A NEUROIMAGING APPROACH TO THE RELATIONSHIP BETWEEN ATTENTION AND SPEED-ACCURACY TRADEOFF

by

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People are able to trade off speed and accuracy when performing a task; that is, they can either focus on performing accurately at the cost of being slow, or on being fast at the cost of decreased accuracy. Performance can be varied along this speed-accuracy tradeoff (SAT) continuum. The present set of studies were designed to, first, test the effects of speed versus accuracy emphasis on attentional processes and the underlying neural activity, and, second, to investigate how the brain achieves the desired level of SAT. In Experiment 1 it was found that attentional adjustments on trials following difficult trials or error trials, and their associated neural activation in the anterior cingulate and lateral prefrontal cortices were modulated by SAT. The conflict adaptation effect and associated anterior cingulate and prefrontal activation were greater under speed emphasis, whereas post-error slowing and associated anterior cingulate and prefrontal activation were greater under accuracy emphasis. Experiment 2 tested how people achieve a desired level of SAT by measuring neural activity in response to cues instructing participants whether to either emphasize speed or accuracy during a subsequent set of trials. Increased activation to speed cues was found in brain regions related to the preparation and execution of actions, which was furthermore sustained throughout speeded performance. This suggests that the level of baseline activation in these areas increased under speed emphasis. Moreover, transient, response-related activation of the dorsal premotor cortices was increased during accuracy emphasis. The results of Experiment 2 support computational theories of decision making according to which evidence for one or another decision builds from a baseline to a threshold, and different levels of SAT are achieved by varying the distance between this baseline and threshold. Together, these studies provide novel data that help us better understand how people are able to regulate their performance.

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1.0 INTRODUCTION

1.1 SAT DURING SIMPLE DECISIONS

The faster you try to do something, the greater the chance of making an error. Conversely, greater focus on accuracy comes at the cost of slower performance. The ability to vary performance along a continuum on which performance is accurate but slow on one end of the spectrum, and fast but relatively inaccurate on the other end is referred to as speed-accuracy tradeoff (SAT). It has long been known that people are able to strategically trade off speed and accuracy (e.g., Busemeyer & Townsend, 1993; Meyer, Osman, Irwin, & Yantis, 1988; Osman et al., 2000; Rinkenauer, Osman, Ulrich, Müller-Gethmann, & Mattes, 2004; Wickelgren, 1977). This ability is far from uniquely human, as it has been observed in species as phylogenetically distant as bumblebees (Chittka, Dyer, Bock, & Dornhaus, 2003) and ants (Franks, Dornhaus, Fitzsimmons, & Stevens, 2003).

There have been various theories as to how people are able to achieve different levels of SAT. These can be divided into "mixture models", and "decision criterion models" (Osman et al., 2000; Rinkenauer et al., 2004). The central claim of the mixture models is that people trade speed and accuracy by varying the proportions of two qualitatively different types of responses: first, chance-level guesses, and second, accurate responses based on analysis and evaluation of the stimulus. Decision criterion models (or "accumulation models", or "sequential-sampling models") are based on the notion that stimuli are identified and responded to by the accumulation of evidence in favor of one or another decision. Different levels of SAT are achieved by varying how much evidence is needed to reach the threshold. When a low amount of evidence is needed, responses are fast but tend to be less accurate.

Although the mixture models are now mostly considered obsolete, it might be worth discussing them briefly. As discussed by Rinkenauer et al. (2004) and Osman et al. (2000), two

different mixture models have been proposed which differ in how guesses are made. The "fast-guess model" postulates that a proportion of the responses are fast, chance-level guesses. In contrast, the "slow-guess" model (or "deadline" model) hypothesizes that people base their decisions on the accumulation of evidence in favor of a response, which is executed as soon as there is enough evidence for one response over its competitors. However, when a time deadline has been reached, which the participant sets on each trial, and there is not enough evidence for one response over the others, the participant makes a random guess; different SAT levels are reached by varying the deadline (Ruthruff, 1996). Both of these models make specific predictions about the distribution of RTs of correct and erroneous trials under various levels of SAT, and neither the fast-guess model nor the slow-guess model have been met with substantial empirical support (as discussed by Osman et al., 2000; Rinkenauer et al., 2004). First of all, the finding that erroneous RTs do vary with the level of SAT is inconsistent with the fast-guess model (Osman et al., 2000), which predicts all error trials to be the same fast guesses. Secondly, the slow-guess model predicts that error RTs are slower than correct RTs, whereas it is commonly observed that errors have fast RTs; typically, the error proportion decreases with slower RTs (e.g., Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; Gratton, Coles, & Donchin, 1992; Ratcliff, Van Zandt, & McKoon, 1999; Ridderinkhof, 2002b). Thirdly, guesses typically do not have chance-level error rates as predicted by the mixture models (Meyer, Irwin, Osman, & Kounios, 1988; Meyer, Osman et al., 1988). It has quite often been reported that very fast responses in interference tasks in fact have a *below* chance level accuracy (e.g., Gratton et al., 1992, Experiment 1), showing that errors are not based on random guesses but are in fact based on processing of the stimulus - just the wrong stimulus dimension.

In contrast, all decision criterion models share a baseline, a threshold, and a growth function. Another feature shared by all decision criterion models is that they do not have any problem accounting for the findings summarized in the previous paragraph (Ruthruff, 1996). These models explain identifying and responding to a stimulus as the accumulation of evidence for one or the other option, and a decision is made once the threshold of the corresponding decision has been reached. Different levels of SAT are achieved by varying the distance between baseline and threshold, thus varying the amount necessary to reach the threshold. If distance between baseline and threshold is large, it takes longer to reach that threshold, thus responses will be slow. However, with a large distance between baseline and threshold, noise or small

incorrect activations induced by the processing of irrelevant stimuli have a smaller chance of reaching threshold, thus responses will also be more accurate.

Several proposed decision criterion models exist. For instance, some theorists have postulated that the buildup of evidence proceeds according to a "random walk model", which describes the accumulation of evidence as a random walk between two (or more) alternatives (e.g., Nikolić & Gronlund, 2002). At stimulus presentation, a random walk, based on sampling at discrete time points, starts; a response is made once that walk reaches one threshold or another. Ratcliff and colleagues (e.g., Ratcliff, 2002; Ratcliff & Rouder, 1998; Ratcliff et al., 1999) have expanded upon this notion with their "diffusion" models, according to which evidence is accumulated continuously over time, rather than sampled at discrete time points as is the case in random walk models. But, like in the random walk models, different levels of SAT are obtained by varying the distance between the starting point and the response boundary. When accuracy is emphasized, the boundaries are far apart, and it will take longer to reach the threshold; under speed emphasis, the boundaries are close to the starting point and therefore the accumulation of evidence will reach the threshold earlier (Ratcliff & Rouder, 1998).

The random walk models and diffusion models are single-counter models, whereas decision criterion models are multiple-counter models (Ratcliff & Smith, 2004; Smith & Ratcliff, 2004; Usher & McClelland, 2001). Single-counter models have one counter (and thus one baseline) with multiple thresholds corresponding to each option; multiple-counter models have as many counters as there are options, each with their own threshold and baseline. Multiple-counter models include the accumulator model (Smith & Vickers, 1988), the Poisson counter model, and the leaky, competing accumulator model (Usher & McClelland, 2001), among others.

The leaky competing accumulator model (Usher & McClelland, 2001) is based on a parallel distributed processing approach (McClelland, Rumelhart, & the PDP Research Group, 1986; Rumelhart, McClelland, & the PDP Research Group, 1986). This model assumes, first, that accumulated evidence is subject to decay (leakage), and second, that the different accumulators are mutually inhibiting one another. It has been suggested that the leaky competing accumulator model and the diffusion model can account equally well for the observed performance data (including RT distributions), and that it is therefore difficult to discriminate between the two approaches (Ratcliff & Smith, 2004; Smith & Ratcliff, 2004).

The notion that evidence accumulates from a baseline to a threshold is consistent with various neurophysiological observations in nonhuman primates (Gold & Shadlen, 2001; Schall, 2003, 2004). Hanes & Schall (1996) recorded in the frontal eye field while monkeys performed a stop-signal task, and found that the rate at which neural activity increased was predictive of the subsequent reaction time (RT) (while each response was associated with a constant level of activation, suggesting a constant threshold throughout this task). Other studies have manipulated the difficulty of the discrimination, for instance by using a dot motion-discrimination task. Such studies have found that the rate at which neural activation accumulates is smaller with more difficult discriminations, resulting in a longer RT. This has been observed in area MT (Ditterich, Mazurek, & Shadlen, 2003), the lateral intraparietal cortex (Roitman & Shadlen, 2002), and the frontal eye field (Kim & Shadlen, 1999). An fMRI study in humans, in which people had to decide whether a blurred stimulus was a face or a house, found that a part of the DLPFC appears to be responsive to the absolute difference between the activation of house-selective and face-selective regions of the temporal cortex (Heekeren, Marrett, Bandettini, & Ungerleider, 2004), suggesting that this region might be involved in the decision between the two alternatives. However, despite a host of data on evidence accumulation and (perceptual) decisions, no neurophysiological or cognitive neuroimaging study has explicitly tried to determine how different levels of SAT are attained.

1.2 COGNITIVE CONTROL AND THE PREFRONTAL CORTEX

The ability to strategically trade speed and accuracy based on the perceived context (depending on the requirements of the task, the environment, the anticipated outcome, and so on) is a form of executive control. Executive control refers to those faculties that allow us to regulate the processing of information in favor of context-appropriate behavior (Logan, 1985). Thus, executive control allows us to overcome direct, automatic stimulus-response mappings and flexibly adapt our behavior to the context. Many instances of executive control have been described and studied, including the selection of appropriate stimuli and responses and the suppression of irrelevant ones, memory storage and retrieval, and the maintenance of relevant

information over short periods of time to guide future actions. Curiously, SAT has not been addressed by neurophysiological or cognitive neuroimaging studies to date.

Generally, executive control is thought to depend on the dorsolateral areas of the prefrontal cortex (DLPFC), which have been argued to represent context information (Cohen, Dunbar, & McClelland, 1990; E. K. Miller, 2000; E. K. Miller & Cohen, 2001; see Wood & Grafman, 2003, for a related proposal). As explained earlier, decisions, including perceptual decisions, are thought to be made by a buildup of evidence for one option or another. It has frequently been observed that selection of a stimulus leads to the amplification of the neural activity associated with the processing of this relevant stimulus, and suppression of other, irrelevant stimuli; such observations have supported biased competition theories of cognitive functioning (Desimone & Duncan, 1995), according to which representations are mutually inhibitory at local levels and excitatory between different kinds of representations or stimulus dimensions (which is also consistent with decision criterion models).

Context representations in the DLPFC have been proposed to be the source of the attentional signal related to the amplification of relevant information and the suppression of irrelevant information (Desimone & Duncan, 1995; B. T. Miller & D'Esposito, 2005; E. K. Miller & Cohen, 2001). Whether this signal is excitatory, and activates task-relevant representations, or inhibitory and suppresses task-irrelevant representations, continues to be a matter of debate. Usher & McClelland (2001) have argued that a purely "inhibitory" view becomes problematic with a greater number of response options, as with a greater number of responses a greater number of context representations is needed; when assuming lateral inhibition, this need not be a problem. An "excitatory view" might therefore be more parsimonious. Nevertheless, various authors have argued in favor of top-down inhibition in various forms of cognition (Allain, Carbonnell, Burle, Hasbroucq, & Vidal, 2004; Anderson et al., 2004; Brunia, 1993).

In short, the DLPFC is thought to bias the accumulation of evidence in favor of appropriate behavior by its top-down connections to representations in posterior cortex.

1.3 THE SIMON TASK

The Simon task is a task that is often used in cognitive psychology to study the effects of cognitive control. It is central to this thesis; therefore, some further discussion about this task is warranted. When making a manual speeded forced-choice button press response, participants react faster and more accurately when the stimulus appears on the same side as the appropriate response (congruent), than when it appears on the opposite side (incongruent) (Fitts & Seeger, 1953; Simon, 1969). This is even the case when the stimulus location is irrelevant for the response, and the participant has to respond to the form, color, pitch, or some other dimension of the stimulus (Craft & Simon, 1970; Simon, 1969; Simon & Berbaum, 1990; Simon & Rudell, 1967); this latter phenomenon is known the Simon effect.

Based on a large amount of data, it is typically assumed that the Simon effect is caused by the parallel activation of two "routes" from perception to action. In the "conditional" route, the appropriate response is activated by a relatively slow intentional process. In the "unconditional" route, the response that corresponds to the stimulus location is activated in a relatively quick and automatic fashion (de Jong, Liang, & Lauber, 1994; Eimer, 1995; Kornblum, 1994; Kornblum, Hasbroucq, & Osman, 1990; Ridderinkhof, 2002b; Wiegand & Wascher, 2005).

Processing along the unconditional route is thought to occur because of dimensional overlap (Kornblum, 1994; Kornblum et al., 1990). That is, the response set and the irrelevant stimulus share a dimension – location – and this shared dimension primes the associated response. Participants encode and maintain the task-relevant responses using a spatial code, and because the irrelevant stimulus varies along the dimension of this spatial code, these two (response set and irrelevant stimulus dimension) automatically become associated with one another. As a result, the location of each relevant stimulus quickly and automatically activates the corresponding response. In contrast, participants are only able to activate the correct response relatively slowly, based on strategic, task-dependent mechanisms (de Jong et al., 1994; Eimer, 1995; Ridderinkhof, 2002b; Wiegand & Wascher, 2005).

In fact, interference tasks such as the Stroop, Simon, and similar tasks are based on the notion that an irrelevant stimulus dimension activates an associated response by a fast and automatic process, while the relevant stimulus-response mapping proceeds much slower –

although there are other forms of dimensional overlap which might also contribute to the specific performance on such a task (Kornblum et al., 1990; Kornblum, Stevens, Whipple, & Requin, 1999). During incongruent trials, the two responses conflict with one another (Coles, Gratton, Bashore, Eriksen, & Donchin, 1985), and it takes time for the correct stimulus-response mapping to override the incorrect response activation. As this fast activation of the incorrect response sometimes reaches response threshold, there are more errors in the incongruent condition than in the congruent condition; moreover, these errors tend to be fast (e.g., Gratton et al., 1988; Ridderinkhof, 2002b).

An important source of supporting evidence for this notion comes from the lateralized readiness potential (LRP). The LRP is computed by taking the difference wave of the ipsilateral and contralateral event-related potentials of the electrodes over the left and right motor cortices, separately for left hand and right hand responses, and then averaging the difference wave across left and right hand responses (de Jong, Wierda, Mulder, & Mulder, 1988; Gratton et al., 1988). The LRP, being a difference wave, thus provides a measure of how much one or the other response hand is activated, free of stimulus-related processing, and how this relative activation evolves over time. Such LRP evidence has shown that during incongruent trials, the incorrect response is activated relatively early, followed by a later activation of the correct response. EMG and measures of response force have provided similar evidence (Burle, Possamaï, Vidal, Bonnet, & Hasbroucq, 2002; Hasbroucq, Possamaï, Bonnet, & Vidal, 1999) but constitute a more peripheral measure of response activation.

So, just like the Stroop task, the Eriksen task, and similar interference tasks, the Simon effect is caused by a fast, more-or-less automatic activation of the response associated with the irrelevant stimulus dimension. However, unlike in the Stroop task, the Eriksen task, and similar tasks, there is no interference between the relevant and irrelevant stimulus dimensions (Kornblum, 1994; Kornblum et al., 1990; Kornblum et al., 1999; Zhang, Zhang, & Kornblum, 1999). That is, in the incongruent condition in Stroop-like tasks, there is a conflict between the relevant and irrelevant stimulus dimensions, above and beyond the conflict between the responses associated with these stimulus dimensions. The existence of such "stimulus conflict" is supported by experimental designs that have, apart from the standard congruent and incongruent conditions, used a condition in which both stimulus dimensions conflict with each other but are mapped onto the same response. This condition therefore contains as much response conflict as

the traditional congruent condition, since both the relevant and irrelevant stimulus dimension are mapped onto the same response in both conditions. Such a stimulus-incongruent condition has been shown to elicit slower responses than the standard congruent condition, but faster and more accurate responses than the standard, response-incongruent condition. This has been shown in the Stroop task (De Houwer, 2003; Schmidt & Cheesman, 2005; Van Veen & Carter, 2005), the Eriksen task (B. A. Eriksen & Eriksen, 1974; C. W. Eriksen & Schultz, 1979; Spieler, Balota, & Faust, 2000; Van Veen & Carter, 2002b; Van Veen, Cohen, Botvinick, Stenger, & Carter, 2001), and the global-local task (Weissman, Giesbrecht, Song, Mangun, & Woldorff, 2003). In contrast, in the traditional Simon task there is no such feasible stimulus-incongruent condition, as there is no inherent conflict between the location and the relevant stimulus dimension (color, shape, pitch, and so on) – unlike for instance between the color red and the word "blue" in the Stroop task.

1.4 PERFORMANCE MONITORING

The amount of attention exerted has frequently been observed to vary on a trial-by trial basis, as a function of trial type. Two kinds of between-trial attentional adjustments are central to this discussion, namely, the conflict adaptation effect, and post-error slowing. These effects, respectively, refer to the observation that the interference effect is greatly reduced (or completely eliminated) on trials following (correct) conflict trials, and to the observation that people respond slower and more accurately on trials that follow error trials. Both phenomena have been linked to adjustments in cognitive control.

It has been proposed that these adjustments of control are linked to the same underlying mechanism, a monitoring system that is responsive to the detection of conflict between mutually incompatible responses or other types or representations, and which engages the systems involved with exerting control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004; Van Veen & Carter, 2002a). Conflict detection is thought to be carried out by the anterior cingulate cortex (ACC), while control is implemented in the DLPFC as explained earlier.

1.4.1 The conflict adaptation effect

The interference effect in Stroop-like and Simon-like tasks is modulated by trial sequence; that is, the interference effect tends to be increased on trials following congruent trials, and reduced on trials following incongruent trials. More specifically, RTs and error rates to incongruent trials typically are reduced when preceded by another incongruent trial (iI) compared to incongruent trials preceded by congruent trials (cI). Conversely, RTs to congruent trials are decreased when preceded by another congruent trial (cC) compared to when preceded by an incongruent trial (iC). This was first shown in the Eriksen task by Gratton et al. (1992, Experiment 1), replicated elsewhere (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Ullsperger, Bylsma, & Botvinick, 2005), and has also been observed in the color word Stroop task (Egner & Hirsch, 2005b; Kerns et al., 2004), and, most importantly for present purposes, the Simon task (Hommel, Proctor, & Vu, 2004; Jentzsch & Leuthold, 2005; Praamstra, Kleine, & Schnitzler, 1999; Stürmer & Leuthold, 2003; Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002; Wühr & Ansorge, 2005). It has also been shown in Simon analogues (Soetens, 1998; Stoffels, 1996a, 1996b), a Stroop analogue using famous faces and written names as stimuli (Egner & Hirsch, 2005a), and a priming task (Kunde, 2003).

Botvinick et al. (2001) proposed to call this phenomenon the “Gratton Effect”, following the mention of this phenomenon by Gratton et al. (1992) using the Eriksen task. More recently, the term “conflict adaptation effect” has become more common (e.g., Egner & Hirsch, 2005b; Mayr, Awh, & Laurey, 2003).

Gratton et al. (1992) proposed that fluctuations in attentional engagement or cognitive control induced by expectancy were responsible for this effect. Botvinick et al. (2001) elaborated upon this theory by proposing that a DLPFC-dependent cognitive control component is regulated on a trial-by-trial basis by an ACC-based monitor which responds to the amount of conflict that exists between active representations in the information processing system. Cognitive control, as explained earlier, refers in this case to the ability to "bias" information processing by supporting the processing of task-relevant stimuli and suppressing the processing of task-irrelevant stimuli. So, increased engagement of control enables one to ignore irrelevant information.

The data and models discussed thus suggest the following sequence of events during conflict adaptation in the Simon task. During incongruent trials, the presence of conflict is

detected by the ACC. This area engages context representations in the DLPFC. On the subsequent Simon trial, the increased DLPFC activation engages the (relevant) color representation more strongly, and/or inhibits the (irrelevant) spatial information or the associated response, thus leading to a smaller susceptibility to the irrelevant stimulus dimension. There is much less conflict during congruent trials than there is during incongruent trials, so considerably less ACC activation. Therefore, control is relaxed during trials following congruent trials, leading to greater susceptibility to the irrelevant stimulus dimension. To summarize, the sequence of events, as a function of the trial type on trial *n* on performance during trial *n* + 1, is therefore as follows:

- iI. Conflict during incongruent trial *n* is detected by the ACC. ACC activation engages DLPFC on the subsequent incongruent trial *n* + 1. During trial *n* + 1, we are better able to ignore the distracting information, so there is less response conflict and relatively fast and accurate performance.
- cI. Since trial *n* is congruent and low on response conflict, the ACC is not engaged to trial *n*, thus, control is also less engaged on trial *n* + 1. We are therefore very susceptible to the irrelevant stimulus dimension on trial *n* + 1, and because this trial is incongruent, response conflict is high and performance is impaired.
- cC. Trial *n* is congruent, therefore there is, again, no ACC activation during this trial, and no ACC-induced engagement of control during trial *n* + 1. We are therefore more susceptible to the irrelevant stimulus dimension on trial *n* + 1, however, since this trial is another congruent trial, the increased processing of the irrelevant spatial information will actually facilitate the correct response, resulting in very fast and accurate performance.
- iC. Conflict during incongruent trial *n* is detected by the ACC, resulting in increased DLPFC activation during the subsequent trial *n* + 1, so we are less influenced by the irrelevant spatial information on trial *n* + 1. However, since trial *n* + 1 is congruent and the processing of the irrelevant spatial information is reduced, the facilitating effect of this information is removed, resulting in somewhat slower RTs.

Evidence from distributional analyses has shown that the increased error rates to cI trials, relative to iI trials, is specifically due to fast slips (e.g., Gratton et al., 1992; Stürmer et al., 2002). Similarly, LRP data have shown that the initial incorrect response activation, that is typically

observed to incongruent trials, is greater for cI trials than it is for iI trials (Stürmer & Leuthold, 2003; Stürmer et al., 2002). This provides supportive evidence for the notion that response conflict is greatest during cI trials.

Botvinick et al. (2001) implemented this theory in several connectionist models, and showed that the behavior of these models on these tasks provides a good fit to the empirically observed behavioral and neuroimaging data. Botvinick et al. conceptualized conflict as energy in a recurrent constraint network (Botvinick et al., 2001; Jones, Cho, Nystrom, Cohen, & Braver, 2003). Hopfield's energy (Hopfield, 1982; Rumelhart, Smolensky, McClelland, & Hinton, 1986) is a measure of activity taken over all units in the set of interest and how well these units are compatible with one another. A high-energy state might be the result of two or more mutually inhibitory units having high levels of activity. Given a certain input, such a network will settle into a locally optimal solution. Thus, the ACC is proposed to respond to such high-energy states and to engage PFC control mechanisms to deal with such conflicts.

Various converging lines of evidence including neuroimaging research have supported the notion that the ACC responds to high-conflict or high-energy states (e.g., Braver, Barch, Gray, Molfese, & Snyder, 2001; Carter et al., 2000; Casey et al., 2000; Durston et al., 2003; Durston, Thomas, Worden, Yang, & Casey, 2002). As far as the conflict adaptation effect is concerned, Botvinick et al. (1999), in the Eriksen task, and Kerns et al. (2004) and Egner & Hirsch (2005b), in the Stroop task, investigated neural activation as a function of trial type sequence, and found that the ACC activation was greatest to cI trials, compared to cC, iC, or iI trials. It has furthermore been found that cognitive control in trials following conflict trials was related to activation of the DLPFC (Egner & Hirsch, 2005b; Kerns et al., 2004), and that the extent to which the ACC was activated predicted both the DLPFC activation and the behavioral conflict adaptation (Kerns et al., 2004).

Egner & Hirsch (2005a) used a Stroop analogue in which participants were presented with the names and faces of politicians or actors, and were asked to respond to either the name or the face, the other stimulus being the distractor. Egner and Hirsch observed modulation of the fusiform face area by the conflict adaptation effect when faces were targets rather than distractors, and observed a tight coupling between the DLPFC and this area. These data suggested that the role of the DLPFC in the conflict adaptation effect is to support task-relevant stimuli rather than inhibit task-irrelevant stimuli, consistent with a biased competition view of

attentional selection. It is however possible that this does not hold true in the Simon effect; it is possible that overcoming the incorrect response activation due to overlap between location and response hand involves different processes than those involved in overcoming interference from automatic word reading; perhaps involving the direct top-down inhibition of the activated spatial representation or associated response (cf. Stürmer & Leuthold, 2003). However, for the sake of simplicity, I will assume lateral inhibition, as suggested by the data of Egner & Hirsch (2005a), in the remainder of this document.

Thus, in sum, according to the conflict-control loop notion, the conflict adaptation effect occurs because conflict leads to greater engagement of control.

1.4.2 Post-error slowing

People are very efficient and fast at correcting their own slips of action; in speeded response tasks, the time between the erroneous and the corrective responses is typically less than 200 ms, making error correction to be one of the fastest cognitive processes (e.g., Cooke & Diggles, 1984; Rabbitt, 1966a; Rodríguez-Fornells, Kurzbuch, & Münte, 2002). In speeded forced-choice response tasks there are large similarities between the data on error and conflict detection, in particular in the neural basis of these two processes. ERP research has shown that slips of action are immediately followed by a large-amplitude, sharp negative waveform which peaks 50-100 ms following button press (e.g., Dehaene, Posner, & Tucker, 1994; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Luu & Tucker, 2001; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Ridderinkhof et al., 2002; Rodríguez-Fornells et al., 2002; Van Veen & Carter, 2002b), and/or 100-150 ms after EMG onset (e.g., Gehring & Fencsik, 2001; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Kopp, Rist, & Mattler, 1996; Scheffers & Coles, 2000; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). This component is usually referred to as error negativity (N_e) or error-related negativity (ERN).

Dipole modeling studies have consistently modeled this component as having a generator in the ACC (Alain, McNeely, He, Christensen, & West, 2002; Debener et al., 2005; Dehaene et al., 1994; Gehring, Himle, & Nisenson, 2000; Holroyd, Dien, & Coles, 1998; Liotti, Woldorff, Perez, & Mayberg, 2000; Miltner et al., 2003; Nieuwenhuis et al., 2003; Rüsseler, Kuhlicke, &

Münte, 2003; Van Veen & Carter, 2002b). Converging evidence from functional MRI studies has shown that action slips are accompanied by ACC activation (Braver et al., 2001; Carter et al., 1998; Debener et al., 2005; Garavan, Ross, Kaufman, & Stein, 2003; Kerns et al., 2004; Menon, Adleman, White, Glover, & Reiss, 2001; Rubia, Smith, Brammer, & Taylor, 2003; Ullsperger & von Cramon, 2001; Van Veen, Holroyd, Cohen, Stenger, & Carter, 2004).

Control is also thought to be more strongly engaged on trials following action slips. People respond slower and more accurate following error trials, a phenomenon usually referred to as post-error slowing (Kleiter & Schwarzenbacher, 1989; Laming, 1979; Rabbitt, 1966b; Rabbitt & Rodgers, 1977). Post-error slowing is frequently thought to be dependent on a control mechanism. Part of the evidence for this comes from studies of clinical populations; post-error slowing is reduced in illnesses characterized by impaired cognitive control, including schizophrenia (Alain et al., 2002; Kerns et al., 2005) and attention deficit hyperactivity disorder (Schachar et al., 2004). Furthermore, post-error slowing appears to be associated with increased DLPFC activation on the trial following the error (Kerns et al., 2004).

Above and beyond post-error slowing, the same sort of top-down control as is engaged following conflict trials has also been argued to be engaged following error trials. It has been argued that such top-down control is reflected in a type of distributional analysis referred to as delta plot (Ridderinkhof, 2002a, 2002b). Delta plots are obtained by binning the data based on RT quantiles, and plotting the interference effect against RT (Ridderinkhof, 2002a, 2002b). Ridderinkhof has proposed that increased top-down control is reflected as a more negative-going slope in the slow part of the delta plot (cf. APPENDIX D). This slope has been found to be more negative-going on trials following errors than on trials following correct trials (Ridderinkhof, 2002b). Thus, assuming that this slope indeed reflects the engagement of control mechanisms, this constitutes further evidence that control is increased following error trials.

The ERN has often been related to the degree of post-error slowing; that is, the greater the amplitude of the ERN, the slower participants tend to be on the trial following the error (Debener et al., 2005; Gehring et al., 1993). Consistently, functional MRI studies have repeatedly shown that the amplitude of the error-related ACC activation is positively related to post-error slowing (Garavan, Ross, Murphy, Roche, & Stein, 2002; Kerns et al., 2004). In a recent study involving the simultaneous recording of ERPs and functional MRI, Debener et al.

(2005) observed a within-subject, trial-to-trial coupling between the ERN amplitude, error-related ACC activation, and post-error slowing.

The ERN is generally thought of as being related to the motivational significance of an error. Errors on trials during which participants are more motivated to perform correctly - either because they are told they will receive a higher reward for correct trials, or because they are told they are being evaluated by a researcher and compared to their peers - elicit greater ERNs (Hajcak, Moser, Yeung, & Simons, 2005). Importantly, the ERN is greater the more emphasis participants are asked to put on accuracy over speed in their performance (Falkenstein et al., 2000; Gehring et al., 1993).

Botvinick et al. (2001) implemented post-error slowing in their neural network simulation by assuming that the ACC-based conflict monitor influences response priming. Botvinick et al. hypothesize a separate unit that has an excitatory influence on the response units; upon the detection of the conflict that occurs during error trials, activation of this unit is decreased, thereby reducing the input into the response layer of their model. Thus, following an error, the level of baseline activation of the response units is decreased, causing slower and more accurate performance on the post-error trial.

1.4.3 Anterior cingulate activation as reflected in the ERP

As stated earlier, the ERN has often been modeled as having a generator in the ACC (e.g., Dehaene et al., 1994; Holroyd et al., 1998). It has also been shown that in the Eriksen task, the ERN has a similar scalp topography as the frontocentral N2, which is elicited by high-conflict trials before a response is made (Kopp et al., 1996). Van Veen & Carter (2002b) proposed that both components reflect the detection of conflict by the ACC, and that the differences in timing between these two components – that is, the N2 occurs prior to the response, the ERN immediately follows it – can be explained by examining the similarities and differences between the timing of response activation during high-conflict correct trials and error trials. During correct high-conflict trials, there is an initial activation of the incorrect response (e.g., Gratton et al., 1988; Gratton et al., 1992; Kopp et al., 1996); the correct response conflicts with this early activation but manages to override it, leading to the moment of maximum conflict to occur prior

to the (correct) response. In contrast, error trials are characterized by fast, impulsive responses and a fast tendency to correct the error. Thus, the error itself results from the initial activation of the incorrect response reaching response threshold (Gratton et al., 1988; Kopp et al., 1996; Rabbitt & Rodgers, 1977; Rabbitt & Vyas, 1981). Therefore, the moment of maximum conflict between the erroneous response and the competing correct response occurs immediately after the error. This explanation can therefore explain why ACC activation occurs prior to the response during correct trials, but immediately following the response on error trials (Van Veen & Carter, 2002b). This notion has subsequently been implemented in connectionist models (Yeung, Botvinick, & Cohen, 2004; Yeung & Cohen, 2006).

Interestingly, the N2 and ERN appear to be differentially influenced by SAT. The N2 has been shown to be increased under speed emphasis (Band, Ridderinkhof, & van der Molen, 2003; Jodo & Kayama, 1992); conversely, the ERN has been shown to be increased under accuracy emphasis (Falkenstein et al., 2000; Gehring et al., 1993). Thus, the notion that both of these components are generated by the ACC predicts that conflict-related and error-related ACC activation are similarly differentially modulated by SAT.

1.4.4 Alternative accounts

Several alternative (competing or complementary) accounts have been proposed regarding the ACC and the conflict adaptation effect. Some of these alternative accounts, for as far as they are relevant to the present thesis, are discussed below.

1.4.4.1 The conflict adaptation effect and repetition priming Mayr et al. (2003), using the Eriksen task, noted that it is possible that the conflict adaptation effect is caused by stimulus or response repetitions. Typically, participants are faster to respond to a stimulus when the preceding stimulus is identical or requires a similar response, and approximately half of all cC and iI trials involve direct stimulus repetitions, which might contribute to the conflict adaptation effect. Removing exact repetition trials, they found that the conflict tadaptation effect had been eliminated; they therefore proposed that priming mechanisms might be solely responsible for the conflict adaptation effect. Other researchers, however, have found that controlling for repetition

effects does not remove the conflict adaptation effect in the Stroop task (Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004), and even in the Eriksen task (Ullsperger et al., 2005).

In fact, in their earlier series of studies on the conflict adaptation effect in the Simon task, Stürmer et al. (2002, experiments 3 and 4), also tested this same notion, but found that removing exact stimulus repetitions from their data reduced the conflict adaptation effect only slightly (Mayr et al. do not cite Stürmer et al., 2002). Stürmer et al. thus rejected the notion that stimulus priming by itself is solely responsible for the conflict adaptation effect. These findings for the Simon task have been replicated elsewhere (Wühr & Ansorge, 2005).

1.4.4.2 The conflict adaptation effect and the theory of event files The notion that the conflict adaptation effect is based on binding is based on the "theory of event files" (Hommel, 2004; Hommel, Müsseler, Aschersleben, & Prinz, 2001), which in turn is based on the earlier notion of object files (Kahneman, Treisman, & Gibbs, 1992). The theory of event files maintains that in order to respond appropriately to a stimulus, the different stimulus and response dimensions need to be "bound" into a single representation, an "event file". Critical is the notion that once bound into an existing event file, it takes time to "unbind" a representation. Also critical is the notion that once one or more features are bound into an active event file, repetition of those features will prime the entire event file.

To illustrate this point, all the possible stimulus repetitions in a Simon task are illustrated in Table 1, which contains two target stimulus values (S1 and S2), two appropriate responses (R1 and R2) and two possible locations (L1 and L2):

Table 1. Possible trial-to-trial ensembles in the Simon task.

1.	cC	S1R1L1 – S1R1L1 and S2R2L2 – S2R2L2	repetition
2.	cC	S1R1L1 – S2R2L2 and S2R2L2 – S1R1L1	alternation
3.	cI	S1R1L1 – S1R1L2 and S2R2L2 – S2R2L1	repetition
4.	cI	S1R1L1 – S2R2L1 and S2R2L2 – S1R1L2	alternation
5.	iC	S1R1L2 – S1R1L1 and S2R2L1 – S2R2L2	repetition
6.	iC	S1R1L2 – S2R2L2 and S2R2L1 – S1R1L1	alternation
7.	iI	S1R1L2 – S1R1L2 and S2R2L1 – S2R2L1	repetition
8.	iI	S1R1L2 – S2R2L1 and S2R2L1 – S1R1L2	alternation

Note that in cases 1 and 7, the stimulus and associated correct response are an exact repetition (complete repetitions); in cases 3 and 5, the target stimulus and associated correct response are repetitions but the location is alternated (partial alternations); in cases 4 and 6, the target stimulus and correct response are alternated but the location is repeated (partial repetitions), and only in cases 2 and 8, no features are repeated (complete alternations).

Thus, for cC trials in case 1, no new event file has to be made. Instead, the already existing and active event file is primed. Therefore, RTs in this condition will be fast. For cC trials in case 2, there is no overlap in the features between the previous and current trial. Therefore, while all of the features have to be bound into a new event file, none of its ingredients have to be "unbound"; RTs in this condition will therefore be fast, but slower than RTs in case 1.

For cI trials in case 3, the target stimulus and appropriate response are already bound into an existing event file and therefore have to be "unbound" for a new, appropriate event file. For cI trials in case 4, the location is bound into an existing event file, so only the location has to be unbound to form a new event file. Thus, RTs in cases 3 and 4 will be slow.

Similarly, for iC trials in case 5, the target stimulus and appropriate response are already bound into an existing event file and have to be "unbound". For iC trials in case 6, the location has to be unbound from the existing event file and bound into a new one. Thus, RTs will be slow in cases 5 and 6.

Finally, iI trials in case 7 are complete stimulus repetitions, and therefore the existing event file is primed. In contrast, iI trials in case 8 are complete stimulus alternations, so RTs in both of there conditions will be relatively fast although faster in case 7.

Hommel et al. (2004) admit that it is difficult to distinguish between an event file interpretation of the conflict adaptation effect and a conflict-control based interpretation, because both theories predict fast RTs to cC trials and very slow RTs to cI trials, with RTs to iC and iI trials somewhere in between. They suggest, however, that an event-file interpretation might be more parsimonious (Hommel et al., 2004).

1.4.4.3 ACC functioning and the reinforcement learning theory Holroyd & Coles (2002) proposed a different interpretation of conflict-related and error-related ACC activation, as part of their "reinforcement learning" theory. They proposed that behavior is monitored by an "adaptive critic", localized in the basal ganglia. This adaptive critic determines whether events are better or

worse than expected, and signals this with a phasic increase or a decrease, respectively, in dopaminergic activity in the ACC. According to this theory, different cognitive processes compete for access to the motor system, and the function of the ACC is to select between these different cognitive processes based on how it's been trained by the dopamine signal from the basal ganglia. Holroyd and Coles assume that error-related ACC activation is assumed to be generated by a phasic reduction in dopamine influx; the inhibitory influence of the dopaminergic innervation in the ACC is briefly disrupted, fine-tuning the ACC to do a more appropriate selection job on future trials.

Holroyd & Coles (2002) based themselves for a large part on findings involving an ERP component somewhat resembling the ERN (or N2) which has been found to be elicited by error feedback stimuli. Several studies have modeled this component as having an ACC-based generator (Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997; Ruchsow, Grothe, Spitzer, & Kiefer, 2002). Holroyd & Coles assume that this component is functionally equivalent to the ERN; they assume this component also indicates a phasic reduction of dopamine influx in to the ACC. In fact, the model is also consistent with some studies using psychopharmacological manipulations; specifically, dopamine agonists increase the ERN amplitude while antagonists decrease it (e.g., De Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; Tieges, Ridderinkhof, Snel, & Kok, 2004; Zirnheld et al., 2004),

However, several lines of research are not consistent with this theory, or have not given consistent results. Several functional MRI studies, using a task that has previously been shown to elicit a reliable error feedback-related negativity (Miltner et al., 1997) have failed to find significant ACC engagement to error feedback stimuli (Nieuwenhuis, Slagter, Alting von Geusau, Heslenfeld, & Holroyd, 2005; Van Veen et al., 2004). This casts considerable doubt on the plausibility of these dipole models; in the absence of supporting findings, it is no longer parsimonious to assume that the error feedback negativity is generated by the ACC. This, in turn, casts doubt on this otherwise interesting theory.

Other lines of research have given inconsistent results. For instance, a strong prediction of the theory is that people suffering from Parkinson's disease would show decreased ERNs. However, while some reports support this notion (Falkenstein et al., 2001), others do not (Holroyd, Praamstra, Plat, & Coles, 2002). It should also be noted that whereas the effects of dopaminergic drugs on the ERN appear consistent across studies, noradrenergic drugs have the

similar effects (De Bruijn et al., 2004; Riba, Rodriguez-Fornells, Morte, Münte, & Barbanoj, 2005); therefore the effects of dopaminergic drugs on the ERN might not be specific to dopamine.

1.4.4.4 ACC functioning and the error likelihood model Brown & Braver (2005) proposed that rather than detecting conflict, the ACC learns to respond to stimuli that are associated with a high error rate. They tested this prediction using a change-signal task (modeled after the stop-signal task) in which participants had to make a forced-choice response to a stimulus ("go" stimulus) unless a second stimulus, presented shortly after the imperative stimulus, indicated the opposite response was required ("change" stimulus). By varying the stimulus onset asynchrony between the go and change stimuli, error rates per participants were set to be 4% in an easy condition and 50% in a difficult condition. Prior to the go and change stimuli, a cue instructed participants whether the upcoming stimulus set was going to be difficult or easy. According to Brown & Braver's (2005) model, the ACC receives input about the identity of the stimulus and learns to respond to which stimuli are associated with error commission. As predicted by their model, they found that the ACC activation is greatest to difficult change stimuli and smallest to easy go stimuli. According to Brown & Braver, a model in which the ACC responded to the conflict between the two responses would have predicted that the ACC should respond equally strongly to change stimuli under hard and easy conditions, due to equivalent levels of conflict in their model.

It should be noted here that the architecture of the conflict model of Brown & Braver (2005) differs in important ways from the architecture of the models used by Botvinick et al. (2001). For instance, easy and difficult go and change stimuli are assumed to be represented separately by the perceptual layer. Furthermore, evidence from LRPs from similar tasks strongly suggests that response conflict does increase with longer intervals between the two stimuli (de Jong, Coles, Logan, & Gratton, 1990). Thus, we might question the validity of their response conflict model.

It should also be noted that while Brown & Braver's (2005) model assumes that the ACC responds to the perceptual representations of stimuli, the ACC receives very little input from the occipital and temporal cortices; its main cortical input comes from parietal and frontal areas including the motor areas (Goldman-Rakic, 1988). Lastly, the error likelihood model strongly

predicts that conflict-related ACC activation should always be proportional to the error rates in each experimental condition, yet this is not always the case (Van Veen & Carter, 2005). Nevertheless, Brown & Braver's error likelihood model was shown to be able to account for learning effects, which conflict models typically do not.

1.5 PSYCHOPHYSIOLOGICAL INVESTIGATIONS OF SAT

Several studies have investigated some of the psychophysiological differences between performing at different levels of SAT during response interference tasks and related paradigms. One such line of inquiry concerns the effects of different levels of SAT on motor preparation (Band et al., 2003; Osman et al., 2000; Rinkenauer et al., 2004; Van der Lubbe, Jaśkowski, Wauschkuhn, & Verleger, 2001). Osman et al. (2000), in an ERP study, had participants emphasize either speed or accuracy when performing an Eriksen task. They distinguished two types of SAT, namely, SAT as depending on instruction and SAT as expressed in trial-to-trial variability (i.e., within any block, regardless of SAT emphasis, fast responses will be less accurate than slower responses). Specifically, these authors divided the time from stimulus onset to the response into two; the interval between stimulus onset and the onset of the LRP (S-LRP), and the interval between the onset of the LRP and the moment that the response was made (LRP-R interval). This distinction is made quite frequently, as these two measures reflect different aspects of cognitive processing; they can be independently manipulated (e.g., Osman et al., 2000; Rinkenauer et al., 2004). It is usually assumed that the LRP-R interval mostly reflects response processing, whereas the S-LRP interval mostly reflects stimulus processing.

Osman et al. found that instruction only affected the LRP-R interval, which was longer under accuracy emphasis than under speed emphasis; conversely, trial-to-trial RT variability (as determined by a per-block, per-condition median split) only affected the S-LRP interval. Consistent with the notion that between-trial variability in stimulus-related processes, the authors also found that the latency of the P300 of the event-related potential, which is often taken as a measure of stimulus-related processing (e.g., Kutas, McCarthy, & Donchin, 1977), was influenced by trial-to-trial variability in SAT, but not by SAT instruction. These data are consistent with the notion that strategically trading speed and accuracy involves a change in the

distance between the start and the threshold of a decision process based on evidence accumulation, as predicted by the various accumulator models. However, Osman et al. note that the finding that trial-to-trial variability in SAT has effects on different measures might not be consistent with such models in so far as they would predict random trial-to-trial variability in SAT to be caused by variability in that same parameter, the distance between onset and threshold.

As far as the effects of instructed SAT are concerned, which is what we are mostly concerned with here, similar data were obtained by Van der Lubbe and colleagues (Van der Lubbe et al., 2001). They noted that both in the Simon task and in a location-based task (similar to the Simon task except that participants were asked to respond to the location of the stimulus, ignoring its identity), speed emphasis shortened the LRP-R interval while leaving the S-LRP interval unaffected. Van der Lubbe et al. also noted that in both tasks, the posterior contralateral negativity (PCN), an ERP component related to spatial orienting, was unaffected by the SAT manipulation. In both of these tasks, and also in a simple single-response task, the response force of the correct response was increased under speed emphasis, replicating other work (Jaśkowski, Van der Lubbe, Wauschkuhn, Wascher, & Verleger, 2000; Sangals, Sommer, & Leuthold, 2002). In this study, however, time pressure did not affect the initial activation of the incorrect response hand as indexed by the LRP (Van der Lubbe et al. do not report response force measures of the incorrect response hand).

In contrast to these two studies, Rinkenauer and colleagues (Rinkenauer et al., 2004) obtained slightly different results. These authors noted that in the study by Osman et al. (2000), SAT was manipulated by simple instruction, and the overall SAT effect on RTs and error rates of this instruction wasn't very large. Similarly, although Van der Lubbe et al. (2001) manipulated SAT by changing a response deadline, the overall effect of this manipulation on SAT was again relatively small. In order to encourage greater tradeoff effects, Rinkenauer et al. had participants perform at three SAT levels, using a changing deadline procedure which resulted in performance at 97.5%, 82%, or 66% accuracy. They did this during three different tasks; a line discrimination task, a lexical task (noun gender), and an Eriksen task. In contrast to what was observed in the Osman et al. (2000) and Van der Lubbe et al. (2001) studies, Rinkenauer et al. observed that high speed stress did in fact shorten the S-LRP interval, in all three different tasks. More specifically, in all three tasks, the S-LRP interval decreased with increased speed stress, but the LRP-R

interval "saturated" at medium and high speed stress (Rinkenauer et al., 2004). However, after correcting for the contribution of lucky guesses (by subtracting out the LRP based on error trials), they found that the SAT effects on the S-LRP interval almost disappeared in the Eriksen task, while in the perceptual and lexical tasks, the S-LRP interval still decreased with increased speed stress. Guesses might contribute to the S-LRP if upon presentation of the stimulus, the participant were to arbitrarily start preparing one of the responses (Sangals et al., 2002), whereas in paradigms that rely more on perceptual discrimination, they are more likely to occur at processing stages related to making those perceptual discriminations, and likewise for different paradigms such as syntactic discrimination tasks and so on. This supports the notion suggested by Osman et al. (2000) and Rinkenauer et al. (2004) that the locus of SAT is likely to be task-specific; in response interference tasks such as the Eriksen and Simon tasks, SAT is likely to occur at motor-related processing stages.

Others have investigated performance at different levels of SAT using cue-target paradigms. In a task studied by Band and colleagues (Band et al., 2003), participants viewed an arrow cue pointing either left or right, after which they saw a target stimulus of a particular color. Targets of one color, which appeared in 80% or trials per block, indicated that a response congruent with the direction of the arrow had to be made; the other color indicated in certain blocks ("go/no-go" blocks) that a response had to be withheld, and in other blocks ("go/change" blocks) that a response opposite to the arrow had to be made. SAT was manipulated on a between-subject basis. In both go/no-go blocks and go/change blocks, speed emphasis increased both the contingent negative variation (CNV) and the LRP elicited by the arrow cue, suggesting that participants increased the activation of the response congruent with the arrow under speed emphasis. Further analyses showed that the N2 observed in change and no-go trials was enhanced under speed emphasis, replicating prior research regarding N2 modulation by SAT in a go/no-go task (Jodo & Kayama, 1992). Also, LRP results indicated that these effects of motor preparation were carried over into the response to the target; the incorrect motor activation to no-go and change trials was enhanced under speed emphasis, whereas the correct motor activation to go trials was facilitated under speed emphasis.

Note that these results are not the same as those found by Van der Lubbe et al. (2001), who found that the degree of incorrect response activation was not modulated by SAT. A likely explanation is that in the cueing paradigm used by Band et al. (2003), a cue indicated that one

response was going to be likely, and participants therefore prepared this response; Band et al. found that this response preparation prior to the imperative stimulus was enhanced under speed emphasis. In the Simon and location tasks used by Van der Lubbe et al. (2001), no advance selective response preparation was possible, and participants had most likely both responses equally prepared, on average (i.e., an increase in baseline activity of both responses under sped emphasis). Over and above this enhanced baseline activity, the LRP (which is a difference wave, after all) did therefore not differ in the Van der Lubbe et al. tasks.

1.6 SAT, RESPONSE INTERFERENCE, AND ATTENTION

The reviewed evidence suggests the following about how SAT modulates response preparation processes in interference tasks. First, the level of baseline activation is increased under speed emphasis and reduced under accuracy emphasis, such that it takes a longer time and more accumulation of evidence to reach the response threshold; the notion that thresholds are relatively constant despite variation in RTs is suggested by data by Hanes & Schall (1996), who showed that the accumulation of evidence for an eye movement in the frontal eye field reaches a constant level of activation prior to the response regardless of the actual RT (although it could be argued that the monkeys in that task were performing at a constant level of SAT). Transient, response-related activation is therefore associated with greater amplitudes under accuracy emphasis. In contrast, LRP evidence has shown that, in interference tasks, the initial activation of the incorrect response that is often observed during high-conflict trials is not influenced by different levels of SAT (Osman et al., 2000; Van der Lubbe et al., 2001). Note that this activation to the irrelevant stimulus dimension is relative to baseline, so in absolute terms we can assume that an increase in baseline activation under speed emphasis would result in a greater amplitude of the incorrect response in absolute terms (see APPENDIX A).

Similarly, fast errors during speeded response tasks are thought to be the result of this initial incorrect response activation reaching response threshold; such errors are followed by the immediate tendency to correct the error. We have proposed that ACC activation during error trials represents the conflict between this initial error activation and the subsequent corrective response. If the level of baseline activation is increased under speed emphasis, we can assume

the both the (transient) error response and the (transient) corrective response to be relatively decreased under speed emphasis compared to baseline. Therefore, while the conflict between the erroneous response and the corrective response might or might not be equal between speed and accuracy emphasis in absolute terms, the transient, baseline-to-peak level of conflict can be assumed to be greater under accuracy emphasis since the level of baseline activation is lower under accuracy emphasis (see APPENDIX A).

As reviewed, despite the fact that computational approaches can account for performance data of SAT manipulations, not much is known about how SAT itself is implemented in the brain – that is, those studies that have studied SAT as a form of executive control have usually studied the effects of already having established a desired level of SAT, as opposed to how people achieve this desired level in the first place. On the other hand, different levels of SAT are known to influence other measures; the ERN is greater under accuracy emphasis (Falkenstein et al., 2000; Gehring et al., 1993), the N2 is greater under speed emphasis (Band et al., 2003; Jodo & Kayama, 1992), the interval between LRP onset and response is enhanced under accuracy emphasis (Osman et al., 2000; Rinkenauer et al., 2004).

In the two experiments presented in this document, two aspects of SAT were explored. One experiment addressed the neural mechanisms of selecting a desired level of SAT (Experiment 2). Another experiment addressed the effects that different SAT levels might have on other types of attentional control (Experiment 1) – specifically, the effects of SAT on the conflict adaptation effect and on post-error slowing. SAT was manipulated in Experiment 1 in a "blocked" design, by instructing participants before onset of each block whether to emphasize speed or accuracy during performance. In Experiment 2, SAT was manipulated using a "cued" or an "event-related" design, by presenting the participant with cues instructing them to emphasize speed or accuracy on the set of trials that followed each cue, such that the processes associated with selecting a desired level of SAT could be studied by analyzing the neural responses to the speed and accuracy cues.

The Simon task was chosen in this set of experiments rather than similar tasks, such as the Eriksen and Stroop tasks, for two reasons. First, as opposed to the Eriksen and Stroop task, the conflict that is responsible for the interference effect in performance is thought to occur only between the responses; in the Stroop and Eriksen, there is additional conflict between the stimulus representations (Kornblum et al., 1990; Kornblum et al., 1999; Van Veen & Carter,

2005; Van Veen et al., 2001). Second, the conflict adaptation effect appears to be extremely robust in the Simon task (cf. Stürmer & Leuthold, 2003; Stürmer et al., 2002), whereas in the Eriksen task, it is not always found (cf. Mayr et al., 2003).

2.0 EXPERIMENT 1

2.1 PREDICTIONS

Selecting at which level of SAT to perform is a form of executive control. The purpose of Experiment 1 was to test how different levels of SAT influence other manifestations of executive control. Since the effect of manipulating SAT in a controlled fashion influences response processes, at least in interference tasks (Osman et al., 2000), it can be hypothesized that the level of SAT at which a participant is performing should influence instances of executive control that are influenced by response processes. The conflict monitoring theory postulates that executive control is influenced by the conflict between active but incompatible responses. The conflict adaptation effect and post-error slowing are two such control-dependent effects that are thought to be driven by this kind of conflict. Therefore, these effects should be influenced by SAT, if this interpretation of the conflict theory and SAT effects are correct.

Experiment 1 was, in essence, a replication of the study performed by Kerns et al. (2004), using SAT as an addition within-subject manipulation, and using the Simon instead of the Stroop task; before the onset of each block, participants were asked to emphasize either speed or accuracy in their performance. The design used by Kerns et al. allowed them to study conflict, errors, the conflict adaptation effect, post-error slowing, and the neural processes associated with these phenomena. Recall that Kerns et al. found that:

- conflict and errors engaged a similar area of the ACC (cf. Van Veen & Carter, 2002b; Carter et al., 1998);
- conflict-related ACC activation was greater during cI than during iI trials (also found by Botvinick et al., 1999; Egner & Hirsch, 2005b);

- there was increased control-related activation of the DLPFC on trials following conflict trials, related to the conflict adaptation effect (see also Egner & Hirsch, 2005a; Egner & Hirsch, 2005b);
- this control-related DLPFC activation was related to the amplitude of the ACC activation on the preceding conflict trial (see also Egner & Hirsch, 2005a; Egner & Hirsch, 2005b);
- there was increased control-related activation of the DLPFC following error trial, as well (related to post-error slowing);
- this control-related DLPFC activation was again related to the magnitude of the ACC activation on the preceding error trial (Kerns et al., 2004).

A similar design, using SAT as a within-subject factor, allows for the study of whether and how these trial-to-trial control adjustment effects, and the neural processes associated with them, are modulated by different levels of SAT.

Thus, Experiment 1 was designed to test predictions inspired by several notions published earlier, regarding how different levels of SAT influence different measures of conflict and control, specifically, the conflict adaptation effect and post-error slowing. These predictions are as follows.

First, the basic previously reported effects should be verified. Thus, the basic SAT and Simon effects should be confirmed; performance should be faster but less accurate under speed than under accuracy emphasis, verifying the SAT effect, while performance should be faster and more accurate to congruent than to incongruent trials, verifying the Simon effect. In addition, the conflict adaptation and post-error slowing effects should be replicated; interference should be smaller following incongruent than following incongruent trials, and participants should respond slower following error trials.

Second, the findings reported by Kerns et al. (2004) should be replicated. Thus, conflict and errors should engage a similar area of the ACC; conflict-related ACC activation should be greater during cI than during other (correct) trial types; control associated with the conflict adaptation effect and post-error slowing should be associated with activation of the DLPFC; ACC activation during conflict or error trial *n* should be correlated with DLPFC activation on trial $n + 1$ within participant.

Third, based on the dipole models of the ERN and N2 as being generated by the ACC (Van Veen & Carter, 2002a, 2002b), and the observed differential modulation of these components by SAT, opposite effects of SAT on conflict-related and error-related ACC activation should be expected. A strong prediction of these dipole models thus holds that ACC activation as measured by fMRI should be modulated by the same experimental manipulations that modify N2 and ERN, in the same direction. Specifically, since the N2 is enhanced under speed emphasis (Band et al., 2003; Jodo & Kayama, 1992), ACC activation during correct high-conflict trials should also be greater during speed emphasis. In contrast, since the ERN is enhanced under accuracy emphasis (Falkenstein et al., 2000; Gehring et al., 1993), ACC activation during error trials should be greater under accuracy emphasis.

Fourth, the conflict monitoring theory (e.g., Botvinick et al., 2001; Van Veen & Carter, 2002a), strongly predicts that conflict, ACC activation, subsequent DLPFC activation, and control-related changes in performance should covary with one another. Thus, manipulations that result in greater ACC activation should lead to greater DLPFC activation, which should lead to a greater expression of control; conversely, manipulations that result in smaller ACC activation should lead to smaller DLPFC activation, which should lead to a smaller expression of control.

Fifth, an important prediction derived from the computational models of the mechanisms of SAT discussed earlier involves the amplitude of the response signals. If it indeed takes a longer time, or greater amount of accumulated evidence, to reach the response threshold under accuracy emphasis (as is assumed by the decision criterion models), response-related activation in the premotor and/or primary motor cortices contralateral to the correct response during correct trials should be greater under accuracy emphasis.

Sixth, the effects of responding on areas of the motor system should replicate the results of LRP studies. That is, conflict-related activation in the premotor and motor cortices contralateral to the incorrect response should be increased to cI trials, but this activation should not be modulated by SAT, as is the case for the LRP deflection associated with the initial activation of the incorrect response during high-conflict trials (Osman et al., 2000; Van der Lubbe et al., 2001).

2.2 MATERIALS AND METHODS

2.2.1 Research participants

Twenty-four healthy adults (16 females, 8 males), all right hand-handed, and all between 20 and 33 years of age (M = 25, SD = 4) participated in this experiment. They received a monetary fee of $75 for participating. All had normal or corrected-to-normal vision. Prior to the test, participants provided written informed consent in accordance with the Institutional Review Board of the University of California at Davis.

2.2.2 Task procedure

Experimental control was implemented using the E-Prime experimental software suite (Psychological Software Tools, Pittsburgh, PA). A fixation point and the grey outline of two squares left and right of the fixation point were present on screen throughout each block. During each trial, one of these two squares lit up in either red or green (duration 150 ms); the intertrial interval was 2850 ms. Participants were instructed to respond with a left or right index finger button press to the color of each stimulus, ignoring whether it was presented to the left or right of fixation.

Trial types were defined as follows. When the presentation side of the stimulus (left/right of fixation) and the response hand (left/right) matched, the stimulus was congruent; when the stimulus was mapped onto the opposite response hand, the stimulus was incongruent. An example of a trial sequence is shown in Figure 1.

Thus, for participants instructed to respond to green with the left hand and to red with the right hand, Figure 1 depicts a sequence consisting of a congruent, incongruent, and congruent trial.

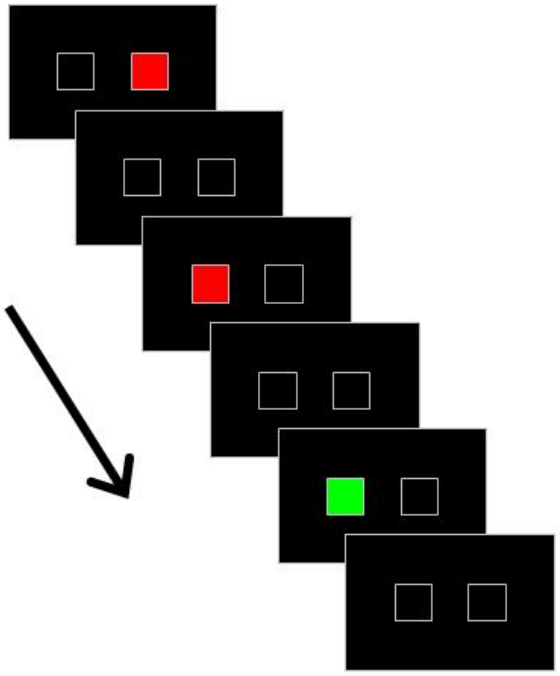

Figure 1. Example of a trial sequence in Experiment 1.

For the analysis of the conflict adaptation effect, trial types were defined by the preceding trial type, as follows:

- cC trials were congruent trials immediately preceded by a correct congruent trial;
- cI trials were incongruent trials immediately preceded by a correct congruent trial;
- iC trials were congruent trials immediately preceded by a correct incongruent trial;
- iI trials were incongruent trials immediately preceded by a correct incongruent trial.

For each overt error, the RT difference between the trial immediately following and the trial immediately preceding the error was calculated in order to analyze post-error slowing.

There were 4 blocks of 150 trials each (100 congruent trials, 50 incongruent trials, presented in random order with the constraint that the first 4 trials of each block were congruent trials). Each block started and ended with a 12000 ms fixation screen. Before each block, participants were asked to either emphasize speed or accuracy during performance. Participants started with either two accuracy blocks or two speed blocks. Stimulus-response mapping and the order of speed/accuracy emphasis blocks were fully counterbalanced across participants.

2.2.3 Imaging procedures

2.2.3.1 Scanning procedures Functional images were acquired with a Signa Advantage 1.5 Tesla whole-body MRI system (General Electric, Waukesha, Wisconsin), with an elliptical end-capped quadrature radio frequency and local gradient head coil (Medical Advances, Milwaukee, Wisconsin), using T2*-weighted gradient-recalled echo, echo planar imaging (EPI) with a repetition time (TR) of 1500 ms, an effective echo time (TE) of 40 ms and a flip angle (FA) of 90°. Eighteen interleaved oblique axial slices were acquired, with the bottom slice going through the AC-PC line. These slices had a 22 cm field of view and consisted of a 64 by 64 matrix and a slice thickness of 4.0 mm (no interslice gap), yielding a voxel size of 47.3 mm^3.

2.2.3.2 Image processing Data was first corrected for distortion artifacts (by registering them to the inplane FSE images) and movement using AFNI software (Cox, 1996), and then further preprocessed and analyzed using BrainVoyager software (Brain Innovation, Maastricht, the Netherlands). As each block started with a 12000 ms fixation screen, the first 8 images at each slice location were discarded from the analysis. The remaining 308 images were preprocessed using interscan slice time correction, 3D motion correction, 3D Gaussian spatial filtering (FWHM = 6 mm), and temporal high-pass filtering using a low cutoff frequency of 3 cycles/block (or .00649351 Hz). For each participant, three-dimensional images (SPGRs) of the brain were acquired at the end of the experiment. The functional data were aligned to these and then transformed into Talairach space.

2.2.3.3 Event-related analyses Functional data were analyzed using a general linear approach. Predictors were obtained by convolving the onset of each based on a canonical BOLD response (e.g., Logothetis & Wandell, 2004). Thus, these analyses identified transient hemodynamic activity locked to each trial type. Multiple linear regression as implemented in BrainVoyager using a least-squares approach was used to obtain parameter estimates. These parameter estimates were then further analyzed by testing specific contrasts using participant as a random factor. All tests were thresholded at a per-voxel $p < .001$ (uncorrected), using a contiguity threshold of 250 mm^3.

To identify conflict-related activity, 7 regressors were constructed per block, corresponding to the cC, cI, iC and iI trials, overt error trials, correct trials following error trials, and the first (corresponding) trial of each block. Conflict-related activity was then identified by testing the parameter estimates for the cI condition (the high-conflict condition) against those for the cC, iC, and iI conditions taken together (contrast: cC cI iC iI -1 3 -1 -1), separately for the speed and accuracy conditions. This contrast should identify brain regions that respond more strongly to the cI condition than to the cC, iC, and iI conditions, and therefore respond to the presence of strong response conflict.

To identify error-related activity, two regressors were constructed per block; one for correct trials, and one for overt error trials. To identify error-related activity, the parameter estimates obtained for these two regressors were tested against each other. This contrast would identify brain regions that respond more strongly to errors that to correct trials.

To identify control-related activity on trials following conflict trials, an approach was used similar to the one taken by Kerns et al. (2004). This approach is based on the notion that greater control on an incongruent trial should lead to a faster RT, while conversely, greater control on a congruent trial should lead to a slower RT. On a per-block basis, high-control trials were defined as the fastest quartiles of iI trials and the slowest quartile of iC trials, while low-control trials were defined as the slowest iI trials and the fastest iC trials. Per block, five regressors were constructed; one for low-control iI trials, one for high-control iI trials, one for low-control iC trials, one for high-control iC trials, and a covariate of noninterest for error trials. In order to identify control-related activity, the parameter estimates for the fast iI and slow iC regressors were compared to the slow iI and fast iC regressors, separately for speed and accuracy emphasis. Note that this analysis is equivalent to an interaction between quantile (fast, slow) and condition (iC, iI).

To identify control-related activity following error trials, 7 regressors were constructed per block, corresponding to the cC, cI, iC and iI trials, overt error trials, correct trials following error trials, and the first (corresponding) trial of each block (identical to the conflict analyses). Control-related activity was then identified by testing the parameter estimates for the post-error trials against those for the post-correct trials (cC, cI, iC, and iI conditions) taken together (contrast: cC cI iC iI post-error trials -1 -1 -1 -1 4).

2.3 RESULTS

2.3.1 Performance data: Effects of SAT and trial type.

Mean RTs and error rates are displayed in Figure 2 and Figure 3, respectively, as a function of SAT emphasis, Preceding Trial Type (congruent: c; incongruent: i), Current Trial Type (congruent: open circles; incongruent: grey squares), and whether trials were S-R Repetitions or S-R Alternations. Error bars represent one standard error of the mean (SEM). See Table 8 and Table 9 in APPENDIX B for more details.

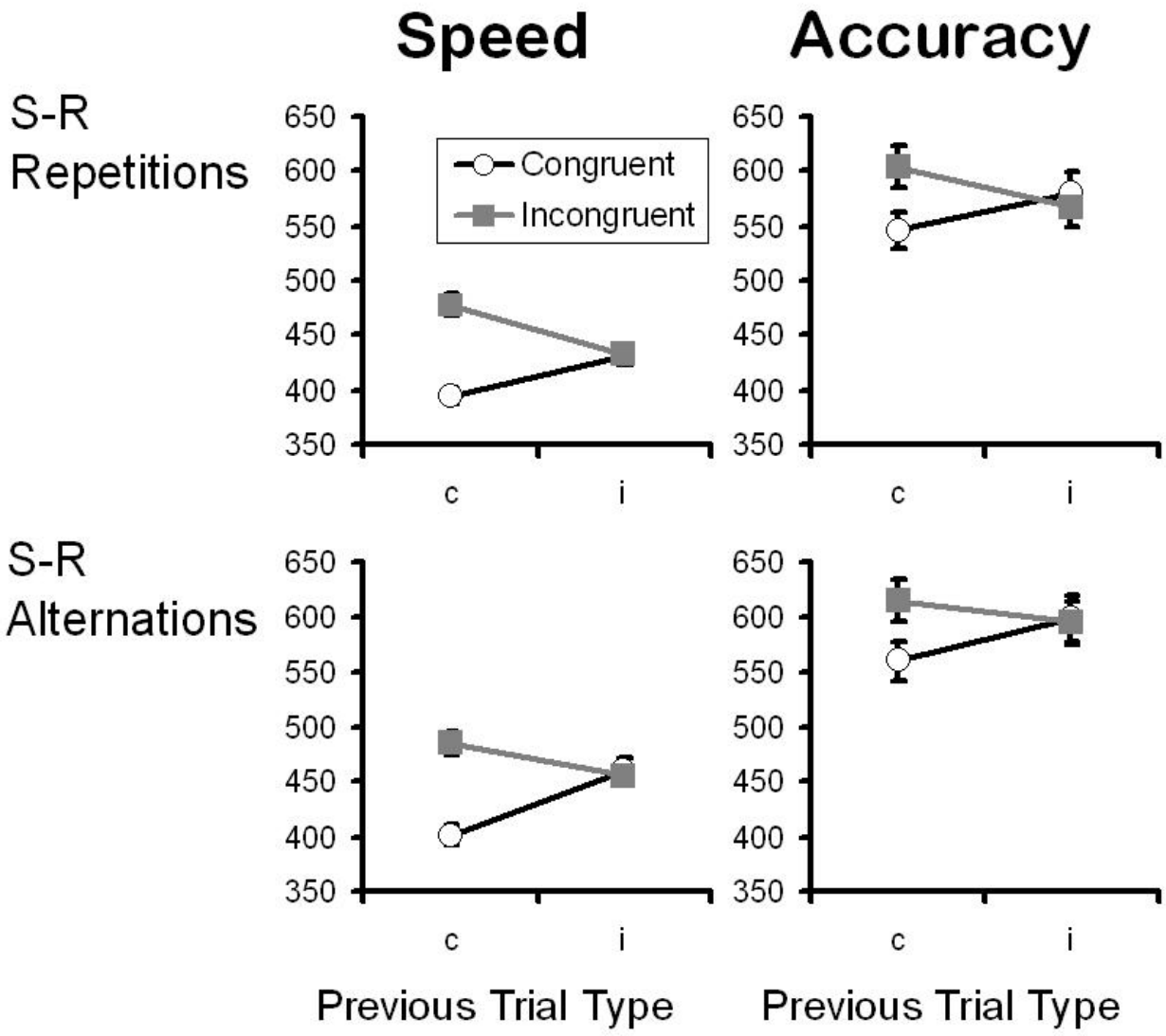

Figure 2. Reaction times in Experiment 1.

Taken together, there was no interaction between repetition (repetition, alternation) and the "completeness" of the repetition (complete, partial); this was neither the case for RTs, $F(1, 23) = .205$, $p = .655$, nor for error rates, $F(1, 23) = 1.422$, $p = .245$. Thus, repetition effects did not differ between partial and complete repetitions and alternations. This indicates that in the current data set, the correct way to control for priming effects when investigating the conflict adaptation effect is to take both complete and partial SR repetitions into account, rather than complete repetitions only.

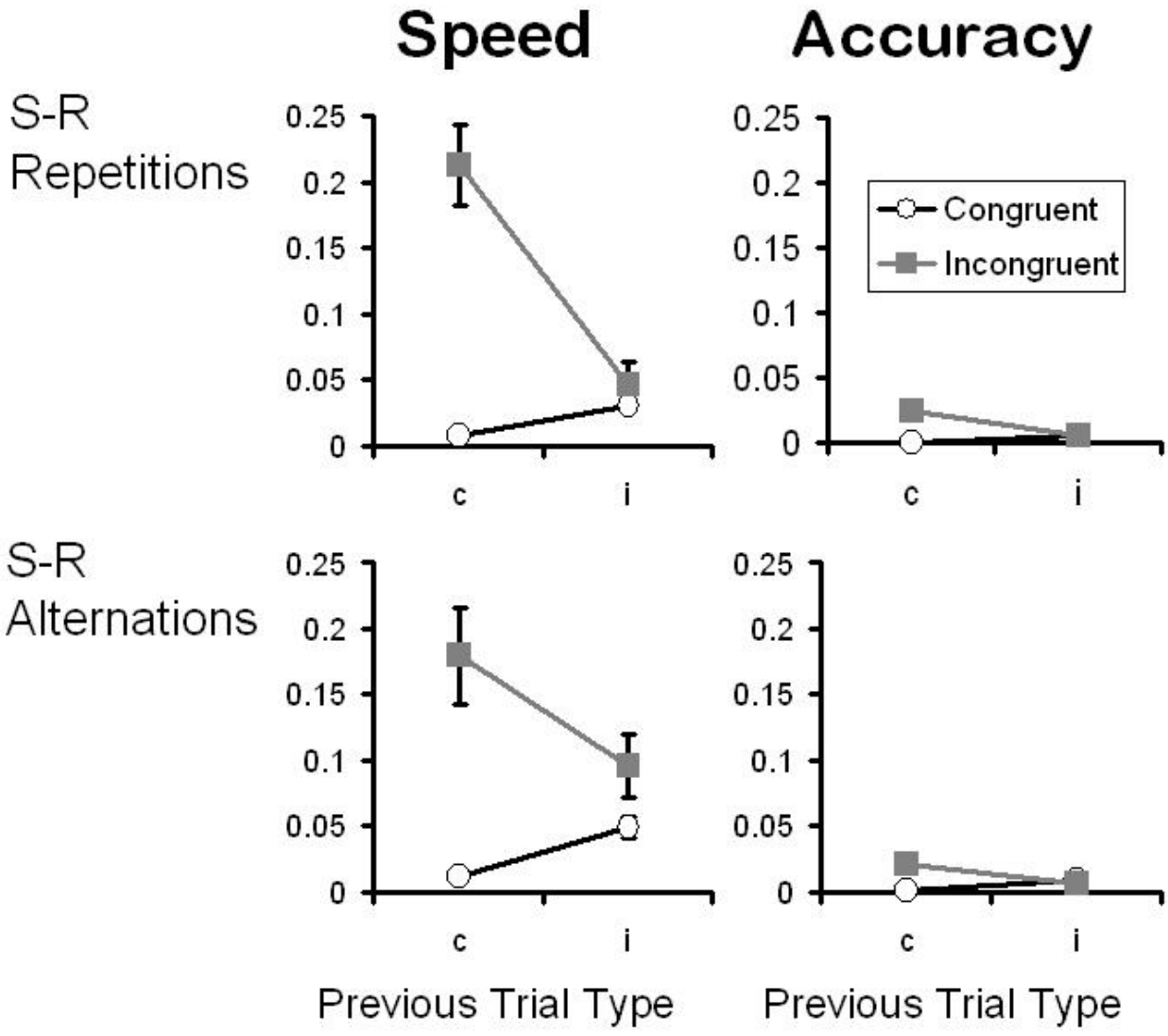

Figure 3. Error rates in Experiment 1.

Typically, variances under accuracy emphasis are greater than under speed emphasis; this is the case in the present data set, as well. Therefore, analyses of RTs were performed on log-transformed data; this proved to remove heteroscedasticity from the data completely. Analyses of error rates are performed on arcsine-transformed data. This transformation reduced heteroscedasticity but did not remove it. The results of the 4-way ANOVA between repetition (Rep), SAT, previous trial type (PT) and current trial type (CT), for both RTs and error rates, are shown in Table 2.

Table 2. Overview of statistical analyses of performance data.

Effect	**RTs (log-transformed)**	**Error Rates (arcsine-transformed)**
Main effects		
SAT	$F(1, 23) = 99.835$, $p < .001$	$F(1, 23) = 104.910$, $p < .001$
Rep	$F(1, 23) = 9.216$, $p = .006$	$F(1, 23) = 3.822$, $p = .063$
PT	$F(1, 23) = 6.038$, $p = .022$	$F(1, 23) = 18.467$, $p < .001$
CT	$F(1, 23) = 118.856$, $p < .001$	$F(1, 23) = 26.778$, $p < .001$
2-way interactions		
PT*CT	$F(1, 23) = 179.298$, $p < .001$	$F(1, 23) = 86.548$, $p < .001$
SAT* PT	$F(1, 23) = .795$, $p = .382$	$F(1, 23) = 3.877$, $p = .061$
PT * Rep	$F(1, 23) = 13.818$, $p = .001$	$F(1, 23) = 20.437$, $p < .001$
SAT*CT	$F(1, 23) = 24.968$, $p < .001$	$F(1, 23) = 19.343$, $p < .001$
SAT*Rep	$F(1, 23) = .822$, $p = .374$	$F(1, 23) = 2.170$, $p = .154$
CT*Rep	$F(1, 23) = .015$. $p = .904$	$F(1, 23) = .555$, $p = .464$
3-way interactions		
SAT*PT*CT	$F(1, 23) = 13.901$, $p = .001$	$F(1, 23) = 26.655$, $p < .001$
Rep*PT*CT	$F(1, 23) = .049$, $p = .827$	$F(1, 23) = 4.132$, $p = .054$
Rep*SAT*CT	$F(1, 23) = .018$, $p = .894$	$F(1, 23) = .007$, $p = .935$
Rep*SAT*PT	$F(1, 23) = 2.954$, $p = .099$	$F(1, 23) = 6.224$, $p = .020$
4-way interaction		
SAT*Rep*PT*CT	$F(1, 23) = .787$, $p = .384$	$F(1, 23) = 3.182$, $p = .088$

2.3.1.1 SAT RTs were faster under speed accuracy than under accuracy emphasis, while error rates were higher under speed than under accuracy emphasis. This verifies that the participants did indeed trade speed and accuracy.

2.3.1.2 Simon interference RTs were faster to congruent than to incongruent trials; error rates, too, were lower to congruent than to incongruent trials. Thus, performance was impaired during incongruent trials compared to congruent trials; this verifies the Simon interference effect (Kornblum et al., 1999; Simon & Berbaum, 1990).

2.3.1.3 Repetition effects RTs were faster to S-R repetition trials than to S-R alternation trials; error rates were marginally different between S-R repetitions and S-R alternations. This verified that priming of S-R mappings did indeed contribute to the pattern of performance.

3.3.1.4 Modulation of repetition effects by SAT While the 2-way interaction between SAT and repetition was not significant, it should be mentioned that an omnibus SAT by repetition repeated measures ANOVA that was performed on the untransformed RT data revealed a trend towards significance, $F(1, 23) = 4.005$, $p = .057$, which suggested that repetition effects might be greater under accuracy than under speed emphasis.

2.3.1.5 Modulation of interference by SAT The 2-way interaction between SAT and condition indicated that the Simon effect was greater under speed than under accuracy emphasis, for both RTs and for error rates. This suggests that conflict was greater under speed emphasis.

2.3.1.6 The conflict adaptation effect The 2-way interaction between current trial type and previous trial type was significant, both for RTs and error rates. This verified the previously reported conflict adaptation effect; the size of the interference effect was greater when preceded by a congruent trial and smaller when preceded by an incongruent trial. As also reported previously, repetition did not influence this greatly; the 3-way interaction between repetition, previous trial, and current trial was not significant for RTs. Post-hoc tests showed that for RTs, the previous trial by current trial interaction remained significant both for S-R repetitions, $F(1, 23) = 79.131$, $p < .001$, and for S-R alternations, $F(1, 23) = 115.989$, $p < .001$.

2.3.1.7 Modulation of the conflict adaptation effect by SAT The 3-way interaction between SAT, previous trial type, and current trial type was significant for both RTs and error rates, showing that the conflict adaptation effect was greater under speed than under accuracy emphasis. The fact that the 4-way interaction which included repetition failed to reach significance suggests that the pattern of results is independent of whether the trials are S-R alternations or S-R repetitions. The interaction between SAT, previous trial, and current trial was furthermore computed separately for S-R repetitions and S-R alternations. For S-R repetitions, the 3-way interaction for RTs between SAT, previous trial, and current trial was significant both for RTs, $F(1,23) = 16.416$, $p < .001$, and for error rates, $F(1,23) = 7.378$, $p = .012$. Computed separately for SR alternations, the 3-way interaction was also significant both for RTs, $F(1,23) = 29.551$, $p < .001$, and for error rates, $F(1,23) = 24.504$, $p < .001$. Thus, these results confirmed the predictions outlined earlier.

2.3.2 Performance data: Post-error slowing

Post-error slowing was calculated for each overt error by taking the difference between the RT of the preceding correct trial and the RT to the subsequent correct trial. Unfortunately, most participants only made a few overt errors under accuracy emphasis; only 7 participants had 3 or more overt errors in this condition, so analyses were limited to these participants. The amount of post-error slowing per SAT condition for these 7 participants is displayed in Figure 4. Standard errors represent one SEM.

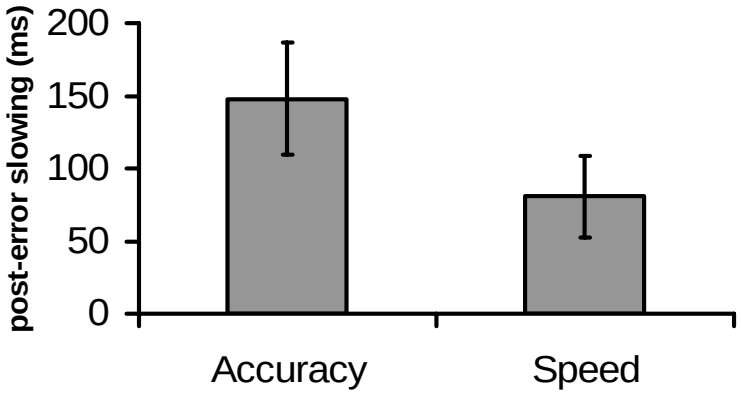

Figure 4. Post-error slowing in Experiment 1.

The difference between the post-error trial and the pre-error trial was significantly greater than 0 both under accuracy emphasis (M = 148 ms, SD = 102), $t(6) = 3.855$, $p = .008$, and under speed emphasis (M = 81 ms, SD = 74), $t(6) = 2.889$, $p = .028$, that participants did indeed performed slower following error commission. Furthermore, post-error slowing was significantly greater under accuracy emphasis than under speed instructions $t(6) = 2.893$, $p = .028$, confirming the predictions stated earlier.

2.3.3 Imaging data

2.3.3.1 Conflict under speed emphasis The contrast of interest (cI trials versus cC, iC, and iI trials taken together) revealed that a number of areas were more engaged to the cI condition than to the other three conditions. These included the ACC extending into the pre-supplementary motor area (pre-SMA), right DLPFC, and bilateral medial and lateral parietal cortices. The activation is displayed in Figure 5 and summarized in Table 3.

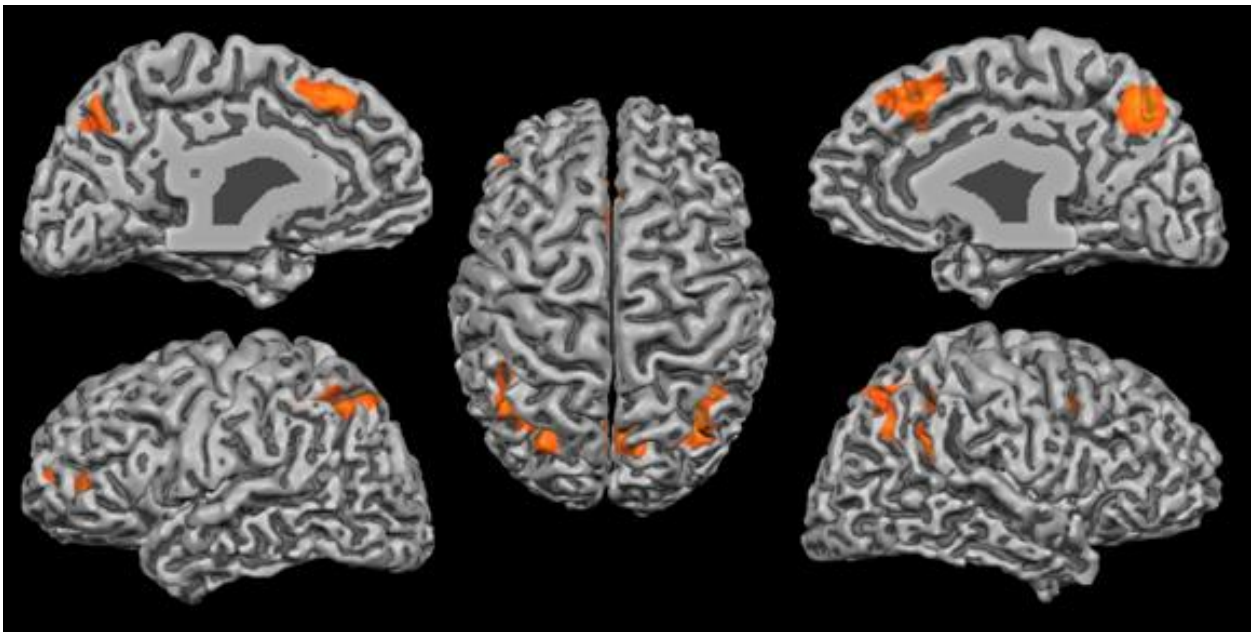

Figure 5. Brain areas engaged by response conflict under speed emphasis.

Table 3. Brain regions engaged by conflict under speed emphasis.

Region of interest	**BA**	**Volume (mm^3)**	**Talairach coordinates**
ACC	32/8/6	3582	5, 25, 46
Right middle frontal gyrus	8/9/6	1583	35, 13, 45
Precuneus	7/19	2700	5, -67, 41
Right inferior parietal cortex, extending into supramarginal gyrus, angular gyrus, superior /middle temporal gyrus	40/7/39/19	6897	38, -56, 35
Left inferior parietal cortex, extending into supramarginal gyrus, angular gyrus, superior temporal gyrus	40/7/39/19	2891	-34, -60,41
Left inferior parietal cortex	40	392	-40, -36, 39
Left superior/middle frontal gyrus	10	467	-22, 55, 14
Left middle/inferior frontal gyrus	10/46	434	-38, 41, 10

Post-hoc tests using the same contrast of interest performed on the average signal of each of these areas verified that each of these areas responded stronger to the cI condition than to the cC, iC, and iI conditions under speed emphasis, all Fs > 20.133, all ps < .001. Post-hoc tests also showed that some of these areas were also engaged by this contrast under accuracy emphasis: The precuneus, $F(1, 23) = 10.511$, $p = .004$, the right inferior parietal cortex, $F(1, 23) = 16.209$, $p < .001$, the large left inferior parietal cortex activation extending into the supramarginal and angular gyri, $F(1, 23) = 12.816$, $p = .002$, the small left inferior parietal cortex activation, $F(1, 23) = 13.133$, $p = .002$, and the left middle/inferior frontal gyrus, $F(1, 23) = 7.355$, $p = .012$. Importantly, conflict-related activation under accuracy emphasis failed to reach significance in the ACC, $F(1, 23) = 0.667$, $p = .422$, in the right DLPFC, $F(1, 23) = 1.999$, $p = .171$, and in the left polar frontal cortex, $F(1, 23) = 0.026$, $p = .873$.

In order to verify whether this conflict-related activation of these areas was greater under speed than under accuracy emphasis, the interaction between the conflict contrast (cC cI iC iI -1 3 -1 -1) and SAT (speed, accuracy) was also analyzed (i.e. the contrast Speed-cC Speed-cI Speed-iC Speed-iI Accuracy-cC Accuracy-cI Accuracy-iC Accuracy-cI -1 3 -1 -1 1 -3 1 1). The results of this analysis showed that speed emphasis resulted in greater conflict-related activation in the ACC, $F(1, 23) = 10.686$, $p = .003$, right middle frontal gyrus, $F(1, 23) = 15.547$, $p < .001$, the right inferior parietal cortex extending into the supramarginal and angular gyri, $F(1, 23) = 9.828$, $p = .005$, and the left polar frontal cortex, $F(1, 23) = 8.009$, $p = .009$. Thus, the conflict-related activation of the ACC, right DLPFC, right inferior parietal cortex, and left polar frontal cortex was greater under speed than under accuracy emphasis, although the right inferior parietal cortex responded significantly under both speed and accuracy emphasis.

No significant interaction was found in the precuneus, $F(1, 23) = 1.486$, $p = .235$, the left inferior parietal cortex extending into the supramarginal and angular gyri, $F(1, 23) = 1.636$, $p = .214$, the smaller left parietal activation, $F(1, 23) = 0.555$, $p = .464$, and the left middle/inferior frontal gyrus, $F(1, 23) = 2.673$, $p = .116$. Since significant conflict-related activity was observed in these areas under both speed and accuracy emphasis, but did not interact with the level of SAT, this suggests that conflict engaged these areas to similar extents under speed and accuracy emphasis.

2.3.3.2 Conflict under accuracy emphasis Under accuracy emphasis, the contrast of interest (cI trials versus cC, iC, and iI trials taken together) revealed that a number of areas were more engaged to the cI condition than to the other three conditions. These included the bilateral premotor areas and bilateral medial and lateral parietal cortices, but did not include ACC or DLPFC at the chosen statistical threshold. The activation is displayed in Figure 6 and summarized in Table 4.

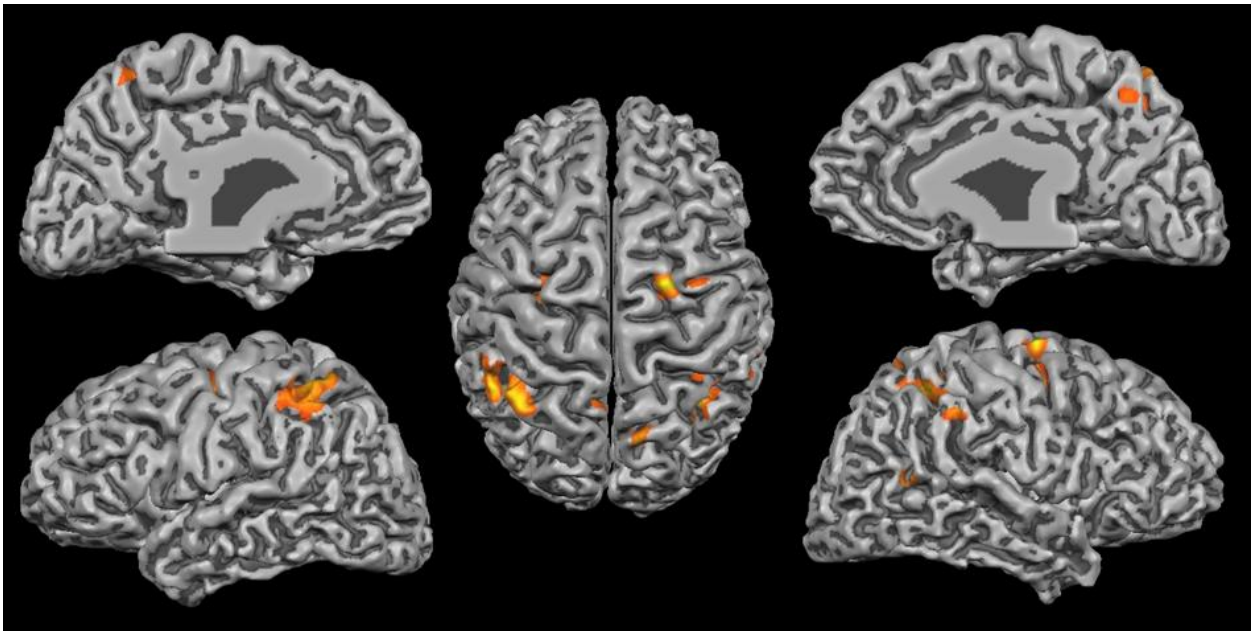

Figure 6. Brain areas engaged by response conflict under accuracy emphasis.

Table 4. Brain regions engaged by conflict under accuracy emphasis.

Region of interest	**BA**	**Volume (mm^3)**	**Talairach coordinates**
Right dorsal premotor cortex	6	745	23, -7, 62
Right dorsal premotor cortex	6	495	33, -6, 52
Left dorsal premotor cortex	6	496	-26, -9, 52
Left Precuneus	7	501	-7, -56, 54
Right Precuneus	7	347	3, -61, 44
Right inferior parietal cortex	40	2249	37, -50, 45
Right superior parietal cortex	7/19	1478	17, -68, 51
Left inferior parietal cortex	40	4382	-43, -47, 47
Right supramarginal gyrus	40	288	57, -41, 35
Right middle temporal gyrus	39	424	49, -57, 11

Post-hoc tests using the same contrast of interest, performed on the average signal of each of these areas, verified that each of these areas responded more strongly to the cI condition than to the cC, iC, and iI conditions under accuracy emphasis, all Fs > 18.535, all ps < .001. Post-hoc tests also showed that some of these areas were also engaged by this contrast under speed emphasis: The more superior right premotor cortex region of interest (ROI), $F(1, 23) = 4.661$, $p = .027$, the other right premotor cortex ROI, $F(1, 23) = 6.843$, $p = .015$, the left premotor cortex, $F(1, 23) = 7.612$, $p = .013$, the right precuneus, $F(1, 23) = 27.363$, $p < .001$, the right inferior parietal cortex, $F(1, 23) = 15.626$, $p < .001$, the right superior parietal cortex, $F(1, 23) = 10.850$, $p = .003$, the left inferior parietal cortex, $F(1, 23) = 5.915$, $p = .023$. No significant effect was observed in the right supramarginal gyrus, $F(1, 23) = 0.564$, $p = .461$, or the left precuneus, $F(1, 23) = 0.184$, $p = .672$.

2.3.3.3 Response preparation In order to study processes related to preparing and executing the responses, further post-hoc tests were performed on the premotor ROIs identified under accuracy emphasis. For these analyses, a set of regressors was constructed separately for each correct response hand and for each level of SAT. These regressors were low-conflict trials (cC, iC, iI) and high-conflict trials (cI). An additional set of regressors was based on errors, post-error trials, and the very first trial of each block; these were covariates of noninterest. Each ROI was subjected to two two-way interactions, one for the correct response hand, and one for the incorrect response hand, with SAT (speed, accuracy) and conflict (low, high) as factors, and with participant as a random factor.

For the more superior of the two ROIs located in the premotor cortex of the right hemisphere, the analysis based on contralateral (left-hand) responses revealed a significant main effect of conflict, $F(1, 23) = 4.491$, $p = .045$, showing that motor activation of the correct hand was greater for high-conflict trials; the significant interaction between SAT and conflict, $F(1, 23) = 4.770$, $p = .039$, showed that the difference in activation between low- and high-conflict trials was greater under accuracy emphasis. For ipsilateral (right-hand) responses, a significant main effect for conflict, $F(1, 23) = 10.472$, $p = .004$, showed that high-conflict trials also elicited higher activation of the incorrect hand; however, this activation of the incorrect hand did not differ between speed and accuracy emphasis, $F(1, 23) = .206$, $p = .654$.

For the more inferior of the two right hemisphere premotor areas, only a significant main effect of conflict was observed for ipsilateral (right-hand) responses, $F(1, 23) = 9.360$, $p = .006$), showing again that these was greater activation of the incorrect hand during high-conflict than during low-conflict trials, however, again, no significant interaction between SAT and conflict was observed for ipsilateral responses, $F(1, 23) = .168$, $p = .685$. No significant main effect of conflict, or a significant interaction between SAT and conflict was observed for contralateral responses for this ROI, all Fs < 1.882, all ps > .183.

For the premotor ROI in the left hemisphere, no significant main effect of conflict, or a significant interaction between SAT and conflict was observed for ipsilateral (left-hand) responses for this ROI, all Fs < .968, all ps > .335. However, for contralateral (right-hand) responses, a significant main effect of conflict was observed, $F(1, 23) = 30.790$, $p < .001$, showing that activation of the correct hand was greater for high-conflict trials than low-conflict trials. A trend towards a significant interaction between SAT and conflict, $F(1, 23) = 2.046$, $p = .052$, suggested that this difference in activation was greater under accuracy emphasis.

In sum, transient activation of the incorrect hand to high-conflict trials does not differ between SAT conditions for any of the premotor areas. However, transient activation of the correct hand tends to be greater under accuracy instructions, particularly for high-conflict trials.

2.3.3.4 Errors Because of the relative infrequency of overt error trials under accuracy instructions, the difference between error and correct trials was computed across the entire experiment (independent of SAT). Whether certain regions were more engaged under accuracy than speed emphasis, or vice versa, was determined on a post-hoc basis. Figure 7 displays all areas identified by the error vs correct analysis. In Figure 7, the medial view of the left hemisphere is presented in the top left; the lateral view of the left hemisphere is presented in the bottom left; the medial view of the right hemisphere is presented in the top right; the lateral view of the right hemisphere is presented in the bottom right. The top view of both cerebral hemispheres is presented in the center. These areas are summarized in Table 5.

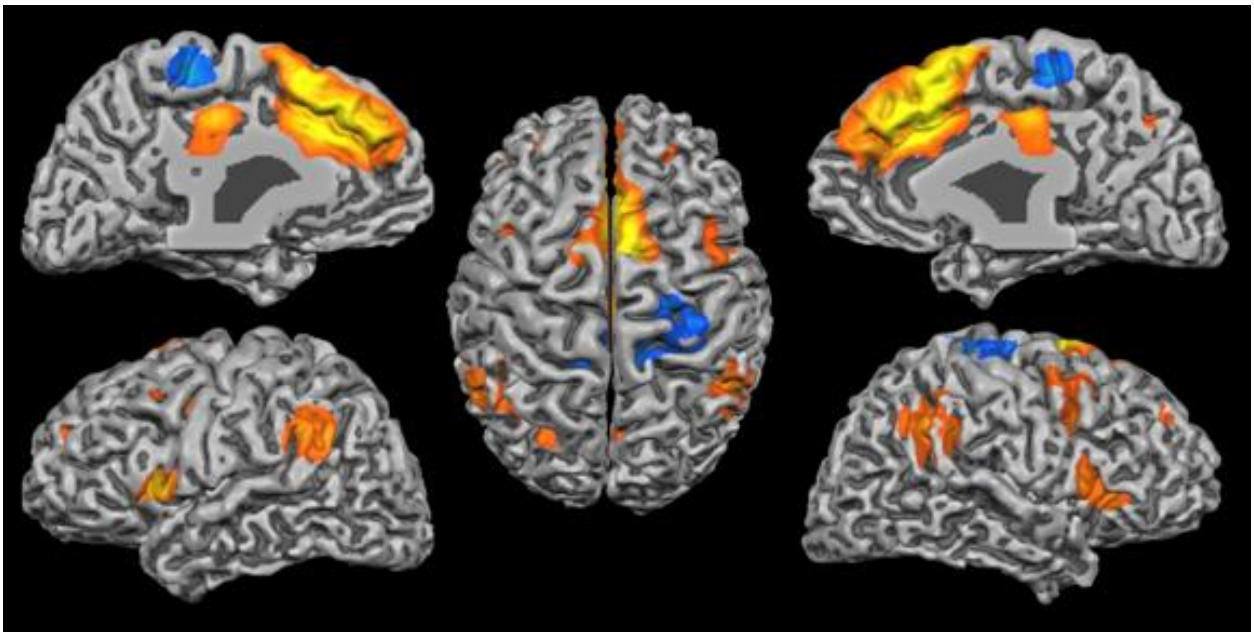

Figure 7. Brain areas engaged by errors.

As in the analyses of the performance data, for those participants with 3 or more overt errors under accuracy emphasis (N = 7), per-ROI repeated measures ANOVAs were carried out, with SAT (speed, accuracy) and accuracy (error, correct) as factors, to identify areas for which the difference between errors and correct trials was greater under accuracy than under speed emphasis. Although this might seem like relatively few trials, it should be noted that the error-related ACC activation can be reliably detected at a single-trial level (Debener et al., 2005).

Significant interactions between these factors were observed in the ACC/medial superior frontal gyrus activation, $F(1, 6) = 8.680$, $p = .026$, and the right lateral inferior parietal cortex extending into the supramarginal and superior temporal gyri, $F(1, 6) = 14.938$, $p = .008$, while trends towards significance were observed in the right middle/inferior frontal gyrus, $F(1, 6) = 4.29$, $p = .084$, and the left superior frontal gyrus, $F(1, 6) = 5.813$, $p = .051$. For all other regions, no significant interactions were observed, all Fs <3.568, all ps > .108, although it should be noted here that the relatively small number of participants included in this analysis might have impaired the statistical power.

Table 5. Brain regions engaged by errors.

Region of interest	**BA**	**Volume (mm^3)**	**Talairach coordinates**
Errors > correct			
ACC, superior frontal gyrus	32/24/8/9/6	33065	2, 26, 42
Right middle/inferior frontal gyrus	9/44/6	2885	41, 7, 43
Left middle frontal gyrus	9/6	1573	-38, 8, 40
Right lateral inferior parietal cortex, supramarginal gyrus, superior temporal gyrus	44/22	6870	50, -47, 31
Left lateral inferior parietal cortex, supramarginal gyrus, superior temporal gyrus	44/22	4563	-56, -48, 30
Posterior cingulate cortex	31/23/24	3159	1, -21, 34
Right middle/superior frontal gyrus	8/9	326	23, 45, 34
Left superior frontal gyrus	9/10	556	-26, 47, 27
Right Precuneus	7/18/19	436	6, -69, 32
Right inferior frontal gyrus / anterior insula	44/45	6458	43, 19, 8
Left inferior frontal gyrus / anterior insula	44/45	7020	-41, 16, 10
R caudate / thalamus		2125	9, -2, 15
Correct > errors			
Bilateral precentral/postcentral sulcus, right premotor / paracentral lobule	4/3/6/7	7455	11, -30, 59
Right putamen		1077	26, -5, 15
Left putamen		334	-22, 6, 8

Although the ACC responds more strongly to errors under accuracy than under speed emphasis, it is possible that this is caused by a habituation-like process; people make more errors under speed emphasis, and arguably the error-related signal could have habituated more, reducing the average error-related ACC signal under speed emphasis. In order to investigate this possibility, another post-hoc test was performed on the ACC signal for those participants with 3 or more errors under accuracy emphasis, with three regressors for the speed blocks; one for correct trials, one for the first few error trials, and one for the remaining error trials. For each participant, the amount of error trials included in the "early errors" regressor corresponded to the amount of errors s/he made under accuracy emphasis; by comparing the error-related signal under accuracy emphasis to the "early" error-related signal under speed emphasis, one can control for the amount of trials that account for each regressor.

Taking into account only the "early error" regressor for speed emphasis rather than the "late error" regressor, the two-way interaction between SAT (speed, accuracy) and accuracy (correct, error) remained significant, $F(1, 6) = 61.356$, $p < .001$, showing that even when controlling for the number of error trials, the error-related ACC activation was greater under accuracy than under speed emphasis. Limiting this interaction to the "late error" rather than the "early error" regressor, this interaction again remained significant, $F(1, 6) = 19.132$, $p = .004$; directly comparing the "early error" and "late error" parameter estimates did not reveal a significant difference, $t(6) = 0.771$, $p = .470$. Thus, habituation or a habituation-like process cannot account for the increased error-related ACC activation under accuracy emphasis.

2.3.3.5 Cognitive control following conflict Regions that implement conflict-induced control were identified by an interaction between quantile (fast, slow) and condition (iI, iC), specifically, regions with activity greater to slow iI and fast iC, than to fast iI and slow iC. Under speed emphasis, this analysis revealed activation in the left middle frontal gyrus of the DLPFC (BA 9/46, average Talairach coordinates: X = -36, Y = 33, Z = 25; Volume = 817 mm^3), and the right inferior parietal cortex (BA 40, average Talairach coordinates: X = 52, Y = -28, Z = 32; Volume = 1311 mm^3). These results are displayed in Figure 8. Control-related activity under speed emphasis is displayed in yellow/orange; control-related activity under accuracy emphasis is displayed in blue.

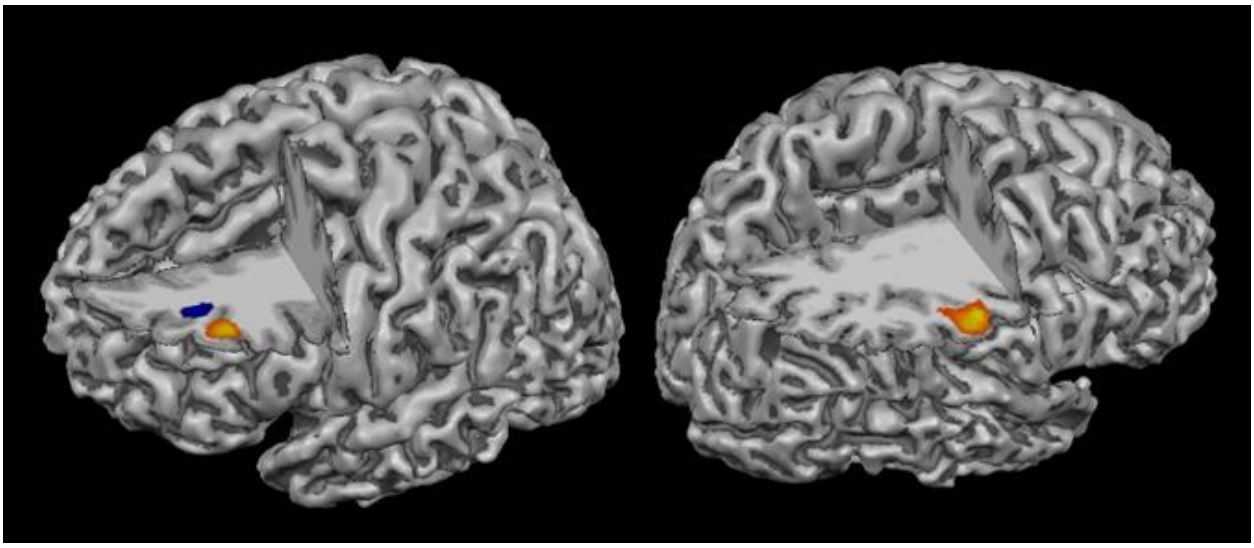

Figure 8. Neural activity engaged by conflict-induced control.

Post-hoc tests were performed on each of these regions separately. For the left DLPFC ROI associated with speed emphasis, paired *t*-tests verified that under speed emphasis, this area was indeed significantly more engaged to fast iI trials than to slow iI trials, $t(23) = -2.256$, $p = .034$, and more engaged to slow iC trials than to fast iC trials, $t(23) = 3.14$, $p = .001$. A significant post-hoc interaction between quantile (fast, slow) and condition (iC, iI) also verified that this area indeed reflected control under speed emphasis, $F(1, 23) = 36.796$, $p < .001$. These same post-hoc tests performed on this ROI on the data obtained under accuracy emphasis showed a different pattern of results; these showed that this area responded stronger to slow than fast responses, both for iC trials, $t(23) = 2.588$, $p = .016$ and for iI trials, $t(23) = 2.485$, $p = .021$, thus reflecting response time rather than control. Consistently, the interaction between quantile and condition was not significant under accuracy emphasis, $F(1, 23) = .292$, $p = .594$. The 3-way interaction between SAT, quantile, and condition was significant, $F(1, 23) = 10.133$, $p = .004$, verifying that this area distinguished between high and low control under speed, but not under accuracy emphasis.

For the right parietal ROI, paired *t*-tests showed that under speed emphasis, this area was more engaged to slow than to fast iC trials, $t(23) = 4.276$, $p < .001$, although it did not respond significantly more strongly to fast than to slow iI trials, $t(23) = -1.104$, $p = .281$ (though note that the signal was in the expected direction). An interaction between quantile and condition was

indeed significant, $F(1, 23) = 30.542$, $p < .001$. These same post-hoc tests performed on this same region for data obtained under accuracy emphasis showed that this area responded stronger to slow than fast responses, both for iC trials, $t(23) = 2.214$, $p = .037$ and for iI trials, $t(23) = 2.701$, $p = .013$, thus again reflecting response time rather than control under accuracy emphasis. A nonsignificant interaction (for accuracy data) between quantile and condition verified this, $F(1, 23) = 1.011$, $p = .325$. The 3-way interaction between SAT, quantile, and condition was significant, $F(1, 23) = 7.099$, $p = .014$, verifying that this area distinguished between high and low control under speed, but not under accuracy emphasis.

Under accuracy emphasis, the interaction between quantile and condition also revealed activation in the left middle frontal gyrus of the DLPFC (BA 9/46, average Talairach coordinates: X = -25, Y = 36, Z = 29; Volume = 278 mm^3), adjacent to but not overlapping with the area engaged under speed emphasis. Paired *t*-tests performed on this region showed that while this area, under accuracy emphasis, responded more strongly to fast than to slow iI trials, $t(23) = 6.595$, $p < .001$, it did not distinguish significantly between fast and slow iC trials, $t(3) = -.058$, $p = .954$, verified by a significant interaction between quantile and condition, $F(1, 23) = 24.971$, $p < .001$. However, a similar pattern of results obtained for these same post-hoc tests performed on the data obtained for this region under speed emphasis; under speed emphasis, it responded more strongly to fast than to slow iI trials, $t(23) = 3.772$, $p = .001$, but did not distinguish between fast and slow iC trials, $t(23) = -.946$, $p = .354$, verified by a significant interaction between quantile and condition, $F(1, 23) = 11.069$, $p = .003$. Indeed, no significant three-way interaction between SAT level, quantile, and condition was observed, $F(1, 23) = .049$, $p = .827$, suggesting that this area was not differently engaged under accuracy and speed emphasis.

In sum, these results are consistent with the notion that control, implemented in the DLPFC, is increased following conflict trials, and that this engagement of control is increased under speed emphasis.

2.3.3.6 Cognitive control following errors The next analysis focused on control during post-error slowing. While Kerns et al. (2004) identified neural activation related to post-error slowing by comparing slow post-error trials to fast post-error trials, limiting themselves to congruent trials only, this approach was not possible in the current data set to analyze differences between

speed and accuracy emphasis because of the relatively few error trials under accuracy emphasis. Therefore, neural activity related to post-error slowing was analyzed by comparing the beta coefficients associated with correct trials following errors, to those associated with correct trials following correct trials. For this analysis, 7 regressors per block were constructed, based on cC trials, cI trials, iC trials, iI trials, the first trial of each block, error trials, and post-error correct trials, convolved using a canonical hemodynamic response function. In order to identify neural activation associated with post-error slowing, the betas associated with the post-error correct trials regressor were compared to the betas associated with the cC, cI, iC and iI regressors (post-error contrast: cC cI iC iI post-error trials: -1 -1 -1 -1 4), using participant as a random factor, a statistical threshold of $p < .001$ and a contiguity threshold of 250 mm^3.

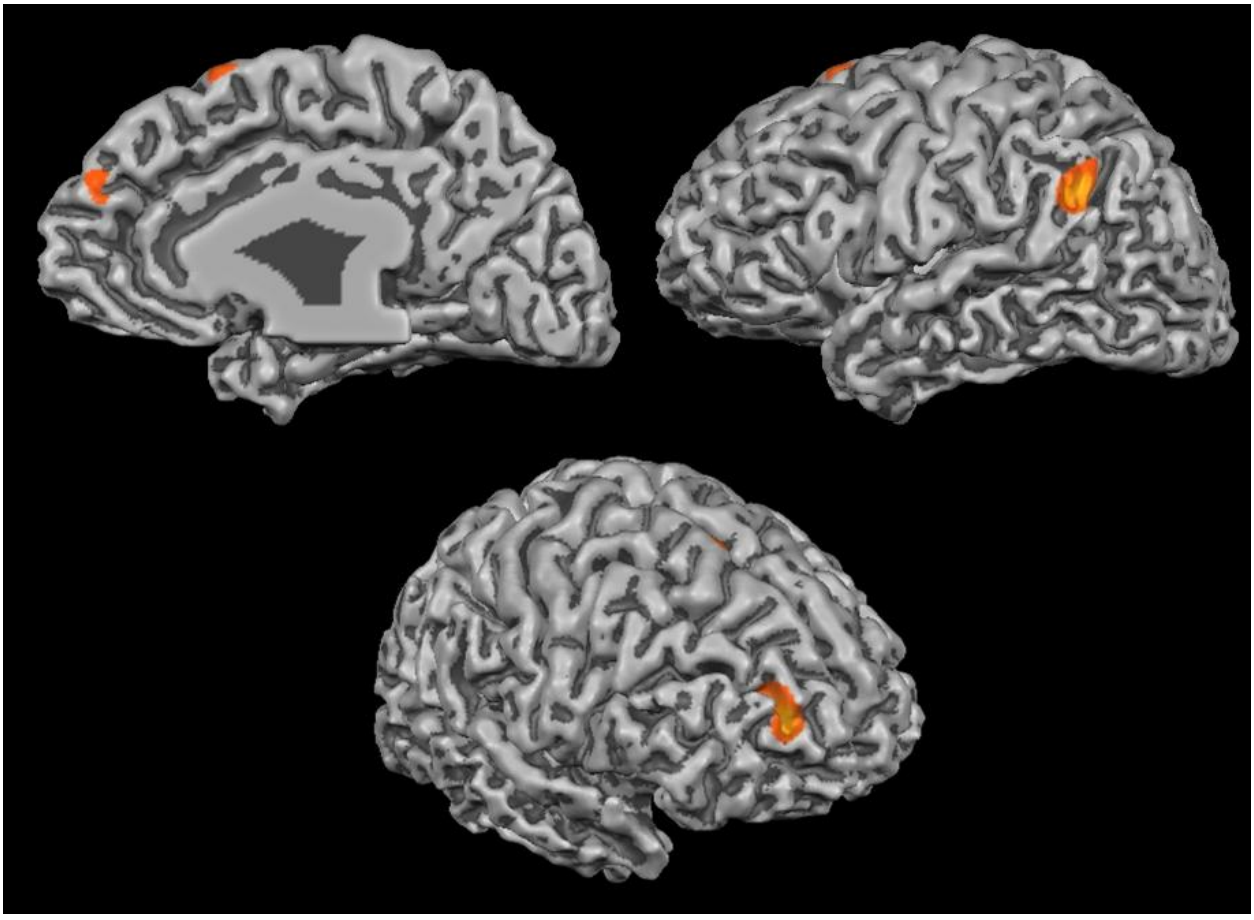

Figure 9. Neural activity related to post-error slowing.

This analysis revealed increased activation in four cortical areas: the right superior/medial frontal gyri (BA = 9/10/46; X = 25, Y = 51, Z = 25; Volume = 2715 mm^3), the medial superior frontal gyrus (BA = 9; X = 1, Y = 51, Z = 33; Volume = 794 mm^3), the left tempo-parietal junction (BA = 39/40; X = -56, Y = -57, Z = 31; Volume = 3859 mm^3), and the right pre-SMA (BA = 6; X = 5, Y = 17, Z = 64; Volume = 364 mm^3). The results are displayed in Figure 9.

Post-hoc tests were then carried on the average signal of each of these areas to verify whether this activation was greater under speed or accuracy emphasis. This was done by examining the interaction between SAT and the above-mentioned "post-error contrast", using participant as a random factor. Only the 7 participants who made 3 or more errors under accuracy emphasis were included in this analysis.

Significant interactions were obtained for the right lateral DLPFC, $F(1, 6) = 11.569$, $p = .014$, the left tempo-parietal junction, $F(1, 6) = 27.270$, $p = .002$; a trend towards significance was observed in the right pre-SMA, $F(1, 6) = 4.094$, $p = .089$. No significant difference was observed in the medial superior frontal gyrus, $F(1, 6) = 1.596$, $p = .253$.

Post-hoc tests (again restricted to 7 participants) performed on the average signal of these ROIs show that in the right DLPFC, activation related to post-error trials is indeed greater than activation during trials following correct trials under accuracy emphasis, $F(1, 6) = 27.436$, $p = .002$, but not under speed emphasis, $F(1, 6) = 2.413$, $p = .171$. Similarly, the left tempo-parietal junction was engaged more to trials following error trials than to trials following correct trials under accuracy emphasis, $F(1, 6) = 12.568$, $p = .012$, but not under speed emphasis, $F(1, 6) = .254$, $p = .632$.

Again, it should be noted here that the relatively small number of participants included in this analysis might have impaired the statistical power. Nevertheless, these data suggest that the activation related to post-error slowing observed in the left tempo-parietal junction and the right DLPFC was greater under accuracy emphasis.

In order to test whether the approach taken by Kerns et al. (2004) was valid, further post-hoc tests were performed on both the DLPFC ROI associated with post-error slowing and the DLPFC ROI associated with the conflict adaptation effect under speed emphasis, limiting the analyses to blocks performed under speed instructions. For these analyses, median-split regressors were constructed on post-error congruent and incongruent trials, resulting in fast post-

error congruent trials, slow post-error congruent trials, fast post-error incongruent trials, and slow post-error incongruent trials. Regressors to error trials were taken into account as covariates of noninterest. The four regressors of interest were, for both ROIs, subjected to a quantile by condition repeated-effects ANOVA, restricting the analyses to participants who had 10 or more observations for both conditions (N = 11). For the post-error ROI, a significant main effect of quantile, $F(1, 10) = 9.068$, $p = .013$, showed that the BOLD response was greater for slow RTs than for fast RTs, for both congruent and incongruent conditions. No significant main effect of condition, $F(1, 10) = .439$, $p = .523$, nor a significant interaction, $F(1, 10) = .809$, $p = .390$, was obtained for this ROI.

For the conflict adaptation effect ROI, no significant interaction was observed, $F(1, 10) = 2.712$, $p = .131$, nor a significant main effect for quantile, $F(1, 10) = 1.598$, $p = .235$ However, a trend towards a significant main effect for condition was observed, $F(1, 10) = 3.413$, $p = .094$. Post-hoc tests indicated that slow post-error congruent trials were associated with greater activation of this ROI than fast post-error congruent trials, $t(10) = 3.075$, $p = .012$, a finding that replicates the post-hoc analysis of Kerns et al. (2004). Post-error fast incongruent trials elicited nonsignificantly greater activation than post-error slow trials, $t(10) = -.252$, $p = .806$.

2.3.3.7 Between-area trial-to-trial correlations Kerns et al. (2004) showed that ACC activation during conflict and error trials predicted DLPFC activation during subsequent post-conflict and post-error trials, even when the activation of a temporal area was taken into account as a covariate of noninterest. A concern with this approach is that DLPFC activation during the conflict trial itself might be a confounding factor. In the present analyses, this was taken into account.

Kerns et al. (2004) calculated their correlations by taking the sum of scans 3, 4, and 5 following a Stroop stimulus as an index of the activation of an area during that trial. Likewise, in the present analyses, activation of an area during a certain trial was defined as the average of scans 3, 4 and 5 following that trial of the average *z*-scored time course of that area.

The analysis of the correlation between ACC and DLPFC in the conflict adaptation effect focused on the area of the ACC as identified by its increased activation to cI trials under speed emphasis, and the area of the DLPFC as identified by its difference in activation by comparing high-control to low-control post-conflict trials. Per participant, the activation of the left DLPFC

following conflict trials and to subsequent post-conflict trials was regressed onto the ACC activation; this was done separately for speed and accuracy blocks. Following Kerns et al. (2004), *t*-tests were then performed on the beta estimates to examine whether the ACC and PFC activity were significantly associated across the entire group of participants. Thus, controlling for DLPFC activation on conflict trial *n*, ACC activation on conflict trial *n* was significantly related to DLPFC activation on post-conflict trial $n + 1$ under speed emphasis, $t(23) = 3.818$, $p = .001$. The same was true for data obtained under accuracy emphasis; for that analysis, it did not matter whether the left DLPFC area obtained under speed emphasis, $t(23) = 4.216$, $p < .001$, or under accuracy emphasis, $t(23) = 5.574$, $p < .001$, was taken into account.

The analysis of the correlation between ACC and DLPFC during errors and post-error slowing focused on the area of the ACC as identified by its activation to error trials, and the area of the DLPFC related to post-error slowing. Again, the DLPFC activation to the error trial itself was taken into account as a covariate of noninterest. Under speed emphasis, error-related ACC activation was significantly related to subsequent DLPFC activation on the subsequent trial, controlling for DLPFC activation on the error trial itself, $t(23) = 3.870$, $p = .001$. However, under accuracy emphasis, this relationship failed to reach significance, $t(6) = 1.228$, $p = .226$; this is most likely due to the relatively small number of observations under accuracy emphasis, and it is worth noting that the correlation was in the expected direction.

2.4 DISCUSSION

In this experiment, the effects of different levels of SAT on conflict and control and their underlying neural processes were investigated in a Simon response interference task. It should first be emphasized that the behavioral results corroborated previously reported findings. Performance was impaired to incongruent compared to congruent trials, verifying the often-reported Simon effect (Craft & Simon, 1970; Simon, 1969; Simon & Berbaum, 1990; Simon & Rudell, 1967). Participants were faster but less accurate when instructed to emphasize speed in their performance, compared to when they were instructed to emphasize accuracy; this verified that participants did, indeed, trade speed and accuracy (Wickelgren, 1977).

The Simon interference effect was increased for trials preceded by congruent trials, and was considerably reduced for trials preceded by incongruent trials, verifying the expected conflict adaptation effect (Stürmer & Leuthold, 2003; Stürmer et al., 2002; Wühr & Ansorge, 2005). This did not depend on S-R repetition effects, and was observed for both S-R repetitions and S-R alternations, replicating earlier findings indicating that such effects do not influence the conflict adaptation effect in the Simon task (Stürmer & Leuthold, 2003; Stürmer et al., 2002; Wühr & Ansorge, 2005). On trials following overt error trials, participants were slower than on the trials preceding the errors, verifying the post-error slowing effect.

Importantly, the results confirmed the novel predictions stated earlier. Both the conflict adaptation effect and the post-error slowing effect were modulated by SAT, in opposite directions. The conflict adaptation effect was greater when participants were instructed to emphasize speed rather than accuracy in their performance. This phenomenon is hypothesized to be driven by the response conflict, and since one can assume response conflict to be increased under speed emphasis, the conflict adaptation effect was therefore expected to be greater under speed emphasis. The neuroimaging data are consistent with this interpretation; conflict-related activation of the ACC, which is hypothesized to monitor the amount of response conflict (Van Veen et al., 2001), was greater under speed than under accuracy emphasis. This increase of conflict-related ACC activation under speed emphasis is consistent with findings concerning the frontocentral N2 in conflict tasks. This component appears to be increased when participants are asked to emphasize speed (Band et al., 2003; Jodo & Kayama, 1992), and it has repeatedly been modeled as being generated by the ACC (Bokura, Yamaguchi, & Kobayashi, 2001; Lange, Wijers, Mulder, & Mulder, 1998; Liotti et al., 2000; Nieuwenhuis et al., 2003; Van Veen & Carter, 2002b; West, 2003; Yeung et al., 2004). It is worth pointing out that consistent with a conflict interpretation of ACC functioning, conflict-related ACC activation was greatest to cI trials, replicating earlier work (Botvinick et al., 1999; Kerns et al., 2004).

The (left) DLPFC was engaged on high-control trials following conflict trials, both under speed and under accuracy emphasis. This replicates earlier findings about the DLPFC in the conflict adaptation effect (Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004), and highlights the role the DLPFC plays in exerting cognitive control. Importantly, it was more engaged under speed emphasis, consistent with the prediction that as conflict increases, control on the subsequent trial should also be increased. Furthermore, ACC activation on conflict trials

predicted DLPFC activation on subsequent control trials; both under speed and accuracy emphasis, ACC activation during conflict trials was correlated with the subsequent DLPFC activation on control trials, replicating earlier work (Kerns et al., 2004).

Interestingly, this control-related DLPFC activation was left-lateralized, rather than right-lateralized like it was in these earlier studies (Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004). One possible, though speculative, explanation for this concerns the nature of the irrelevant stimulus dimension. In the studies by Kerns et al. (2004) and Egner & Hirsch (2005b), who used the color-naming Stroop task, the irrelevant stimulus dimension was the color word. In the study described by Egner & Hirsch (2005a), the right DLPFC ROI was identified by an analysis of the "face-target" task, in which the irrelevant stimulus dimension consistent of written names. Thus, in all of those studies, the irrelevant stimulus dimension was a word. In contrast, in the current study, the irrelevant stimulus dimension was location. Since word processing is typically thought to depend on the left hemisphere while location processing is typically thought of depending on the right hemisphere, it is possible that control to overcome an irrelevant stimulus dimension does not engage the DLPFC of the hemisphere dominant for that stimulus dimension, to reduce distraction.

In contrast to the conflict adaptation effect, post-error slowing was greater under accuracy emphasis. This is also consistent with the predictions stated earlier. In addition, the neuroimaging data showed that error-related ACC activation was greater under accuracy emphasis. This is consistent with the finding that the ERN is greater when accuracy is emphasized (Falkenstein et al., 2000; Gehring et al., 1993), considering that the ERN has consistently been modeled as having a generator in the ACC (e.g., Debener et al., 2005; Dehaene et al., 1994; Van Veen & Carter, 2002b), and with earlier findings that have found a relationship between ERN amplitude and post-error slowing (Debener et al., 2005; Gehring et al., 1993). Post-error slowing was furthermore related to activation of the right DLPFC, replicating Kerns et al. (2004); also replicating Kerns et al., error-related ACC activation predicted control-related DLPFC activation on the next trial.

In contrast, and not replicating Kerns et al. (2004), was the finding that DLPFC activation related to the conflict adaptation effect and DLPFC activation related to post-error slowing were not identical. In the neural network analyses of Botvinick et al. (2001), post-error slowing and the conflict adaptation effect were implemented in different ways: the conflict adaptation effect

is due to an increase of the activation of task-relevant stimulus representations, while post-error slowing is due to a decrease of the activation of motor representations. This begs the following questions: first, why did Kerns et al. (2004) identify the same area of the DLPFC as being associated with both the conflict adaptation effect and with post-error slowing, whereas these two phenomena were associated with different areas (in different hemispheres) in the present experiment? Second, why is post-error slowing associated with DLPFC activation in the first place, both in the data of Experiment 1 and in the data by Kerns et al. (2004)?

The answer to the first question must lie in the fact that different analyses were used to obtain these different results. Kerns et al. identified the DLPFC activation related to the conflict adaptation effect in a similar way as was done in the current study. However, they identified post-error slowing-related DLPFC activation by comparing slow post-error congruent trials to fast post-error congruent trials by performing a post-hoc analysis on the DLPFC ROI identified by the conflict adaptation effect analysis, and concluded that the same area of the DLPFC was involved in both the conflict adaptation effect and post-error slowing. In the present Experiment 1, this approach was not used because of the relatively few errors made under accuracy emphasis. Note that a potential problem with the approach taken in Experiment 1 is that the regressor for post-error trials and that for error trials were completely correlated; therefore, it is possible that variance associated with the neural response to errors was a confounding factor. (On the other hand, the DLPFC ROI attributed to post-error trials did not overlap with any activation identified by the error trials versus correct trials contrast.)

A analysis similar to the one performed by Kerns et al. (2004), performed on the keft DLPFC conflict adaptation effect ROI in the present data set, limited to speed emphasis, gave similar results: slow post-error congruent trials were associated with greater activation of this area than fast post-error congruent trials. However, there was no difference between slow and fast post-error incongruent trials for this area. On the other hand, in the right DLPFC ROI, slow post-error trials elicited greater activation than fast post-error trials independent of condition.

Thus, in answer to the second question, it is possible that while the left DLPFC identified by the conflict adaptation effect modulates the processing of representations in posterior areas by increasing the activation of task-relevant stimulus representations (Egner & Hirsch, 2005a), the area in the right DLPFC influences the priming of the response system. This possibility thus assumes that (at least) two processes influence performance on post-error trials. Recall that

Ridderinkhof (2002b) has argued that post-error top-down control is reflected in the slow portion of the delta plot. If one assumes that Ridderinkhof's (2002b) interpretations of the delta plot are correct, both forms of control indeed contribute to post-error performance. Thus, following this assumption, the data by Kerns et al. (2004) do not allow one to identify the post-error DLPFC activation to be related to one or the other process; in the data by Kerns et al. (2004), the post-error DLPFC activation could either reflect a component responsible for decreasing the activation of the motor system, or it could reflect a component responsible for increasing the activation of task-relevant stimulus representations while some other process was taking care of reducing the activation of the motor representations.

We might therefore propose that the post-error slowing component is associated with the right DLPFC in the present dataset (consistent with the observation that slow post-error trials elicited greater activation than fast post-error trials regardless of condition), whereas the post-error increase in top-down control is associated with the left DLPFC ROI in the current data set (consistent with the observation that slow post-error congruent trials elicited greater activation than fast post-error congruent trials, and with the observation that fast post-error incongruent trials elicited greater activation than slow post-error incongruent trials, albeit nonsignificantly). Obviously, future research is needed to test this further. One implication is that post-error slowing should be correlated with the right DLPFC between participants, whereas the left DLPFC should be associated with the decrease in the slope of the delta plot; however, any distributional analysis requires a lot of observations per subject, and even under speed emphasis, there were not enough post-error observations for all participants to calculate a delta plot.

Two possible functional hypotheses can account for the observation that post-error slowing is associated with right DLPFC activation. It is, first, possible that the increased DLPFC activation inhibits the activation of the response system in a top-down fashion. It might do so, for instance, by activation of its excitatory connections to the reticular nucleus of the thalamus, which, in turn, has an inhibitory effect on the underlying "specific" thalamic nuclei, thereby reducing the baseline activation of the response system (cf. Brunia, 1993, 1997). An alternative possibility is that the identified area of the DLPFC plays a role in perceptual decision making (cf. Heekeren et al., 2004). The increased activation under accuracy emphasis would indicate that following errors, its level of baseline activation has been reduced, and more perceptual evidence

is therefore needed to reach a decision threshold. The current data are not able to distinguish between these two possibilities.

Analysis of activation of the dorsal premotor areas, separately for response hand, showed motor preparation of the incorrect (ipsilateral) response hand to correct cI trials, consistent with LRP studies published earlier (Stürmer & Leuthold, 2003; Stürmer et al., 2002). The amplitude of this activation did not appear to differ between speed and accuracy emphasis. This is consistent with the findings from LRP data obtained using the Eriksen and Simon tasks (Osman et al., 2000; Rinkenauer et al., 2004; Van der Lubbe et al., 2001), which have also shown that in these tasks, the amplitude of the initial incorrect activation typically observed to incongruent trials in interference tasks is not modulated by different levels of SAT. In addition, in the present study, motor preparation of the correct (contralateral) response hand was indeed greater under accuracy emphasis. Thus, the present findings are consistent with the notion that greater accuracy emphasis is achieved by increasing the distance between baseline and threshold of (pre)motor representations, such that more activation is needed to reach the threshold, as proposed by the various decision criterion models (e.g., McClelland, 1979; Nikolić & Gronlund, 2002; Ratcliff, 2002; Ratcliff & Rouder, 1998; Ratcliff & Smith, 2004; Ratcliff et al., 1999; Usher & McClelland, 2001).

3.0 EXPERIMENT 2

3.1 PREDICTIONS

In Experiment 1, SAT was manipulated by instructing the participants before the onset of each block to emphasize either speed or accuracy. One could therefore say that SAT was manipulated via a "blocked design". While this design is appropriate for studying the results of already having established a desired level of SAT, it does not allow one to make inferences about how people actually establish that level of SAT in the first place. Thus, in Experiment 2, this issue was approached by, rather than varying SAT on a block-by-block basis, presenting participants with cues at various times during the experiment that instructed them to emphasize either speed or accuracy on the subsequent set of trials. Thus, analysis of differential fMRI activation in response to these SAT cues should reveal areas involved in changing a baseline or threshold.

Predictions are follows. First and most straightforward is the prediction that the performance data from Experiment 1 should replicate. Importantly, the conflict adaptation effect should be greater under speed emphasis, while post-error slowing should be greater under accuracy emphasis. Second, for areas that respond differentially to the SAT cues and that are involved in setting a baseline or threshold, this difference in activation should be sustained throughout the subsequent trial set. Third, since the "locus" of SAT during response interference tasks appears to be mostly at the level of response preparation and execution (Osman et al., 2000; Rinkenauer et al., 2004), these activations should include motor areas, including but not limited to the primary motor and/or premotor cortices. If speed emphasis involves an increase in the level of baseline activation, these areas should show increased sustained activation throughout performance emphasizing speed. This would also be consistent with the results of Experiment 1, which indicated that accuracy emphasis was associated with increased activation of the premotor areas.

3.2 MATERIALS AND METHODS

3.2.1 Research participants

Twenty healthy adults (10 females, 10 males), all right hand-handed, and all between 20 and 31 years of age (M = 25, SD = 4) participated in this experiment. They received a monetary fee of $75 for participating. All had normal or corrected-to-normal vision. Prior to the test, participants provided written informed consent in accordance with the Institutional Review Board of the University of California at Davis.

3.2.2 Task procedures

As in Experiment 1, participants performed the Simon task, using a similar experimental setup. The gray outline of two squares was visible throughout each block, along with a fixation point. Participants performed three blocks of trials. Each block started with a fixation screen, containing the two squares and a fixation point. SAT instruction cues consisted of either a large uppercase "A" or a large uppercase "S", indicating that the participant was to emphasize accuracy or speed, respectively, on the upcoming "miniblock" of Simon trials. This cue lasted 1000 ms, followed by a 5000 ms fixation screen. During this fixation screen and during the entire miniblock, the fixation point in the center of the screen was either a small uppercase "A" or a small uppercase "S", depending on the condition. Each "miniblock" could consist of a series of 4, 8, or 20 Simon trials. For each condition (speed and accuracy emphasis), there were 7 miniblocks of 4 trials, 4 miniblocks of 8 trials, and 1 miniblock of 20 trials. Thus, each block contained a total of 24 miniblocks, and a total of 160 Simon trials. These miniblocks, including SAT instruction cues, were presented in random order. As in Experiment 1, during each trial, one of the squares lit up either green or red for 150 ms, and was followed by a 2850 intertrial interval. The exception was the last stimulus of each miniblock, which was followed by a 14850 ms fixation point, after which the next cue was presented (so, the SOA from the last stimulus of a miniblock to the next cue was 15000 ms). Each block lasted 15 minutes and 24 seconds.

Also as in Experiment 1, when the stimulus location corresponded to the response hand, the trial was congruent; when the stimulus location was mapped onto the opposite response hand, the trial was incongruent. An example of a trial sequence is depicted in Figure 10.

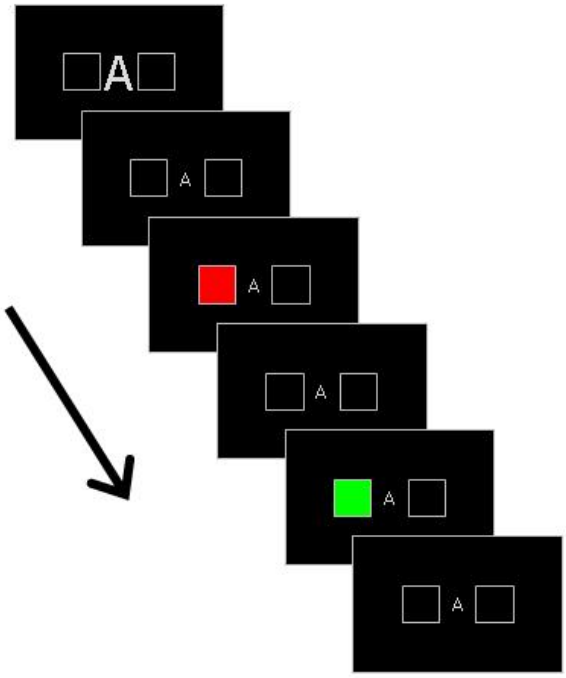

Figure 10. Example of a trial sequence in Experiment 2.

Thus, for participants instructed to respond to green with the left hand and right with the right hand, Figure 10 displays a cue telling a participant to emphasize accuracy, followed by two congruent trials.

For the analysis of the conflict adaptation effect, trial types were again defined as follows:

- cC trials were congruent trials immediately preceded by a correct congruent trial;
- cI trials were incongruent trials immediately preceded by a correct congruent trial;
- iC trials were congruent trials immediately preceded by a correct incongruent trial;
- iI trials were incongruent trials immediately preceded by a correct incongruent trial.

For the analyses involving the conflict adaptation effect, the first stimulus of each miniblock was ignored. The post-error slowing effect was analyzed by taking, for each error, the

difference between the RTs of the trial immediately following the error and the trial immediately preceding it.

3.2.3 Imaging procedure

3.2.3.1 Scanning procedures Functional images were acquired with a Trio 3 Tesla whole-body MRI system (Siemens AG, Munich, Germany), using T2*-weighted gradient-recalled echo, echo planar imaging (EPI) in the axial plane with a repetition time (TR) of 1500 ms, an effective echo time (TE) of 25 ms and a flip angle (FA) of 90°. Twenty-eight interleaved oblique axial slices with a 22 cm field of view were acquired, consisting of a 64 by 64 matrix with a slice thickness of 4.0 mm (no interslice gap), yielding a voxel size of 47.3 mm^3.

3.2.3.2 Image processing Data was preprocessed and analyzed using BrainVoyager software (Brain Innovation, Maastricht, the Netherlands). As each block started with a 12000 ms fixation screen, the first 8 images at each slice location were discarded from the analysis. The remaining 608 images were preprocessed using interscan slice time correction, 3D motion correction, 3D Gaussian spatial filtering (FWHM = 6 mm), and temporal high-pass filtering using a low cutoff frequency of 3 cycles/block (or .00328947 Hz). For each participant, three-dimensional images (MPRAGEs) of the brain were acquired at the end of the experiment. The functional data were aligned to these and then transformed into Talairach space.

In order to determine which areas responded differentially to the two cue types, 2 by 6 repeated measures ANOVAs were conducted per voxel on the first 6 scans after each cue, baseline-corrected (as percent signal change) to the first of these scans. SAT condition (speed, accuracy) and Scan (1-6) were used as factors, and participant as a random factor. Temporal autocorrelation in the fMRI time series was estimated and corrected using an autoregressive function. The per-voxel statistical threshold for this analysis was F_{crit} (5, 95) < 4.503, corresponding to $p < .001$. A voxel cluster threshold of 250 mm^3 was used.

To determine whether these areas were activated in a sustained fashion throughout each miniblock, over and above the transient responses these areas might have in response to the Simon stimuli, post-hoc general linear analyses were performed on each ROI. These analyses contained sustained regressors for speed and accuracy miniblocks, and transient responses to

low-conflict trials (cC, iI, iC), high-conflict trials (cI), error trials, and correct trials preceded by error trials, each of the separately for speed and accuracy emphasis. To test whether activity was sustained throughout each block, the beta weights for the "sustained" regressors for speed and accuracy blocks were tested against each other with paired t-tests.

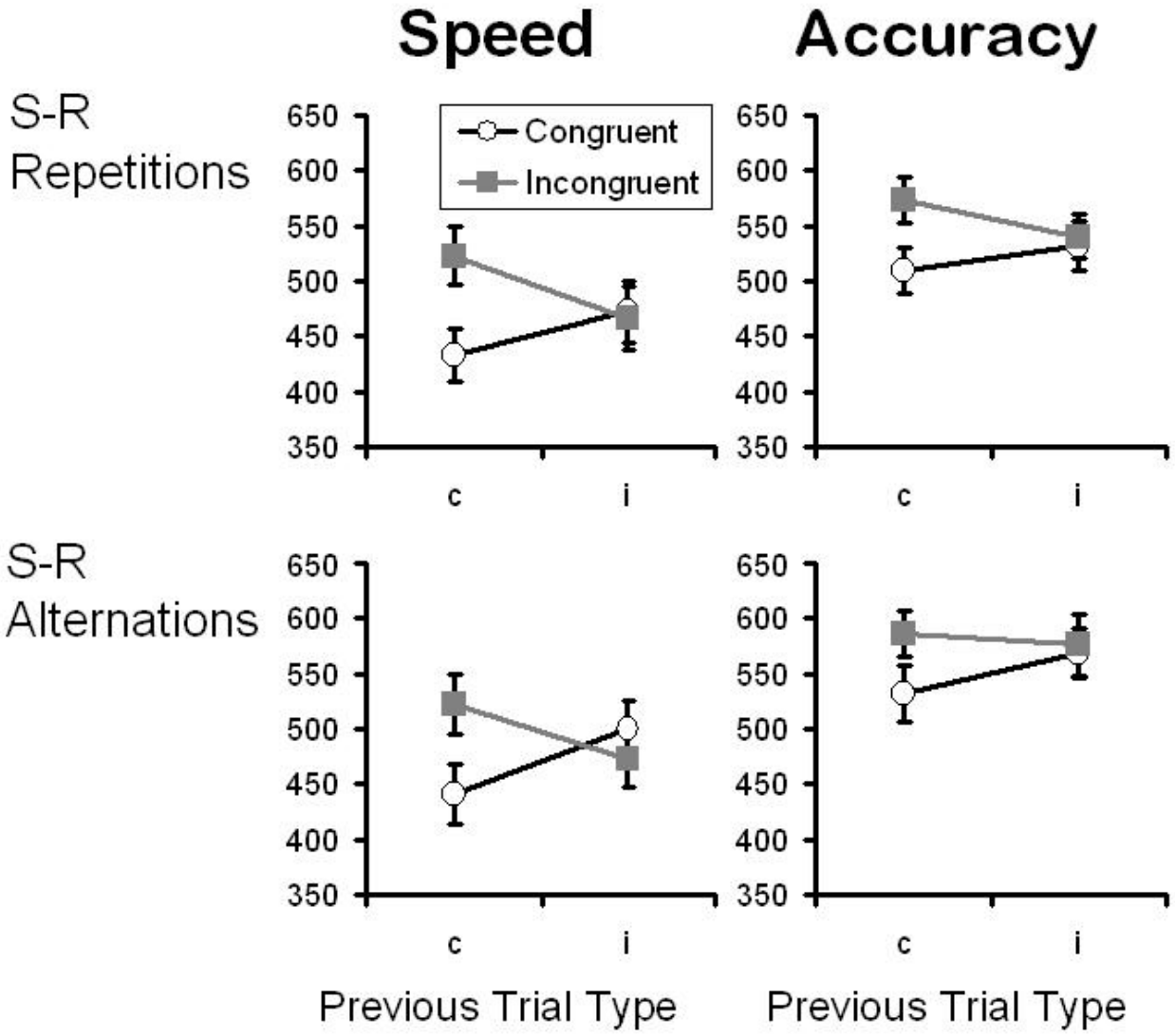

Figure 11. Reaction times in Experiment 2.

3.3 RESULTS

3.3.1 Performance data: Effects of SAT and trial type

Mean RTs and error rates are displayed in Figure 11 and Figure 12, respectively, as a function of SAT emphasis, Preceding Trial Type (congruent: c; incongruent: i), Current Trial Type (congruent: open circles; incongruent: grey squares), and whether trials were S-R Repetitions or S-R Alternations. Error bars represent one SEM. See Table 10 and Table 11 in APPENDIX B for more details.

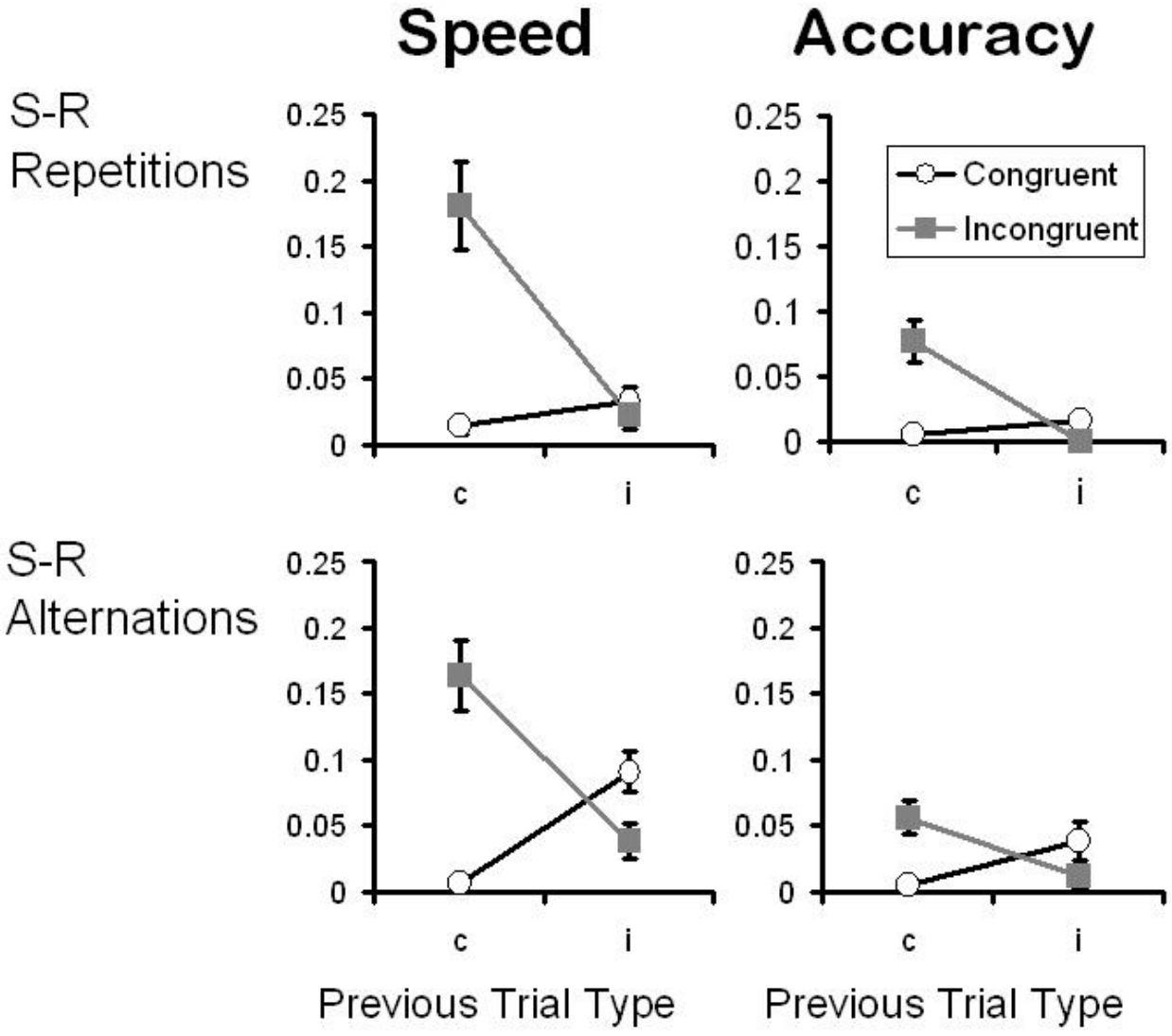

Figure 12. Error rates in Experiment 2.

The results of the repeated-measures 4-way ANOVA between Repetition (Rep), SAT, previous trial type (PT) and current trial type (CT), for both RTs and error rates, are shown in Table 6. Although variances are often greater under accuracy emphasis than under speed emphasis (e.g., in Experiment 1), this did not appear to be the case in the current data set. Therefore, no transformation on the RTs was deemed necessary. Analyses of error rates were performed on arcsine-transformed data. This transformation reduced heteroscedasticity but did not remove it.

Table 6. Overview of statistical analyses of performance data.

Effect	**RTs**	**Error Rates (arcsine-transformed)**
Main effects		
SAT	$F(1, 19) = 7.907$, $p = .011$	$F(1, 19) = 27.142$, $p < .001$
Rep	$F(1, 19) = 11.301$, $p = .003$	$F(1, 19) = 1.675$, $p = .211$
PT	$F(1, 19) = .115$, $p = .739$	$F(1, 19) = 27.751$, $p < .001$
CT	$F(1, 19) = 68.620$, $p < .001$	$F(1, 19) = 24.466$, $p < .001$
2-way interactions		
PT*CT	$F(1, 19) = 147.834$, $p < .001$	$F(1, 19) = 60.787$, $p < .001$
SAT * PT	$F(1, 19) = 1.884$, $p = .186$	$F(1, 19) = 3.562$, $p = .074$
PT * Rep	$F(1, 19) = 6.160$, $p = .023$	$F(1, 19) = 8.136$, $p = .010$
SAT*CT	$F(1, 19) = .124$, $p = .729$	$F(1, 19) = 8.815$, $p = .008$
SAT*Rep	$F(1, 19) = 22.335$, $p < .001$	$F(1, 19) = .774$, $p = .390$
CT*Rep	$F(1, 19) = 3.791$. $p = .066$	$F(1, 19) = 3.915$, $p = .063$
3-way interactions		
SAT*PT*CT	$F(1, 19) = 16.688$, $p = .001$	$F(1, 19) = 10.977$, $p = .004$
Rep*PT*CT	$F(1, 19) = .007$, $p = .936$	$F(1, 19) = .216$, $p = .647$
Rep*SAT*CT	$F(1, 19) = 1.509$, $p = .234$	$F(1, 19) = .337$, $p = .569$
Rep*SAT*PT	$F(1, 19) = .200$, $p = .659$	$F(1, 19) = 1.263$, $p = .275$
4-way interaction		
SAT*Rep*PT*CT	$F(1, 23) = 1.185$, $p = .290$	$F(1, 23) = .785$, $p = .387$

Table 6 displays the results of the 4-way interaction on the RT data and the arcsine-transformed error rates in Experiment 2, with as factors SAT (speed, accuracy), Previous Trial Type or PT (congruent, incongruent), Current Trial Type or CT (congruent, incongruent), and Stimulus-Response Repetition or Rep (repetition, alternation).

3.3.1.1 SAT RTs were faster under speed accuracy than under accuracy emphasis, while error rates were higher under speed than under accuracy emphasis. This verifies that the participants did indeed trade speed and accuracy.

3.3.1.2 Simon interference RTs were faster to congruent than to incongruent trials; error rates, too, were lower to congruent than to incongruent trials. Thus, performance was impaired during incongruent trials compared to congruent trials; this verifies the Simon interference effect (Simon, 1969; Simon & Berbaum, 1990; Simon & Rudell, 1967).

3.3.1.3 Repetition effects RTs were faster to S-R repetition trials than to S-R alternation trials, though error rates did not significantly differ between S-R repetitions and S-R alternations. This verified that priming of S-R mappings did indeed contribute to the pattern of performance.

3.3.1.4 Modulation of repetition effects by SAT The 2-way interaction between SAT emphasis and repetition was significant for RTs, revealing that repetition effects were greater under accuracy than under speed emphasis.

3.3.1.5 Modulation of interference by SAT The 2-way interaction between SAT emphasis and condition failed to reach significance for RTs, although it did so for error rates. This suggested that the Simon interference effect was greater under speed than under accuracy emphasis. This suggests that conflict was greater under speed emphasis.

3.3.1.6 The conflict adaptation effect The 2-way interaction between current trial type and previous trial type was significant, both for RTs and error rates. This verified the previously reported conflict adaptation effect; the size of the interference effect was greater when preceded by a congruent trial and smaller when preceded by an incongruent trial. As also reported

previously, repetition did not influence this greatly; the 3-way interaction between repetition, previous trial, and current trial was not significant, neither for RTs nor error rates. Post-hoc tests showed that for RTs, the previous trial by current trial interaction remained significant both for S-R repetitions, $F(1, 19) = 103.112$, $p < .001$, and for S-R alternations, $F(1, 19) = 37.342$, $p < .001$.

3.3.1.7 Modulation of the conflict adaptation effect by SAT The 3-way interaction between SAT, previous trial type, and current trial type was significant for both RTs and error rates, showing that the conflict adaptation effect was greater under speed than under accuracy emphasis. The fact that the 4-way interaction which included Repetition failed to reach significance suggests that the pattern of results is independent of whether the trials are S-R alternations or S-R repetitions. The interaction between SAT, previous trial, and current trial was furthermore computed separately for S-R repetitions and S-R alternations. For S-R repetitions, the 3-way interaction for RTs between SAT, previous trial, and current trial was significant both for RTs, $F(1, 19) = 9.041$, $p = .007$, and for error rates, $F(1, 19) = 7.114$, $p = .015$. Computed separately for SR alternations, the 3-way interaction was also significant both for RTs, $F(1, 19) = 12.291$, $p = .002$, and for error rates, $F(1, 19) = 8.616$, $p = .008$. Thus, these results confirmed the predictions outlined earlier.

3.3.2 Performance data: Post-error slowing

Post-error slowing was calculated for each overt error by taking the difference between the RT of the preceding correct trial and the RT to the following correct trial. Unfortunately, most participants only made a few overt errors under accuracy emphasis; only 10 participants had 3 or more overt errors in this condition, so analyses were limited to these participants. The amount of post-error slowing per SAT condition for these 10 participants is shown in Figure 13. Error bars represent 1 SEM.

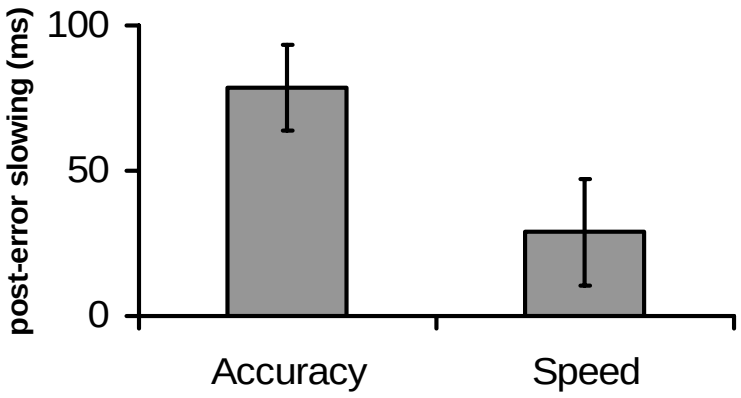

Figure 13. Post-error slowing in Experiment 2.

The difference between the post-error trial and the pre-error trial was significantly greater than 0 under accuracy emphasis (M = 75 ms, SD = 45), $t(9) = 5.259$, $p = .001$, while showing a trend towards significance under speed emphasis (M = 33 ms, SD = 56), $t(9) = 1.885$, $p = .092$, verifying the post-error slowing phenomenon. Furthermore, post-error slowing was significantly greater under accuracy emphasis than under speed emphasis $t(9) = 2.263$, $p = .050$, confirming the predictions stated earlier.

3.3.3 Imaging data

The SAT by scan repeated measures ANOVA identified a number of regions, listed in Table 7, which were differentially activated to the speed and accuracy cues. These differentially active regions included the bilateral DLPFC, the bilateral dorsal premotor areas, the supplementary motor area (SMA), the ACC, the posterior cingulate cortex (PCC), the left ventral premotor cortex, and the bilateral basal ganglia and thalamus.

Figure 14 displays the cortical region differentially activated by the speed and accuracy cues. The medial view of the left hemisphere is displayed on the top left; the lateral view of the left hemisphere is displayed on the bottom left; the medial view of the right hemisphere is displayed on the top right; the lateral view of the right hemisphere is displayed on the bottom right. A top view of both cerebral hemispheres is presented in the center.

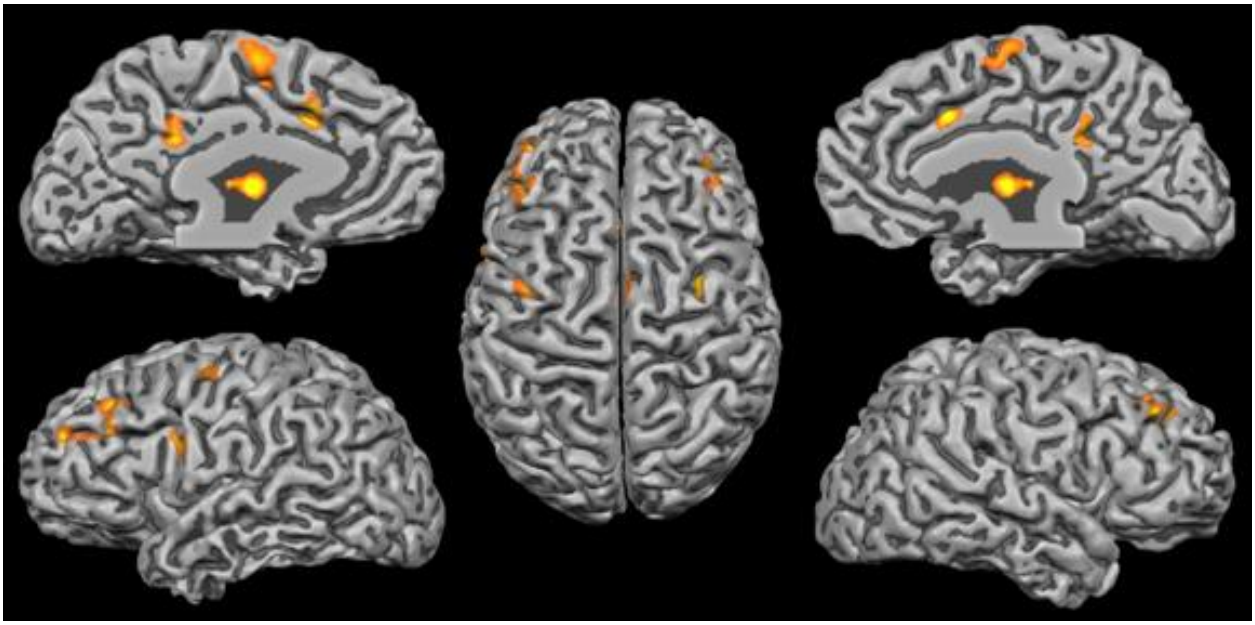

Figure 14. Cortical areas differentially engaged by speed versus accuracy cues.

Figure 15 displays the activity in he basal ganglia and thalamus on axial slices. Images are shown in neurological convention (left = left, right = right). The left image displays activation at Z = 16; the right image displays activation at Z = 4.

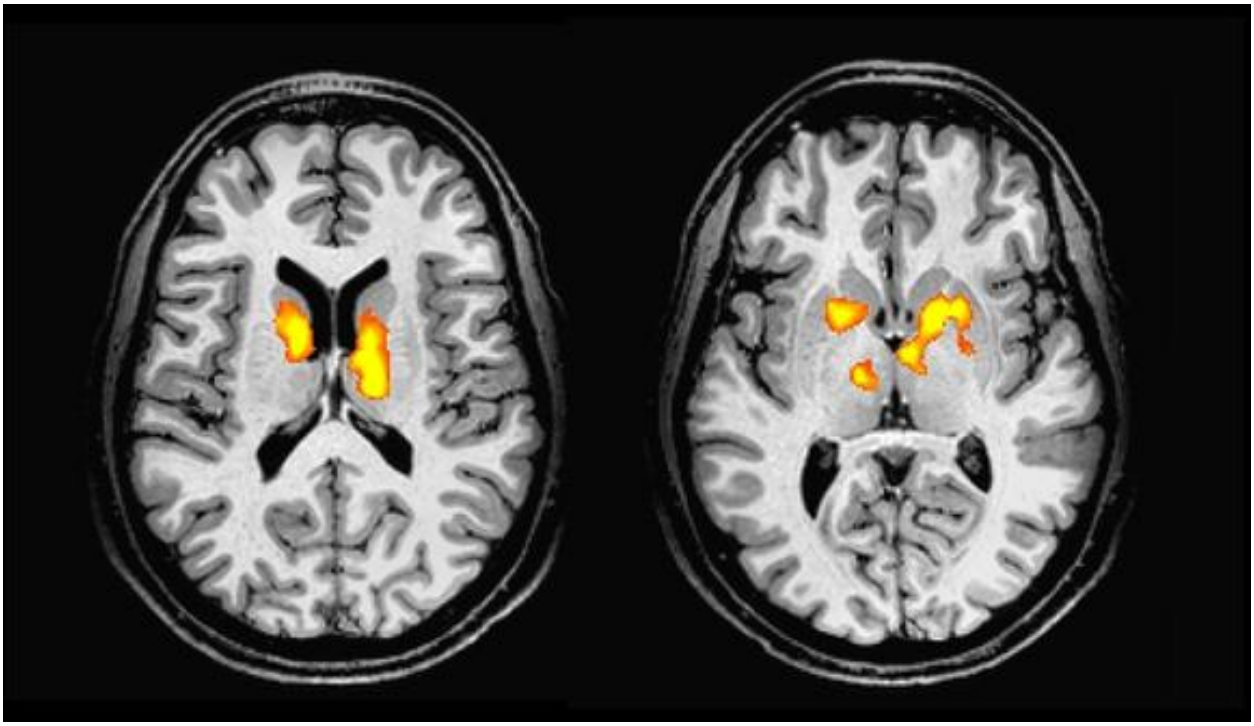

Figure 15. Subcortical areas differentially engaged by speed versus accuracy cues.

Table 7. Brain areas differentially engaged by speed versus accuracy cues.

Region of interest	**BA**	**Volume (mm^3)**	**x, y, z**
SMA	6	1017	-2, -6, 60
Left dorsal premotor cortex	6	277	-38, -7, 55
Right dorsal premotor cortex	6	578	34, -7, 59
ACC	6/32/24	792	0, 15, 39
Left DLPFC	8/9	986	-39, 39, 37
Right DLPFC	8/9	676	37, 38, 42
Left ventral premotor cortex	6/44	546	-53, 3, 27
Posterior cingulate cortex	31	985	0, -40, 31
Basal ganglia / thalamus		9511	1, -2, 12

The time courses of activation in response to the cues for each of these areas are displayed in Figure 16. Activation following accuracy cues is shown in gray diamonds; activation following speed cues is shown in open circles. Error bars represent one SEM.

The results of the post-hoc tests also suggested that activity was sustained throughout each miniblock for most of these areas; controlling for transient activity in response to the Simon stimuli, the betas for the sustained regressor for speed were greater than those for the sustained regressor for accuracy for the right premotor cortex, $t(19) = 2.519$, $p = .021$, the left premotor cortex, $t(19) = 2.201$, $p = .040$, the supplementary motor area, $t(19) = 3.264$, $p = .004$, the ACC, $t(19) = 3.121$, $p = .006$, the right DLPFC, $t(19) = 3.762$, $p = .001$, the left DLPFC, $t(19) = 2.362$, $p = .029$, and the basal ganglia/thalamus, $t(19) = 3.076$, $p = .006$. The left ventral premotor area only showed a trend towards significance, $t(19) = 1.993$, $p = .061$, while activation differences in the posterior cingulate cortex were not significant, $t(19) = 1.462$, $p = .160$. The latter pattern of activation, suggesting that the posterior cingulate is not engaged tonically throughout each miniblock, is consistent with the pattern of the time course to the cue (see Figure 16), which suggests that the enhanced posterior cingulate activation in response to speed cues is transient, and returns to baseline.

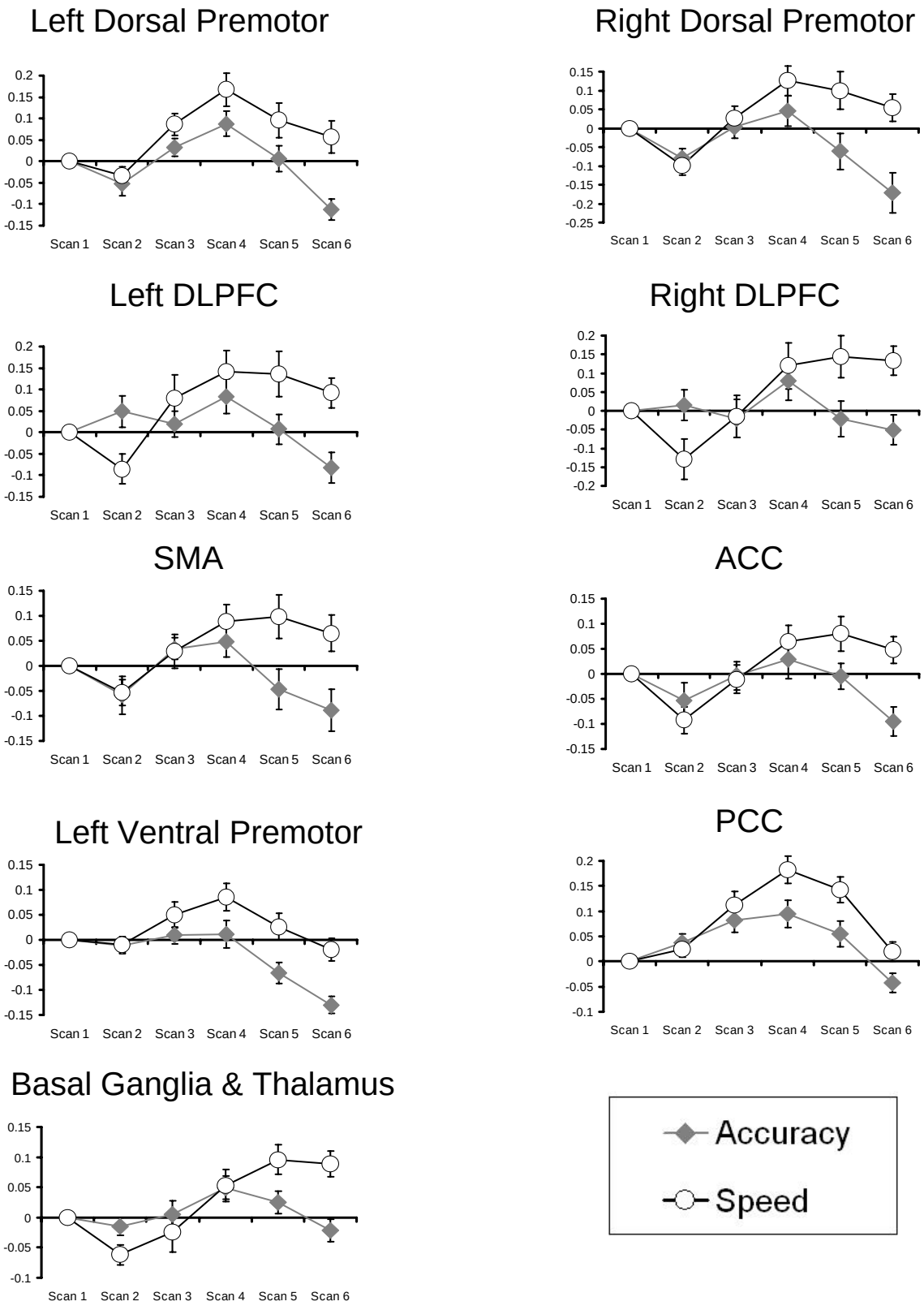

Figure 16. Activation time course of areas differentially engaged by speed vs accuracy cues.

To assess whether the transient activation related to making an overt response was greater under accuracy emphasis, the average, z-scored fMRI signal from the dorsal premotor cortices was analyzed using a deconvolution analysis. Note that no percent-from-baseline calculation was made so that the baseline activation level could be studied. The regressors used for these analyses were based on 8 time points following each response with the contralateral hand, separately for speed and accuracy emphasis, plus error trials and ipsilateral responses as covariates of noninterest, again each separately for speed and accuracy emphasis. Again, an autoregressive function was used to estimate and correct autocorrelation. The obtained results for contralateral responses were then averaged across the left and right hemispheres. (An approach whereby motor-related activation is averaged across right hand and left hand responses is common in research using LRPs, EMG, and squeeze force; e.g., Coles et al., 1985; de Jong et al., 1988; Gratton et al., 1988; Kopp et al., 1996.)

The result is shown in Figure 17. Response-related activation under accuracy emphasis is shown in gray diamonds; response-related activation under speed emphasis is shown in open circles. Error bars represent one SEM.

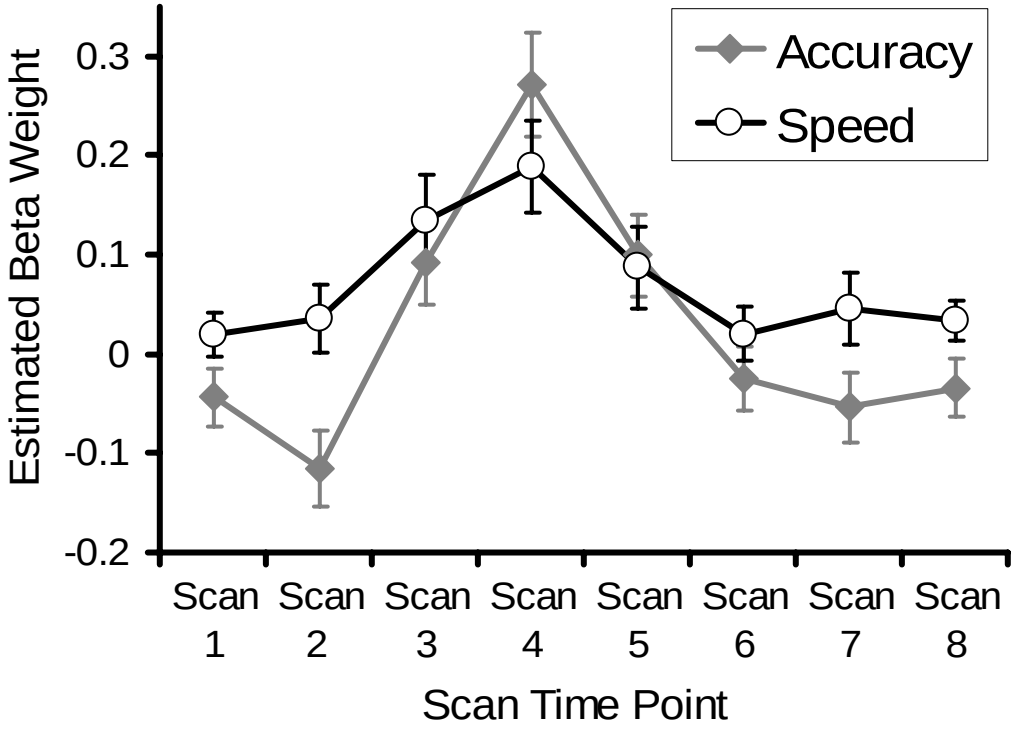

Figure 17. Average deconvoluted response-related time course of the dorsal premotor areas.

The significant interaction between SAT (speed, accuracy) and scan time point (scans 1 through 8) indicated that transient activation of the dorsal premotor cortices was greater under accuracy emphasis, $F(7, 133) = 4.88$, $p < .001$. To test whether the baseline was decreased under accuracy emphasis, the baseline was defined as the average of time points 1 and 8; this baseline activation was indeed significantly lower under accuracy than under speed emphasis, $t(19) = 3.14$, $p = .005$.

3.4 DISCUSSION

Behavioral results replicated those of Experiment 1. The basic Simon and SAT effects were replicated (Craft & Simon, 1970; Wickelgren, 1977). Post-error slowing and the conflict adaptation effect were both observed, also replicating earlier work, (Laming, 1979; Rabbitt, 1966b; Rabbitt & Rodgers, 1977; Stürmer & Leuthold, 2003; Stürmer et al., 2002; Wühr & Ansorge, 2005). As has also been reported earlier, the conflict adaptation effect was found not to depend on repetition effects (Stürmer & Leuthold, 2003; Stürmer et al., 2002; Wühr & Ansorge, 2005). Importantly, the novel findings of Experiment 1 were replicated: the conflict adaptation effect was again found to be greater under speed than under accuracy emphasis (and as in Experiment 1, this was found not to depend on repetition effects), whereas the post-error slowing effect was again found to be greater under accuracy emphasis.

Several brain areas showed differential activation to the speed and accuracy cues; all of these areas were more engaged by speed than by accuracy cues. These areas encompassed the bilateral dorsal premotor cortex (PMC), the left ventral PMC, the supplementary motor area (SMA) in the medial wall, the ACC, bilateral dorsolateral prefrontal cortex (DLPFC), and bilateral basal ganglia/thalamus. These areas are all known to be involved in the making of decisions and in the preparation and execution of movements. As discussed earlier, computational approaches have conceptualized the difference between performance under speed versus accuracy emphasis as a change in the distance between baseline activity and threshold; the smaller this distance, the more speed is emphasized (Ratcliff & Rouder, 1998; Smith & Ratcliff,

2004; Usher & McClelland, 2001). Thus, the increased baseline activation during speeded performance is consistent with the assumptions of these computational models.

The PMC and SMA have long been known to play an important role in the preparation and execution of movements (Ball et al., 1999; Ghez, 1991; Picard & Strick, 1996, 2001) The basal ganglia, too, are known to be involved in motor processes (Graybiel, Aosaki, Flaherty, & Kimura, 1994). The premotor areas in the ACC, PMC and SMA are thought to be the major contributors to the readiness potential leading up to a motor response (Ball et al., 1999; Leuthold & Jentzsch, 2001), while the PMC (together with the primary motor areas) is thought to be the major generator of the LRP (Dehaene et al., 1998; Leuthold & Jentzsch, 2001, 2002). Specifically, these areas are thought to represent actions at a relatively abstract level of representation, as opposed to the primary motor cortices which represent movement at a more mechanistic and concrete level (Ghez, 1991).

Both the DLPFC and the premotor regions of the frontal lobe (including those areas of the PMC, SMA, and ACC identified in the present study) have been argued to form integral, segregated cortico-striato-thalamo-cortical circuits (Alexander & Crutcher, 1990; Alexander, DeLong, & Strick, 1986; Strick, 2004). The premotor circuits connecting the SMA, the PMC, and the cingulate motor areas to the striatum and thalamus include the putamen of the striatum, and the ventrolateral nuclei of the thalamus (Alexander et al., 1986; Hatanaka et al., 2003; Strick, 2004). The DLPFC circuit includes the caudate nucleus of the striatum, and the ventral anterior and dorsomedial nuclei of the thalamus (Alexander et al., 1986). In the present data, basal ganglia activation included bilateral caudate nucleus (including both head and tail), putamen, and the globus pallidus, while thalamic activation included the dorsomedial, ventrolateral, anterior, and ventral anterior nuclei. Thus, the current data show that speed emphasis not only increases the baseline activation of the cortical representations, but of the entire cortico-striato-thalamo-cortical circuits that underlie the motor and context representations involved in this task.

Note that apart from the "motor" ACC circuit, which includes the premotor areas of the ACC (Hatanaka et al., 2003; Strick, 2004), a "limbic" ACC circuit has also been described (Alexander et al., 1986). This loop includes the anterior nucleus of the thalamus and the ventral striatum of the basal ganglia. However, while the anterior nucleus of the thalamus was indeed differentially engaged by the speed versus accuracy cues, the ventral striatum was not (this was also the case at considerably reduced statistical thresholds). Thus, while the present dataset

provides evidence for involvement of the ACC motor circuit, there is only partial support for involvement of the limbic ACC circuit in the manipulation of SAT.

The caudate nucleus has been identified as playing a role in establishing a selective response bias as well. Lauwereyns and colleagues (Lauwereyns, Watanabe, Coe, & Hikosaka, 2002) found that this nucleus is involved in creating a selective, reward-based response bias in the macaque (i.e., increasing the level of baseline activation of one of the relevant responses). The present results extend such findings to the notion that the striatum does not only generate a response bias that is not selective for one or another response, but can increase the level of baseline activation of all relevant responses. Future research might investigate whether activation of the caudate in SAT is reward-based or not (cf. Delgado, Locke, Stenger, & Fiez, 2003). Assuming that participants perceive making errors as a kind of punishment (as has on occasion been done; e.g., Holroyd & Coles, 2002) one could argue that on the one hand, participants anticipate greater such punishment when instructed to emphasize speed; on the other hand, they might find making errors more of a punishment when asked to emphasize accuracy and avoid errors altogether.

Of particular interest is the fact that increased sustained activation under speed emphasis was also found in bilateral areas of the DLPFC. There are, again, two hypotheses regarding the functional role played by the DLPFC in emphasizing speed versus accuracy.

First, it is possible that the DLPFC plays a role in the top-down activation of posterior representations. As discussed earlier, the DLPFC is often thought to be critically involved in attentional processes by increasing the activation of task-relevant representations in posterior cortices (Egner & Hirsch, 2005a; B. T. Miller & D'Esposito, 2005; E. K. Miller, 2000; E. K. Miller & Cohen, 2001). The present data suggests that the DLPFC could perform a similar function during SAT as it does during other forms of executive control; it provides top-down support to increase the level of baseline activation of motor representations. This pattern of activation is also consistent with findings implicating a role for the DLPFC not just in task preparation (MacDonald, Cohen, Stenger, & Carter, 2000; Sohn, Ursu, Anderson, Stenger, & Carter, 2000), but also in movement preparation (Pochon et al., 2001; Ramnani & Miall, 2003); preparing to make a speeded response is a process that involves an increase in the baseline activation of the appropriate response representations as well (Jennings & Van der Molen, 2005).

At a superficial glance, if this interpretation is correct, this pattern of activation in the DLPFC might appear somewhat counterintuitive. In everyday speech, we typically treat emphasizing accuracy, paying closer attention, and "being careful" as more-or-less related concepts. These data, however, clearly contradict this notion, as this would have predicted greater activation under accuracy emphasis. But, since the baseline activation of motor representations is increased under speed emphasis, an area involved in executive control that provides top-down support to activate motor representations would be expected to display elevated activation during speed emphasis – which is what was found in the present experiment. These findings are thus consistent with the notion that the DLPFC represents context (Cohen, Servan-Schreiber, & McClelland, 1992; Curtis & D'Esposito, 2003; E. K. Miller, 2000; E. K. Miller & Cohen, 2001) and has an excitatory influence on posterior representations (Egner & Hirsch, 2005a; B. T. Miller & D'Esposito, 2005).

Based on their Talairach coordinates, the engaged areas of the SMA and dorsal PMC can be assumed to correspond to the "SMA proper" and the caudal dorsal PMC, rather than the pre-SMA or the rostral dorsal PMC. Picard & Strick (1996) have suggested that the vertical plane through the anterior commissure (Y = 0) can be used as a heuristic to differentiate between the pre-SMA and SMA proper. Elsewhere, these authors have suggested a similar heuristic to distinguish between the rostral and caudal parts of the dorsal premotor areas (Picard & Strick, 2001). In contrast to their more rostrally situated counterparts, neither the SMA proper nor the caudal dorsal PMC is thought to receive substantial input from the DLPFC (Picard & Strick, 2001). Therefore, assuming that the DLPFC does influence the activity in these areas in a top-down fashion, it most likely does so indirectly, via its connections with the basal ganglia. This would be consistent with the notion that the caudate nucleus creates a response bias (cf. Lauwereyns et al., 2002). Alternatively, the DLPFC increases the baseline activation of the premotor system by its connections to the thalamus, or via its connections with the ventral PMC or ACC (cf. Picard & Strick, 2001).

A second notion regarding the role of the DLPFC in emphasizing speed versus accuracy comes from findings suggesting it plays a role in perceptual decisions. According to this notion, the DLPFC (or a part thereof) makes perceptual decisions by integrating evidence from sensory processing areas. Evidence for this notion comes mostly from neurophysiological studies in nonhuman primates; however, there is functional MRI evidence in humans as well. Heekeren et

al. (2004) presented house and face stimuli in varying levels of degradation and asked participants to determine whether the stimulus was a face or a house. Areas in the ventral temporal cortex were identified that responded selectively to faces versus houses. Heekeren et al. found a region in the left DLPFC that was more active to easy than to difficult discriminations, and also responded to the absolute difference in activation between the two ventral temporal regions. They explained these data by assuming that evidence is being accumulated in the face- and house-selective ventral temporal regions, while the DLPFC takes the absolute difference between these sources of evidence to make a perceptual decision. It is thus possible that the DLPFC in the present data identifies the stimulus by comparing the accumulating evidence in favor of one or the other color and location, thus deciding what response to make. If this interpretation of the change in baseline activation in the DLPFC is correct, this would imply that SAT takes place at a pre-response level of decision making.

Based on the results of Experiment 2, one cannot distinguish between these two different possibilities. It would seem that the notion that a baseline increase in the DLPFC is associated with smaller buildup of evidence under speed emphasis is not consistent with the results of Experiment 1, which showed that DLPFC activation is greater under speed emphasis. If the increase in baseline activation of the DLPFC would have represented a change in the baseline of an evidence accumulator for a decision, one would have predicted greater DLPFC activation under accuracy emphasis. However, the DLPFC areas engaged in the two experiments by these two conditions did not overlap with one another. Therefore, firm conclusions cannot be drawn regarding this issue.

It is important to note that the increased DLPFC activation under speed emphasis does not constitute perceptual attention or selection, in the sense of a top-down control component that engages task-relevant perceptual representations (as it has been suggested to do in the conflict adaptation effect, Egner & Hirsch, 2005a). We can exclude this possibility because the Simon interference effect was greater under speed emphasis, while greater perceptual attention under speed emphasis would have predicted the opposite. This is not consistent with the assumptions made by Yeung et al. (2004) in their attempt to model increased ACC activation (as indexed by the ERN) under accuracy emphasis (Falkenstein et al., 2000; Gehring et al., 1993). They modeled emphasizing accuracy as an increase in threshold (not a decrease of baseline activation) in combination with increased sustained perceptual attention (i.e., an increase in the

activation of the context layer). In their model, the increased sustained attention increases the tendency to correct the error, which in their model accounts for the increased ERN. However, since such sustained attention under accuracy emphasis would most likely be associated with increased sustained DLPFC activation following accuracy cues, the present data do not support this model.

Furthermore, the results of Experiment 1 were also inconsistent with the model described by Yeung et al. (2004). As this model assumes increased perceptual attention under accuracy emphasis, it predicts a reduced interference effect and a reduced activation of the incorrect response during incongruent trials under accuracy emphasis. This is because such an increase in sustained attention should result in reduced susceptibility to the irrelevant stimulus dimension, and therefore a smaller preparation of the incorrect response during incongruent trials. This is not what was observed in Experiment 1, nor has it been observed in LRP studies of both the Simon and Eriksen tasks (Osman et al., 2000; Rinkenauer et al., 2004; Van der Lubbe et al., 2001); both in Experiment 1 and in these LRP studies, the magnitude of preparation of the incorrect response during conflict trials was not modulated by SAT. Thus, a more likely explanation for the greater conflict-related ACC activation under speed emphasis is an increased baseline activation of the response system.

4.0 CONCLUSIONS

The behavioral results of Experiments 1 and 2 were remarkably similar. SAT modulated the conflict adaptation effect and post-error slowing in opposite ways. The conflict adaptation effect was greater under speed emphasis; post-error slowing was greater under accuracy emphasis. These results are in agreement with the predictions made earlier: as observed in Experiment 1, greater conflict-related ACC activation under speed emphasis led to greater engagement of control and control-related DLPFC activation during post-conflict trials under speed emphasis, resulting in a greater conflict adaptation effect under speed than under accuracy emphasis; conversely, greater error-related ACC activation under accuracy emphasis led to greater increased control and control-related DLPFC activation on post-error trials during accuracy emphasis, resulting in greater post-error slowing during accuracy emphasis.

The neuroimaging data were also consistent with the predictions. The results of Experiment 2, which showed increased baseline activation of parts of the response system during speed emphasis, provided evidence for the notion that speed is emphasized by decreasing the distance between baseline and threshold activity (Ratcliff & Rouder, 1998; Smith & Ratcliff, 2004; Usher & McClelland, 2001). Additionally and relatedly, this experiment provided supportive evidence for the notion that response conflict is increased under speed emphasis (despite the fact that transient activation of the incorrect response is not modulated by SAT). This is because an increased level of baseline activation between two competing responses would imply a higher level of energy (see APPENDIX A). Consistent with both of these implications, the results of Experiment 1 showed that conflict-related ACC activation, and subsequent control-related DLPFC activation, were greater when participants emphasize speed; error-related ACC activation, and subsequent control-related DLPFC activation, were greater when participants emphasized accuracy.

On the surface, the bilateral dorsal PMC appeared to behave oppositely in both experiments. That is, on the one hand, conflict-related activation of these regions in the blocked design of Experiment 1 was increased under accuracy, and transient response-related activation was increased under accuracy emphasis in Experiment 2. On the other hand, however, sustained activation of these regions in Experiment 2 was increased under speed emphasis. This pattern of activation is consistent with the predictions of accumulation models (Smith & Ratcliff, 2004; Usher & McClelland, 2001).

The ACC did not behave like this; speed emphasis led to greater ACC activation both in the case of conflict in Experiment 1, and in the case of cue-related activation of Experiment 2. This argues against the notion that this structure has a role in response preparation comparable to that of the dorsal PMC. These findings are more consistent with a role for the ACC in performance monitoring.

An obvious and seemingly inherent contradiction in the present data sets concerns the relationship of DLPFC activation to SAT. In Experiment 1, it was observed that post-error slowing (which constitutes a shift in SAT towards accuracy; see Laming, 1979) was associated with increased DLPFC activation. In contrast, in Experiment 2, cues instructing participants to place emphasis on speed elicited greater DLPFC activation than cues instructing accuracy emphasis. These results do not appear to be in agreement; why is DLPFC activation greater under accuracy emphasis following errors, but greater under speed emphasis following instruction cues?

The present data do not provide an answer to this question, but it is possible to speculate. First, it is possible that, since the areas of the DLPFC did not overlap, the two areas have different functions. Thus, as explained earlier, one area might implement top-down control while the other might reflect the buildup of evidence towards a perceptual decision.

It is, however, also possible that the function of the DLPFC in the representation of context is more general than has been assumed throughout this thesis. Context representations become engaged when automatic processing needs to be controlled; it might be the case that people automatically, depending on the nature of the task, tend to gravitate towards one particular place on the SAT curve, and need control to place themselves on another part of this curve regardless of whether this change would place more emphasis on speed or accuracy. Thus, speed emphasis in the current data might require more control because participants are naturally

more inclined to place more emphasis on accuracy in the Simon task. In contrast, more control might be needed following errors under accuracy emphasis than under speed emphasis because it is more important to slow down following errors under accuracy emphasis. The notion that SAT cues modulate DLPFC according to where on the SAT curve a participant's natural tendency falls is an empirical one. There are several ways to test this notion. One could, for instance, compare DLPFC activation in healthy individuals that have a tendency to place greater emphasis on accuracy, to a clinical population that has a tendency to be more impulsive and who are more automatically inclined to emphasize speed (e.g., children diagnosed with attention deficit hyperactivity disorder). Alternatively, one could compare DLPFC activation to speed cues in the present task to a task in which participants are more inclined to emphasize speed, and require context to emphasize accuracy (although the problem with this latter suggestion is that it is thought that where a person falls on the SAT curve is dependent on one's sensitivity to error commission, e.g., Laming, 1979; if such a task could be found, it would elicit low error rates).

Other, competing theories can account for the present findings to varying extents. In order to account for the present findings, the explanation of the conflict adaptation effect that the event file theory offers (Hommel et al., 2004) would have to assume that event files degrade over time, and that slower responses are therefore less likely to be influenced by event files established on previous trials. This is consistent with several sources of evidence (Hommel, 1994), for instance with the finding that the conflict adaptation effect is reduced with increasing interstimulus time (Wühr & Ansorge, 2005). Thus, as far as the conflict adaptation effect is concerned, the event file theory can account for the present findings. This assumption, however, is not consistent with the tendency for repetition priming effects to be greater under accuracy emphasis. Recall that in Experiment 2, stimulus-response repetition priming effects were significantly greater under accuracy emphasis – and although repetition priming effects in the analysis of the log-transformed data in Experiment 1 were not significantly modulated by SAT, an analysis of the untransformed data approached significance. So, the notion that both repetition priming effects and the conflict adaptation effect result from the same cognitive process is not supported by the current data, which suggest that these phenomena are differentially modulated by SAT.

Brown and Braver's (2005) "error likelihood" theory of ACC functioning also finds partial support in the current datasets. As explained earlier, this theory predicts that ACC

activation is greatest in situations in which errors are most likely. This is indeed what was found; conflict-related ACC activation was greatest under speed emphasis. The theory might even be consistent with the findings of greater sustained ACC activation throughout speeded performance, and with the findings that ACC activation is greater under accuracy emphasis. However, this theory predicts that ACC responses are greater to stimuli that have previously been associated with high error rates. Therefore, this theory predicts that after having performed 2 speed blocks, responses to incongruent trials would be high, whereas responses would be low after having performed two accuracy blocks. This is not what the present results showed (see APPENDIX C); ACC activation did not depend on block order. Furthermore, ACC activation was greater during speed blocks following accuracy blocks than during accuracy blocks following speed blocks, even when constraining this analysis to the first few trials following the first two blocks.

The issue of the role of the increased DLPFC activation following speed cues is an important one for understanding the cognitive architecture involved in trading speed and accuracy. Previously, two interpretations for this activation were discussed: top-down control on motor representations, and perceptual decision making. If this activation indicates enhanced top-down control that increases the baseline activation of the response system, this places the "locus" of SAT at the representational level of response preparation and execution (cf. Osman et al., 2000; Rinkenauer et al., 2004). If, on the other hand, this activation is related to making perceptual decisions, this places the locus at both a response-level and a pre-response level of representation.

As explained earlier, Rinkenauer et al. (2004) compared different levels of SAT across a line discrimination task, a syntactic gender discrimination task, and the Eriksen task, and found that (after correcting for fast guesses), speed emphasis in the line and syntactic gender discrimination tasks shortened the interval between stimulus onset and LRP onset. In contrast, speed emphasis in the Eriksen task shortened the interval between LRP onset and response. Rinkenauer et al. suggested that the types of representations engaged by SAT might be task-dependent. Thus, SAT during tasks that require finer perceptual decisions, and that do not have difficult response requirements, might occur by the manipulation of the level of baseline activation of the perceptual processes involved rather than that of the response baseline. But, since making a perceptual decision involves the DLPFC (Heekeren et al., 2004), it is possible

that SAT during perceptual or similar decisions might also involve a modulation of DLPFC activation. A possible future follow-up experiment might thus manipulate SAT during the face/house discrimination task by Heekeren et al. Such an experiment should be able to test both of these notions; if SAT were to influence perceptual representations, one would expect a baseline modulation of the relevant temporal areas. If SAT in this task involved perceptual decisions, one would expect a baseline increase in the same DLPFC area as identified by Heekeren et al. If, on the other hand, SAT and perceptual decision making were independent, one would expect separate areas of the DLPFC to be involved with SAT and perceptual decision-making.

Executive control refers to those faculties that allow us our remarkable cognitive and behavioral flexibility. The ability to trade speed and accuracy is an important aspect of this flexibility; we can choose to focus on whatever mode of responding we judge to be more important given the situation. The present two experiments have increased our understanding of these abilities by identifying the neural processes that underlie the ability to strategically control the position along the SAT continuum, and the effects that this has on attention and performance.

APPENDIX A

HYPOTHESIZED TIME RESPONSE COURSES

As explained earlier, high-conflict trials are typically characterized by a fast activation of the (incorrect) response associated with the irrelevant stimulus dimension; the activation of the correct response is relatively later and competes with this activation. Likewise, errors during speeded response tasks are typically instances in which this initial activation reaches response threshold; the subsequent activation of the correct response is manifested as the immediate tendency to correct the error. The ACC is thought to respond to the level of conflict tat exists between these two activations.

The assumption that the levels of baseline activation are increased under speed emphasis and decreased under accuracy emphasis has implications for the level of conflict that exists between these two activations. This can be illustrated by a schematic depiction of the response activation and the level of conflict that exists between the active responses. A cartoon depiction of the time course of response activation during correct conflict trials is depicted in Figure 18; in this figure, the initial activation of the correct response is displayed in red, the subsequent activation of the correct response is displayed in green. Note that the level baseline of activation is increased under speed emphasis, such that correct responses under speed emphasis are associated with a smaller transient (baseline-to-peak) activation of the correct response; note also that the transient (baseline-to-peak) activation of the initial, incorrect response is not modulated by SAT,

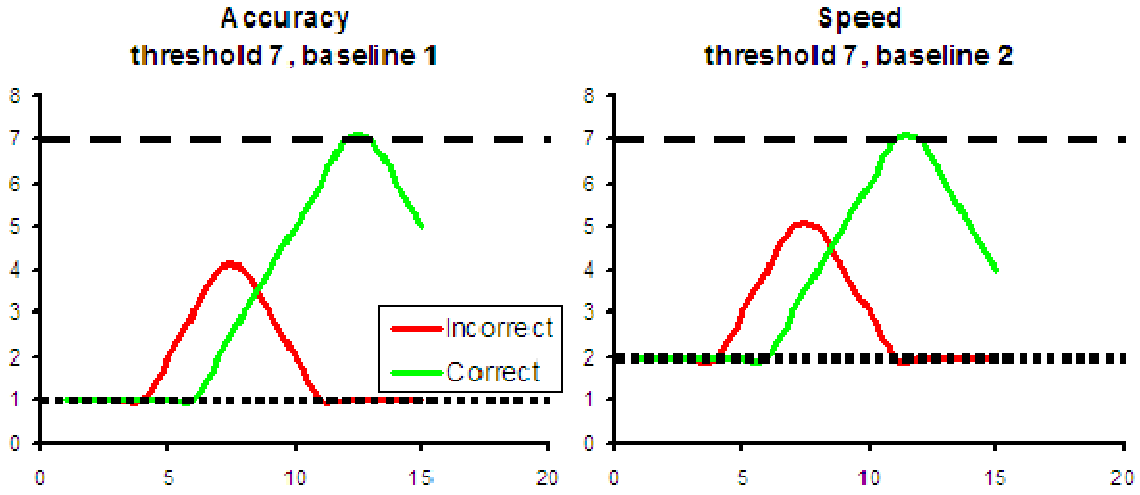

Figure 18. Cartoon of response activation time courses for conflict trials.

A cartoon of the time course of activation of the incorrect and correct responses during error trials is depicted in Figure 19. Note again that the level of baseline activation if increased under speed emphasis; note also that the transient (baseline-to-peak) activations of both the incorrect and subsequent corrective responses are smaller under speed emphasis than under accuracy emphasis.

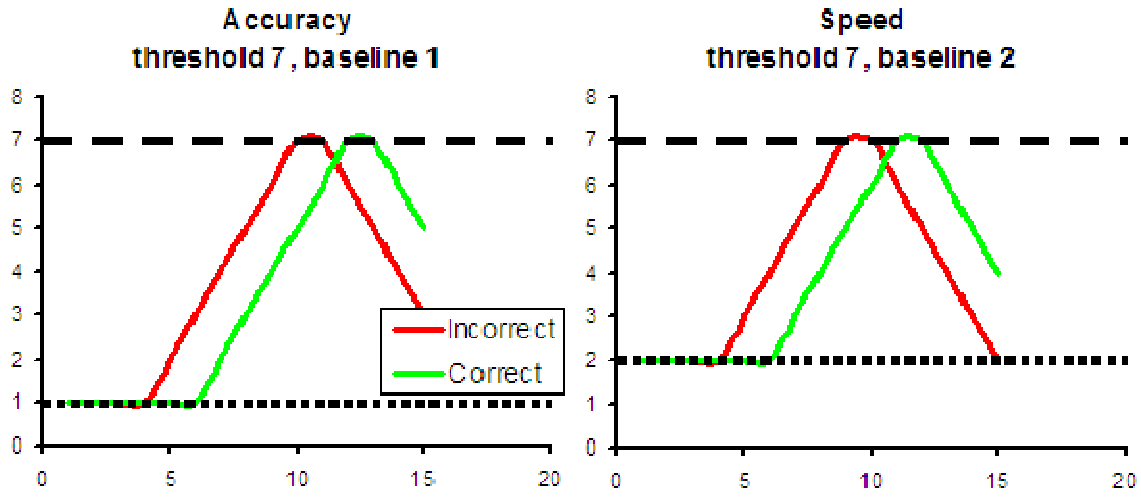

Figure 19. Cartoon of response activation time courses for error trials.

If the level of conflict is then computed for each of these time courses, by taking the product, as proposed by Botvinick et al (2001) following Hopfield (1982), the following results are obtained, displayed in Figure 20:

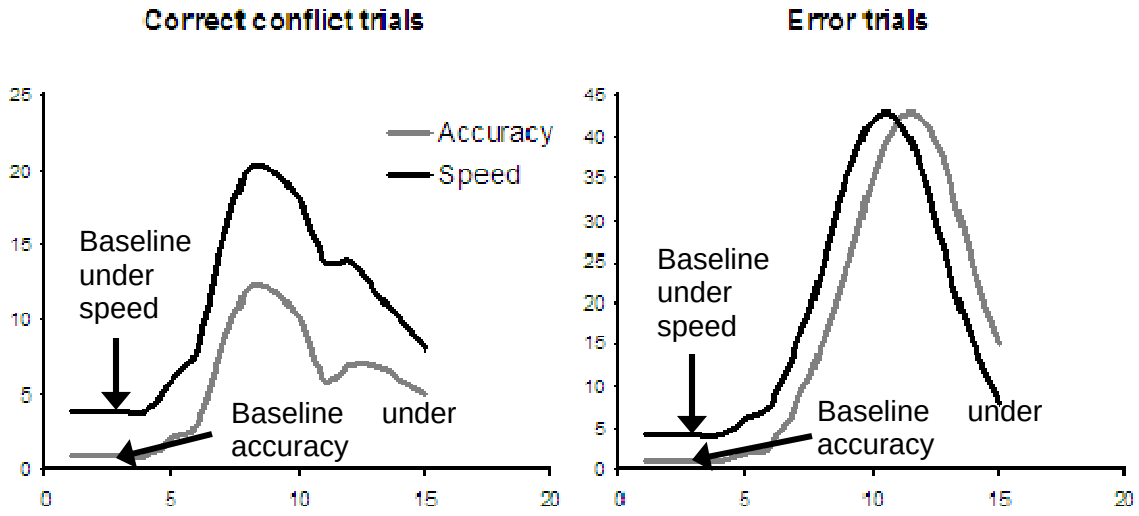

Figure 20. Cartoon of the conflict time course during conflict and error trials.

In Figure 20, the conflict between incorrect and correct response under accuracy emphasis is displayed in grey, while the conflict under speed is displayed in black. Note how the transient (baseline-to-peak) conflict during correct conflict trials is greater under speed emphasis, but that the transient (baseline-to-peak) activation during error trials is greater under accuracy emphasis.

APPENDIX B

TABLES OF PERFORMANCE DATA

This appendix contains the summarized performance data for each experiment. Table 8 contains the mean RT per condition from Experiment 1. Table 9 contains the mean error rates per condition from Experiment 1. Table 10 contains the mean RT per condition from Experiment 2. Table 11 contains the mean error rates per condition from Experiment 2. Standard deviations per condition are given in brackets.

Table 8. RT data from Experiment 1.

		Speed		**Accuracy**	
		Congruent	**Incongruent**	**Congruent**	**Incongruent**
Repetition	**Preceded by congruent**	401 (36)	483 (53)	546 (83)	604 (96)
	Preceded by incongruent	441 (47)	436 (39)	579 (95)	566 (79)
Alternation	**Preceded by congruent**	402 (40)	485 (50)	560 (89)	615 (93)
	Preceded by incongruent	461 (56)	455 (39)	599 (101)	595 (99)

Table 9. Error rate data from Experiment 1.

		Speed		Accuracy	
		Congruent	**Incongruent**	**Congruent**	**Incongruent**
Repetition	**Preceded by congruent**	.01 (.01)	.21 (.15)	0 (0)	.02 (.03)
	Preceded by incongruent	.03 (.03)	.05 (.08)	.01 (.01)	0 (.02)
Alternation	**Preceded by congruent**	.01 (.01)	.18 (.18)	0 (0)	.02 (.03)
	Preceded by incongruent	.05 (.04)	.10 (.12)	.01 (.02)	.01 (.02)

Table 10. RT data from Experiment 2.

		Speed		Accuracy	
		Congruent	**Incongruent**	**Congruent**	**Incongruent**
Repetition	**Preceded by congruent**	432 (106)	523 (120)	509 (92)	573 (92)
	Preceded by incongruent	472 (126)	467 (126)	531 (100)	540 (90)
Alternation	**Preceded by congruent**	441 (119)	522 (122)	533 (115)	586 (91)
	Preceded by incongruent	499 (118)	473 (109)	569 (101)	576 (125)

Table 11. Error rate data from Experiment 2.

		Speed		**Accuracy**	
		Congruent	**Incongruent**	**Congruent**	**Incongruent**
Repetition	**Preceded by congruent**	.01 (.03)	.18 (.15)	.01 (.01)	.08 (.07)
	Preceded by incongruent	.03 (.05)	.02 (.05)	.02 (.02)	0 (0)
Alternation	**Preceded by congruent**	.01 (.01)	.16 (.12)	0 (.01)	.06 (.06)
	Preceded by incongruent	.09 (.07)	.04 (.06)	.04 (.06)	.01 (.04)

APPENDIX C

ACC ACTIVATION AND PREVIOUS SAT CONDITION

Although most of the present data is consistent with the "error likelihood" model of ACC functioning, as recently proposed by Brown and Braver (Brown & Braver, 2005), some aspects of these data might not immediately be reconcilable with this model in its present form. Specifically, the error likelihood model would predict that, for participants who start out with speed emphasis and therefore make a lot of errors to incongruent trials, a large conflict-related ACC response should be observed on the subsequent accuracy emphasis blocks. This is because, according to this model, the ACC responds to the stimulus representation itself, and the stimuli in the present experiments do not change between SAT conditions. Thus, when a participant is asked to emphasize accuracy after they've just performed the task emphasizing speed, those same stimuli have been associated with a high error rate; therefore, the ACC should be expected to respond strongly to those stimuli for these participants.

In order to test this, ACC activation in Experiment 1 was re-analyzed using SAT block order (speed first, accuracy first) as an additional, between-group factor. While SAT interacted significantly with the conflict contrast (cC cI iC iI -1 3 -1 -1), as described in the Results section of Experiment 1, adding SAT block order to this analysis did not significantly change this interaction; the 3-way interaction between SAT, SAT block order, and the conflict contrast was not significant, $F(1, 22) = .019$, $p = .892$. Restricting this analysis to the ACC response under accuracy emphasis, conflict-related activation under accuracy emphasis was not significantly different between people who started and those who ended with accuracy emphasis $F(1, 22) = 2.546$, $p = .125$,(albeit in the right direction), whereas the error likelihood model would predict that it should be greater for people who finished with accuracy emphasis blocks.

It is possible, however, that after switching from speed emphasis to accuracy emphasis, the ACC quickly adapts to the new low error rate. To test this possibility, a new regresion analysis was performed, with predictors based on the cC, iC, iI, error, post-error, and first trial for each block, plus a regressor based on the first 5 cI trials and the remaining cI trials, separately for speed and accuracy emphasis. The error likelihood model would predict that after switching from speed emphasis to accuracy emphasis, the ACC would respond strongly to cI trials in the beginning but on later trial respond less strongly. The conflict theory, in contrast, would predict that the ACC would respond more strongly to cI trials under speed emphasis. The contrast testing early cI trials to cC, iC, and iI trials was indeed significant, $F(1, 22) = 6.950$, $p = .016$, and interacted significantly with SAT, $F(1, 22) = 10.050$, $p = .004$, replicating the earlier analysis; however, again, block order did not significantly contribute to this interaction, $F(1, 22) = .048$, $p = .829$. Limiting the analysis to accuracy emphasis conflict-related activation early on during performance was again not significantly different between those that started with accuracy emphasis and those that ended with it, $F(1, 22) = 1.129$, $p = .267$ (although it should again be noted that pattern was in the right direction). Thus, conflict theory accounts for the present findings considerably better than the error likelihood model.

APPENDIX D

DISTRIBUTIONAL ANALYSES: DELTA PLOTS

It has recently been hypothesized that control during interference tasks is reflected in delta plots, which show the interference effect (incongruent - congruent) as a function of RT (Burle et al., 2002; de Jong et al., 1994; Mattler, 2003; Ridderinkhof, 2002a, 2002b; Ridderinkhof, van den Wildenberg, Wijnen, & Burle, 2004). In order to obtain a delta plot, the RTs per condition are first divided into quantiles; then the quantiles for the incongruent and congruent conditions are subtracted from one another. That is, the fastest congruent quantile is subtracted from the fastest incongruent quantile, the second-fastest congruent quantile is subtracted from the second-fastest incongruent quantile, and so forth. Each of these difference scores is then plotted against the average RT; thus, the difference score for the fastest quantiles is plotted against the average RT of the fastest incongruent and fastest congruent quantiles, and so forth.

Increased interference control reduces the interference effect, resulting in relatively smaller differences between incongruent and congruent RTs. Since control operates more effectively on slower than on faster RTs, differences in control between conditions should be expressed more in the interference effect for slower RTs than for faster RTs. Thus, control is expressed in the *slope* associated with the slower RT portion of the delta plots, with greater control being manifested as a (relatively) more negative-going delta plot slope for slower RTs.

Delta plots have been used as argued to provide evidence for increased control on trials following response conflict and errors (Burle et al., 2002; Ridderinkhof, 2002b). Additionally, they have been shown to be sensitive to individual differences in interference control, including clinical disorders. For instance, delta plots are more negative for participants with smaller

interference effects (Ridderinkhof, 2002a). Furthermore, children with AH/HD, a disorder that is also thought to involve impaired control, show less negative-going delta plots compared to age-matched controls (Ridderinkhof, Scheres, Oosterlaan, & Sergeant, 2005). Thus, delta plots can be used not only to detect between-condition, but also between-group differences in cognitive control.

Delta plots were analyzed for both experiments by binning each condition (cC, cI, iC, and iI, separately for speed and accuracy, correct trials preceded by correct trials only) into quintiles. This way, delta plot for interference following congruent trials and following incongruent trials could be constructed separately for speed and accuracy emphasis. In order to obtain the delta plots, the first quantile of the cC condition was subtracted from the first quantile of the cI condition and plotted against the average of these two quantiles, the first quantile of the iC condition was subtracted from the first quantile of the iI condition and plotted against the average of these two quantiles, and so on.

Results for Experiment 1 and Experiment 2 are shown in Figure 21 and Figure 22, respectively. Results for speed emphasis are displayed in open circles; results for accuracy emphasis are displayed in gray diamonds. The interference effect for trials following congruent trials is displayed in dotted lines; the interference effect for trials following incongruent trials is displayed in solid lines.

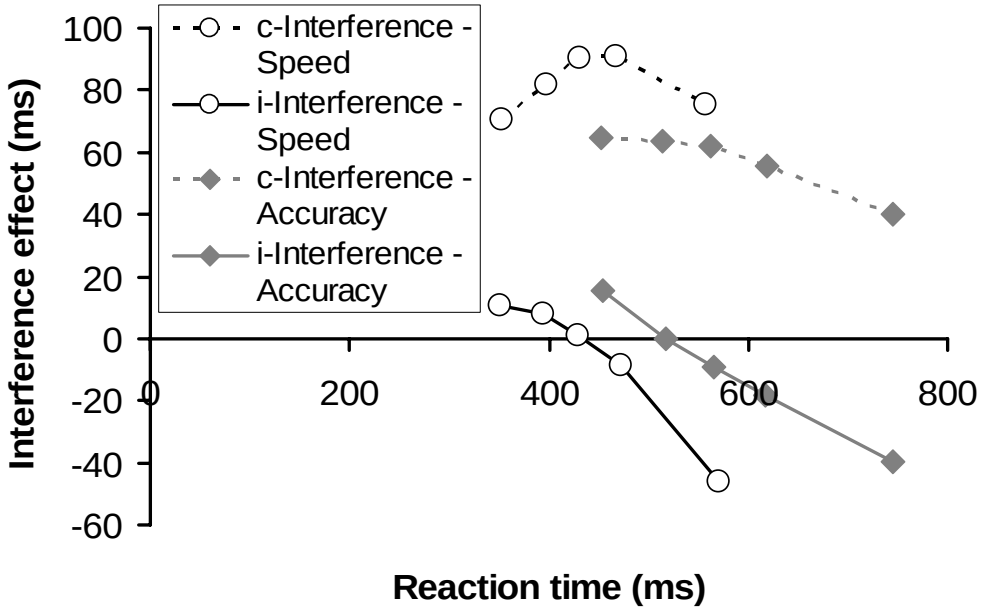

Figure 21. Delta plots for Experiment 1.

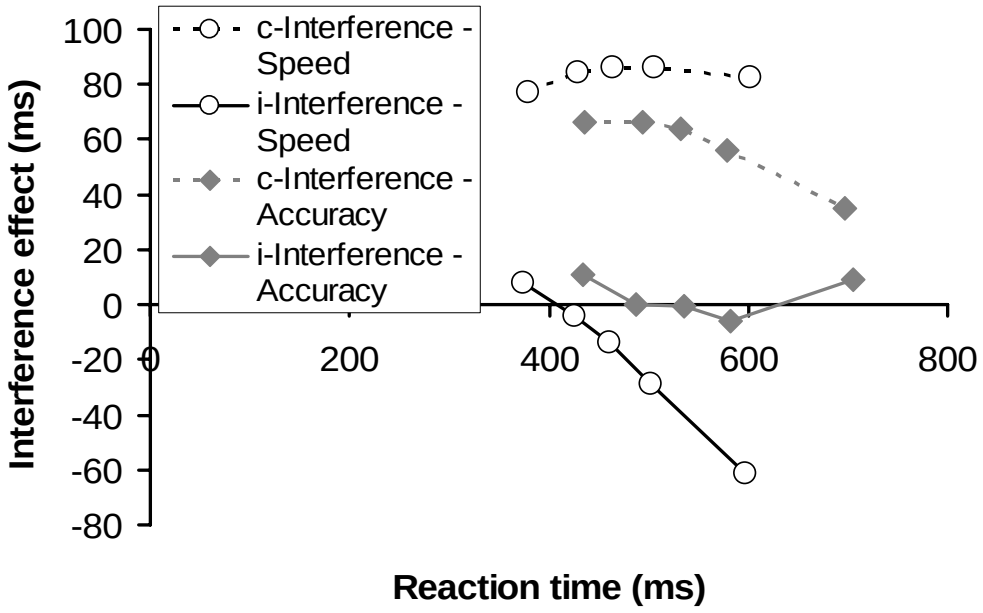

Figure 22. Delta plots for Experiment 2.

Planned contrasts were performed on the slopes associated with the fourth and fifth quintile, calculated for each condition, for each participant. The slope for trials following incongruent trials under speed emphasis was significantly more negative-going than in the other three conditions, both in Experiment 1, $F(1, 23) = 4.730$, $p = .040$, and in Experiment 2, $F(1, 19) = 7.974$, $p = .011$. The slopes for the remaining three conditions did not differ significantly from one another in Experiment 1, $F(1, 23) = .147$, $p = .864$, although there was a trend towards significance in Experiment 2, $F(1, 19) = 2.680$, $p = .082$.

Thus, results from delta plot analysis, assuming that Ridderinkhof's (2002b) interpretation of these distributional analyses is correct, are consistent with the interpretation for the behavioral and neuroimaging findings of Experiment 1, namely, that the increase of control following incongruent trials is greater under speed emphasis.

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