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Trends in Cognitive Sciences

Feature Review

Neural mechanisms of domain-general inhibitory control

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Inhibitory control is a fundamental mechanism underlying flexible behavior and features in theories across many areas of cognitive and psychological science. However, whereas many theories implicitly or explicitly assume that inhibitory control is a domain-general process, the vast majority of neuroscientific work has hitherto focused on individual domains, such as motor, mnemonic, or attentional inhibition. Here, we attempt to close this gap by highlighting recent work that demonstrates shared neuroanatomical and neurophysiological signatures of inhibitory control across domains. We propose that the regulation of thalamocortical drive by a fronto-subthalamic mechanism operating in the β band might be a domain-general mechanism for inhibitory control in the human brain.

Highlights

Inhibitory control is a fundamental mechanism for adaptive behavior and cognition that features in theories across psychology and cognitive science.

Although inhibitory control is thought to regulate processes ranging from actions to memories, most neuroscientific work studies these domains of inhibition separately.

Recent crossdomain comparisons have converged on the view that inhibitory control arises from common, domain-general neural mechanisms.

We propose that an input inhibition mechanism, acting via fronto-subthalamic inhibitory pathways may contribute to domain-general inhibitory control by suppressing thalamic drive of cortical activity.

We argue that β-band activity in local circuits constitutes a potential domain-general signature of inhibitory control.

The gap between motor and cognitive inhibition

Enacting complex, goal-directed behavior requires the ability to flexibly adjust ongoing thoughts and actions in response to changes in goals or circumstances. To achieve this, humans engage cognitive control functions to override habitual behavior and to adjust their thoughts and actions according to their goals. **Inhibitory control** (see Glossary) – the ability to suppress activated, but outdated or otherwise inappropriate representations or processes – is one of these cognitive control functions. Inhibitory control features in many cognitive theories as a process that controls diverse mental operations. Indeed, several influential cognitive control frameworks posit that inhibition is one of three 'general purpose control mechanisms that regulate the dynamics of human cognition' [1–3].

Consequently, inhibitory control is found in models across the entire spectrum of cognitive and psychological science (Figure 1). Inhibitory control processes ostensibly underlie the ability to stop inappropriate actions [4–7], the ability to suppress outdated or unwanted mnemonic representations [8–13], and the ability to avoid distractions from an active focus of attention [14–17]. Inhibitory control is also a key variable in many models of personality [18–20] and its normal [21–24] and abnormal [25–27] development. Prominent models of language processing also feature inhibitory control, for example during the suppression of semantic or phonological competition or during language processing in bilinguals [28–33].

In line with this breadth of cognitive processes that ostensibly involve inhibitory control, its deficits purportedly contribute to symptoms of many neuropsychiatric disorders. Prominent among them are attention-deficit hyperactivity disorder (ADHD) [34], substance use disorders [35,36], post-traumatic stress disorder [37,38], anxiety disorders [39,40], eating disorders [41], Parkinson's disease [42], and many others.

The importance of inhibitory control across such diverse mental contexts begs an obvious, but neglected question: is inhibitory control truly a unitary, domain-general process? Does stopping an action recruit the same mechanism(s) as, for example, suppressing an unwanted memory, an inappropriately activated word, or an outdated attentional representation?

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Cognitive neuroscience provides a powerful approach to answer this question: identifying a neural circuit or signature for inhibitory control in one domain would allow testing whether this same circuit underlies inhibitory control in other domains. Until recently, however, few studies have compared inhibitory control mechanisms across domains. Instead, most cognitive neuroscience work on inhibitory control has investigated this function within separate areas of behavior and cognition. For example, the last three decades have witnessed the development of a large body of research on how inhibitory control influences the motor system to stop overt physical actions (for reviews, see [6,43–45]). Paralleling this, during a similar period, other cognitive and cognitive neuroscience research has focused on inhibitory control of memory (for reviews, see [11,13,46]) and other domains such as affect and attention [47,48]. Such work has – until recently – rarely demonstrated mechanistic connections between the inhibitory control processes in these separate domains, leaving the existence of a domain-general inhibitory control mechanism unaddressed.

This disconnect is especially notable because the vast majority of neuroscientific work on inhibitory motor control uses the stop-signal task (other tasks, such as the Go/NoGo or anticipated response inhibition tasks are also used). The stop-signal task is popular partially because an influential cognitive model (the 'horse-race' model, [4,49] allows a parameterization of the pro- and antikinetic processes involved in action stopping, providing a helpful benchmark that may be related to purported neural signatures of inhibitory control [50]. Notably, the seminal paper formulating this horse-race model was titled 'On the ability to inhibit thought and action: a theory of an act of control'. This title highlights the longstanding aspiration that the processes simulated in stop-signal tasks may indeed be domain-general – that is, that the knowledge gained from studying inhibitory motor control in the stop-signal task may ultimately generalize to the inhibitory control of mental operations more broadly.

However, whereas recent work has indeed shown a relationship between the cortical regions activated during motor response inhibition and inhibitory dysfunction in complex real-life situations [51], mechanistic neural models of how these activated cortical regions may inhibit both motor and nonmotor activity have only recently been entertained [52–55]. Here, we aim to specify key elements of a mechanistic account of how domain-general inhibitory control could be achieved. In particular, we propose a broad mechanism that may contribute to how both motoric and cognitive processes are canceled by suppressing driving thalamic input to those cortical regions – activity essential to sustain and enact the targeted processes. This input inhibition process differs from and complements another common conceptualization of the mechanisms of inhibitory control in terms of target inhibition (Box 1).

First, we briefly introduce the neuroanatomy and neurophysiology of the inhibitory motor control circuit. We focus on work using stop-signal tasks in humans, which provides the most comprehensive evidence for fronto-subthalamic involvement and associated β-band (~13–30 Hz) signatures in the local field potential. We then outline how that same neuroanatomic motif – inhibitory control of thalamocortical drive via fronto-subthalamic pathways, signified by neural β-band dynamics – may contribute to cognitive inhibition. Finally, we present recent preliminary evidence supporting the possibility of such shared mechanisms underlying motor and nonmotor inhibition.

Neuroanatomy of inhibitory motor control: fronto-subthalamic pathways

We begin with a brief review of the neural basis of inhibitory motor control in the human brain (for more complete reviews of the topic, see [5,45,56,57]), because the fronto-subthalamic pathways and β-band activity that are key to the current proposal have been examined most thoroughly in this domain. We then propose that analogous neural mechanisms may contribute to inhibiting nonmotor processes.

Glossary

Cued cognitive inhibition: Inhibition of cognitive representations or processes (e.g., a memory content) that occurs because participants are explicitly instructed to inhibit them by the experimental task.
(Hemi)ballism: The pathological presence of excessive, involuntary movements after damage to the basal ganglia, especially the subthalamic nucleus.
Incidental cognitive inhibition: Inhibition of cognitive representations or processes (e.g., a memory content) that occurs because of the incidental activation of the inhibitory fronto-subthalamic circuitry, typically by a salient event.
Inhibitory control: The ability to suppress activated, but outdated or otherwise inappropriate representations or processes.
Memory intrusions: A cue briefly elicits recollection of its associated target memory, which then has to be canceled or purged.
Stop-signal reaction time (SSRT): A latent variable derived from behavior in stop-signal tasks that expresses the speed of the compound stopping process, which is otherwise not overtly observable.
Subthalamic nucleus: A small, diencephalic region that is functionally part of the basal ganglia and crucial for (motor) inhibition. It is the final nucleus that is common to both inhibitory basal ganglia pathways before they reach the output nuclei of the basal ganglia. Damage to the nucleus can result in (hemi)ballism and modulation of its activity is effective in treating movement disorders such as Parkinson's Disease.
Thalamocortical drive: Persisting excitatory innervation by the thalamic nuclei that sustains firing in their projection targets, for example in cortex or hippocampus.

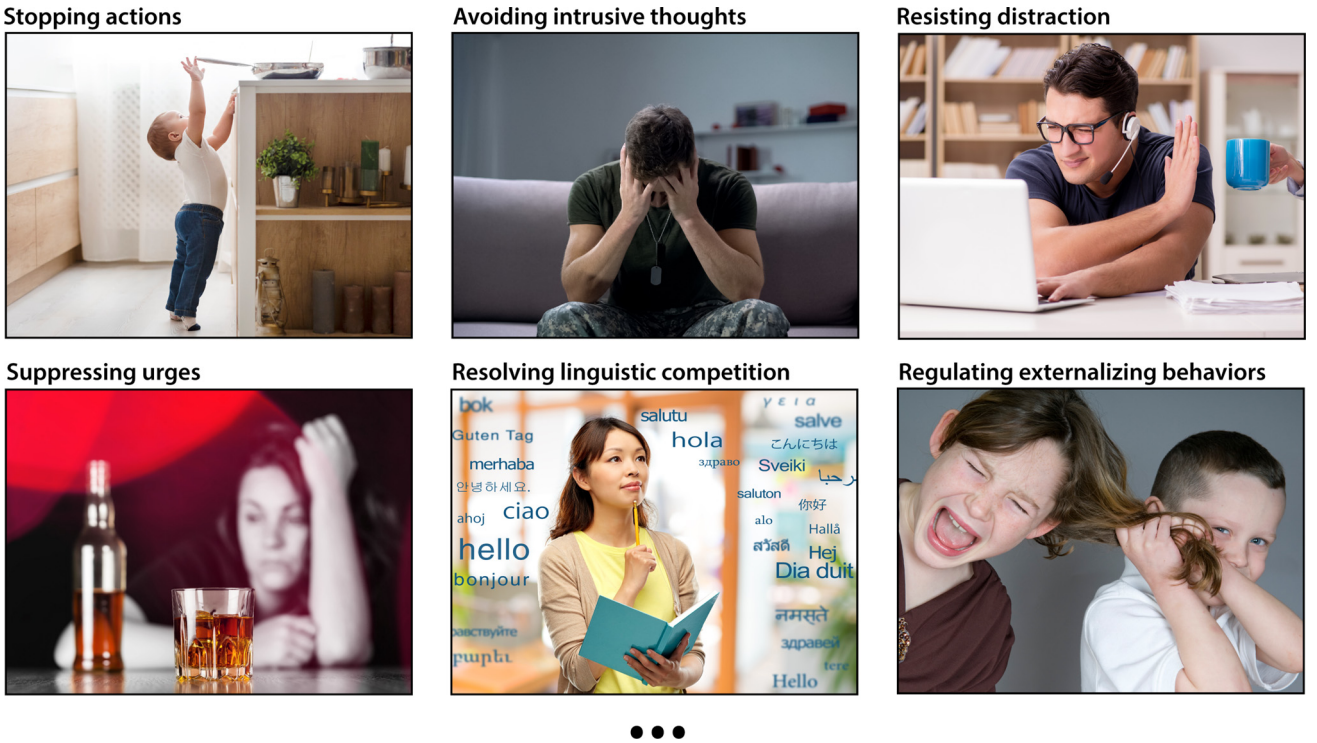

Figure 1. The many facets of inhibitory control. Inhibitory control processes are purportedly involved in the regulation of many motor and cognitive processes. Images licensed via shutterstock.

At rest, persisting excitatory innervation by the ventral thalamic nuclei sustains firing in the motor cortex as its default state [58–60], a process known as **thalamocortical drive**. This sustained thalamocortical drive does not induce overt motor action because subcortical basal ganglia, in turn, exert steady inhibition that prevents this from occurring [56]. In healthy individuals, initiating a movement shifts this delicate balance of inhibition and excitation towards disinhibition, upregulating thalamocortical drive [61]. To refine these movements, three loop-like corticobasal ganglia–thalamocortical circuits more precisely modulate the excitation–inhibition balance [56,62]: the prokinetic, net-excitatory direct pathway and the antikinetic, net-inhibitory indirect, and hyperdirect pathways [63–65]. All three pathways ultimately converge on the output nuclei of the basal ganglia: the internal part of the globus pallidus (GPi), and the substantia nigra pars reticulata (SNr). These output nuclei can inhibit the motor segments of the ventral thalamus [66] and in doing so, suppress thalamocortical drive, shifting the balance back towards inhibition. Thus, whether a given pathway activates the GPi/SNr or deactivates it at the end of the corticobasal ganglia chain determines whether that pathway achieves net inhibition or net excitation: activating GPi/SNr net-inhibits the drive from ventral thalamus to motor cortex and suppresses movement, deactivating GPi/SNr net disinhibits thalamocortical drive and invigorates movement.

The three foregoing pathways crucially differ in the upstream structures that innervate the GPi/SNr. In both inhibitory pathways, a small diencephalic structure known as the **subthalamic nucleus** (STN) excites the GPi/SNr, net-inhibiting thalamocortical drive and, consequently, movement. Damage to the STN produces disinhibitory **(hemi)ballism** [67], illustrating its key causal role in motor inhibition. Beyond their effects on thalamocortical drive, the three pathways

Box 1. Target inhibition versus input inhibition

Research on inhibitory control often distinguishes between the source of top-down inhibitory control and the target representation that is to be stopped. Control mechanisms can reduce the influence of a currently active target representation or process on ongoing behavior and thought in many ways. A common mechanism widely hypothesized in research on the inhibitory control of cognition is one in which the control source acts directly on the representation or process to be canceled, reducing its activation via an active process that renders the representation less accessible (Figure I, left). We term this broad approach target inhibition, reflecting the action of control on the very representation whose activity requires regulation. For example, in resolving the meaning of the homograph 'Bank', an inhibitory process might suppress the contextually inappropriate meaning (financial institution) in favor of the contextually appropriate one (river's edge), by a process that removes activation from the inappropriate meaning. Ample evidence establishes disruptive effects of inhibition on to-be-rejected representations, which often show persisting deficits in accessibility on later trials or tests, taken to indicate aftereffects of the subtractive process [11,13]. Neural evidence for persisting aftereffects of suppressing particular representations also exists; for example, suppressing retrieval of a visual object, given a cue, renders that visual object harder to see on a later perceptual test, an effect accompanied by a persisting reversal of adaptation effects in sensory cortex for the suppressed content [188,189].

Another approach to inhibiting processes, however, instead involves preventing the accumulation of activity in the system needed to produce the response by suppressing driving input into the units representing them (Figure I, right). In this input inhibition approach, the control mechanism does not act directly on the representation or process to be stopped, but rather acts upstream of it to prevent the propagation of vital input needed to sustain or enhance its activity. Consequently, in an input inhibition approach, it need not follow that the to-be-stopped process or representation will show persistently reduced accessibility on subsequent trials or tests, as no subtractive process acts directly on it, but instead operates by suppressing input into it. Notably, however, input inhibition is not *per se* incommensurate with the presence of inhibitory after-effects. For example, inhibiting thalamic inputs to the motor cortex, allows steady inhibitory input from the basal ganglia to dominate activity in motor cortex, effectively inhibiting it (see upcoming section, 'Neuroanatomy of inhibitory motor control: fronto-subthalamic pathways') In the current proposal, we conceptualize the action of the fronto-subthalamic inhibitory control pathway as acting via a process of thalamic input inhibition (by reducing thalamo-cortical drive), rather than via target inhibition. It remains an open question, however, whether input inhibition and target inhibition are mediated by different aspects of the proposed inhibitory control circuit, or whether other, distinct control pathways complement input inhibition in the regulation of action and thought.

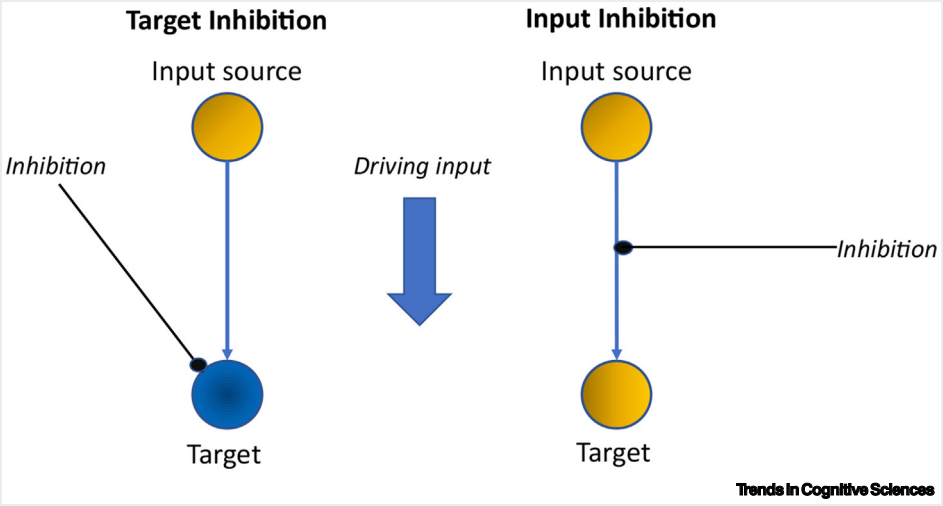

Figure I. Target inhibition versus input inhibition.

also differ according to whether these upstream structures include the striatum (which is true for the direct and indirect pathways) or not (which is true for the hyperdirect pathway). Whether a pathway includes the striatum, en route to the GPi/SNr, strongly influences how targeted and selective the impact of that pathway is on thalamocortical motor activity. For example, individual striatal neurons can target specific neurons in the basal ganglia output nuclei (GPi/SNr, [68],

thereby selectively influencing specific thalamocortical motor loops. In contrast, the STN contains many neurons that broadly connect to those output nuclei [65]). Thus, without striatal fine-tuning (i.e., in the hyperdirect pathway), subthalamic net-inhibition of thalamocortical motor activity occurs rapidly, broadly and nonselectively; with striatal fine tuning (as would occur in the indirect pathway), net inhibition occurs more slowly and selectively (Box 2).

Building on the preceding observations, one influential model of inhibitory motor control in the human brain posits that when a person rapidly stops an action, the prefrontal cortex recruits the hyperdirect pathway via monosynaptic projections from prefrontal cortex to the STN [43]. Other pathways may be involved, however, when more refined control is required. For example, increasing evidence indicates that the indirect pathway also contributes to stopping action, likely in a slower and more selective fashion [45,69,70].

Historically, however, discussion about the neural mechanisms of inhibitory motor control has centered on the specific prefrontal cortical areas that invigorate antikinetic basal ganglia pathways (though not every proposal holds that a given cortical region inhibits action via the basal ganglia; many do not specify how cortical regions influence motor cortex). Early proposals suggested that ventromedial prefrontal cortex implemented inhibitory control, based on extensive nonhuman work showing that lesions of that region caused disinhibited, impulsive behaviors [71–73]. Later work suggested that these findings arose because those lesions had severed white matter tracts passing through this structure, connecting remote regions that actually implemented motor inhibition [74,75]. By then, functional magnetic resonance imaging (fMRI) research during the 1990s already revised this picture, with multiple studies demonstrating right-hemisphere dominant prefrontal activity outside of ventromedial PFC in humans performing a Go-NoGo task [76–79]. While the Go-NoGo paradigm can be limited in its ability to reliably operationalize inhibitory motor control [80,81], an influential lesion study of action stopping confirmed right-lateralized (pre)frontal cortical involvement using the stop-signal task and the horse-race model [82]. This study found an association between elongated **stop-signal reaction times (SSRT)** and lesion volume in

Box 2. Selective versus nonselective inhibitory control

Inhibitory control of motor – and perhaps nonmotor – activity can be exerted selectively or nonselectively, with a trade-off between the two. Nonselective inhibition often arises when actions must be stopped rapidly, reactively, and unpredictably. Such nonselective inhibition yields a broad physiological suppression of motor excitability: successfully stopping an action; for example, in the stop-signal task, suppresses corticospinal excitability even for task-unrelated muscles [54,190,191] and increases β activity over task-unrelated aspects of the motor cortex [112,192]. Foreknowledge of an upcoming stop-signal or of the exact effector to be stopped decreases this nonselective suppression, leading to more selective effects [193–195]. In the motor domain, this difference in selectivity may map onto the hyper- and indirect pathways, with the hyperdirect pathway implementing nonselective inhibition, and the indirect pathway implementing selective inhibition [45,196]. Notably, hyperdirect pathways exist from many regions of the prefrontal cortex to the STN, at least in nonhuman primates [197] (see also [198] for evidence from human probabilistic tractography). The same is true in humans, at least for two out of the three areas commonly implicated in inhibitory control: the rlFC [88] and the DLPFC [89].

Striking parallels between nonselective and selective motor inhibition exist in inhibitory control over memory. For example, several reports demonstrate that actively suppressing retrieval broadly compromises hippocampal function, rather than simply suppressing the individual to-be-avoided memory [199,200]. For example, suppressing retrieval of a response word (e.g., roach), given the cue (e.g., ordeal) from a previously studied word pair (e.g., ordeal roach), not only harms memory for the response itself, but also entirely unrelated pictures encoded 5–10 s before or after the retrieval suppression attempt [199]. This amnesic shadow effect has been linked to systemic hippocampal suppression that degrades encoding and stabilization processes generally, perhaps paralleling the previously discussed nonselective effects of action stopping on the motor cortex. In contrast, selectively retrieving a target memory inhibits competing memories (i.e., retrieval-induced forgetting), impairing their retention, but does not disrupt memory for temporally adjacent items [199], not does it suppress hippocampal activity [201]. Whether these instances of global and selective inhibition in memory and motor inhibition are produced by similar frontothalamic mechanisms remains to be examined.

the right inferior frontal gyrus (rIFG) and the middle frontal gyrus in the dorsolateral prefrontal cortex (DLPFC). Subsequent fMRI and magnetoencephalographic work confirmed that action-stopping activates rIFG, DLPFC, and the pre-supplementary motor area (pre-SMA, [83,84]; see [85] and [52] for recent meta-analyses). Ongoing discussions in this field focus on the exact role of each of the cortical areas involved in this network [52,86,87] (Box 3), whether they trigger stopping via hyperdirect or indirect connections to the STN [88,89], and whether one or multiple inhibitory processes contribute to action stopping [45,70,90]. We point the reader to recent reviews for an up-to-date picture of these issues (see preceding text).

Relative to research on the prefrontal cortex, less controversy surrounds the evidence that action stopping recruits STN. Although imaging this nucleus via fMRI is difficult [91,92], direct recordings of the intracranial local field potential in humans consistently show that successful action stopping engages STN [88,93–100]. Two recent studies provide especially compelling evidence regarding the STNs role in inhibitory motor control. First, electrical stimulation of the STN antidromically propagated to rIFG at very short latencies (~2 ms), demonstrating a monosynaptic connection between both nodes (Box 1). The latency of sequential activation of this fronto-subthalamic pathway after a stop signal was directly related to SSRT [101]. Second, modulating the STN via deep brain stimulation eliminated the typical physiological effects of action stopping on corticomotor excitability, as assayed by transcranial magnetic stimulation of motor cortex [102] (Box 1).

In sum, whereas the exact functions of different prefrontal cortical areas remain to be established, clear evidence supports the causal role of one (or more) fronto-subthalamic circuits in the implementation of inhibitory motor control (Figure 2).

Neurophysiology of inhibitory motor control: β activity as an inhibitory signature

Neural activity in the above-described fronto-subthalamic pathways for inhibitory motor control is dominated by signatures in the β-band (Figure 2B). We here provide a short overview of these signatures, while pointing the reader to comprehensive reviews for further detail [45,103–105].

Broadly speaking, initiating a movement involves reducing the inhibition of the motor system that is present at baseline (see preceding text). Marking this motor system disinhibition, β-band signals in motor cortex reduce during action initiation, relative to their dominance at rest [106–108].

Box 3. Prefrontal cortical contributions to inhibitory control

Research on inhibitory control over actions and thoughts historically has adopted differing emphases about which prefrontal regions originate top-down control signals. Whereas research on retrieval stopping has emphasized the right DLPFC [46,53,157], research on action stopping has emphasized right VLPFC [43]. Numerous studies demonstrate robust rDLPFC activation during retrieval suppression and effective connectivity analyses indicate that this structure causally modulates hippocampal activity [159,201–203]. However, a recent fMRI-based within-subjects comparison of action and retrieval stopping revealed both right anterior DLPFC (BA 9/46/10) and rVLPFC involvement across both domains (see Figure 5A in main text), with both regions showing: (i) correlations with both SSRT and suppression-induced forgetting; (ii) significant cross-task decoding; and (iii) dynamically varying effective connectivity with hippocampus or with motor cortex, depending on the nature of the content to be suppressed. Dynamic causal modeling established that a model including both structures strongly outperformed models featuring only either rDLPFC or rVLPFC as the source of inhibitory control. A complementary conjunctive meta-analysis further demonstrated that domain-general inhibitory control activates both anterior rDLPFC and rVLPFC. Consistent with the view, rDLPFC, like rVLPFC (i) originates hyperdirect projections to STN that could support rapid stopping [89,197]; (ii) exhibits increased activity during action-stopping in intracranial recording in humans [109,204] and nonhuman primates [205]; and (iii) when stimulated intracranially in nonhuman primates, can induce animals to withhold their motor responses [205]. Hence, there is as yet no strong empirical basis for attributing rDLPFC or rVLPFC as the primary origin of domain-general inhibitory control.

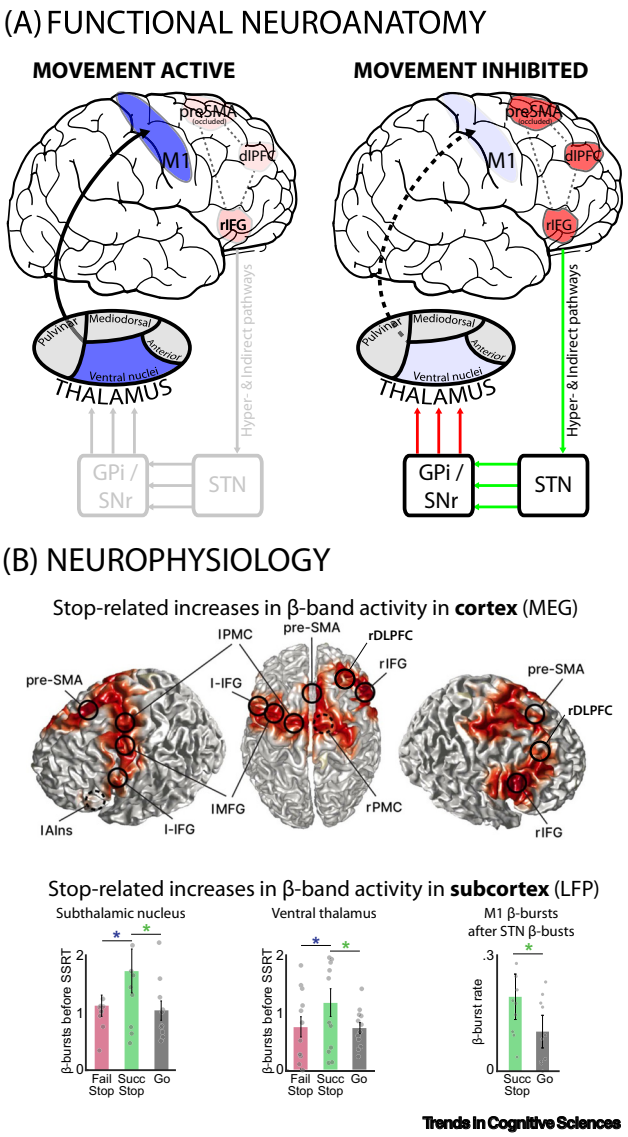

Figure 2. The fronto-subthalamic inhibitory control circuit during action stopping. (A) Stop-signals trigger activity in (pre)frontal cortex (rIFG, DLPFC, and pre-SMA). Pathways from these regions activate STN, which excites the output nuclei of the basal ganglia (GPi), thus inhibiting thalamocortical drive of motor cortex. (B) β-Band activity in the critical nodes of the circuit during action-stopping. Top: cortical β-band activity measured by magnetoencephalography during successful action-stopping shows concentration around rIFG, pre-SMA, and DLPFC (middle frontal gyrus) and motor cortex. Adapted, with permission, from [84]. Bottom: β-band activity measured using intracranial recordings from subcortex shows increases in β during successful stopping in STN and thalamus, and β increases in primary motor cortex (M1) immediately after STN β, with permission, from [100]. *P < 0.05. Abbreviations: DLPFC, dorsolateral prefrontal cortex; GPi, globus pallidus; pre-SMA, pre-supplementary motor area; rIFG, right inferior frontal gyrus; STN, subthalamic nucleus.

Conversely, studies using many neurophysiological methods consistently show that when actions have to be stopped and the motor system has to be returned towards inhibition, β-band activity in all regions of the fronto-subthalamic circuit rapidly increases. In intracranial recordings [109] and in magnetoencephalography (MEG) [84], stop-related β power increases are found in rIFG, DLPFC, and pre-SMA. Studies using scalp recordings have found that this stop-related β-band activity over (pre)frontal cortex is rapidly followed by an upregulation of β over motor cortex [110–112] along with a broad suppression of corticomotor excitability [113]. Lesions to rIFG abolish these (pre)frontal β signals [87]. Based on our theoretical model, one might expect a similar pattern arising from lesions to pre-SMA or DLPFC (Notably, some have argued that these (pre)frontal β bursts cannot index inhibitory control, based on recordings made during eye-movement stopping in monkeys [114]. While frontal β bursts were in fact increased when eye movements were successfully stopped, they were not more prominent at shorter stop-signal delays. According to these authors, since stopping is more likely at shorter delays, there should be more β

bursts. However, in stop-signal tasks, higher stop success at shorter stop-signal delays is typically due to differences in the latency of the go process, not due to a stronger stop-process, rendering this logic questionable).

Moreover, a subcortical cascade of increased β activity in the STN and the motor thalamus precedes the increased β in primary motor cortex at the end of this cascade, an outcome which purportedly reflects successfully re-instated motor inhibition (Figure 2B) [45]. These neurophysiological studies suggest a processing cascade according to which β activity first increases in prefrontal regions of the fronto-subthalamic network, propagates to the basal ganglia, and ultimately inhibits thalamocortical motor drive – returning the motor system to its default inhibited state. As such, β-band activity is a useful index of inhibitory control across the fronto-subthalamic system.

Cognitive activity in thalamocortical circuits

The current Review proposes that the fronto-subthalamic inhibition of thalamocortical drive just described for the motor system could be a domain-general mechanism for inhibitory control. We base this proposition on the observation that thalamocortical drive also supports many types of non-motor functions [115,116] – a proposal long-held by theoretical [117,118] and computational models [119–122]. Indeed, recent studies in humans and nonhuman primates confirm that the thalamus is not merely a 'relay station'; instead, just like in the motor system, thalamic activity drives both local neural activity and network-wide dynamics in nonmotor parts of the neocortex and other areas of the brain (for reviews, see [123–125]. These thalamo-(sub)cortical interactions underpin processes such as attentional engagement, working memory, and long-term memory formation. We focus on these three processes in the following, because there is preliminary support, both neuroanatomically and empirically, for the possibility that they undergo inhibitory control via fronto-subthalamic circuitry. (Whether other processes are inhibited via the same mechanism remains to be seen in future research.)

During sustained attention, the macaque pulvinar drives neural activity in the frontal eye fields and lateral intraparietal cortices [126–129]. The pulvinar also projects directly to primary visual cortex [130]. In line with these findings, human neuroimaging has found that several thalamic nuclei, including the anterior pulvinar, broadly upregulate cortical excitation during perceptual decision making [131,132] (Figure 3A). This upregulation also is reflected in increases of attention-related occipital alpha oscillations in humans, which are driven by the pulvinar [133] (Figure 3A). Similarly, mediodorsal thalamic nuclei interact with prefrontal cortex during decision making in both primates [134] and humans [135], where mediodorsal thalamic activity adaptively partitions cortical activation patterns (for a review, see [136]). The same mediodorsal nuclei have also been found to drive prefrontal cortical activity during the updating of perceptual representations in humans [137], as well as during memory retrieval [138] (Figure 3B). Finally, during long-term memory formation, the anterior nuclei of the thalamus, which exhibit strong functional connectivity to the hippocampus [139–142] show strong phase relationships with frontal cortical areas that are highly indicative of functional connections [143,144]. Stimulating the anterior thalamus intracranially increases gamma activity in the human hippocampus during the formation and retrieval of memories, alongside a functional improvement in those abilities [145,146] (Figure 3C).

These studies represent converging evidence for the key role of thalamocortical drive in primate cognition – similar to the thalamocortical dynamics underlying movement. Studies of this kind show that the thalamus is not merely a relay, but that thalamocortical drive underlies nonmotor activity in many scenarios, similar to the motor system (for reviews, see [123,136,147]). Here, we therefore propose that inhibitory control may act via the same fronto-subthalamic circuitry that inhibits thalamocortical motor drive to also inhibit nonmotor thalamocortical drive. If so, this circuit motif constitutes an

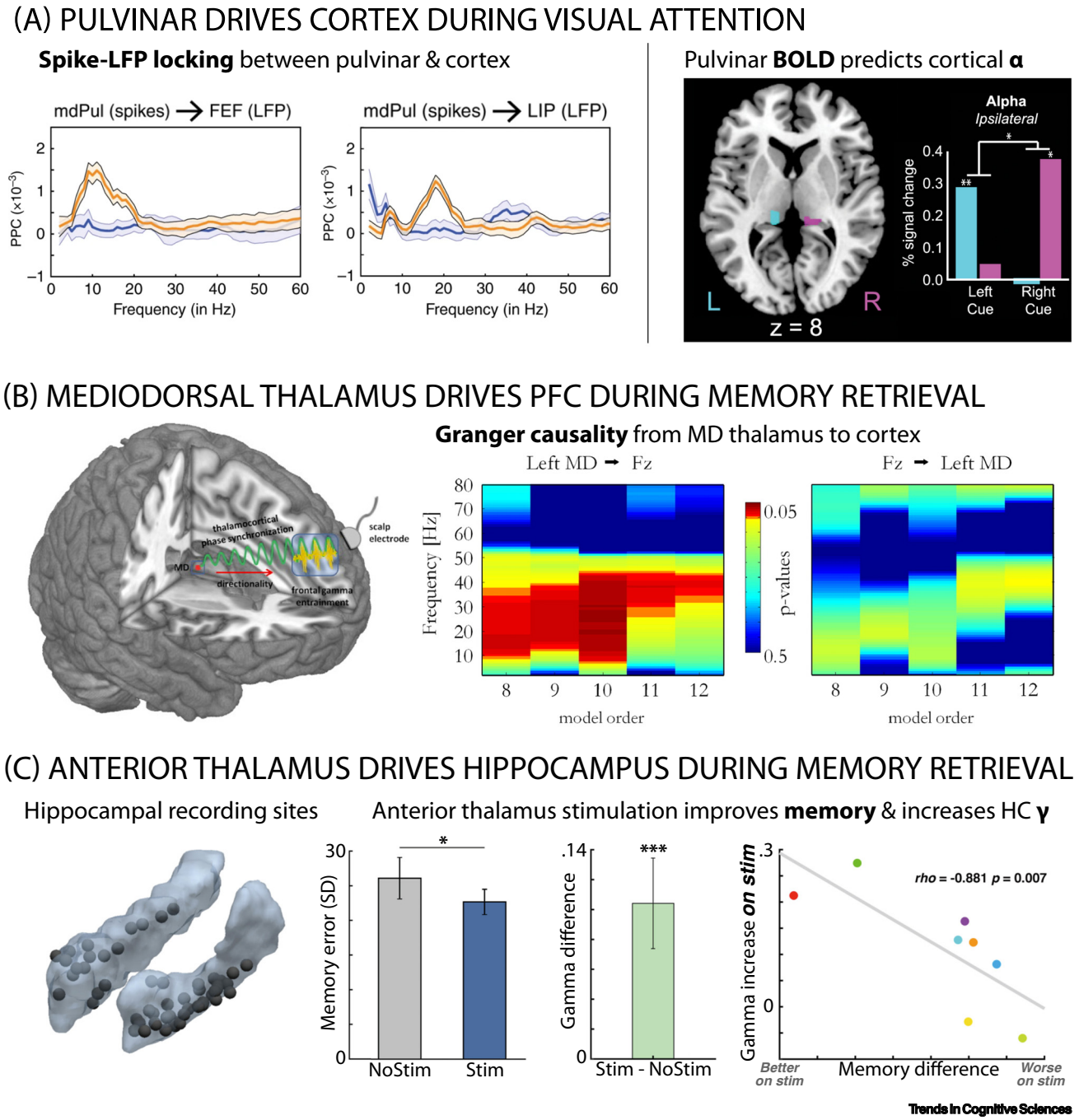

Figure 3. Examples of thalamocortical drive underpinning cognitive processes. (A) The pulvinar drives cortical areas during periods of attentional engagement. Left panels: increased spike-local field potential phase coupling between mediodorsal pulvinar (mdPul) spikes and the local field potential of the frontal eye fields (FEF) and lateral intraparietal cortex (LIP) in macaques during active attentional engagement. Adapted, with permission, from [126]. Right panel: human pulvinar BOLD activity predicts attentional-related increases in occipital alpha activity. Adapted, with permission, from [133]. *$P < 0.05$, **$P < 0.01$. (B) Human mediodorsal thalamus granger-predicts frontocentral EEG signals during correct retrieval from long-term memory. Adapted, with permission, from [138]. (C) Anterior thalamus stimulation in humans increases hippocampal activity during memory retrieval, thereby improving mnemonic precision. Left: hippocampal recording sites during anterior thalamic stimulation experiment. Center left: memory error decreases ON anterior thalamic stimulation compared to OFF. Center right: hippocampal gamma activity during retrieval increases ON anterior thalamic stimulation. Right: stimulation-related increase in hippocampal gamma activity predicts the reduction of memory error across subjects. Adapted, with permission, from [145]. *$P < 0.05$, ***$P < 0.001$.

important mechanistic component of domain-general inhibitory control. We begin this argument in the next section by outlining anatomical connections from the output nuclei of the basal ganglia (GPi/SNr) to the aforementioned nonmotor nuclei of the thalamus. These connections parallel the known inhibitory connections from the basal ganglia to the motor thalamus.

Basal-ganglia projections to nonmotor thalamus

The GPi and SNr send projections not only to ventral thalamic motor nuclei, but also to nonmotor nuclei. Comprehensive mapping of the corticobasal ganglia–thalamic network in mice found that SNr broadly projects to multiple thalamic nuclei, yielding at least six parallel networks that refine multidomain cortical activity via the basal ganglia–thalamic route [148]. Indeed, evidence suggests that all of the previously-mentioned thalamic nuclei that engage in thalamocortical drive to maintain cognitive representations in the primate (pulvinar, mediodorsal and anterior groups, see preceding text) also receive inputs from the GPi/SNr complex: The pulvinar receives inputs from the SNr [149,150] and shows functional connectivity with that region in the human brain [151]. The mediodorsal thalamus receives inputs from the GPi/SNr complex in both the rodent [152] and primate brain [153]. Both the anterior thalamic nucleus group, as well as the nucleus reuniens (another brain region densely connected to the hippocampus and important to memory formation [154,155] receive similar projections from the SNr/GPi complex [152,156]).

These anatomical pathways could conceivably provide a neuroanatomical basis for the potential fronto-subthalamic inhibition of non-motor thalamocortical activity (Figure 4). Whether these specific basal ganglia–thalamic projections fulfill the same function as in the motor system (i.e., whether excitation of the output nuclei of the basal ganglia inhibits the corresponding downstream segments of the thalamus) is an important avenue for future neuroanatomic research.

Cognitive inhibition via the fronto-subthalamic circuit

We now describe recent studies that show preliminary evidence that fronto-subthalamic inhibitory circuitry may affect nonmotor processes, and more generally, that motor and cognitive inhibition may involve a domain-general neural mechanism operating in the β band. These studies can be roughly classified into two categories (Figure 5). (i) **Cued cognitive inhibition**: in such studies, participants are explicitly instructed to inhibit the cognitive representation (e.g., a memory content) by the experimental task. (ii) **Incidental cognitive inhibition:** in such studies, the inhibition of an active cognitive representation is caused by the incidental activation of the inhibitory fronto-subthalamic circuitry, typically via a salient event.

Cued cognitive inhibition is typically studied using the Think-No-Think (TNT) (Figure 5A) and directed-forgetting (DF) paradigms. Such tasks typically instruct participants to explicitly stop active cognitive representations (typically memory items), either during – or, in the case of the TNT, after they have been encoded. The TNT task in particular involves training cue-target associations (e.g., between word or picture pairs), followed by a TNT phase in which some cues are paired with an instruction to suppress retrieval of the corresponding associate (No-Think). Afterwards, a final recall phase assesses how effectively participants suppressed the memory items (for reviews, see [11,13,157]). In a typical DF task, a 'forget' cue follows the initial presentation of a specific memory item, and memory performance is subsequently assessed in comparison to items that were paired with a 'remember' cue. A recent study [52] had participants perform both the TNT task and a motoric stop-signal task (SST) to compare the neural mechanisms involved using fMRI. Critically, stop signals in the SST and No-Think signals in the TNT task activated overlapping regions in the rDLPFC, rIFG, and pre-SMA (Figure 5A), regions robustly confirmed in a companion meta-analytic conjunction of 40 independent stop signal and 16 TNT studies. They found that both rDLPFC and rIFG showed dynamically varying connectivity with either motor

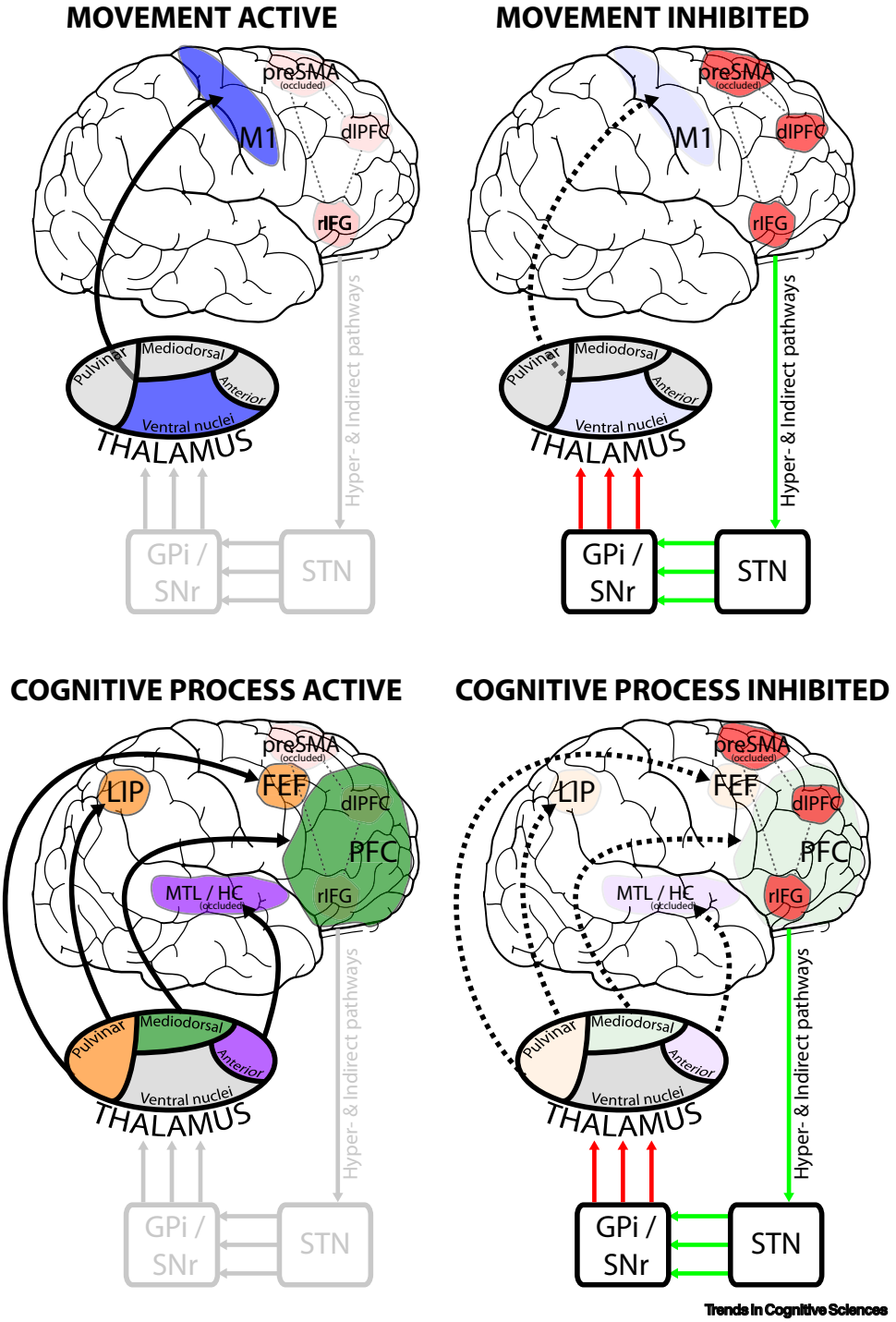

Figure 4. A possible neural circuit for domain-general inhibition. Upper row: established route for inhibitory motor control. Thalamocortical drive invigorates movement, which can be inhibited by the fronto-subthalamic circuit (driven by activity in red regions) via inhibitory projections from the output nuclei of the basal ganglia to the motor thalamus. Lower row: expanding the circuit to include nonmotor activity. Mediodorsal thalamocortical drive to PFC has been shown to modulate decision making and perceptual processes (and may modulate other functions of PFC), the pulvinar drives

(Figure legend continued at the bottom of the next page.)

cortex or hippocampus, depending on whether participants inhibited a movement (SST) or a memory (TNT task). Finally, significant BOLD signal reductions occurred in either the hippocampus or in the motor cortex, depending on whether the participant's goal was to stop memory retrieval or to stop physical action.

Several additional lines of work support the possibility that mnemonic and motoric inhibition both draw upon the fronto-subthalamic circuit posited here. For example, parallels in electrophysiological markers of inhibition also arise across mnemonic and motoric domains. Stopping actions (SST) and stopping memory retrieval (TNT task) both elicited right frontal β-band signals [158], a key neurophysiological signature of the fronto-subthalamic circuit. Moreover, right DLPFC exerts a top-down Granger-causal influence on hippocampus, via similar β oscillatory activity [159]. Relatedly, a recent study [160] compared neural activity recorded during action stopping and directed forgetting. Again, frontal β-band activity arose during both action stopping and memory inhibition, as revealed by crosstask neural decoding. Action-stopping indices (SSRT) correlated with behavioral markers of successful directed forgetting (Figure 5B). Finally, during No-Think trials, STN activity increases [161] – especially when participants report **memory intrusions** (i.e., when a cue briefly elicits recollection of its associated target memory, that then has to be canceled/purged) (see also [55] for meta-analytic evidence for overlapping engagement of subcortical basal ganglia regions in stop signal and TNT tasks). Engagement of the STN during the suppression of memory intrusions converges with a study of corticomotor excitability during the TNT task, which found that memory intrusions are accompanied by a nonselective reduction of corticospinal excitability at task-unrelated muscles [162] – the same signature that indicates STN activity during action stopping [102]. This latter finding suggests that rapidly activating the fronto-subthalamic circuit during inhibition in one domain (in this case, memory) also could trigger inhibition incidentally in other domains (in this case, the motor system). Nevertheless, inhibitory control can also be targeted in goal directed, selective fashion at specific domains: for instance, action stopping suppresses motor cortical responding to a greater extent than does retrieval suppression, and retrieval stopping suppresses hippocampal activity more so than motor stopping [52]. It is tempting to speculate that this domain-specific targeting parallels the different modes of deployment of inhibitory motor control in the stop-signal task, which can be implemented with varying selectivity, depending on how rapid and reactive it has to be (Box 2). Intrusions may be an example of a situation in which mnemonic inhibition has to be rapidly deployed and is hence less selective than usual. A global suppression of thalamocortical drive affecting the hippocampus and other structures during intrusions could act in concert with memory-specific target inhibition mechanisms, such as hippocampal suppression via the nucleus reuniens of the thalamus [45,163]. These questions need more explicit answers in future work.

Studies of incidental cognitive inhibition add additional evidence towards the proposal that fronto-subthalamic circuitry contributes to inhibiting nonmotor processes. In these studies, salient stimuli – rather than instructions to inhibit – are used to activate the fronto-subthalamic circuitry. Examples of salient stimuli include unexpected perceptual events [164,165], action errors [166–168], and incongruent stimuli that elicit response conflict [169,170]. Such salient stimuli are known to automatically trigger inhibitory control, halting ongoing processing to purchase the cognitive system additional time and resources to reassess ongoing behavior [54]. In the motor system, this salience-induced inhibitory activity is readily apparent: salient stimuli slow responses that follow them, suppress

frontal eye fields and lateral intraparietal cortex during visual attention, and the anterior thalamus may drive the medial temporal lobe/hippocampal complex during memory retrieval. The representations supported by those loops may be subject to inhibitory control via the same neural circuit as movement. Abbreviations: dlPFC, dorsolateral prefrontal cortex; PFC, prefrontal cortex; pre-SMA, pre-supplementary motor area; rIFG, right inferior frontal gyrus.

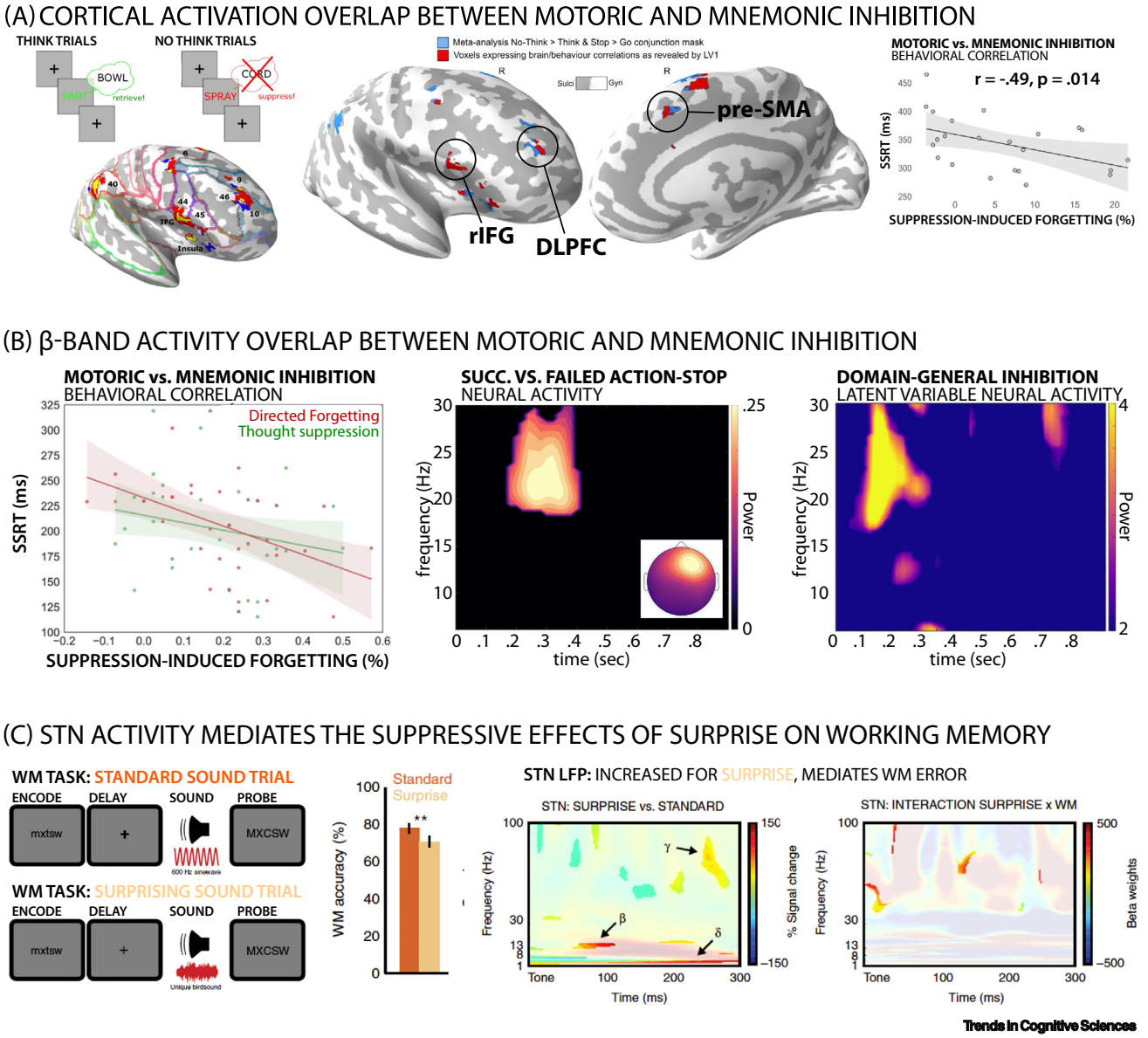

Figure 5. Examples of studies showing overlap between motor and cognitive inhibition. (A) Cortical activity during action stopping (measured by the stop-signal task) and the inhibition of memories (measured by the Think-No-Think task, depicted in top left) shows overlap in critical nodes of the fronto-subthalamic network, including rDLPFC, rIFG, and pre-SMA (bottom left). Moreover, behavioral indices of action-stopping (stop-signal reaction time) and mnemonic suppression were correlated (right), with that correlation being reflected in pre-SMA, rDLPFC, and rIFG BOLD activity (middle two panels). Reproduced, with permission, from [52]. (B) β-Band activity over frontal cortex during both action stopping and directed forgetting of memory contents. Left: stop-signal reaction times and magnitude of directed forgetting effect are correlated. Center: β-Band activity after stop signals in the stop-signal task. Right: correlation between a latent variable expressing motor and nonmotor inhibition and neural activity also reveals β cluster. Reproduced, with permission, from [160]. (C) STN activity mediates the effects of surprise on working memory. Left: task diagram; participants maintained a string of letters in working memory across a delay interval which was, on some trials, disrupted by an unexpected sound. Left center: unexpected sounds led to lower working memory (WM) accuracy and incorrect WM trials had sounds that carried higher surprise values. Right center: unexpected sounds were followed by increased STN activity. Right: increased STN activity after unexpected sounds related to decreases in WM accuracy. Reproduced, with permission, from [171]. $^{**}P < 0.01$. Abbreviations: rDLPFC, right dorsolateral prefrontal cortex; pre-SMA, pre-supplementary motor area; rIFG, right inferior frontal gyrus; STN, subthalamic nucleus.

corticospinal excitability, and activate both the cortical nodes of the fronto-subthalamic network and the STN (for review, see [54]). Importantly, studies of incidental cognitive inhibition have recently begun to reveal that salient events also suppress active cognitive processes via fronto-subthalamic circuitry. The first study of this kind showed that surprising sounds during the delay between a working memory stimulus and probe reduced memory performance [171] (Figure 5C). A trial-to-trial mediation model further established that STN activation may have contributed to this decrement: greater surprise-related activity in the STN local field potential predicted worse working memory performance. A second experiment used a stop-signal task as a functional localizer for scalp-recorded electroencephalography (EEG) activity that distinguished successful from failed stop trials. Independent components analysis then showed that the neural generator underlying this inhibitory motor control signature also mediated surprise effects on working memory. These experiments suggest that both cortical and subcortical aspects of the fronto-subthalamic inhibitory control circuit contribute to how surprise affects working memory.

A subsequent study of attentional inhibition [172] used the latter approach as well. Scalp signatures of inhibitory motor control were identified using the stop-signal task as a functional localizer and then related to attentional inhibition. Specifically, as participants attended to visual stimulus material, steady-state visual evoked potentials (SSVEP) were recorded to index their degree of attention to the target material. Periodically, surprising sounds arose during the task, suppressing the SSVEP. As in the previous work on working memory, activity of the scalp signature of inhibitory motor control (identified using the stop-signal task as a functional localizer) mediated the disruptive effect of surprise on the neural marker of attention (i.e., the SSVEP). This suggests that the attentional shift away from the current locus towards the salient event is achieved by inhibiting the current attentional representation, via the fronto-subthalamic circuitry itself.

Although the foregoing studies of incidental cognitive inhibition provide initial empirical evidence for our proposal that fronto-subthalamic circuitry contributes to nonmotor inhibitory control, they are purely correlational. Causal evidence remains exceedingly rare. One recent study, however, provides a first demonstration. The investigators studied the causal role of the subthalamic nucleus by quantifying how much surprise suppressed the SSVEP in patients with implanted STN deep-brain stimulators (DBSs). Modulating the STN via DBSs significantly reduced the suppressive impact of surprising sounds on the SSVEP [173]. This finding shows that STN causally contributes to suppressing the SSVEP during surprise – in line with the interpretation that the STN mediates the inhibition of attentional representations.

β-Band activity as a universal index of the inhibition of local neural circuits?

Two recent research directions have additional important implications for the role of β-band activity in domain-general inhibitory control. Although this work does not test the role of fronto-subthalamic circuitry in inhibitory control, its main findings are commensurate with the current theoretical model. This work suggests that β-frequency bursts constitute a universal signature of local circuit inhibition – inhibition specifically effected through changes in thalamocortical drive.

First, abundant research shows that β bursts in primary somatosensory cortex impair tactile stimulus detection [174,175]. This has been interpreted as a sign of somatosensory inhibition, paralleling the relationship between β bursts in primary motor cortex and motor inhibition (see preceding text) [110,112]. Detailed biophysical modeling of the currents giving rise to these somatosensory β bursts suggests that they originate from changes in long-distance signaling from thalamus [174,176]. This further supports the idea that changes in thalamocortical drive, reflected in β-band activity, may be a domain-general mechanism for inhibition.

Second, in line with this, β bursts may contribute to inhibiting working memory contents [177–180]. Specifically, PFC regions showing increased gamma and spiking activity during working memory maintenance exhibited increased β bursting once those same items had to be removed from working memory. The authors concluded that 'the interplay between oscillations and spiking observed here seems congruent with an inhibitory role of beta' [197].

These two findings raise the exciting possibility that cortical β bursts represent a universal signature of the local inhibition of any type of neural information in local cortical circuits. Here, we suggest activation of the fronto-subthalamic circuit can rapidly implement this type of inhibition.

Implications

Inhibitory control has long been conceptualized as a unitary construct, relevant to controlling a broad range of motor or non-motor representations or processes. As reviewed here, evidence increasingly suggests that shared neural mechanisms underlie action stopping (motor inhibition) and the inhibition of at least some nonmotor processes (such as those underlying mnemonic or attentional processes). Although inhibitory control is often invoked intentionally (cued inhibition), incidental factors such as surprise or saliency also can trigger inhibition (incidental inhibition), and their effect on cognition may be explained through shared mechanisms. This work establishes a potential neuroscientific foundation for a presumption long held in cognitive theory: that inhibitory control constitutes a crucial domain-general process that broadly regulates motoric and nonmotoric domains, in diverse situations.

We further suggest a novel component mechanism of domain-general inhibitory control, that complements proposals about the role of target inhibition (Box 1): input inhibition. According to this proposal, stopping motoric or cognitive processes may be partly achieved by a domain-general mechanism that interrupts driving input to cortical regions necessary to enact those processes. We propose that this input inhibition arises through fronto-subthalamic inhibition of thalamocortical drive. The characteristics of this circuit may allow inhibitory control over both motor and nonmotor activity, in either a selective or a nonselective fashion, depending on circumstances. Although the exact processing cascade within this circuit remains up for debate, future studies should test hypotheses implied by this mechanistic proposal, including how its operation relates to targeted inhibition of interfering representations.

Beyond these exciting basic science implications, fronto-subthalamic involvement in motor and nonmotor inhibition carries prominent clinical implications. In particular, it may explain long-observed links between stop-signal performance and nonmotoric clinical symptoms, such as substance use [51], impulsivity [18], or intrusive thoughts [181]. This hypothesis could explain why prominent cognitive side effects often arise after treatments for hypokinetic symptoms of movement disorders that inactivate the fronto-subthalamic inhibitory control circuit, such as DBS or subthalamotomy. Indeed, some researchers have described such side effects as the reduced ability to inhibit nonmotor activity, yielding pathologically increased impulsivity [182]. Indeed, if the same circuit inhibits motor and nonmotor processes, therapeutically releasing the excess motor system inhibition by DBS may accidentally and pathologically disinhibit cognitive function.

Concluding remarks

We here present recently emerging and converging evidence suggesting that inhibitory control over motor and nonmotor representations builds on a common neural mechanism. In addition, we propose an important component pathway that may contribute to domain-general inhibitory control: a fronto-subthalamic circuit operating in the β band. Evidence suggests that the activity of this circuit contributes to inhibiting movement, mnemonic representations, and attention.

Outstanding questions

How does the proposed fronto-subthalamic input inhibition mechanism relate to target inhibition (Box 1)? Do these inhibitory control mechanisms recruit different circuits, and if so, how do they interact to achieve successful control?

What are the boundary conditions of the input and output of the fronto-subthalamic circuits? Which types of stimuli, beyond salient events and stop signals, activate the circuit? Which types of processes can (not) be inhibited via the circuit?

What are the specific roles (if any) of the cortical areas (pre-SMA, DLPFC, and rlFC) during inhibitory control? Do they fulfill the same function in both cognitive and motor inhibition?

At what prethalamic level do the purported inhibitory circuits deviate? Are there parallel tracks from specific neurons in STN to specific neurons in the basal ganglia output nuclei (SNr and GPi)? From the SNr/GPi to the thalamus? Or do all prethalamic paths feature the same neurons?

Similar to the motor system, does nonselective cognitive inhibition work via the hyperdirect and selective cognitive inhibition via the indirect pathway?

Is nonselective inhibition only nonselective within domain? Or, does nonselective recruitment of inhibitory motor control also inhibit cognition (and vice versa) and if so, are within and crossdomain inhibition of a similar magnitude?

Do the neuroanatomic connections from SNr/GPi to the nonmotor thalamic nuclei subserve the same function as their connections to motor thalamus?

Are other thalamocortical circuits (and cognitive processes) than the ones outlined here susceptible to similar inhibition?

If cortical β is a universal signature of inhibition due to changes in thalamocortical drive (e.g., of the motor or somatosensory cortex), then why is prefrontal β increased after stop signals?

Whether this mechanism inhibits other representations as well is one of many remaining open questions (see Outstanding questions).

Future studies should causally manipulate this hypothesized circuit by targeting its subcortical aspects via DBS or its cortical aspects via transcranial magnetic stimulation. Advances in recording methods also offer promise. In healthy individuals, high-field fMRI may facilitate noninvasive mapping of subcortical circuits (including the STN [92], whereas in patients with implanted DBS devices, novel hardware interfaces open the door for precise physiology via recordings of local field potential from these regions [183]). Furthermore, innovations for isolating precise, millisecond-resolved neural representations underlying cognition have emerged: SSVEP can track attention [172], multi-variate pattern analysis can track sensory memory [184], and representational similarity analysis can track active task rule representations [185,186] and individual items in memory [187]. Combining these methods with the causal (in)activation of fronto-subthalamic inhibitory control circuitry would allow more unequivocal inferences regarding the role of this circuit in suppressing cognition more broadly. We hope that the theory presented here yields novel and testable hypotheses to this effect.

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Declaration of interests

No interests are declared.

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