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P R E S S

Response inhibition in the stop-signal paradigm

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Response inhibition is a hallmark of executive control. The concept refers to the suppression of actions that are no longer required or that are inappropriate, which supports flexible and goal-directed behavior in ever-changing environments. The stop-signal paradigm is most suitable for the study of response inhibition in a laboratory setting. The paradigm has become increasingly popular in cognitive psychology, cognitive neuroscience and psychopathology. We review recent findings in the stop-signal literature with the specific aim of demonstrating how each of these different fields contributes to a better understanding of the processes involved in inhibiting a response and monitoring stopping performance, and more generally, discovering how behavior is controlled.

People can readily stop talking, walking, typing and so on, in response to changes in internal states or changes in the environment. This ability to inhibit inappropriate or irrelevant responses is a hallmark of executive control. The role of inhibition in many experimental paradigms is debated, but most researchers agree that some kind of inhibition is involved in deliberately stopping a motor response. Here, we focus on the stop-signal paradigm [1], which has proven to be a useful tool for the study of response inhibition in cognitive psychology, cognitive neuroscience and psychopathology. We review recent developments in the stop-signal paradigm in these different fields. The focus is primarily on the inhibition of manual responses. Studies of oculomotor inhibition are discussed in Box 1.

Successful stopping: inhibition and performance monitoring

In the stop-signal paradigm, subjects perform a go task such as reporting the identity of a stimulus. Occasionally, the go stimulus is followed by a stop signal, which instructs subjects to withhold the response (Figure 1). Stopping a response requires a fast control mechanism that prevents the execution of the motor response [1]. This process interacts with slower control mechanisms that monitor and adjust performance [2].

The race between going and stopping
Performance in the stop-signal paradigm is modeled as a race between a 'go process', which is triggered by the presentation of the go stimulus, and a 'stop process', which

is triggered by the presentation of the stop signal. When the stop process finishes before the go process, the response is inhibited; when the go processes finishes before the stop process, the response is emitted. The latency of the stop process (stop-signal reaction time [SSRT]) is covert and must be estimated from a stochastic model, such as the independent race model [3] (Box 2). SSRT has proven to be an important measure of the cognitive control processes that are involved in stopping. Cognitive neuroscientists use SSRT as a criterion to determine whether neural processes participate directly in response inhibition (Box 1). Psychopathologists use SSRT to study inhibitory deficits in different patient groups (see later). Developmental scientists found that SSRT is elevated in younger children and older adults, compared with young adults. In addition, a comparison of SSRT and go reaction time (RT) showed that going and stopping develop and decline independently [4–6].

Monitoring and adjusting go and stop performance
Successful performance in the stop-signal paradigm also involves monitoring go and stop performance and adjusting response strategies to find an optimal balance between the conflicting demands of the go task ('respond as quickly as possible') and the stop task ('stop the response'). Several studies indicate that subjects change response strategies proactively when they expect stop signals to occur, trading speed in the go task for success in the stop task [2,7]. Many studies indicate that subjects also change response strategies reactively after stop-signal trials [8–11]. Some show that go RT increases after unsuccessful inhibition, reminiscent of the post-error slowing observed in choice reaction tasks. Others show that go RT increases after successful stopping, which is inconsistent with error-correction but indicates a shift in priority to the stop task after a stop signal. Recent studies show that stimulus repetition might be a crucial variable: responding after successful stopping is typically slower when the stimulus from the stop trial is repeated, as if the stimulus was associated with stopping, and retrieval of that association impaired go performance [8]. This stimulus-specific slowing can persist over many intervening trials [10] and might support the development of automatic inhibition [12].

Interim conclusions
Cognitive psychologists have identified the computational mechanisms underlying performance in the stop-signal paradigm, identifying a fast-acting stop process that pro-

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Box 1. Inhibitory control and monitoring of eye movements

Important insights into the cognitive and neural mechanisms involved in stopping have come from stop-signal studies of eye movements in macaque monkeys while recording single-cell activity (for a review, see Ref. [54]). The fine temporal resolution of single-cell recording enables stronger inferences about the role of different brain regions in response inhibition and monitoring: cells that modulate before SSRT can contribute directly to response inhibition; cells that modulate after SSRT cannot contribute directly and might contribute to monitoring and control instead. Several studies have shown that activity of movement- and fixation-related neurons in frontal eye fields in dorsolateral prefrontal cortex [55] and superior colliculus in midbrain [56] was modulated before SSRT on successful stop trials (Figure Ia). This indicates that certain neurons in these regions are part of a circuit that directly controls the inhibition of eye movements [55,56]. By contrast, activity of neurons in supplementary eye fields (SEF), which are an ocularmotor extension of SMA, and anterior cingulate cortex (ACC) in medial frontal cortex did not modulate until after SSRT (Figure Ib). Instead, neurons in SEF and ACC signal feedback about errors, rewards and, possibly, conflict. This indicates that SEF and ACC are not directly involved in the inhibition of movements, but instead are involved in monitoring performance [57,58]. Consistent with this idea, unsuccessful inhibition is associated with negatively polarized local-field potentials recorded in ACC of monkeys stopping eye movements [59].

Stopping of eye movements is also studied in humans. Behavioral studies showed that stopping eyes and hands is qualitatively similar, although SSRT is shorter for eye movements [60,61]. An fMRI study showed greater activation in FEF on stop-signal trials and greater activation in SEF on unsuccessful stop trials [62]. However, this study used only one stop-signal delay in the fMRI phase of the experiment, which might have influenced performance [2].
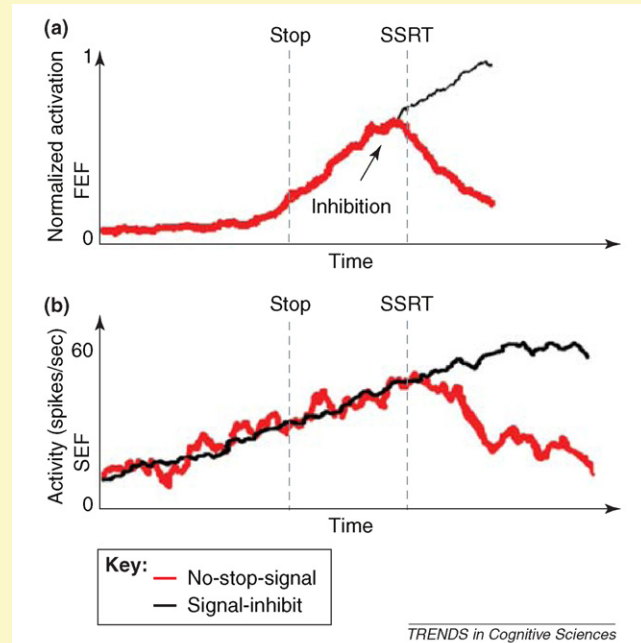

Figure I. Neural activity in countermanding saccades. (a) Neural activity of FEF movement neurons on no-stop-signal trials and signal-inhibit trials [55]. (b) Neural activity in SEF neurons on no-stop-signal trials and signal-inhibit trials.
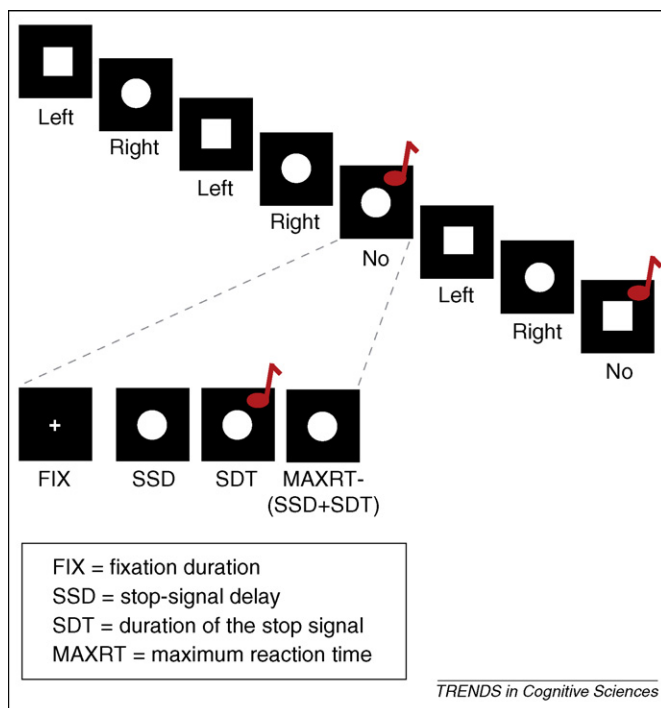

Figure 1. Depiction of a trial course in the stop-signal paradigm. Tasks and task parameters in this figure are adapted from STOP-IT, which is a free-to-use stop-signal task program [74]. In the go task, subjects respond to the shape of a stimulus (a 'square' requires a left response and a 'circle' requires a right response). On one-fourth of the trials, the go stimulus is followed by an auditory stop signal after a variable stop-signal delay (SSD). Subjects are instructed to respond as quickly and accurately as possible to the go stimulus on no-stop-signal trials. They are instructed to try to withhold their response on stop-signal trials, but not to wait for the stop signal to occur. On both no-stop-signal trials and stop-signal trials, the stimulus remains on the screen until subjects respond or until the maximal RT has elapsed.

duces immediate inhibition and slower monitoring and adjustment processes that optimize performance. In recent years, many insights into the underlying cognitive and neural mechanisms of response inhibition have come from cognitive neuroscience and psychopathology. In the next sections, we review the most important findings in these fields and show how they contribute to a better understanding of control processes involved in stopping and monitoring stop performance.

Neural substrates of stopping and monitoring

Going is associated with activation of a cortico-basal-ganglia-thalamocortical circuit [13]. Recent studies using a variety of methods indicate that stopping is associated with activation of a fronto-basal-ganglia circuit that includes inferior frontal gyrus (IFG; ventrolateral prefrontal cortex), middle frontal gyrus (dorsolateral prefrontal cortex), medial frontal gyrus (MFG) and basal ganglia [14–18]. Results are sometimes inconsistent between studies, possibly because they used different methods for isolating inhibition-related regions.

A fronto-basal-ganglia circuit for response inhibition

Recent neuroimaging research has indicated that right IFG is involved in stopping (e.g. Refs [14,15,18]), and possibly other kinds of inhibition (Box 3). This region shows increased activation when stopping is successful, and the magnitude of the activation correlates negatively with SSRT [14,17,19]. Some studies showed that right IFG is activated to some degree on unsuccessful stop trials (e.g. Ref. [17]), but not on no-stop-signal trials (e.g. Refs [17,18]). Successful stopping is also associated with pre-supple-

Box 2. The independent and interactive race models of response inhibition

Logan and Cowan [3] developed an independent race model (Figure Ia) that described the probability of responding on a stop-signal trial, p(respond | signal), the latency of go RTs that escape inhibition and SSRT. According to the model, p(respond | signal) depends primarily on three factors: SSD, go RT and SSRT. First, increasing SSD increases p(respond | signal): the stop process starts later and, therefore, finishes later relative to the go process (Figure Ib). Second, for every SSD, increasing go RT decreases p(respond | signal) because the probability that the stop process finishes before the go process increases (Figure Ic). Third, for every SSD, increasing SSRT increases p(respond | signal) because the probability that the stop process finishes after the go process increases (Figure Id). Importantly, the independent race model provides methods for estimating SSRT. The model assumes that the stop process begins at SSD, which is observed. The point at which the stop process finishes can be estimated from the observed go-RT distribution on no-signal trials and the observed p(respond | signal) for a given SSD (Figure Ia). SSRT can be estimated by subtracting SSD from the finishing time (for reviews of estimation methods of SSRT, see Refs [1,63]).

The independent race model assumes stochastic independence between the go and stop process. However, complete independence between the go and stop process is unlikely. Boucher *et al.* [64] proposed an interactive race model, in which the go and stop processes are independent for much of their latencies and interact strongly near the end (Figure II). The go process is initiated by the go stimulus and a go unit is activated after an afferent delay. The stop process is initiated by the stop signal and a stop unit is activated after an afferent delay. Once the stop unit is activated, it inhibits go processing strongly and quickly. In this model, SSRT primarily reflects the period before the stop unit is activated, during which stop and go processing are independent, so its predictions correspond to those of the independent race model [64].
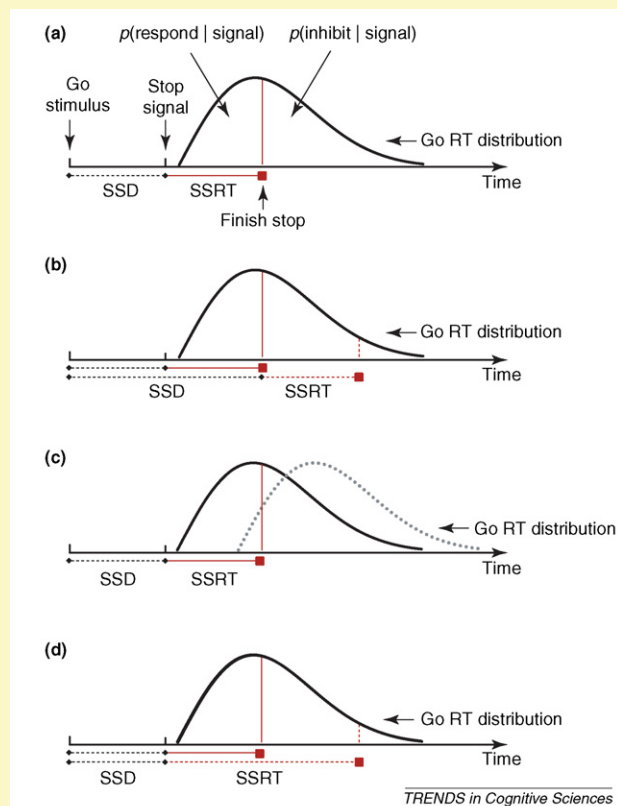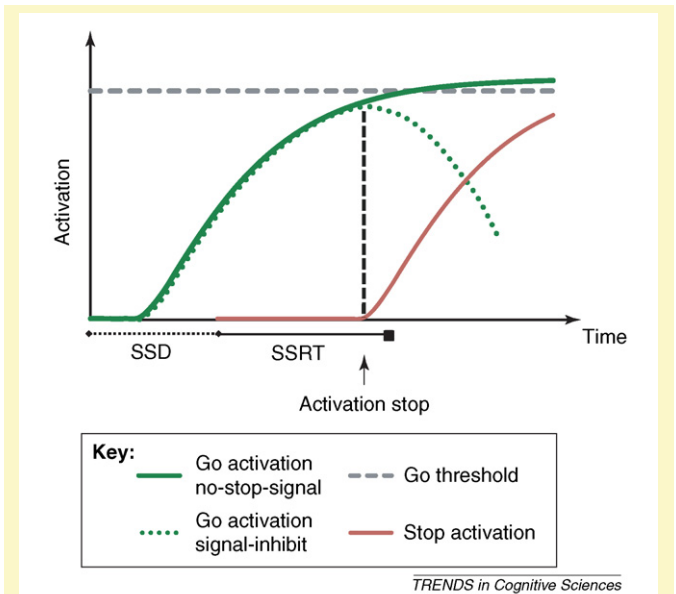

Figure II. Graphic representation of the assumptions of the interactive race model [64], indicating how go activation on a signal-inhibit trial is inhibited when the stop unit is activated.

Figure I. The independent race model. **(a)** Graphic representation of the assumptions of the independent race model [3], indicating how the probability of responding [p(respond | signal)] and the probability of inhibiting [p(inhibit | signal)] depend on **(b)** SSD, **(c)** the distribution of go RTs and **(d)** SSRT. p(respond | signal) is represented by the area under the curve to the left of each red vertical line.

mentary-motor area (pre-SMA) activation, but unlike right IFG, the magnitude of activation in pre-SMA did not correlate with SSRT [14]. According to some researchers, these findings indicate that right IFG contributes to response inhibition and not to monitoring performance or adjusting behavior [18], whereas pre-SMA is involved

Box 3. One or many inhibitory mechanisms?

An important issue is whether the inhibitory mechanism that is involved in the stop-signal paradigm is also involved in other inhibitory paradigms. Behavioral results indicate a functional relationship between stop-signal inhibition and interference control in the Stroop task and the flanker task. Incongruent trials produce interference and prolong SSRT in these tasks [65,66]. Individual-difference studies show correlations between stop-signal inhibition and interference control [67]. Neuroimaging studies show activation in right IFG and pre-SMA in different inhibition tasks [68,69]. However, this need not imply that the same inhibitory circuit is involved. rTMS of the right IFG influenced response inhibition but not interference control in a flanker task with stop signals [21]. Future research should clarify whether functional dependence between different kinds of inhibition implies similar neural mechanisms.

A related issue is whether the same inhibitory mechanism is involved in stopping responses with different effectors. SSRTs are similar for interrupting speech and interrupting manual responses [70], but SSRT is typically shorter for eye movements than for hand movements (Box 1). fMRI data indicate that right IFG and pre-SMA are involved in inhibition of hand movements and suppression of speech, but STN was activated only for inhibition of hand movements [70]. However, activation of STN is hard to detect in fMRI. One fMRI study compared inhibition of eye and hand movements directly, and found common activation in right IFG and medial frontal regions (among other regions) [71]. Inhibition of hand movements was associated with activation in more ventral and posterior parts of right IFG, whereas inhibition of eye movements was associated with activation in more dorsal and anterior parts of right IFG. However, no eye movements were recorded in the scanner, so it is not clear whether the common activation is because of inhibition or performance monitoring. Future research should clarify how general the inhibitory circuits are.

in monitoring or resolving the conflict between the opposing task demands in the stop-signal paradigm [14,20]. However, the poor temporal resolution of functional magnetic resonance imaging (fMRI) makes it difficult to determine the specific role of right IFG and pre-SMA (Box 1).

The involvement of right IGF and pre-SMA in stopping is further supported by results of transcranial magnetic stimulation (TMS) and lesion studies. Repetitive TMS (rTMS) of right IFG (but not left IFG or right middle frontal gyrus) impaired stopping but not going [21,22]. By contrast, rTMS over right dorsal premotor cortex influenced going but not stopping. These findings support the theoretical distinction between stop and go processes in formal race models. Response inhibition is impaired in patients with lesions to right IFG but not left IFG [23]; moreover, the magnitude of the lesion to right IFG correlated with SSRT but not with go RT. Similarly, lesions to right SMA and pre-SMA impaired stopping without greatly influencing going [24].

Several subcortical regions might also play an important part in stopping. fMRI studies showed inhibition-related activation in basal ganglia, including the subthalamic nucleus (STN) [17] and striatum [18,25]. Lesions to the basal ganglia impaired stop performance for both humans and rodents [26–28], whereas deep-brain stimulation of STN in Parkinson patients enhanced inhibitory control [29]. Lesions to STN and stimulation of STN in Parkinson patients influence both go RT and SSRT [29,30]. However, the effects of STN stimulation on go RT and SSRT might be functionally independent [29].

Combined, these studies indicate that right IFG, pre-SMA and basal ganglia are part of a fronto-basal-ganglia inhibition network, although the exact role of these regions is debated. Some researchers proposed that activation in right IFG or pre-SMA leads to a suppression of motor output through a projection to STN [14,17,31]. When STN is activated, the internal segment of the globus pallidus becomes activated and motor output is suppressed. In most stop-signal situations, this suppression is very general and might affect all response tendencies including activation in muscles that are irrelevant to the task [32–34].

Neural substrates of monitoring
Unsuccessful inhibition is associated with an error-related negativity (ERN) in the electroencephalogram [35], which is reminiscent of the ERN that is typically observed after choice errors in reaction tasks. Event-related fMRI studies showed that (mainly) parietal and frontal brain regions are more activated when inhibition is unsuccessful [15,16,18,19]. Unsuccessful inhibition is associated with greater activation of medial frontal regions, including anterior cingulate cortex (ACC), pre-SMA and middle frontal regions. Some studies report that ACC is also activated on successful stop-signal trials (e.g. Ref. [17]), which indicates that this region is involved in the monitoring of stopping performance. Consistent with this idea, single-cell studies show that ACC modulation occurs after SSRT, which is too late to be involved directly in inhibiting the response; instead, the neurons signal reward and error (Box 1). Medial frontal regions are commonly associated with detection of errors and detection of conflict between responses and action plans (monitoring behavior), whereas

middle frontal regions are commonly associated with adjusting behavior after conflict or errors [36]. Some researchers proposed that activation of middle frontal regions reflects adjusting response strategies to balance the opposing demands of the go and stop tasks [18].

Combined, behavioral data (post-error slowing) and neural data (ERN and activation of medial and middle frontal regions) indicate that monitoring and adjusting performance in the stop-signal paradigm might be similar to monitoring and adjusting performance in paradigms that do not involve inhibition of motor responses. Moreover, the neural mechanisms involved in monitoring can be distinguished from the neural mechanisms involved directly in stopping (Box 1). However, it is unclear the extent to which activation associated with monitoring actually reflects memory-retrieval effects (Box 4).

Inhibitory disorders and psychopathology
Response-inhibition deficits have been linked to several psychopathological and neurological disorders. Some disorders, such as autism [37] and schizophrenia [38], are associated with general cognitive impairments in addition to inhibitory deficits. Other disorders, such as attention-deficit/hyperactivity disorder (ADHD) (for a metanalysis,

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Box 4. Open questions and future directions

Recent stop-signal studies provided important insights in how stopping might be achieved. Several open questions remain.

- How do working memory and long-term memory contribute to successful stopping? Short-term memory might be necessary for maintaining task rules and action plans. Retrieval of short-term and long-term associations might also contribute to stop performance. After many repetitions, associations between stimuli and stopping might enable automatic response inhibition, which reduces the need for cognitive control processes [11]. Automatic inhibition is more likely to develop in the go/no-go paradigm, in which stimuli are consistently associated with going and stopping, than in the stop-signal paradigm, in which stimuli are inconsistently associated with going and stopping [11]. It will be useful to link the neural substrates of inhibition to the neural substrates of memory, and to explore the links between inhibition deficits and memory deficits.

- How does the ability to switch contribute to successful stopping? Successful stopping implies shifting attention from the go signal to the stop signal, and upon detection of the stop signal, activating an alternative task goal (the stop goal) or action plan [20,72,73]. The links between neural substrates of response inhibition, shifting attention and replacing task goals and action plans should be explored.

- How selective can stopping be? This article focuses on non-selective stopping, in which subjects inhibit all responses when a stop signal occurs. Some research has examined selective stopping, in which subjects inhibit only some responses or inhibit only when some signals occur (e.g. Refs [34,60]). Much more is known about non-selective stopping. Future research should focus on selective forms of stopping and determine how one response can be stopped without inhibiting all other concurrent processing.

- The further development of formal models. The independent race model is very general, addressing only finishing times of stop and go processes without addressing how the processes unfold computationally or neurally. The interactive race model is constrained by neural data but it is currently specific to stopping eye movements. No current models address the monitoring processes involved in the stop-signal paradigm. Future research should focus on the development of general models that account for stopping and monitoring computationally and neurally.

see Ref. [39]) and compulsive disorders [40], are described specifically as inhibitory disorders. In the following sections, we review stop-signal studies of inhibitory disorders. These reveal important insight into the underlying cognitive and neural mechanisms of response inhibition and monitoring.

Attention-deficit/hyperactivity disorder
Probably the most studied clinical group in the stop-signal literature is children with ADHD. ADHD is typically associated with poor control of impulses. Many stop-signal studies have shown slower SSRT in people with ADHD [39] and in their relatives [41]. These findings led researchers to propose that stop-signal inhibition might be an endophenotype of ADHD [41,42]. In children with ADHD, slower SSRT is often accompanied by slower go RT, indicating a general deficit in cognitive control [38]. By contrast, in adults with ADHD, SSRT is impaired but go RT is not, indicating a selective deficit in inhibition. The inhibitory deficit ADHD is linked to functional and structural differences in the fronto-basal-ganglia inhibitory network (for reviews, see Refs [42,43]). However, the amplitude of non-inhibitory (attentional) components in the electroencephalogram is also reduced in ADHD, which indicates that the response-inhibition deficit might have multiple origins [44].

Performance monitoring might also be impaired in ADHD. Children with ADHD slow their go RTs less after unsuccessful stopping than control subjects [9]. Post-error adjustments were not correlated with SSRT, indicating a dissociation of response inhibition and monitoring or adjustment. It is not clear whether the behavioral impairment reflects deficits in monitoring or deficits in adjustment. However, neural data indicate a monitoring deficit: unsuccessful stopping in ADHD is associated with a reduced amplitude of the ERN [45] and a reduced magnitude of activation in posterior and anterior parts of the cingulate gyrus and in left ventrolateral prefrontal cortex [46,47]. Combined, behavioral and neural data indicate an error-monitoring deficit in ADHD that can be dissociated from the response-inhibition deficit.

Poor inhibitory control over obsessions, compulsions, tics and urges
Inhibitory deficits are associated with disorders other than ADHD. SSRT is prolonged in people with obsessive-compulsive disorder (OCD) and their first-degree relatives, people with trichotillomania (repetitive hair pulling) and people with Tourette's syndrome (e.g. Refs [40,48,49]). These inhibitory deficits result in poor control of behavior, which is characteristic of these disorders. Typically, go RT is not affected, indicating a selective deficit in inhibition. The inhibitory deficit in OCD has been linked to reduced grey matter in the orbitofrontal and right inferior frontal regions [49]. A recent fMRI study of people with OCD found reduced activation in inferior and orbital fronto-striatothalamic brain regions when inhibition was successful and reduced activation in mesial and dorsolateral prefrontal cortices when inhibition was unsuccessful. This indicates that response inhibition and performance monitoring are both impaired in OCD [50]. However, these results should be interpreted cautiously because sample size was small.

Poor inhibitory control is also characteristic of substance-abuse disorders. SSRT is prolonged in chronic cocaine users [51], chronic methamphetamine users [52] and alcohol-dependent subjects [48], compared with normal control subjects, indicating a response-inhibition deficit. It is not clear whether these SSRT differences reflect pre-morbid differences in inhibitory control, post-morbid abnormalities because of chronic chemical abuse or both. However, prolonged SSRTs in high-risk adolescents predict alcoholism and other substance-abuse disorders [53], which indicates that prolonged SSRTs in chronic substance users might reflect pre-morbid differences in inhibitory control.

Concluding remarks
The stop-signal paradigm has become a popular tool for the study of response inhibition in cognitive psychology, cognitive neuroscience and psychopathology. Through this paradigm, findings from different fields of research have stimulated each other, leading to integrative, converging conclusions about cognitive control processes involved in stopping and performance monitoring. Cognitive psychologists modeled response inhibition as a race between a go process and a stop process. Cognitive neuroscientists showed that these processes might activate brain regions differently, and psychopathologists showed selective deficits in inhibition. Studies in each field indicate that successful stop performance requires effective performance monitoring and behavior adjustment, in addition to an efficient stop process, to find an optimal balance between the opposing task demands of the stop-signal paradigm.

Inhibitory processes and monitoring have been dissociated behaviorally and neurally. The most important challenge for the future is to determine how inhibitory processes and monitoring jointly contribute to successful stop performance and how the neural substrates of these processes carry out the required computation. Successful stopping might require some general processes that are not unique to response inhibition, in addition to some specific processes that are unique. Future research should further differentiate these processes and their associated brain circuits. This will require combining different methods and using techniques that have sufficient temporal resolution to distinguish between processes that occur before and after SSRT (Box 4).

References
1 Logan, G.D. (1994) On the ability to inhibit thought and action: a user's guide to the stop signal paradigm. In *Inhibitory Processes in Attention, Memory and Language* (Dagenbach, D. and Carr, T.H., eds), pp. 189–239, Academic
2 Verbruggen, F. and Logan, G.D. Proactive adjustments of response strategies in the stop-signal paradigm. *J. Exp. Psychol. Hum. Percept. Perform.* (in press)

3 Logan, G.D. and Cowan, W.B. (1984) On the ability to inhibit thought and action: a theory of an act of control. *Psychol. Rev.* 91, 295–327

4 van den Wildenberg, W.P.M. and van der Molen, M.W. (2004) Developmental trends in simple and selective inhibition of compatible and incompatible responses. *J. Exp. Child Psychol.* 87, 201–220

5 Williams, B.R. *et al.* (1999) Development of inhibitory control across the life span. *Dev. Psychol.* 35, 205–213

6 Rush, B.K. *et al.* (2006) Accounting for cognitive aging: context processing, inhibition or processing speed? *Aging Neuropsychol. Cogn.* 13, 588–610

7 Stuphorn, V. and Schall, J.D. (2006) Executive control of countermanding saccades by the supplementary eye field. *Nat. Neurosci.* 9, 925–931

8 Verbruggen, F. *et al.* (2008) Short-term aftereffects of response inhibition: repetition priming or between-trial control adjustments? *J. Exp. Psychol. Hum. Percept. Perform.* 34, 413–426

9 Schachar, R.J. *et al.* (2004) Evidence for an error monitoring deficit in attention deficit hyperactivity disorder. *J. Abnorm. Child Psychol.* 32, 285–293

10 Verbruggen, F. and Logan, G.D. (2008) Long-term aftereffects of response inhibition: memory retrieval, task goals, and cognitive control. *J. Exp. Psychol. Hum. Percept. Perform.* 34, 1229–1235

11 Emeric, E.E. *et al.* (2007) Influence of history on saccade countermanding performance in humans and macaque monkeys. *Vision Res.* 47, 35–49

12 Verbruggen, F. and Logan, G.D. Automatic and controlled response inhibition: associative learning in the go/no-go and stop-signal paradigms. *J. Exp. Psychol. Gen.* (in press)

13 Alexander, G.E. *et al.* (1990) Basal ganglia-thalamocortical circuits - parallel substrates for motor, oculomotor, prefrontal and limbic functions. *Prog. Brain Res.* 85, 119–146

14 Aron, A.R. *et al.* (2007) Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *J. Neurosci.* 27, 3743–3752

15 Rubia, K. *et al.* (2003) Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage* 20, 351–358

16 Li, C.S.R. *et al.* (2006) Imaging response inhibition in a stop-signal task: neural correlates independent of signal monitoring and post-response processing. *J. Neurosci.* 26, 186–192

17 Aron, A.R. and Poldrack, R.A. (2006) Cortical and subcortical contributions to stop signal response inhibition: role of the subthalamic nucleus. *J. Neurosci.* 26, 2424–2433

18 Chevrier, A.D. *et al.* (2007) Dissociation of response inhibition and performance monitoring in the stop signal task using event-related fMRI. *Hum. Brain Mapp.* 28, 1347–1358

19 Rubia, K. *et al.* (2007) Linear age-correlated functional development of right inferior fronto-striato-cerebellar networks during response inhibition and anterior Cingulate during error-related processes. *Hum. Brain Mapp.* 28, 1163–1177

20 Nachev, P. *et al.* (2007) The role of the pre-supplementary motor area in the control of action. *Neuroimage* 36, T155–T163

21 Chambers, C.D. *et al.* (2007) Dissociable mechanisms of cognitive control in prefrontal and premotor cortex. *J. Neurophysiol.* 98, 3638–3647

22 Chambers, C.D. *et al.* (2006) Executive "brake failure" following deactivation of human frontal lobe. *J. Cogn. Neurosci.* 18, 444–455

23 Aron, A.R. *et al.* (2003) Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nat. Neurosci.* 6, 115–116

24 Floden, D. and Stuss, D.T. (2006) Inhibitory control is slowed in patients with right superior medial frontal damage. *J. Cogn. Neurosci.* 18, 1843–1849

25 Vink, M. *et al.* (2005) Function of striatum beyond inhibition and execution of motor responses. *Hum. Brain Mapp.* 25, 336–344

26 Gauggel, S. *et al.* (2004) Inhibition of ongoing responses in patients with Parkinson's disease. *J. Neurol. Neurosurg. Psychiatry* 75, 539–544

27 Rieger, M. *et al.* (2003) Inhibition of ongoing responses following frontal, nonfrontal and basal ganglia lesions. *Neuropsychology* 17, 272–282

28 Eagle, D.M. and Robbins, T.W. (2003) Inhibitory control in rats performing a stop-signal reaction-time task: effects of lesions of the medial striatum and d-amphetamine. *Behav. Neurosci.* 117, 1302–1317

29 van den Wildenberg, W.P.M. *et al.* (2006) Stimulation of the subthalamic region facilitates the selection and inhibition of motor responses in Parkinson's disease. *J. Cogn. Neurosci.* 18, 626–636

30 Eagle, D.M. *et al.* (2008) Stop-signal reaction-time task performance: role of prefrontal cortex and subthalamic nucleus. *Cereb. Cortex* 18, 178–188

31 Mostofsky, S.H. and Simmonds, D.J. (2008) Response inhibition and response selection: two sides of the same coin. *J. Cogn. Neurosci.* 20, 751–761

32 Coxon, J.P. *et al.* (2006) Intracortical inhibition during volitional inhibition of prepared action. *J. Neurophysiol.* 95, 3371–3383

33 Coxon, J.P. *et al.* (2007) Selective inhibition of movement. *J. Neurophysiol.* 97, 2480–2489

34 Aron, A.R. and Verbruggen, F. Stop the presses: dissociating a selective from a global mechanism for stopping. *Psychol. Sci.* (in press)

35 Kok, A. *et al.* (2004) ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology* 41, 9–20

36 Ridderinkhof, K.R. *et al.* (2004) Neurocognitive mechanisms of cognitive control: the role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain Cogn.* 56, 129–140

37 Geurts, H.M. *et al.* (2004) How specific are executive functioning deficits in attention deficit hyperactivity disorder and autism? *J. Child Psychol. Psychiatry* 45, 836–854

38 Enticott, P.G. *et al.* (2008) Response inhibition and impulsivity in schizophrenia. *Psychiatry Res.* 157, 251–254

39 Lijffijt, M. *et al.* (2005) A meta-analytic review of stopping performance in attention-deficit/hyperactivity disorder: deficient inhibitory motor control? *J. Abnorm. Psychol.* 114, 216–222

40 Chamberlain, S.R. *et al.* (2006) Motor inhibition and cognitive flexibility in obsessive-compulsive disorder and trichotillomania. *Am. J. Psychiatry* 163, 1282–1284

41 Schachar, R.J. *et al.* (2005) Inhibition of motor responses in siblings concordant and discordant for attention deficit hyperactivity disorder. *Am. J. Psychiatry* 162, 1076–1082

42 Aron, A.R. and Poldrack, R.A. (2005) The cognitive neuroscience of response inhibition: relevance for genetic research in attention-deficit/hyperactivity disorder. *Biol. Psychiatry* 57, 1285–1292

43 Kieling, C. *et al.* (2008) Neurobiology of attention deficit hyperactivity disorder. *Child Adolesc. Psychiatr. Clin. N. Am.* 17, 285–307

44 Bekker, E.M. *et al.* (2005) Disentangling deficits in adults with attention-deficit/hyperactivity disorder. *Arch. Gen. Psychiatry* 62, 1129–1136

45 Liotti, M. *et al.* (2005) Abnormal brain activity related to performance monitoring and error detection in children with ADHD. *Cortex* 41, 377–388

46 Rubia, K. *et al.* (2005) Abnormal brain activation during inhibition and error detection in medication-naive adolescents with ADHD. *Am. J. Psychiatry* 162, 1067–1075

47 Pliszka, S.R. *et al.* (2006) Neuroimaging of inhibitory control areas in children with attention deficit hyperactivity disorder who were treatment naive or in long-term treatment. *Am. J. Psychiatry* 163, 1052–1060

48 Goudriaan, A.E. *et al.* (2006) Neurocognitive functions in pathological gambling: a comparison with alcohol dependence. Tourette syndrome and normal controls. *Addiction* 101, 534–547

49 Menzies, L. *et al.* (2007) Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain* 130, 3223–3236

50 Woolley, J. *et al.* (2008) Brain activation in paediatric obsessive-compulsive disorder during tasks of inhibitory control. *Br. J. Psychiatry* 192, 25–31

51 Fillmore, M.T. and Rush, C.R. (2002) Impaired inhibitory control of behavior in chronic cocaine users. *Drug Alcohol Depend.* 66, 265–273

52 Monterosso, J.R. *et al.* (2005) Deficits in response inhibition associated with chronic methamphetamine abuse. *Drug Alcohol Depend.* 79, 273–277

53 Nigg, J.T. *et al.* (2006) Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *J. Am. Acad. Child Adolesc. Psychiatry* 45, 468–475

54 Schall, J.D. and Boucher, L. (2007) Executive control of gaze by the frontal lobes. *Cogn. Affect. Behav. Neurosci.* 7, 396–412

55 Hanes, D.P. *et al.* (1998) Role of frontal eye fields in countermanding saccades: visual, movement, and fixation activity. *J. Neurophysiol.* 79, 817–834

56 Pare, M. and Hanes, D.P. (2003) Controlled movement processing: superior colliculus activity associated with countermanded saccades. *J. Neurosci.* 23, 6480–6489

57 Ito, S. *et al.* (2003) Performance monitoring by the anterior cingulate cortex during saccade countermanding. *Science* 302, 120–122

58 Stuphorn, V. *et al.* (2000) Performance monitoring by the supplementary eye field. *Nature* 408, 857–860

59 Emeric, E.E. *et al.* (2008) Performance monitoring local field potentials in the medial frontal cortex of primates: anterior cingulate cortex. *J. Neurophysiol.* 99, 759–772

60 Boucher, L. *et al.* (2007) Stopping eye and hand movements: are the processes independent? *Percept. Psychophys.* 69, 785–801

61 Logan, G.D. and Irwin, D.E. (2000) Don't look! Don't touch! Inhibitory control of eye and hand movements. *Psychon. Bull. Rev.* 7, 107–112

62 Curtis, C.E. *et al.* (2005) Canceling planned action: an fMRI study of countermanding saccades. *Cereb. Cortex* 15, 1281–1289

63 Band, G.P.H. *et al.* (2003) Horse-race model simulations of the stop-signal procedure. *Acta Psychol. (Amst.)* 112, 105–142

64 Boucher, L. *et al.* (2007) Inhibitory control in mind and brain: an interactive race model of countermanding saccades. *Psychol. Rev.* 114, 376–397

65 Verbruggen, F. *et al.* (2004) The interaction between stop signal inhibition and distractor interference in the flanker and Stroop task. *Acta Psychol. (Amst.)* 116, 21–37

66 Verbruggen, F. *et al.* (2005) Effects of stimulus-stimulus compatibility and stimulus-response compatibility on response inhibition. *Acta Psychol. (Amst.)* 120, 307–326

67 Friedman, N.P. and Miyake, A. (2004) The relations among inhibition and interference control functions: a latent-variable analysis. *J. Exp. Psychol. Gen.* 133, 101–135

68 Aron, A.R. *et al.* (2004) Inhibition and the right inferior frontal cortex. *Trends Cogn. Sci.* 8, 170–177

69 Derrfuss, J. *et al.* (2004) Cognitive control in the posterior frontolateral cortex: evidence from common activations in task coordination, interference control, and working memory. *Neuroimage* 23, 604–612

70 Xue, G. *et al.* (2008) Common neural substrates for inhibition of spoken and manual responses. *Cerebral Cortex* 18, 1923–1932

71 Leung, H.C. and Cai, W.D. (2007) Common and differential ventrolateral prefrontal activity during inhibition of hand and eye movements. *J. Neurosci.* 27, 9893–9900

72 Camalier, C.R. *et al.* (2007) Dynamics of saccade target selection: race model analysis of double step and search step saccade production in human and macaque. *Vision Res.* 47, 2187–2211

73 Verbruggen, F. *et al.* (2008) How to stop and change a response: the role of goal activation in multi-tasking. *J. Exp. Psychol. Hum. Percept. Perform.* 34, 1212–1228

74 Verbruggen, F. *et al.* (2008) STOP-IT: Windows executable software for the stop-signal paradigm. *Behav. Res. Methods* 40, 479–483