Neuroscience and Biobehavioral Reviews 36 (2012) 1107–1118

Contents lists available at SciVerse ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review

Intentional inhibition in human action: The power of 'no'

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ARTICLE INFO

Article history:
Received 13 July 2011
Received in revised form 5 December 2011
Accepted 20 January 2012

Keywords:
Intention
Inhibition
Volition
Action
Cognitive control
Anarchic hand syndrome

ABSTRACT

The capacity to inhibit and withhold actions is a key feature of human cognition. Withholding action forms the basis of self-control, delayed gratification, social contracts, and trust in others. Most experimental studies of this function come from studying the processing of external stop signals. However, another important aspect of inhibition is 'will-power', i.e., intentional inhibitory control over one's own actions, in the absence of external countermanding signals. We review whether a concept of intentional inhibition is justified, and how it might differ from externally triggered inhibition. Further, we consider three types of neuroscientific evidence that can clarify the brain's mechanisms of inhibition: neuropsychology, neurostimulation and neuroimaging. Finally, we propose a model in which intentional inhibition, unlike externally triggered inhibition, is linked to representing longer range consequences of action decisions. We suggest that the human brain contains a 'neural brake' mechanism that blocks specific ongoing motor activity, and that this mechanism plays a key role in action decisions.

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1. Introduction

1.1. What is intentional inhibition?

Most people recognise the experience of being about to commit an action, and suddenly holding back at the last possible moment. Often there is a distinct experience of cancelling the action as a result of a quite specific decision or process, and for an identifiable reason. Consider two examples. You are writing an to your boss,

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doi:10.1016/j.neubiorev.2012.01.006

perhaps because you are upset or angry. You are just about to click the 'Send' button, when you seem to hear a voice in your head that says "do you really want to send that?", and you hold back. You are posting a letter, and are just about to release your grip on it and let it fall into the post box, when you suddenly get the feeling that you should check whether you put a stamp on the envelope. You tighten your grip and inspect the letter. In both these cases, one intentionally withholds an action whose preparation and path towards execution has already begun. Further, in both cases, there appear to be clear reasons for making the action, and also clear reasons for stopping it.

We will use the term 'intentional inhibition' to refer to this capacity to voluntarily suspend or inhibit an action. Intentional inhibition shares some features with other instances of inhibition in psychology, where participants are instructed to withhold responses when presented with particular stimuli, such as NoGo stimuli and stop signals. For example, there is a prepotent or otherwise salient motivation for action. Further, the preparatory processes that lead to action are already underway when inhibition occurs. However, intentional inhibition has other features that are not shared with other forms of inhibition. By definition, the process or signal that cancels or inhibits the action is not the result of any external signal or instruction, but is generated *internally* by the participant themselves. In this respect, intentional inhibition clearly differs from classic psychological paradigms where an external stop signal is used to trigger inhibition (Logan and Cowan, 1984), or NoGo tasks (Pfefferbaum et al., 1985; Eimer, 1993). Further, intentional inhibition prevents motor output, but clearly does not remove the reason for action. For example, stopping myself from sending the angry does not stop me wanting to express my anger. Finally, intentional inhibition seems linked to three quite specific experiences: an urge to act, a simultaneous experience of a distinct reason to resist the urge to act, and often a feeling of frustration at failure to achieve the desired action.

We suggest that intentional inhibition is a core process of the general capacity that psychologists have termed self-control (Baumeister et al., 2007). In particular, the capacity to withhold a prepotent action, and to adjust or cancel an action after its initial preparation, gives humans the capacity to act flexibly and strategically.

1.2. A factorial structure for action control

Studies of action control classical distinguish between an internally generated and an externally triggered route to action (Goldberg, 1985; Jahanshahi et al., 1995; Jenkins et al., 2000). An action may either be a direct and immediate result of an imperative stimulus, or may occur for reasons that seem unrelated to any obvious stimulus at all, but are instead strongly related to the internal states of the subject. In fact, a standard operational definition of voluntariness refers to actions that lack an immediate preceding stimulus (Passingham et al., 2009). Instead, intentional actions are assumed to follow from desires, goals and intentions of the subject.

These desires and intentions are, of course, generally related to the external environment, and could thus be seen as representations that mediate between the external world and the expression of behaviour. This mediation means that voluntary actions can be remote in time from many of the factors that we consider relevant to their causation, and can thus have 'freedom from immediacy' (Shadlen and Gold, 2004).

The distinction between these two routes is often based on the neuroanatomical distinction between a medial frontal system for internally generated action, centred on the pre-SMA, and a more lateral parietal-premotor system for externally triggered action.

The origin of the observed differences between internally generated and externally triggered actions remains controversial. Whilst some authors argue that internally generated actions depend on evaluation and monitoring of internal states (Passingham et al., 2009); others strongly reject this view, and suggest instead that internal generation is related to the evaluation of a complex environment (Nachev and Husain, 2010). More generally, the nature of voluntary action remains highly controversial, both in neuroscience and in philosophy (Haggard, 2008).

Although the source of the differences is controversial, empirical data point unequivocally to the fact that internally generated and externally triggered actions represent two extremes of a continuum. Here, we suggest that the same continuum may be found in inhibition of behaviour. Our assertion of similar continua for action and inhibition is largely independent of the conceptual issue of how this continuum is understood. Particularly, we believe that the internally generated vs. externally triggered distinction can be made just as clearly for inhibition as for action. If it is true that action can be either internally- or externally triggered, then in principle inhibition of actions could show a similar distinction. A person may withhold an action either because of an external stop signal, or because of an internal decision to do so. The decision to inhibit, like the decision to act, may depend on external stimuli, or on internal reasons and desires. For example, the current situation may make a particular action inappropriate or undesirable, even though it might be highly appropriate in other situations. Indeed, the brain processes balance between enabling instrumental action in some situations, and inhibiting it in others, are thought to underlie the flexible nature of social behaviour (Crockett et al., 2010). For now, it is important to point out that the internal/external dimension for inhibition is *orthogonal* to the internal/external dimension for action. That is, one can intentionally inhibit both actions that one decided oneself to make, or actions that are triggered by environmental signals or objects, as in anarchic hand syndrome (AHS). On this view, the cognitive control of action has a factorial structure, as illustrated in Table 1.

Interestingly, many people also recognise the experience of going ahead with action, while simultaneously acknowledging possible reasons for withholding it, and then later regretting having made the action. One common example is saying an unkind and gratuitous word to someone whom we should respect, and regretting the comment as soon as it is made. Such actions can produce unpleasant consequences, and have high personal and moral cost.

2. Methodological issues in studying intentional inhibition

Despite the importance of intentional inhibition as a cognitive control process, it has received relatively little attention in the psychological or neuroscientific literature. Indeed, one might ask whether we need a concept of intentional inhibition at all. So far, the evidence for intentional inhibition we have given above is only subjective experience, and this is a notoriously unreliable guide to cognitive processing (Nachev and Husain, 2010). Clearly, stronger evidence is required.

Classically, psychologists have postulated internal processes when and only when they are required to explain behaviour (Turing, 1950). This leads to three important methodological difficulties arising from the features of intentional inhibition outlined above. First, intentional inhibition produces no behavioural output. Since behaviour is our standard guide to internal processes, can we be justified in drawing *any* inferences about internal cognitive processes from the absence of behaviour? Moreover, *measurement* of intentional inhibition is problematic because there is no overt behaviour to measure. Behavioural experiments on intentional inhibition may do no more than elicit failures to inhibit. In contrast, neurophysiological and neuroimaging methods can be particularly valuable, since they identify brain processes associated with the

Table 1
Factorial organisation of internal and external control of action and inhibition.

Action	Inhibition	Everyday example	Experimental task
Externally triggered	Externally triggered	Driving towards a green traffic light, which suddenly turns to red	Stop signal reaction time (Logan, 1994)
Externally triggered	Internally generated	Resisting the temptation to take another biscuit from the biscuit tin	Freely choosing whether to respond to a stimulus or not (Karch et al., 2009; Kühn et al., 2009) Omission trials in Continuous Performance Task (Rosvold et al., 1956)
Internally generated	Externally triggered	Suddenly cancelling a nefarious activity when you see that someone is watching you	Being caught in the act by witnesses (Finger et al., 2006) Pausing an action in response to external stimulation (Matsuhashi and Hallett, 2008)
Internally generated	Internally generated	Deciding not to send an angry email just before clicking the 'send' button' Holding back from kissing somebody, despite inviting circumstances	"Veto". (Brass and Haggard, 2007)

inhibition of behavioural output (see Section 4). Second, intentional inhibition necessarily involves an internally generated inhibitory process, rather than an externally triggered one. Most traditional experimental designs rely on studying a participant's behaviour by manipulating external stimuli. Studies of externally triggered inhibition are reviewed in the next section. However, *internal* generators of behaviour cannot be manipulated in the same way, since they are intrinsic to the participant themselves.

A third methodological difficulty arises because intentional inhibition must be an inhibition of *something*. That is, there must be a process which would lead to action had it not been inhibited. But, in the absence of any behaviour, what evidence is there that an action would have occurred? In particular, it may be difficult to distinguish a situation where there was never any intention to act at all, from a situation where an action is prepared but then inhibited. If no action was ever prepared, we should speak of an early decision not to act, rather than of a decision to intentionally inhibit a prepared action. We suggest an important distinction between two possible sources of non-action: (1) *early decisions* whether or not to begin action processing, and (2) *late decisions* whether to inhibit a final motor output (Brass and Haggard, 2008; Kühn et al., 2009). The former can be explained in terms of action selection processes alone, while the latter require an additional process of intentional inhibition. This process would have the specific function of blocking motor output, and suppressing an action that has already been prepared. From now on, we restrict the term *intentional inhibition* of action to the latter case. The distinction between early and late inhibition is clearly identifiable in differences between two tasks classically used to study externally triggered inhibition, namely the NoGo and stop-signal tasks (SST). Inhibition clearly occurs earlier within the action preparation process in the case of NoGo tasks than in SST. We therefore suggest that the early voluntary decision to inhibit may be analogous to the NoGo task, while late voluntary decisions to inhibit may be analogous to the SST.

The SST has been widely used to study the neural basis of response inhibition. Logan et al. (Logan, 1994; Verbruggen and Logan, 2008) have suggested that action and inhibition interact though a cognitive model of competition between different response alternatives (the 'race' model). In this model, action and inhibition are triggered by different external signals, and whichever process first reaches a threshold level of neural activation will determine the action outcome.

Here we compare three experimental methods for studying intentional inhibition: free choice designs, contextual inhibition designs, and artificially induced inhibition. These designs vary in the prominence of an internal decision to inhibit. While no single method solves all the methodological problems described above, in combination they may clarify some aspects of intentional inhibition.

2.1. Free choice action inhibition tasks

The first approach simply involves asking participants to first prepare an action, and then to decide for themselves to either inhibit it, or execute it (Brass and Haggard, 2007; Haggard et al., 2009; Walsh et al., 2010).

This "free decision" approach has been used to study decisions about when (Jahanshahi et al., 1995; Jenkins et al., 2000; Libet et al., 1983) to act, and about what action to make (Deiber et al., 1999; Fleming et al., 2009). This "free choice" design can be criticised in at least three ways. First, the instructions to the participant are problematic. They leave much to the experimenter's communicative intention, and the way the participant understands it. Second, in most experimental studies, little or nothing hinges on whether the participant decides to act or not on any particular trial. Therefore, ecological reasons for action and for inhibition are lacking. Third, the 'decision' to act or inhibit is presumably determined by random variations in the state of the brain just before the decision is required. The alleged 'decision' is not a de novo cognitive process, but a consequence of preceding activity. On this view, free choice decisions simply capitalise on chance (Dennett, 1984; Popper and Eccles, 1984; Ebert and Wegner, 2011). We suggest that people will shift from late to early decisions (see above) about action or inhibition in this situation. When no strong reason to act or inhibit exists, it may be easier to not to begin an action in the first place, than to begin one and suppress it. Therefore, free choice tasks could emphasise early response selection, at the expense of late suppression of actions already prepared. On the other hand, the free decision approach does allow an open decision between two equal alternatives: neither acting nor inhibiting is obviously correct or incorrect. Put another way, these designs clearly separate internally generated action decisions from decisions triggered by external or contextual stimuli.

2.2. Contextual inhibition tasks

A second approach involves a contextual instruction to inhibit, but no overt 'inhibit' signal. For example, in Jacoby's exclusion task, participants are asked to complete a stem, e.g., "tab__", with any word apart from a word that was presented just previously. Thus, if the word *table* is presented first, followed by "*tab__*" the participant must intentionally inhibit the *table* response, in order to achieve a correct response such as *taboo* (Cothran and Larsen, 2008). Similarly, the instruction to perform an antisaccade involves inhibiting a prepotent prosaccade response (Munoz and Everling, 2004), and the Stroop instruction to name the colour of a word involves inhibiting the prepotent response to read it (Stroop, 1935), and the spatial location of a stimulus may strongly influence spatially organised responses, meaning that these responses must be

inhibited when spatial parameters are irrelevant and when stimuli contain incongruent spatial information (Forstmann et al., 2008; Simon, 1969).

Contextual inhibition therefore involves both an external stimulus, and a context which influences the way the stimulus is processed. Often, the context can be treated as a rule, for example in a set of task instructions. Thus, in Stroop tasks, the instructions specify that a word should not be read, but rather the ink colour should be named. Successful performance thus depends both on a preceding process of understanding the context, and on perceiving the stimulus. In our view, applying such rules still involves sensory processing of external stimuli, but is just more complex. Therefore, contextual inhibition is closer to external than to internal inhibition.

2.3. Inducing inhibition artificially: negative motor areas

A third approach uses direct experimental activation of inhibitory brain areas to demonstrate mechanisms for inhibition, but provides limited information about function. In some cases of drug-resistant epilepsy, clinical need suggests neurosurgery. Pre-surgical evaluation may include stimulation of frontal cortex through implanted electrodes. In some cases, stimulation can produce an inhibition of ongoing movement. This body of literature is discussed in Section 2.3. Here, the inhibitory system is clearly activated by an external, artificial input, making this data difficult to relate to internal, voluntary decisions to inhibit. On the other hand, stimulation unequivocally demonstrates that a brain mechanism for suppressing actions does exist, and allows its organisation to be studied systematically.

2.4. Neuropsychology of intentional inhibition

More persuasive evidence regarding intentional inhibition comes from the neuropsychological condition of anarchic hand syndrome (AHS) (Della Sala and Marchetti, 2005). This condition typically follows medial frontal and/or callosal lesions. A bilateral equivalent is known as utilization behaviour (Boccardi et al., 2002). A posterior form of AHS, as found in corticobasal degeneration patients, is somewhat different, and is not discussed here. The key feature of frontal AHS is that the affected hand makes compulsive, object-oriented movements in response to environmental stimuli. Crucially, these movements are well-controlled, purposive actions, but they occur against the patient's will. The patient is aware of their anarchic hand movements, and also fully aware that the movements are goal-directed. However, the patient cannot voluntarily prevent their occurrence. Indeed, they may attempt to prevent the unwanted movements by unusual physical means, such as sitting on the anarchic hand, or restraining it with the unaffected hand. Because patients are aware that the anarchic movements are inappropriate, this represents a case of failed internally generated inhibition.

For example, Della Sala et al. (1991) describe a patient with AHS: "...The right hand frequently carried out complex activities that were not willed by G.C. These activities were clearly goal-directed and were well executed, but undesired by the patient, who used her left hand to try to stop them. For example, when the patient had a steaming cup of tea in front of her, the right hand proceeded to pick it up and bring it to her mouth, even though the patient knew that it was too hot and had just said she would wait a few moments until it had cooled. Nevertheless it needed the intervention of her left hand to replace the cup on the table. The will to do so was not sufficient to modify the directed but inappropriate behaviour of the right hand" (p. 1114).

Is the primary issue in these cases a failure of intentional inhibition, as defined at the beginning of this paper, or is it rather an excessive level of voluntary action, which overloads the patient's normal inhibitory capacity? If inhibition itself were normal, but simply overloaded by a hyperkinetic disorder, then AHS patients should show intact or supra-normal performance on tests of voluntary action. In fact, the converse is found: the failure to inhibit unintended movements to external stimuli has been linked with a reduced strength of volition for intended actions. One recent report notes that the affected hand was slow to initiate endogenous actions (i.e., those not defined by external stimuli), *as well as* being compulsively drawn by external stimuli that were present (Cantagallo et al., 2010; Kritikos et al., 2005). Interestingly, the externally triggered action errors of the anarchic hand were reduced when an additional verbal cue was given to indicate the intended action (Cantagallo et al., 2010). This suggests an interpretation in which the unwanted action, in the absence of verbal reinforcement, emerges *because* an alternative positive intention is insufficiently strongly coded, and not because the external triggers are particularly strongly represented.

Taken together, the AHS findings are not consistent with an explanation based on hyperactivity or excessive volition, but are consistent with a deficit in intentional inhibition. They therefore suggest that an important aspect of voluntary control is the inhibition of rival, externally triggered actions. Efficient volition involves a combination of initiating the desired action and inhibiting other actions. Without intentional inhibition, the will may be surprisingly weak.

Recent experimental results shed light on how the inhibitory process works. Giovannetti et al. (2005) noted that the capture of the anarchic hand's actions by external stimuli was highly perseverative. In a naturalistic task (making coffee), the anarchic hand would *repeatedly* engage in inappropriate stimulus-triggered actions. This suggests that intentional inhibition in the normal brain must be a frequent, iterative function that continues for as long as a motivation for action exists. Second, many more perseverative errors were found when an AHS patient was required to perform a concurrent task, compared to standard conditions. This suggests that intentional inhibition depends on effortful central executive resources: "automatic responses to nearby distractors in AHS are suppressed by a resource-limited system that is affected by task load" (p. 86).

3. Neurostimulation and inhibition

Neuropsychological dissociations offer a valuable existence proof for specific cognitive functions. However, they document failure of a mechanism, rather than success. Further, if the lesions are large, they cannot precisely identify the brain circuits that implement the putative inhibition function. In fact, most conventional methods of neuroscience cannot easily identify the neural bases of intentional inhibition, for several reasons. First, laboratory animals may be unsuitable subjects for studying intentional inhibition. Second, the complexity and flexibility of human decisions to inhibit may be difficult to capture in a laboratory experiment. Third, correlational approaches such as neuroimaging may have difficulty distinguishing intentional inhibition from other processes with which it is associated, such as uncertainty, or regret.

Direct stimulation of the brain offers an important alternative method. Because it is interventive rather than correlational, it can separate any process of inhibition from other processes correlated with inhibition. To our knowledge, this data has not yet been reviewed in detail in the cognitive neuroscience literature. We present here a brief overview of neurostimulation data, highlighting the evidence for two distinct neural systems for inhibiting action. These seemingly separate systems may be related to a parallel distinction drawn from other bodies of evidence (such as neuroimaging and neuropsychology).

Recent neurosurgical literature has identified brain areas where stimulation causes slowing or suppression of ongoing movements (Lüders et al., 1995). Clearly, external stimulation will bypass any internal decision to inhibit, so it can say little about the natural circumstances under which this suppression occurs. On the other hand, stimulation offers a well-controlled method that can reveal how the suppressive mechanism functions.

In cases of presurgical evaluation in drug-resistant epilepsy, subdural electrode arrays may be placed on the cortical surface. Each electrode of the array can be stimulated individually to assess the function of the immediately underlying local area of cortex. Similar stimulation techniques can be used intraoperatively. This method has been famously exploited by Penfield and Welch (1951).

Lüders et al. (1995) screened electrodes implanted on the frontal cortex with progressively increasing current intensities. In some electrodes, they found the classical positive motor or sensory responses reported previously (Penfield and Jasper, 1954). Instead of considering the remaining electrodes as simply silent, Lüders et al. went on to perform tests for negative motor responses (NMRs). In these tests, patients were asked to perform rapid alternating eye, tongue, hand or foot movements. While the movement was ongoing, currents would be applied through one electrode of interest. A NMR was reported in cases of clear arrest or slowing of the ongoing movement. Alternative explanations of arrest, such as loss of consciousness, were excluded.

Since the 1950s, over 20 studies have reported NMR upon direct cortical stimulation. The total frequency of NMRs varies dramatically between studies, perhaps reflecting the difficulty of extensive and comprehensive sampling given the strict clinical restrictions of this unique setting.

Negative motor areas (NMAs) have been reported in two distinct clusters, namely the medial frontal cortex, and the perirolandic area (Lim et al., 1994; Mikuni et al., 2006).

Lüders et al. (1987, 1992) found NMA most consistently in the inferior frontal gyrus (IFG) 'immediately in front of the face motor area'. Several studies reported NMAs in the supplementary motor area (SMA) (Penfield and Rasmussen, 1950; Lüders et al., 1988; Fried et al., 1991; Chauvel et al., 1996; Hanakawa et al., 2001; Chassagnon et al., 2008), and the perirolandic area (Uematsu et al., 1992; Nii et al., 1996).

To our knowledge, recent data on NMRs has not been reviewed systematically. More importantly, this literature has not previously been linked to cognitive function, or to action inhibition, in the context of a cognitive task. Indeed, previous discussions of NMR are largely limited to the neurosurgical literature.

Mikuni et al. hypothesised that NMRs are not truly negative, but simply reflect disruption of ongoing action by non-physiological stimulation of excitatory motor centres (Mikuni et al., 2006). In contrast, we suggest that negative motor areas may be a true mechanism for inhibiting action. On Mikuni et al.'s view, the arrest of an ongoing action is, in reality, artificial triggering of a different action, or artificial capture of normal action mechanisms. However, there is clear evidence against this view. First, not all stimulation sites with positive motor effects are able to produce NMRs. Even complex sequences of purposeful action have been evoked by electrical stimulation (Bancaud et al., 1976; Desmurget et al., 2009). If NMRs were the result of disruption of complex action plans by a positive stimulation, then NMRs should be very widespread, and positive motor signs should be rare. Second, this view would predict NMAs to be co-located with positive motor sites, but this is not always the case. In particular, distribution of NMAs is anterior to electrodes eliciting positive motor signs (Uematsu et al., 1992). Third, NMRs are sometimes found at lower intensity than positive motor effects, though this is the opposite of what one might expect if NMR reflected a disruptive effect (Mikuni et al., 2006).

If NMAs are in fact negative, and NMRs are part of a normal physiological inhibitory mechanism, then stimulation data offer an important, well-controlled window onto action inhibition. Two main points emerge from the literature and are relevant here. First, since this mechanism can suppress *already ongoing* movements, NMAs can be considered a mechanism of inhibition that occurs very late in the chain of events. Therefore, NMAs might contribute to the late stopping of action, as opposed to early decisions not to act. Second, the anatomical organization of NMAs into apparently distinct medial and lateral clusters suggests the interesting possibility that these two regions could correspond to the two types of inhibition distinguished in this review. This anatomical distinction had been suggested before, (Ikeda et al., 1992; Kunieda et al., 2004), but no functional relevance had been proposed. We speculate that frontomedial NMAs could contribute to intentional inhibition, while lateral NMAs could be involved in externally triggered inhibition. Indeed, NMRs have been reported in the IFG and preSMA regions (Lüders et al., 1987, 1988; Penfield and Rasmussen, 1949), which has been consistently associated with external inhibitory processing in tasks such as the stop-signal task (Swann et al., 2009).

Verbruggen and Logan (2008) point out that both pre-SMA and IFG are involved in an 'inhibition network', although the division of labour between these areas is debated. NMA data may shed light on the functional and physiological differences between these two inhibition centres. Any future studies combining experimental methods and cognitive tasks with stimulation of NMAs and recording of NMRs would be very valuable.

4. Neuroimaging of inhibition

Neural stimulation suggests that action suppression mechanisms exist within the lateral and medial frontal cortices. However, no stimulation studies have investigated the operations of these mechanisms in controlled experimental tasks. For that, we must rely on a series of functional imaging studies. These have identified the neural correlates of intentional inhibition, and their relations to other inhibitory and action-generating systems. This section compares fMRI results in a number of different experimental tasks and conditions, in order to clarify the workings of intentional inhibition.

Relatively few neuroimaging studies have focussed on intentional inhibition. We therefore included in our analysis studies involving inhibition of higher order processes (e.g., thoughts). The assumption is that thoughts, cravings and emotions may all be inhibited in the same way as actions are. A review of the recent literature identified 7 studies which clearly involved intentional inhibition of either a motor action that the participant was disposed to make; or suppression of thoughts, desires or emotions.

Linking inhibition of action to inhibition of thought and emotion may seem controversial, but we see clear theoretical grounds to defend it. On one important view, thought requires speech and language processes (Davidson, 1975; McDowell, 1994; Wittgenstein, 1953). Another view suggests that language is an input–output system for central cognition and is therefore a channel to transfer thoughts out of or into the mind (Cummins, 1996).

This view is consistent with studies of embodied language which find somatotopic recruitment of motor brain areas according to the somatotopic content of both nouns and verbs (Den Ouden et al., 2009; Meteyard et al., 2010; Pulvermüller et al., 2005). In the some studies of thought inhibition considered here, it is a thought about action that is inhibited. For example, the intentional suppression of cigarette craving (see Brody et al., 2007) shares some important content with the inhibition of actions such as taking a cigarette, smoking, inhaling, etc.

Several theories of emotion suggest that somatomotor actions, and particularly facial expressions, are an automatic part of experiencing emotion (James, 1884; Lange and Kurella, 1887; Wild

et al., 2001; Sato and Yoshikawa, 2007). Therefore, a tight link between emotion and overt motor responses (such as fear evoked by threatening images) may be expected. Furthermore, inhibition of emotions might be mediated by inhibition of the motor responses associated with the emotions.

We begin by a brief description of the classical free-choice paradigms that have been used in neuroimaging of intentional inhibition.

Brass and Haggard (2007) asked participants to voluntarily prepare and execute a simple keypress action on some trials, but on other trials to prepare the action and then withhold it at the last possible moment. Participants freely chose on each trial whether to act or inhibit. Participants reported the time of their intention to act, even on trials where no action in fact occurred, and this was used for event-related fMRI comparisons between action and inhibition conditions.

In this study, participants were free to decide when they would prepare to press a key, and in which cases they would inhibit their actions. The contrast of inhibition vs. action trials revealed BOLD activity in the dorsal fronto median cortex (dFMC). In addition, the analyses revealed a significant correlation between each participant's percentage of inhibited trials and inhibition-related activity in dFMC.

Kühn et al. (2009) asked participants to freely decide between executing and inhibiting a keypress action. Their task provided a prepotent external drive to act, as the action of pressing the key would avoid an unpleasant sound. Some trials consisted of external instructions to either perform or inhibit the keypress. Other trials allowed participants to freely decide what they would do. Intentional inhibition was identified by contrasting trials with a voluntary decision to inhibit with trials with a voluntary decision to proceed with the prepared action.

The contrast between the 'decide-nogo' and the 'decide-go' conditions revealed BOLD activity in dFMC, close to the area reported by Brass and Haggard (2008), though slightly more ventral. As in Brass and Haggard's earlier study, the authors again found a correlation between individuals' probability of inhibition and inhibition-specific BOLD activity.

The authors argue that, given that dFMC appears in this contrast, it cannot be related exclusively to the decision to inhibit itself. Instead, the authors argue through connectivity analyses that the rostral cingulate zone is responsible for the decision, while the dFMC simply expresses the decision outcome "inhibit".

Chung et al. (2006) asked participants to blink in response to occasional disappearance of a fixation point. Crucially, they were instructed at the beginning of the experiment to inhibit blinking at all other times. Thus, while the fixation point persisted, participants had to inhibit an urge to blink which developed progressively, based on a prior instruction that became progressively more and more remote in time. In this sense, the inhibition of blinking represented a trajectory of increasingly internal control. Blocks of instructed eye-blink inhibition lasted for 20–30 s, and were interrupted by periods of 0.5 s during which participants could blink voluntarily. Concurrent EOG recording was used to identify blinks that were made during periods when participants were instructed to blink and when they were instructed not to blink, and these were used for event-related fMRI analysis. Interestingly, this contrast could be interpreted as identifying either the inhibition preceding a blink, or the failure of that inhibition, leading to the blink occurring.

BOLD activity in the precuneus and superior temporal gyrus was associated with eye-blinks during inhibition periods but not with eye blinks during voluntary blinking periods.

Jaffard et al. (2008) assumed that a period of proactive and sustained inhibition occurs in simple reaction tasks, prior to the warning signal that usually precedes the GO signal in a GO/NOGO task. First, the authors contrasted trials with and without a warning signal, arguing that a warning signal constituted a cue to release proactive inhibition of responding. This contrast revealed three areas; two in the medial prefrontal cortex (BA 9 and 10) and one in the left inferior parietal/middle temporal cortex (BA 39/40). Second, they also contrasted BOLD activity related to the go signal on trials with short (100 ms) or long (500 ms) delay between the warning cue and the go signal. The authors reasoned that longer delays would allow for proactive inhibition to be released, and so the BOLD activity related exclusively to trials with short delay would reflect greater levels of proactive inhibitory control. This second contrast again identified the medial prefrontal cortex with greater levels of inhibition, and also found activation of the left posterior cingulate cortex.

Campbell-Meiklejohn et al. (2008) used a gambling task, and reported activations associated with the decision to quit playing (i.e., inhibit further playing actions), in order to prevent further losses.

The contrast between activations related to decisions to quit gambling, vs. decisions to chase losses revealed increased BOLD activity in areas including the bilateral anterior insula, dorsal anterior cingulate, posterior cingulate, parietal cortices and ventral striatum.

Similar results were obtained for inhibition of higher order processes, Kühn et al. (in preparation) asked participants either to decide for themselves or instructed participants whether to feel or suppress emotions for several seconds during which a negative emotion was triggered by an unpleasant image. In order to identify brain regions associated with the voluntary suppression of emotion, the endogenous choice to suppress was compared with the external instruction to suppress the emotion.

Brain regions for both endogenous and externally triggered inhibition of emotion were highly similar to those areas that had been previously identified for endogenous and externally triggered inhibition of motor actions (e.g., Brass and Haggard, 2007; Kühn et al., 2009). Moreover, effective connectivity analyses showed that dFMC exerts greater control onto pre-SMA during internal compared to external inhibition of emotion, as previously found for motor action (Kühn et al., 2009).

Mitchell et al. (2007) asked participants to suppress or to entertain thoughts (about a white bear). Interestingly, their analysis distinguished between sustained suppression throughout a block, and transient failure of suppression when the thought intruded. Transient suppression was assumed to precede a failure to inhibit (participants pressed a button when they had an intrusive, forbidden thought about a white bear). Whereas sustained suppression activated lateral prefrontal cortex, transient failure of suppression activated a more medial area identified as ACC. As in Chung et al.'s (2006) study their analysis reveals activation associated with voluntary inhibition at the point where this inhibition fails. It seems likely that successful inhibition would be associated with stronger activations in the same areas.

Brody et al. (2007) asked 42 cigarette smokers to inhibit their cravings to smoke while exposed to smoking-related cues, or to allow craving. The observation of cigarette handling and smoking may elicit the activation of the motor imagery of the over learned habits associated with cigarette consumption. One main contrast was used to identify brain areas associated with voluntary inhibition of cravings. The authors compared responses to cigarette cues with and without an instruction to voluntarily inhibit cravings. Inhibition was identified by comparing presentation of smoking-related cues and neutral-cues in an event-related design contrasting inhibition with permitted craving. This analysis revealed greater activations in the medial superior frontal gyrus, the left dorsal anterior cingulate cortex and in the posterior cingulate cortex.

Importantly, in both Brody et al.'s (2007) and Chung et al.'s (2006) studies, inhibition was internal in the sense that it was not

repeatedly instructed in every single trial. The decision to inhibit did not therefore come from an immediate external instruction, but from an 'inhibitory context' given by a temporally remote external stimulus. These studies therefore relate to the important difference between immediate and less-immediate external stimuli, with the specific assumption that decreasing immediacy implies increasing 'internality' (Shadlen and Gold, 2004).

Surveying this set of studies shows that the target of inhibition varied considerably, including several different motor effectors, but also other cognitions (thoughts, emotions). There were clear differences between paradigms in how intention to inhibit was operationalised. In some paradigms, intention to inhibit was treated as a sustained task set, specified by an instruction before a block of trials (e.g., Mitchell et al., 2007). In other paradigms, the intention to inhibit was the outcome of an internal momentary decision that should be made during an individual trial (e.g., Kühn et al., 2009). In yet others, intentional inhibition was part of a strategy to optimise performance, either on the immediate task (e.g., Jaffard et al., 2008), or over a longer run of behaviour (e.g., Campbell-Meiklejohn et al., 2008). Moreover, intentional inhibition was identified using several different kinds of contrasts. Interestingly, some of these contrasts in fact included the presence of an action, based on a presumed process of inhibition which in fact *failed* to inhibit (Chung et al., 2006; Mitchell et al., 2007). Table 2 shows the range of different features covered in these studies. Despite this diversity, intentional inhibition consistently produced activation of the medial prefrontal cortex. This commonality was formally addressed by activation likelihood estimation (ALE) analysis in which coordinates within the frontal lobe were entered. We processed the foci by means of Brainmap GingerALE (http://brainmap.org/ale/). We ran the analysis in the standard space of Montreal Neurological Institute (MNI) and converted coordinates reported in Talairach space using formulas provided by Matthew Brett (http://imaging.mrc-cbu.cam.ac.uk/imaging/MniTalairach). The ALE map was thresholded using a false discovery rate of $p < 0.05$ and a cluster threshold of at least 100 adjacent voxels. The thresholded maps were overlaid onto the "Colin" anatomical template. The resulting ALE map can be seen in Fig. 1. The activation associated with intentional inhibition comprised two adjacent peaks. These are both located in the frontomedian wall, and correspond to Brodmanns area 9 and 32. The posterior cluster is on the border between anterior cingulate cortex (ACC) and the dorsomedian prefrontal cortex (dmPFC), but the anterior cluster is clearly distinct from ACC (Table 3a).

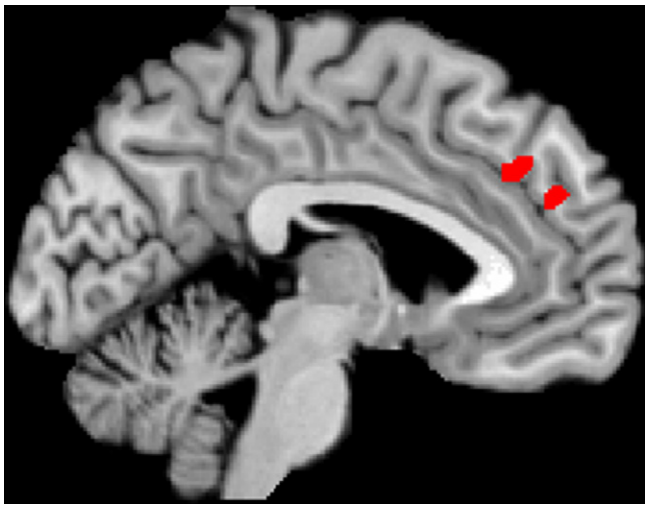

Fig. 1. Results of ALE meta-analysis from 7 intentional inhibition studies. Concurrence can be seen in the medial frontal cortex (BA 9 and BA 32).

4.1. Intentional inhibition is distinct from external inhibition

The clear anatomical substrate for intentional inhibition helps to differentiate it from other forms of inhibition, and inhibition-related processing. First, externally triggered inhibition in stop signal tasks has been associated with two quite different areas, the right inferior frontal gyrus, and the SMA. To formally demonstrate the dissociation between intentional and externally triggered inhibition, we considered BOLD activations found in fMRI studies of the SST. A recent large meta-analysis of the SST (Swick et al., 2011), included the contrasts Stop > Go, Stop > baseline and Successful

Table 2
Neuroimaging studies considered for the meta-analysis of intentional inhibition.

Study	Object of inhibition	Timing of inhibitory signal	Main identified contrast	Areas identified
Brody et al. (2007)	Cigarette cravings	Tonic	Resisting craving in response to cigarette cues, vs. permitted craving	−6, 34, 32 −10, 34, 32 −8, 30, 15 −4, 20, 14 −16, 24, 23
Chung et al. (2006)	Blinking	Tonic, with occasional failures	Inhibition of eye blinking	32, −7, 54 −10, −11, 52 −28, −21, 53 16, −2, 50
Mitchell et al. (2007)	Thinking about a white bear	Tonic (120 s), with occasional failures	Occurrence of a forbidden thought vs. occurrence of a permissible thought	33, 12, 36 24, 15, 36 48, 21, 12
Jaffard et al. (2008)	Key presses	Phasic	Proactive inhibition, just after warning signal or before unwarned target vs. warned target, with long warning signal-target delay	−4, 48, 27 3, 48, 24
Kühn et al. (submitted)	Emotions	Phasic	Voluntary suppression of emotions vs. instructed suppression of emotions	4, 63, 18 −4, 60, 14
Kühn et al. (2009)	Keypress to stop a rolling marble	Phasic	Voluntarily inhibited key press vs. voluntarily pressed key	−7, 42, 21
Brass and Haggard (2007)	Keypress	Phasic	Voluntarily inhibited keypress vs. voluntarily pressed key	−2, 41, 37 −31, 9, −7 32, 18, −10

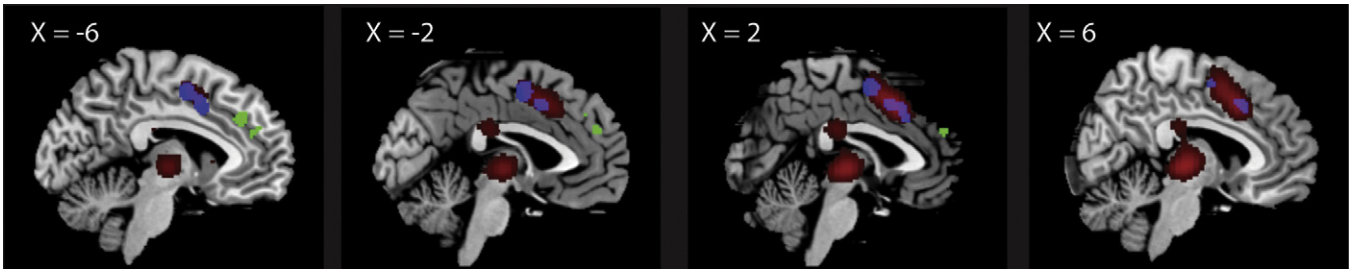

Fig. 2. Results of ALE meta-analysis from 21 stop-signal studies (red) (Swick et al., 2011), 11 response selection studies (blue) and 7 intentional inhibition studies (green). Note overlap in preSMA and SMA of the medial frontal lobe. Also note the clear distinction between the more posterior preSMA coordinates of stop-signal and response selection studies and the more anterior dmPFC coordinates of intentional inhibition studies.

stop > Unsuccessful stop. The resulting ALE map contained major clusters in the left insula, extending into thalamus and putamen; the posterior cingulate (BA 23); right insula, extending to inferior precentral gyri (BA 9) and the superior frontal gyrus (medial BA 6, including the pre-SMA), the right middle frontal gyrus (BA 9), and the right inferior parietal lobule (BA 40). This analysis identifies a network involved in *externally triggered* late inhibition, focussed on a lateral and a medial cluster. Importantly, the network for external inhibition does not overlap with the medial prefrontal areas associated with intentional inhibition. In particular, the medial cluster for external inhibition is clearly posterior to the medial cluster for intentional inhibition.

4.2. Intentional inhibition is distinct from action selection

Second, we have already discussed the relation between intentional inhibition and action selection. In particular, absence of action may result either from an early selection to not act, or from a later suppression of an action already prepared. Therefore, we investigated whether activations associated with intentional inhibition overlapped with frontal lobe activations for action selection. 11 action selection fMRI and PET studies (Cunnington et al., 2002, 2006; van Eimeren et al., 2006; Hyder et al., 1997; Jahanshahi et al., 1995; Jenkins et al., 2000; Lau et al., 2004; Liddle et al., 2001; Müeller et al., 2007; Weeks et al., 2001; Wiese et al., 2004) were considered.

Overall 47 foci of frontal and insula activation, resulting from the contrast self-initiated action > externally triggered response or self-initiated action > rest, were included. The analysis was otherwise identical to that for stop signal tasks. The resulting ALE map in Fig. 2 shows preSMA and SMA activations in action selection (in blue, Table 3c), stop-signal inhibition (in red) and for intentional inhibition (in green). Note that the intentional inhibition activations are more anterior than the other clusters. This analysis suggests that the activation associated with early selection of voluntary actions is distinct from the activation associated with late intentional inhibition of actions that are already prepotent.

In summary, quantitative meta-analysis provides support for dissociating a medial frontal activation for intentional inhibition from (a) the activations for externally triggered inhibition, and from (b) internal action selection decisions. Regarding the distinction between intentional and externally triggered inhibition (a), our meta-analysis provides an empirical basis for the distinction between internal and external inhibition, which we originally

Table 3
Statistical concurrence in the frontal lobe observed across studies on intentional inhibition, externally triggered inhibition (SST, from Swick et al., 2011) and response selection.

Anatomical region	Brodmann area	Coordinates (MNI)			Volume (mm^3)
		x	*y*	*z*	
(a) Intentional inhibition					
Dorsal-frontomedian cortex, extending into anterior cingulate cortex	32/9	−8	35	32	648
Dorsal-frontomedian cortex	9	−2	47	25	352
(b) Externally triggered inhibition (stop signal task, see Swick et al., 2011 for full results)					
Left insula	–	−40	14	0	21,648
Right insula	–	38	16	2	13,776
Right medial frontal gyrus	6	4	14	44	10,640
Right middle frontal gyrus	6	26	40	34	3472
Right inferior parietal lobule	40	58	−40	26	3088
Right lentiform, lateral globus pallidus	–	14	6	0	1944
Left superior temporal gyrus	13	−50	−40	16	1192
Right inferior occipital gyrus	19	44	−70	−8	808
Left superior frontal gyrus	9	−34	36	28	704
Right middle frontal gyrus	6	28	−4	46	600
Left superior parietal lobule	7	−24	−62	42	528
Left precentral gyrus	9	−40	4	32	344
Left middle occipital gyrus	18	−36	−84	0	208
Right superior temporal gyrus	22	46	−26	0	152
Right superior parietal lobule	7	26	−56	46	144
(c) Response selection					
Anterior cingulate cortex	24/32	−5	6	45	4664
Right dorsolateral prefrontal cortex	9	37	34	20	3080
Left insula	13	−39	9	8	712
Left precentral gyrus	4	−37	−10	57	376
SMA	6	11	19	62	192
Left dorsolateral prefrontal cortex	9	−35	35	31	128

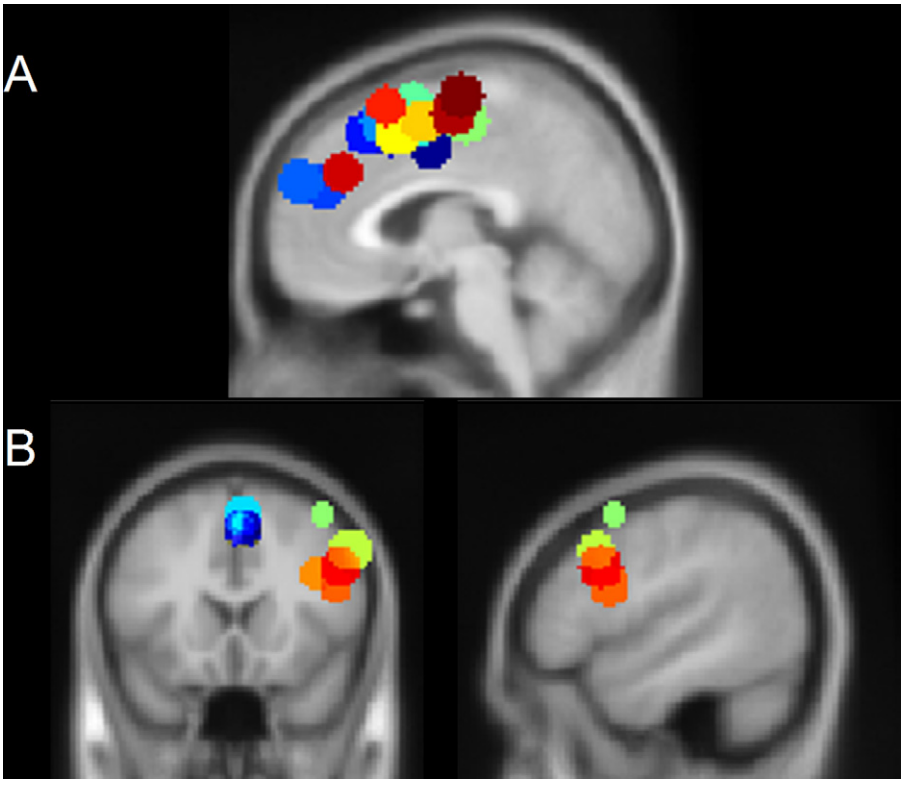

Fig. 3. Approximate reconstruction of brain regions from which negative motor responses (NMR) were elicited by means of electrical stimulation of the brain. Results from each study are plotted as a 10 mm sphere by means of MarsBaR (http://marsbar.sourceforge.net) overlaid onto the MNI template brain. The figure includes (A) 14 medial and (B) 5 lateral foci (depicted as a sphere with a radius of 10 mm) taken from 25 separate studies (of which 17 report medial and 8 report lateral sites in which NMR can be elicited). MNI coordinates are as follows: (A) $x = 2$, (B) $y = 13$, (C) $x = 50$. Left lateral foci were projected to the right hemisphere for display purposes.

suggested above on conceptual grounds. Regarding the distinction between intentional inhibition and action selection (b), Mostofsky and Simmonds (2008) recently suggested that activations in external inhibition tasks may sometimes reflect selection between two alternatives (to act or not), rather than suppression of motor output per se. Analogously, one might ask whether the medial frontal activation for intentional inhibition could reflect an early selection between alternatives of action or non-action, rather than suppression of an action already prepared. Our meta-analysis answers this question with a robust 'no': free selection of actions consistently activated the SMA and pre-SMA, quite distinct from the more anterior clusters in dmPFC associated with intentional inhibition.

Finally, could the activation we found associated with intentional inhibition in fact reflect some alternative process? Epiphenomenal activation is always a risk with neuroimaging, particularly when an area is very frequently activated across many different tasks and paradigms. For example, the ACC, immediately adjacent to the intentional inhibition area, participates in a spectrum of monitoring operations, including conflict monitoring, negative evaluation and self-relatedness (MacDonald et al., 2000). However, we do not believe that intentional inhibition amounts to conflict monitoring. First, when participants are instructed to withhold actions on approximately 50% of trials (Brass and Haggard, 2007), the degree of conflict and the requirement to monitor it are presumably equal on action and inhibition trials. Second, accounts based on monitoring cannot easily explain activations during tonic, sustained inhibition, since this presumably requires little monitoring. Similarly, explanations based on negative evaluation would need to explain why similar activations are found both when people successfully intentionally inhibit action (e.g., Brass and Haggard, 2007; Brody et al., 2007), and when they fail to do so (Chung et al., 2006; Mitchell et al., 2007).

5. Functional role of intentional inhibition and a cognitive model

We have identified mechanisms for intentional inhibition in the human frontal cortex. In this section, we develop a model which suggests how intentional inhibition contributes to the normal generation of action. We treat intentional inhibition as an extension of standard computational models for control and adjustment of simple goal-directed actions (Haruno et al., 2001). These models are typically based on a predictive internal feedback loop which checks whether the predicted consequences of the current command match the intended goal. Any mismatch will trigger a corresponding adjustment of the motor command, and the process will iterate until the goal is met.

Intentional inhibition expands this model in two ways. First, we postulate an additional, outer-loop, which serves to check whether the current action goal itself is or is not appropriate. This additional loop is not simply a hierarchically prior mechanism that selects what action to perform next. Rather, it makes an additional set of predictions about longer term consequences and implications of the current action, in addition to predicting whether it will achieve the proximate goal. Consider the example of the angry with which we began this article. We may want to express our feelings, but a quick check shows that this is not in our long-term interest, so the proximate goal should be abandoned. Most importantly, the hand movement to actually click on the send button should be stopped as soon as possible. To expand the example, the inner feedback loop might use 'send email' as a proximate goal – or P intention (Pacherie, 2008). The inner loop therefore predictively controls the button-click action that sends the email, and adjusts the hand trajectory accordingly. At the same time, the brain contains an additional loop that predicts longer term, distal consequences of

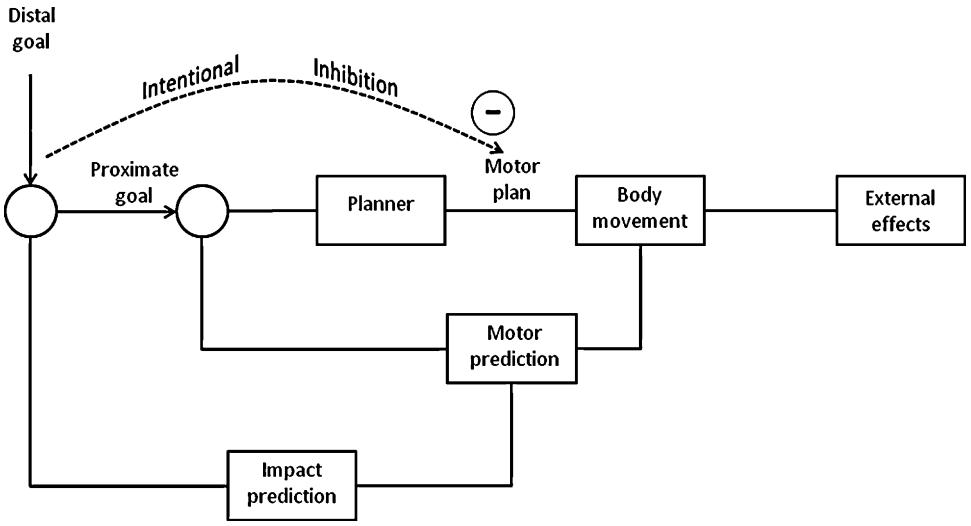

Fig. 4. Intentional inhibition as an extension of computational motor control models. See text for explanation.

sending such an The second loop therefore contains an "impact predictor" that estimates distal consequences, and compares them to general goals – or D-intentions (Pacherie, 2008) – in the normal way. The key feature of the model is the flow of information from a proximal to a distal loop.

If the distal loop shows that the forthcoming action will have undesired impact, this will recruit a specific braking process to suspend and suppress the current action. This process, indicated by a dashed arrow in the figure, implements a flow of feedforward inhibition from the distal to the proximal loop. We emphasize that the intentional inhibition signal is continuously regenerated by the outer loop. The outer loop comparator therefore allows *continuous* and *iterative* checking of whether the current action is appropriate, or should be stopped. Many models emphasize the serial nature of action planning: "think before you act". We suggest that intentional inhibition may be an exception to the broadly serial flow of action control: the brain may monitor and *continually* check the appropriateness of the current action, with the possibility of inhibiting action even after it has begun. Thus, the model of Fig. 3 is hierarchical, but not serial.

Intentional inhibition, then, is the process that links the outer, distal loop and the inner, motor loop, as shown by the dashed arrow of Fig. 4. Negative motor responses may provide an artificial way to activate this mechanism. Further, neuroimaging studies of intentional inhibition may identify the anatomical basis of this mechanism, as we have shown above. Crucially, intentional inhibition does not simply reset the proximate goal to a default non-action state. Resetting the proximate goal to non-action would be equivalent to an action selection process in which non-action is simply another alternative, as in 'early' inhibition decisions. However, we showed above that intentional inhibition and action selection are dissociable mechanisms. Rather, the suppression *first* inhibits the current motor command, to prevent the undesirable action. Then, and only then, is it appropriate to reselect the appropriate action. Put another way, the ability to reconsider and reselect is not useful by itself (indeed it is part of the standard motor control model). But the ability to first stop, and *then* reconsider is a crucial element of flexible cognitive control.

In this sense, the inner, proximal loop comparators, and the outer, distal loop comparator are dramatically different. Whilst the classic internal feedback loop acts by slightly modifying ongoing action plans, the outer, distal loop comparator does not. Rather, the outer loop comparator completely *suspends* the ongoing operation of the inner action control loop, by resetting the motor command to zero. This then allows reprogramming of a novel action.

6. Conclusions and social implications

We have introduced the concept of intentional inhibition, and reviewed the evidence for its existence in the human brain. We have distinguished it from other concepts in action control, and sketched a model for how intentional inhibition interacts with other elements of action control.

Human action is widely recognized to have 'freedom from immediacy' (Shadlen and Gold, 2004), meaning that our actions depend on wide integration of information beyond the current stimulus, such as memory for previous experience and predictions of future outcomes. Specific cognitive abilities and cortical structures have evolved that make flexible, longer range action possible. There is then a possibility that immediate goals and longer range goals can conflict (Dayan et al., 2006). Our review of anarchic hand shows that these conflicts create a problem of self-control. We suggest that intentional inhibition provides one possible mechanism for resolving conflicts between these two action control systems.

We end with some speculations on the potential social significance of intentional inhibition. Humans are social animals with a sophisticated pattern of interactions, based on reciprocity, and the assumption that we will continue to interact with other people in the future, as we interact with them now. We suggest that intentional inhibition becomes particularly important in social settings, because they potentially produce conflict between proximal and distal goals. An individual's proximal goals can be detrimental to others.

To return to our initial example, sending the angry may make the sender feel better right now, but may upset their boss. If the boss is upset, then she might reduce the employee's annual bonus payment, which will clearly compromise their distal, longer term goals. Individuals always balance their own short-term benefits against their own long-term benefits. However, in complex social settings, an individual's long-term benefits critically depend on mediation by other people. Some forms of social interaction may particularly require the ability to intentionally inhibit actions that are immediately appealing. All human societies have a concept of moral responsibility for action which presupposes the capacity for intentional inhibition: the individual could have refrained from an action that they made. This concept of responsibility is a cultural

mechanism for directing the cognitive capacities of intentional action and intentional inhibition to serve wider social needs.

Acknowledgement

We thank Diane Swick for providing the results from the SST meta-analysis.

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