part on task demands[36] (Figure 6). In addition to the IFC, the studies reviewed below point to a role for dorsolateral PFC (dlPFC), pre-SMA, and several structures within the basal ganglia. Although none of these structures is consistently observed to be essential for implementing response inhibition, and hence the

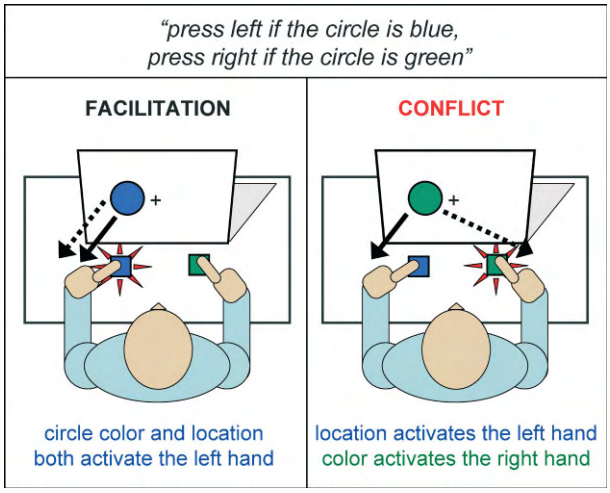

FIGURE 5 | Simon task. Participants press response buttons, either with their left or right hand, according to the color of the circle (dotted arrow). Although the position of the circle is task irrelevant, its action affordance incites a strong tendency to activate the corresponding hand (solid arrow), which leads to longer RT and more response errors in incompatible trials compared to compatible trials.

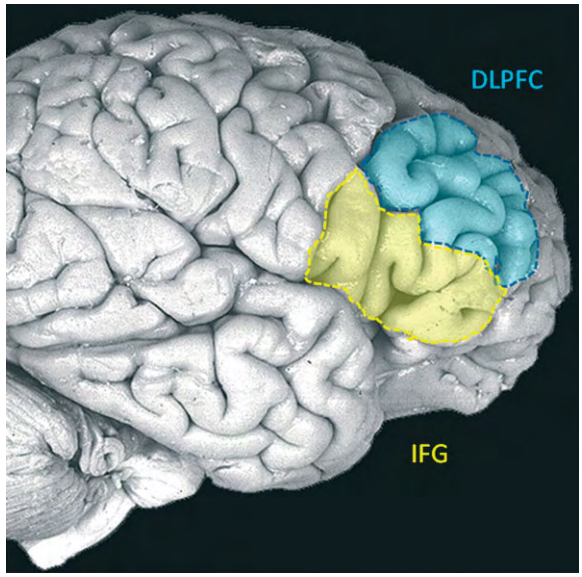

FIGURE 6 | Lateral prefrontal cortex. Lateral surface of the prefrontal brain, delineating the dorsolateral prefrontal cortex (blue) and the inferior frontal cortex (yellow).

outcome of such a review must remain far from conclusive, the emerging patterns begin to delineate a picture in which the dlPFC is active in providing top-down guidance to action-selection areas, the pre-SMA engages response inhibition as an instrument of action selection, the IFC is recruited to aid in implementing response inhibition in more demanding situations, and the basal ganglia keep all responses in check until the final signal is received from upstream. Below, we explore the neurocognitive mechanisms underlying response inhibition in more detail, first for the Go/NoGo and stop-signal paradigms, and then for the conflict paradigm.

Neurocognitive Mechanism of Inhibition in Go/NoGo and Stop-Signal Paradigms

The pre-SMA is optimally situated to transform the information coming in from association cortex into preparation for action—not only for action selection, as reviewed in preceding sections, but also for selective action suppression. This notion is informed by, among others, the pattern of projections that connect the pre-SMA to other cortical and subcortical brain areas.[37,78] The pre-SMA connects not only to lateral prefrontal association cortex (including dlPFC and posterior portions of IFC) and parietal association cortex but also to many of the structures that make up the motor system. The involvement of the pre-SMA in response inhibition has been demonstrated with electrical stimulation studies in monkeys,[117–119] showing that

such stimulation serves to suppress motor actions. In humans, lesions in the SMC region result in deteriorated response inhibition in Go/NoGo and stop tasks.[38,120,121] Neurodisruption of the pre-SMA with rTMS yielded decrements in the ability to inhibit responses upon a stop signal.[122] Single-cell recordings in monkeys[76,81] and scalp encephalographic studies in humans[123] confirm the role of pre-SMA in response inhibition. In fMRI studies using the Go/NoGo task, response inhibition is often found to engage the pre-SMA (as reviewed in recent meta-analyses[40,124]). Similarly, the pre-SMA is often activated in the stop task.[67,125–127] In one study,[126] SSRT was found to correlate with pre-SMA activation, such that individuals who were more proficient at inhibition showed greater activation in the pre-SMA during stopping.

Areas within lateral PFC, including dlPFC and IFC, have been implicated in a large body of evidence as cardinal for response inhibition.[47] The generic top-down guidance function attributed to dlPFC[21] might also apply to guiding response inhibition in accordance with instructions and intentions. The involvement of dlPFC in response inhibition has been demonstrated with electrical stimulation studies in monkeys,[128] showing that when cells in BA46 were stimulated during regular responses, activity in primary motor cortex decreased, resulting in either a delay or the complete suppression of responses. Single-cell recordings in nonhuman primates[128,129] confirmed a role for BA46 in response inhibition, showing that response inhibition elicited by NoGo stimuli was accompanied by firing of cells in the principal sulcus (the monkey homolog of dlPFC). In fMRI studies using the Go/NoGo task in humans, response inhibition is often found to engage dlPFC.[31,45,130–133] Similarly, the dlPFC is often activated in the stop task.[45,125,134] In one fMRI study,[134] SSRT was found to correlate with dlPFC activation, such that individuals who were more proficient at inhibition showed greater activation in BA46 during stopping. In contrast, neurodisruption of the dlPFC using rTMS yielded no effects on the ability to inhibit a prepotent response in the stop-signal task.[50]

Perhaps the most prominently reported frontal brain area involved in response inhibition in humans is the IFC, especially in the right hemisphere. Deficits of response inhibition and interference control as measured using antisaccade tasks or stop tasks have been observed after lesions to the IFC (in particular the right frontal operculum).[48,49] The extent of damage in the right IFC predicted the magnitude of the inhibitory deficit.[49] In monkeys, lesions in a homolog of IFC (BA45) yielded impaired NoGo performance.[116] Neurodisruption of the IFC using rTMS as a virtual lesion technique confirmed observations from patient lesion studies,[46,49] demonstrating that the right IFC is necessary for inhibiting a prepotent response in the stop-signal task.[50] In fMRI studies using the Go/NoGo task, response inhibition is often found to engage the right IFC (as reviewed in recent meta-analyses[40,124]). Similarly, the right IFC is often activated during response inhibition in the stop task.[67,125,126,135,136] SSRT has been found to correlate with right IFC activation, such that individuals who were more proficient at inhibition showed greater activation in the IFC during stopping.[67] The common finding of activation in the right IFC in fMRI studies of the stop-signal task is more consistent than in the Go/NoGo task. This difference may be related to the notion that the demands on inhibitory processing are generally higher in the stop-signal task than in the Go/NoGo task. The activation of right IFC in the Go/NoGo task has been dissociated from the oddball effects of infrequent NoGo events *per se*, thus rendering an explanation in terms of frequency-based salience less likely.[137]

Finally, studies on response inhibition frequently report the involvement of structures within the basal ganglia. Patients with lesions in the basal ganglia have been shown to display impaired response inhibition in the stop task[138] (the exact location and extent of the lesions were not specified). The caudate nucleus is activated more strongly during successful compared to failed stop trials.[139,140] Furthermore, projections from the IFC and pre-SMA to the STN may play a critical role in response inhibition. Given their rapid conduction time, these hyperdirect projections (associated with long-range inhibitory connections[72]) may be particularly important in inhibiting inappropriate responses. For instance, Aron and colleagues suggested an association between SSRT and the density of white-matter fibers between IFC and the STN region.[141] In an fMRI experiment, when SSRT was regressed against the stop-Go contrast, participants with faster SSRTs displayed stronger activation of the IFC and the STN region. These areas coincided with the nodes of a white-matter network as observed in a diffusion-weighted MRI study. In Parkinson's patients with deep brain stimulation in the STN, SSRT is reduced when stimulators are on rather than off.[142] In rats, lesions of the STN did not affect SSRT, but did reduce overall accuracy at stopping.[143] In fMRI studies using the stop task, the STN is activated more prominently during stop trials than during go trials.[67,139] Individuals with more proficient inhibitory control (as expressed in faster SSRTs) showed less STN activation than poor inhibitors.[139]

The patterns of inhibition-related activation described above appear to be coherent and robust. Yet, it should be noted that none of these areas are recruited universally (see reviews elsewhere[36]). The reasons for these inconsistencies are not yet evident, and research is needed to explore the plausibility of a number of possible hypotheses. First, studies may differ in the extent to which they invoke activation of inappropriate responses, and hence in the degree to which effort has to be invested in inhibiting such activation. It might be that the right IFC is recruited most prominently when more effortful inhibition is required, which might explain why IFC activation is seen more consistently in stop tasks than in Go/NoGo tasks. An alternative view holds that while the pre-SMA is consistently engaged across tasks as a final common pathway involved in selecting to withhold a response in implementing response inhibition, the IFC is engaged only when the task demands are more complex, such that the IFC maintains information about stimulus–response associations and provides supplemental guidance in selecting to withhold a response.[36,40]

Neurocognitive Mechanism of Inhibition in the Conflict Paradigm

Selective response inhibition is often thought to be invoked in conflict tasks. When multiple action affordances compete for activation, the engagement of selective inhibition of task-inappropriate actions imposes significant demands on the brain circuits involved in implementing such inhibition.

The inhibition of incorrect, prepotent action affordances as an essential mechanism in resolving response competition during conflict tasks has been implied widely, but theoretical and analytical methods to directly study the inhibition process have only emerged recently.[94,144] Again, timing is everything. According to the activation–suppression model,[29] the rapid activation of an incorrect action affordance by extraneous stimulus features is followed temporally by the engagement and gradual buildup of online suppression of this activation. Based on these temporal dynamics, the model predicts that slower reactions in conflict situations are less likely to be negatively impacted by incorrect action affordances because selective suppression has had more time to accrue. A host of studies now confirm that the interference from incorrect action affordances in conflict tasks levels off or reverses at the slow end of reaction time distributions, consistent with top-down suppression of the action affordance (see Figure 7 for an illustration). Furthermore, the magnitude of the reduction in the interference effect at the slow end of the RT distribution has been shown to be sensitive to the demands on inhibitory control,[94,145] to distinguish

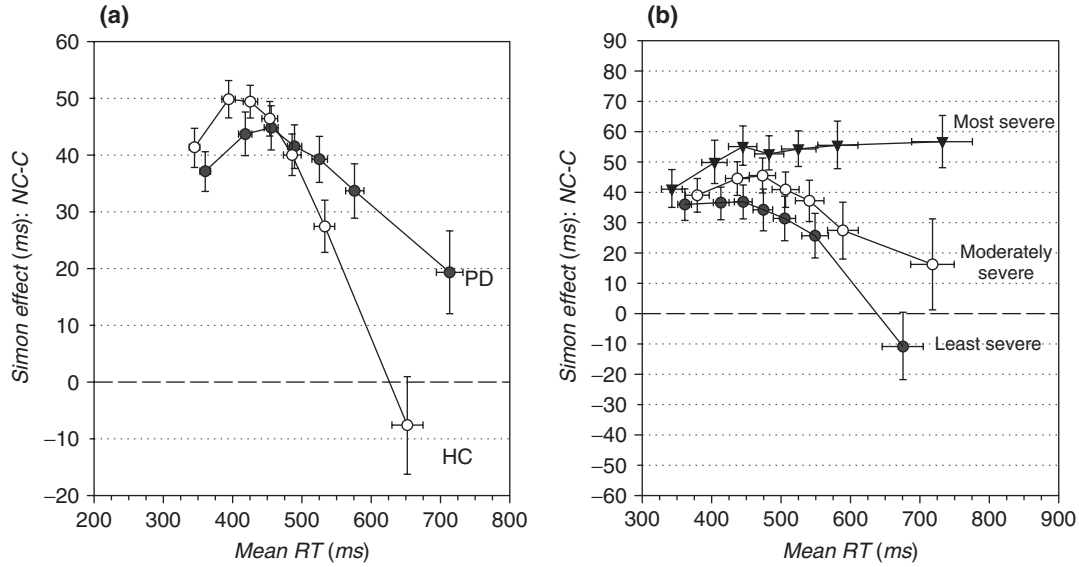

FIGURE 7 | Delta plots. Delta plots illustrate individual differences in the ability to reduce the magnitude of the interference effect over time. Specifically, delta plots depict the Simon effect as a function of reaction time quantile. The slope of the delta plot at the slower end of the RT distribution is indicative of the efficiency of selective response inhibition: the more negative going, the stronger the inhibition (see main text) (adapted with permission from Ref 93 Copyright 2010 The MIT Press). (a) Delta plot illustrating impaired selective suppression in patients diagnosed with Parkinson's disease (PD) compared to age-matched healthy controls (HC). (b) Delta plot illustrating impaired selective suppression in PD patients with severe clinical symptoms compared to patients with less severe symptoms.

individual and group differences in the proficiency of inhibitory control,[7,17,146,147] and to be related to individual differences in the engagement of select prefrontal regions associated with inhibitory control.[32,33,148]

Selective response inhibition in conflict tasks involves a network of brain areas that shows considerable overlap with that involved in inhibition in stop and Go/NoGo tasks. As reviewed below, the key constituents of this network are the lateral PFC, the pre-SMA, and the basal ganglia. Within lateral PFC, a few studies have implicated dlPFC, but the role of the (right) IFC appears more strongly emphasized. The role of pre-SMA in selective inhibition appears relatively less prominent compared to inhibition in stop and Go/NoGo tasks.

As discussed in a preceding section, certain neurons in pre-SMA fired when inhibiting responses in a Go/NoGo task.[81] The same neurons fired also during action reprograming, that is, when the monkey switched between stimulus–response mapping rules, which involved the suppression of a habitual action and the selection of a new response. The pre-SMA may facilitate switching from association-driven to intention-driven action selection by suppressing the habitual response and prompting selection of the controlled response. In humans, the role of the pre-SMA in the same task was confirmed in a paired-pulse TMS study, demonstrating that the pre-SMA influences M1 during action reprograming, but not during normal action selection.[149]

Several studies have implicated the IFC in selective response inhibition in conflict tasks. A neurodisruption study with rTMS in an Eriksen flankers task has failed to confirm that the IFC is necessary for resolving competition between responses.[150] In contrast, rTMS in an imitation congruity task did show that disruption of the IFC incurred a deteriorated ability to inhibit the urge to imitate (incorrect) finger responses.[151] Possibly, the imitation task places greater demands on selective response inhibition than does the flankers task. Particularly, informative results might be obtained from combining rTMS disruption of IFC processing with RT distribution analyses of response inhibition in the Simon task; such studies are currently under way in our laboratory. In neuroimaging studies, comparison of incongruent trials (eliciting two conflicting responses) with congruent trials (affording only one response) has revealed specific activations in IFC.[61,62,151–153] In recent studies using functional and structural imaging, we quantified the extent to which RT distribution measures of selective response inhibition, as described above, were associated with individual differences in both IFC function and structure.[32,33] The results revealed a strong correlation between the model parameters and both fMRI and DTI characteristics of the right IFC. These results appear consistent with the notion that the IFC is engaged most prominently when strong inhibitory effort is required.

Additional studies support a prominent role for the basal ganglia in conflict tasks. For instance, in neuroimaging studies, caudate nucleus activation is found in conflict conditions in both Stroop and Simon tasks.[154] Evidence is also found in studies that show that human neurodegenerative disease of the basal ganglia, such as occurs in Parkinson's disease and Huntington's disease, disrupts inhibitory control mechanisms during the performance of response conflict tasks,[17,49,155,156] although this has not yet been confirmed in studies on patients with lesion in the basal ganglia compared to healthy controls.[157]

CONCLUSION

Online Action Control: Coda and Future Directions

We have reviewed the neurocognitive mechanisms of association-driven and intention-driven routes of action selection and of the inhibition of inappropriate actions. Our review highlighted the pre-SMA as a key node for intention-driven action selection. Direct connections between the pre-SMA and basal ganglia structures (most prominently the anterior dorsal striatum and the STN) serve to keep basal ganglia output in check until intention-driven action selection has completed. Extraneous action affordances may capture the action system non-deliberately. Such association-driven action-selection processes are considered to be rapid, immediate, and non-reflective in nature. When multiple stimulus–action association alternatives compete for activation, the demands on action control are highest, and selecting the appropriate action engages stronger activation of the pre-SMA compared to when response capture is absent. Taken together, our review suggests a role for the pre-SMA as an action-selection director, modulating the action-selection gate through which the available action affordances are translated into actual actions.

The patterns that emerge from our review of the neurocognitive mechanisms underlying response inhibition suggest that the dlPFC provides top-down guidance to action-selection areas, the pre-SMA engages response inhibition as an instrument of action selection, the right IFC is recruited to aid in implementing response inhibition in more demanding situations, and the basal ganglia keep all responses in

check until the final call is received from upstream. Compared to inhibition in stop and Go/NoGo tasks, inhibition in conflict tasks appears to rely more prominently on the right IFC and less prominently on the pre-SMA. In particular, individual differences in the efficiency to implement inhibitory control in humans are associated consistently with functional and structural differences in the right IFC, whereas individual differences in the role of the pre-SMA in suppressing no longer appropriate courses of action have remained somewhat more elusive in conflict tasks.

Future research efforts may aim to further highlight the specific role of the pre-SMA *vis-à-vis* the right IFC, as well as the role of the basal ganglia (in particular the STN) in online action control, more specifically in response capture and response inhibition. Additionally, studies incorporating information about individuals' genetic makeup may allow us to explain and predict the efficiency of action control mechanisms with greater precision, giving rise to the development and articulation of neurobiological models that capture processes essential for resisting the call of the Sirens.

NOTE

[a]In a different context, Braver et al.[11] distinguish proactive and reactive control strategies that correspond roughly to our notions of anticipatory and online control, respectively. We choose not to copy their nomenclature because, as will become apparent in the next section, the terms proactive and reactive apply more specifically to certain dimensions within anticipatory control.

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