STIMULUS AND RESPONSE-RELATED CONFLICT PROCESSING IN THE HUMAN BRAIN

by

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A Dissertation Submitted to the Graduate Faculty of George Mason University in Partial Fulfillment of The Requirements for the Degree of Doctor of Philosophy Psychology

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DEDICATION

This is dedicated to my wife Laren. Thank you for affording me the opportunity to go back to school.
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Cognitive control seeks to limit the conflict created by simultaneous activation of alternative cognitive representations. Conflict monitoring is theorized to be the first of a two-step cognitive control process. Conflicting representations activate control, which then mitigates the consequences of conflict through compensatory attentional allocations. While the current literature has localized these conflict monitoring processes to the medial frontal cortex, the types of conflict that activate the circuit remain unclear.

The three experiments of this dissertation were designed to explore the relationship between conflict instantiated at different levels of information processing and the medial frontal cortex conflict monitoring circuit. The use of modified Eriksen flanker stimuli, psychophysically tuned to each participant, allowed for the controlled elicitation of both response conflict and stimulus uncertainty. By monitoring the behavioral and electrophysiological responses to both types of conflict during a given trial as well as on
a trial-to-trial basis, these studies serve to better characterize both medial frontal cortex mediated conflict monitoring and visual cortex mediated attentional biasing.

Results showed that stimulus uncertainty and response conflict have dissociable, but related effects on conflict monitoring processes. However, the sensitivity of conflict monitoring processes to stimulus uncertainty requires careful quantification of the amount of uncertainty instantiated, an aspect overlooked in prior studies. Trial-to-trial findings showed a relationship between response conflict processing and stimulus uncertainty processing such that prior exposure to either type of conflict facilitated the subsequent performance of a response conflict inducing task. This facilitation occurs via top-down influences on sensory processing following exposure to stimulus uncertainty.

This modulation provides empirical evidence of a biologically plausible mechanism linking cognitive control and biased competition of attention allocation. In addition, these findings suggest a modification of current conflict monitoring theory to include multiple, related conflict monitoring sub processes as opposed to a single executive monitor. These results fit within the framework previously established that implicates the medial frontal cortex in higher-level decision tasks while expanding the empirical proof to lower-level cognitive processes as well as increasing the temporal resolution of the associated processing.
CHAPTER 1: Introduction

The flexibility to change course depending on feedback is an important capability for optimal performance in order to achieve a desired goal. If a strategy does not produce the results required for successful performance, it is essential to both be aware of this, as well as to select and implement a new strategy. Cognitive control is the mechanism responsible for these processes in the brain.

The neural events underlying these cognitive processes are not fully understood. Current models of cognitive control focus on the activating role of conflict between representations within the information processing hierarchy. When multiple alternative cognitive representations are simultaneously active, this conflicting information must be appropriately identified and processed in order to meet task goals.

To date, the major thrust of research has focused on response conflict, processed in the posterior medial frontal cortex. As shown in figure 1, the areas of prior research focus for studies of conflict processing include parts of Broadmann areas 6, 8, 24 and 32.
A classic example of response conflict is the Stroop effect, where prepotent (color word reading) and less potent (color naming) response representations are simultaneously active in the brain, causing conflict. However, conflict can also arise between representations at earlier stages of information processing. For example, conflicts occur within the visual discrimination system when ambiguous or uncertain stimuli are presented for identification. Theories of cognitive control at the later response selection stage allow for bottom-up input from earlier stages, and models of attention biasing at the earlier perceptual stage suggest that top-down biasing can occur. Nonetheless, the question remains as to how these two systems interact to insure optimal performance in the processing of uncertain visual input in the environment.

The goal of the series of novel experiments described in the following chapters is to examine the neural mechanisms of cognitive control in an effort to better understand
how control is activated in the presence of conflicting mental representations, whether “early” or “late” in information processing. Specifically, is conflict that occurs early in the information processing cascade (i.e., visual discrimination conflict or stimulus uncertainty) processed by the same monitoring system as conflict that occurs late in the cascade (i.e. response conflict)? The results reported in the following chapters describe a series of experiments manipulating conflict within the visual and response processing systems, in an effort to create a series of dissociable, conflicting mental representations in the brain.

The interactions between these conflicting representations further describe how stimulus uncertainty elicited from early visual processing and response conflict from later processing interact within the domain of cognitive control. By measuring electrophysiological signals in addition to behavioral responses to different sources of conflict, the neural mechanism responsible for enacting cognitive control can be more fully described.

Theoretical frameworks for both cognitive control and decision making in general will be discussed briefly. A specific behavioral consequence of errors in the activation of cognitive control, the compatibility sequence effect known as the “Gratton Effect,” will be described empirically as well as by using a computational model. The computational framework is thought to be approximated by an influential cognitive theory, conflict monitoring, which has widely been associated with the medial frontal cortex.

The theoretical and neural evidence of conflict monitoring in both humans and non-human primates will be outlined. Finally, the biased competition model of attention
allocation in the visual cortex will be described. This attention allocation framework provides a complimentary mechanism to conflict monitoring. The combination of these two influential theories leads to an integrated description of how stimulus-related and response-related sources of conflict are processed in the brain.

Finally, the role of task context in both stimulus and response conflict processing will be addressed. Together, these experiments seek to describe what, if any, differences there are in the processing of stimulus and response related conflict, and how these differences should modify the current theoretical understanding of both conflict monitoring in the frontal cortex, and biased competition in the occipital cortex.

**Cognitive Control**

Logan (1985) describes cognitive control as the executive processes associated with ensuring optimal performance. Given the prevalence of parallel processing in current models of brain function, limiting the cross-talk (i.e., Allport, 1980) among these processing streams is an important interim step to optimal performance. Thus, the mechanisms of cognitive control are tied closely with attentional theories such as the biased competition model (Desimone and Duncan, 1995) among others (Cohen et al., 1990; Norman and Shallice, 1986).

The goal of cognitive control can be generally conceptualized as limiting the processing of interfering or irrelevant sensory stimuli. Since this process requires the allocation of attention, the capacity for successful control is limited (Moray, 1967), and requires effort on the part of the individual. Because control requires effort to carry out,
the brain requires cognitive systems to govern the engaging and disengaging of control. These sub processes are important to ensure efficient use of mental resources in circumstances requiring cognitive control.

According to the conflict monitoring hypothesis, a theoretical model developed by Botvinick et al. (2001, 2004), cognitive control requires two sub processes in order to successfully govern behavior—an evaluative process and a regulative process. The evaluative process detects conflict and determines when control should be activated, while the regulative process is responsible for the compensatory actions in response to conflict that lead ultimately to changes in behavior. Before describing the evaluative processes associated with conflict monitoring, it is important to discuss the current understanding of how decisions are made in the brain.

**Accumulation Models of Decision Making**

In a noisy environment, information processing theory indicates that the best solution to determine which of two alternative signals is present is to continuously sample the environment, accumulating evidence to support either outcome. This process continues until a criterion level of evidence is accumulated. Once the criterion of evidence is met for one of the alternatives, a choice is made. Many models centering on this concept have been developed (Laming, 1968; Ratcliff and Router, 1998; Usher and McClelland, 2001; Bogacz et al., 2006).

Random walk models of decision making explicitly relate reaction time and errors (Laming, 1968). In this way, these models can account for the speed accuracy tradeoff.
Fast responses show a higher incidence of error, while slower responses lead to improved accuracy. An excessively low (liberal) criterion will lead to fast, erroneous answers based on incomplete evidence accumulation, while an inappropriately high (conservative) criterion will require too much time to accumulate sufficient evidence to elicit a timely response. Typically responses will fall between these two extremes, depending on the criterion level chosen by the participant.

Single unit recording evidence in non human primates has shown that both parietal (Gold and Shadlen, 2002) and medial frontal (Stuphorn et al., 2000) neurons display activation patterns consistent with the accumulation of evidence in decision tasks involving sensorimotor tasks. Gold and Shadlen (2002) reviewed evidence suggesting that the firing rates of stimulus-specific neurons in areas like MT, which is specialized for motion processing, can be used as a measure of the accumulation of evidence for the presence of a particular moving stimulus. A higher firing rate means more accumulated evidence. In addition, a criterion level of neuronal activation is required prior to a response.

Experiments employing an anti-saccade task find similar results for the accumulation of evidence eliciting motor responses (Stuphorn et al., 2000). In this task, monkeys had to inhibit a previously cued saccade upon the presentation of a stop signal. Short intervals between the cue and stop signal coincided with increased ability to stop the eye movement. Stuphorn et al. (2000) found that in a Stop-signal task, activation of gaze-shifting or gaze holding neurons in frontal eye fields (FEF) strongly correlated with the accuracy of a given trial.
When gaze-shifting neurons showed low activation prior to a stop signal, the animals were better able to stop a planned eye movement. However, if gaze-shifting neurons were given adequate time to increase their activation prior to a stop signal, there was a lower probability of successful inhibition of the eye movement, regardless of the activation of gaze-holding neurons in FEF. These results suggest that decisions in the brain are made when the collective activity of a group of certain neurons surpasses a criterion level of activation. Furthermore, it is likely that cognitive control can be employed to influence the processing of one alternative relative to others. This top-down influence on decision making at the neuronal level will be illustrated further in the discussion of the biased competition model of attention allocation described below.

One limitation of this criterion based decision making process is the susceptibility for suboptimal performance when decisions must be made prior to sufficient evidence accumulation. Fast responses are likely to be initiated before sensory evidence reaches a sufficient criterion level for optimal performance. Thus, these fast responses are more likely to lead to errors. In contrast, slower responses allow for additional signal processing, often leading to increased accuracy of response. These theoretical relationships are the essence of the readily observed behavioral speed-accuracy tradeoff (Rabbitt, 1966).

**The Gratton Effect**

One well known instance of the susceptibility of decision making to rapid responses, called the “Gratton Effect”, occurs in the Eriksen flanker task (Eriksen and
Eriksen, 1974). This behavioral effect illustrates how rapid processing in the brain can be overly influenced by inappropriate stimuli when time pressure is increased dramatically (Gratton et al., 1988).

In the Eriksen flanker task (Eriksen and Eriksen, 1974), a central target (e.g., “<”) associated with a specific motor response is surrounded by flankers that either correspond to the target (compatible trials: < < < < < ) or correspond to the alternative response choice (incompatible trials: > > < > > ). Incompatible trials typically produce response conflict, since processing of the flanker stimuli activates the alternative (i.e., incorrect) response to the target stimuli.

The need to resolve the conflict between the simultaneously active response representations of incompatible flanker stimuli requires cognitive control to be employed. As a result of activating these control processes, an increase in mean reaction time (RT) for accurate incompatible stimulus responses is seen. Compatible flanker stimuli, where the flankers and the central stimulus are identical, have expectedly faster mean RTs and higher accuracies than do incompatible stimuli. Results reported by Gratton et al. (1988) suggest the cognitive system relies on strategic integration of rapid parallel and slower focused processing to determine the identity of the central stimulus in a flanker task. The reliance on these separate yet related cognitive processes produces behavioral differences in RT and accuracy between compatible and incompatible flanker stimuli.

When the distribution of RTs for compatible stimuli are segmented into bins, accuracy improves from chance level at the fastest RTs to above 90% for the slowest RTs. This relationship is characterized by the speed-accuracy tradeoff described by
Ratcliff (1985) and Wickelgren (1977) and governed by the accumulation model of decision making. Incompatible flanker stimuli, however, exhibit a different relationship between RT and accuracy. While the fastest responses are at chance level performance, moderately fast responses are at a significantly below-chance level of accuracy. Slow responses to incompatible stimuli show equivalent accuracy to compatible trials, suggesting full and accurate processing of the central target of the flanker stimulus. Figure 2 illustrates the relationships.

![Figure 2: Flanker task accuracy by reaction time](Image source: Gratton et al., 1988)

The dip below chance level performance shows that responses to incompatible stimuli that are slow enough to allow some level of stimulus processing, but not slow enough to allow adequate processing, are overly influenced by the incompatible flankers. Gratton et al. (1988) suggest that the observed relationship between RT and accuracy for
incompatible flanker stimulus processing is due to the presence of at least two phases of stimulus evaluation: an early stage that processes all the elements of the stimulus—both the flankers and the central target—followed by a later stage that selectively processes the central stimulus as per task requirements.

Gratton et al. (1988) theorized that the activation of cognitive control processes, which occur approximately 200ms after stimulus onset in their task, determine if sensory input from the flankers will be filtered. The time course of these control processes will become important when single cell and ERP evidence of conflict monitoring is discussed below.

**Compatibility Sequence Effects**

Subsequent work by Gratton et al. (1992) illustrates how the cognitive control system responsible for filtering the rapid parallel processing of the entire flanker stimulus is sensitive to contextual cues about stimulus compatibility. In this second experimental paradigm, trials were segmented based not only on the type of current stimulus (compatible or incompatible) but also based on the type of immediately prior stimulus (prior compatible or prior incompatible). Thus, there were four types of trials in the experiment; compatible stimuli preceded by compatible stimuli (Cc), compatible stimuli preceded by incompatible stimuli (Ic), incompatible stimuli preceded by compatible stimuli (Ci), and incompatible stimuli preceded by incompatible stimuli (Ii). Figure 3 illustrates the four combinations of prior and current stimuli.
As expected, Gratton et al. (1992) found a main effect for flanker compatibility, with incompatible stimuli eliciting longer mean RTs and increased error rates. However, the identity of the preceding stimulus was shown to influence the processing of the target stimulus. For compatible target stimuli, processing was facilitated in the Cc sequence. This is seen in the reduction of RT as compared to the Ic sequence. In contrast, incompatible stimuli showed a performance cost in the Ci sequence as compared to the Ii sequence. In the Ci sequence, both RT and error rate were increased. Figure 4 illustrates these findings.
Prior trial exposure to either compatible or incompatible flankers determines whether the cognitive system is biased towards either early or later processing of flanker stimuli (Gratton et al., 1992). If the previous stimulus condition was compatible, then participants expect the current stimulus to be compatible. With an expectation of compatibility, there is no reason to exert control to exclude the input from the early processing phase. However, if an incompatible stimulus is expected, incorporating the output of the early processing phase will lead to an inappropriately large contribution of the flankers to the central stimulus identity task. In the case of an incompatible stimulus expectation, cognitive control will be recruited to minimize the influence of the conflicting flankers on target identification.

If an expectation of compatible stimuli has been established, the subsequent processing of a compatible stimulus is enhanced. However, violation of this expectation...
leads to a large cost in RT for correct responses and accuracy. These costs can be seen when comparing the Cc to Ci RT and error rates in figure 4. Conversely, when an expectation of incompatible stimuli has been established, both the facilitation of similar stimuli and the costs for a violation of expectation are reduced. This relationship can been seen when comparing Ic to Ii results in figure 4.

These behavioral findings suggest that cognitive control processes are sensitive to both the current incidence of conflict, as well as the larger dynamic (i.e., trial-to-trial) context of conflict within the environment. The differentiation between within trial and trial-to-trial conflict processing is an important and under-described aspect of current theories of cognitive control in the brain (see Egner, 2007 and Scerif et al., 2006 for notable exceptions).

Recent models of cognitive control have begun to combine within and between trial conflict monitoring processes. The compatibility sequence effects described above have greatly influenced a Bayesian computational model created to explain behavioral consequences seen when subjects expect compatible or incompatible stimuli (Yu et al., 2009). This model characterizes how prior exposure to either compatible or incompatible flanker stimuli might be incorporated into probability estimates of upcoming stimuli and the selection of an appropriate processing strategy.

**Bayesian Model of Flanker Processing**

As described above, the cognitive control necessary for optimal performance of the Eriksen flanker task requires participants to determine when early stimulus processing
is advantageous to central target discrimination. The role of bias in processing flanker stimuli as described by Gratton et al. (1992) suggests that prior evidence is used as part of this determination. A recent computational model (Yu et al., 2009) explicitly describes the role that compatibility bias plays in optimal performance of the flanker task.

This model uses a Bayesian framework to approximate the neural mechanisms of flanker stimulus processing. Briefly, Bayesian methods postulate that current (revised) belief is a product of prior belief, as specified by prior probability, with the likelihood ratio of the information provided by the current event, leading to a posterior probability. Consequently, a prior belief about a state—in this case the compatibility of the central target and flankers—is modified by information input into the system over time. Modifying the prior belief with the observed evidence leads to an adjustment of expectations—the posterior probability or revised belief. Depending on the strength of the observed evidence, the prior belief is either intensified or weakened.

The Yu et al. (2009) model consists of calculations that process a compatibility bias for upcoming flanker stimuli. Compatibility bias is implemented by changing the prior expectation for compatibility to a value greater than 0.5 when compatible stimuli are anticipated. This bias creates a top-down cue to cooperatively integrate the early flanker processing on trials where a compatible stimulus is expected. While not stated explicitly in the model description, this top-down cue modulates attentional allocation among the target and flankers. However, if the system is biased to expect an incompatible stimulus, then the flanker-related processing should be excluded from processing during central target identification.
In the Yu model, manipulations of the parameters related to compatibility bias are shown to produce behavioral results similar to those observed by Gratton et al. (1988, 1992), including both the early response accuracy cost for incompatible stimuli and the context effects due to previous exposure to compatible or incompatible stimuli.

The importance of the model proposed by Yu et al. (2009) is that using a relatively straightforward Bayesian framework, they create a computational neural model that matches the output seen in behavioral studies. The model shows that depending on the degree of bias in the system, the same current trial input can lead to different conclusions. Previous experience is integrated in an effort to optimize task performance and minimize the cognitive control needed on any given trial. This optimization reduces the mental costs associated with cognitive control.

This Bayesian model bears a strong resemblance to conflict monitoring theory (Botvinick et al., 2001, 2004) which will be described in detail in the following section. In fact, Yu et al. (2009) suggest that conflict monitoring theory is a viable approximation of their ideal Bayesian system. The limited neuronal resources in the brain make a full instantiation of the Bayesian model, which entails extensive computations, unlikely.

Full Bayesian computation requires simultaneous processing of all possible outcomes. In the natural environment, the processing of all possible outcomes based on the vast number of available stimuli would quickly lead to an exponential increase in the calculations necessary to arrive at posterior probabilities. Instead of these intensive calculations, the brain takes heuristic “shortcuts” in an effort to conserve resources. Consistent with other theoretical approaches to decision making, such as that of Tversky
and Kahneman (1974), Yu’s model suggests that the brain might use conflict monitoring in the place of the full Bayesian calculations of compatibility bias. This heuristic approximation would limit the calculations necessary while preserving a sensitivity to the information gained via the processing of prior stimuli.

**Conflict Monitoring Theory**

The importance of monitoring conflict during information processing is related to what conflict represents at a cognitive level. In the broadest terms, conflict occurs when multiple representations are active at the same time, and the brain is forced to choose among them in order to respond coherently and in a goal-directed manner. While conflict may be processed prior to selecting a strategy or response, conflict also informs decision making and strategy implementation following a specific task or trial outcome.

Since conflict is aversive, in that it requires mental effort to adequately monitor and address, it registers as a cost (Botvinick, 2007). The changes in strategy that follow the detection of representation conflict can take the form of a speed accuracy trade off in the case of errors (Rabbitt, 1966; Ratcliff, 1985) or a slowing of response after the experience of conflict even without a corresponding error in performance (Gratton et al., 1992). The mechanism responsible for these changes is attention allocation.

**Theoretical Basis of Conflict Monitoring**

Botvinick et al. (2001) theorize that conflict monitoring is the first of a two-step process that leads to cognitive control. When the brain detects conflict, a circuit is activated that leads to compensatory behaviors that seek to limit the negative influence of
conflict on performance. In effect, the detection of conflict is the event that activates the cognitive control system. Once activated, compensatory strategies that modulate attention allocation in response to the processed conflict can be implemented. This network of monitoring and compensating has been shown to include the medial frontal cortex (MFC) and the dorsal lateral prefrontal cortex (dLPFC) respectively (Kerns et al., 2004; Kerns, 2006; MacDonald et al., 2000; Cohen and Ranganath, 2007).

Evidence has shown that brain areas associated with conflict monitoring are more active during trials that require cognitive control (Braver et al., 2001). In go/nogo paradigms where “go” stimuli are more probable, nogo stimuli are thought to elicit conflict due to the need to overcome a prepotent go response bias. In the Eriksen flanker task, incompatible stimuli elicit conflict due to the simultaneous activation of representations associated with both the target and flankers. As described in the Yu et al. (2009) model above, the cognitive system is generally biased towards compatible stimuli given the abundance of coherent stimuli in the natural world. Thus, encountering a conflict-inducing stimulus leads to additional processing and, ultimately, behavioral costs.

**Neural Basis of Conflict Monitoring**

The brain regions implicated in conflict monitoring within the MFC encompasses the border between the anterior cingulate cortex (ACC) and the pre supplementary motor area (preSMA) (see figure 1). The brain regions responsible for the compensatory allocation of attentional resources to resolve conflict are localized to the dLPFC as well as the parietal cortex (MacDonald et al., 2000; Carter et al., 2000; Cohen et al., 2004).
However, evidence of functional specificity within the dlPFC has varied by task. Results have shown only left dlPFC activation (MacDonald et al., 2000), bilateral dlPFC activation (Badre and Wagner, 2004), or hemispheric localization (Stephan et al., 2003) depending on the experimental paradigm employed.

One difficulty in ascribing conflict monitoring roles to each of the ACC and preSMA is the ill-defined border between these two sub regions of MFC. Neuroimaging studies have provided sometimes contradictory evidence of activation in the presence of conflict. The difficulties in developing a unified theory of ACC function is described elsewhere (Bush et al., 2000; Milham, 2005). Additionally, neuronal populations in the MFC exhibit great heterogeneity of response sensitivity (Bush et al., 2002; Ito et al., 2003; Matsumoto et al., 2007; Nishijo, 1997; Stuphorn et al., 2000). Given these difficulties, one way to discern the functional significance of a brain region is to approach the problem hodologically, looking at the connections the MFC shares with other brain regions.

**Anterior Cingulate Cortex**

The ACC has projections to both the motor cortex and the dlPFC (Paus, 2001). This suggests a role for the ACC in translating intention into action. In addition, the dorsal ACC shares connections with temporal brain regions associated with perceptual processing (Van Hoesen et al., 1993). More rostral areas of the ACC are interconnected with the thalamus and brainstem, suggesting an ACC role in affective processing and autonomic arousal (Bush et al., 2000; Critchley et al., 2005).
In addition to the neuroanatomical evidence of an ACC role in conflict monitoring, neuroimaging studies in humans and single unit recording of non-human primates have also provided evidence linking ACC activation to conflict monitoring processing. Carter et al. (1998) showed that dorsal ACC activity increased when participants were presented response conflict inducing stimuli, regardless of the accuracy of the eventual response.

Research has tied the activity in the ACC associated with response conflict directly to changes in behavior such as those described by Gratton et al. (1988). Kerns et al. (2004), Kerns (2006) and Cohen et al. (2007) have both shown ACC activity is related to shifts in behavior on subsequent trials. These context related activations support the Yu et al. (2009) model and are consistent with a role of the MFC in a probability matching function and implicate the ACC in the behavioral compatibility sequence effects described above.

This imaging work shows that ACC is sensitive to conflict, and ACC activation is a precursor to subsequent changes in behavior. However, the human imaging work suffers from a lack of temporal resolution. Using these results, it is difficult to determine if the ACC activation occurs prior to or following response activation. Single cell work (Ito et al., 2003, Stuphorn et al., 2000; Stuphorn and Schall, 2006) shows conflict sensitive neurons are active following motor response.

One complicating factor in the acceptance of conflict monitoring theory is the overlap in cortical activation for situations of outcome evaluation, error commission, response conflict and decision uncertainty (see Ridderinkhof et al., 2004 for a meta
analysis). The location of activation during conflict monitoring tasks (Eriksen flanker task, Stroop color word naming, go/no go response) often straddles the boundary between the ACC and the overlying preSMA. Since each of these circumstances has been shown to elicit activation in similar MFC regions, some extension of the conflict monitoring framework is likely necessary to include all results.

Other recent theories have ascribed strategic, anticipatory functions to the ACC. Separate groups have shown increased ACC activation in anticipation of the need for cognitive control (Roelfs et al., 2006, Aarts et al., 2008, Luks et al., 2007) as well as preceding conditions associated with a higher likelihood of error commission (Brown and Braver, 2005, 2007). In addition, an ACC role in processing costs and benefits of a specific action have been shown in animal studies (Walton et al., 2003, 2006; Kennerley et al., 2006, 2009) and theorized in humans (Botvinick 2007).

These ACC activations with more strategic processing suggest more than simply motor-related conflict processing. As outlined in the Bayesian conflict monitoring model of Yu et al. (2009), perhaps predictive bias modifications provide a link between trial-to-trial conflict monitoring and higher level effort and predictive probability calculations. It remains to be seen if the preSMA and ACC differentially contribute to processing of motor conflict and non motor conflict, respectively.

Given the overlaps in ACC and preSMA activation discussed in the previous functional magnetic resonance imaging (fMRI) evidence, the use of more temporally sensitive methods is warranted to determine the contributions of each brain region to cognitive control. While temporally-sensitive methods like event-related potentials
(ERPs) do not provide the same spatial resolution as fMRI, a large literature exists on the role of frontal ERP components—specifically the medial frontal N2—in conflict monitoring and cognitive control. Together with the previously discussed fMRI evidence that describes where activation occurs within the MFC, these ERP findings help determine when MFC activation occurs during information processing.

*ERP Evidence of MFC Conflict Processing*

A large literature exists on the significance of medial frontal N2 modulations in tasks requiring cognitive control. The medial frontal N2 (figure 5) is an event related potential (ERP) component with a frontal central scalp distribution and maximum negative amplitude between 200-400ms post stimulus onset. For brevity, the medial frontal N2 will be referred to as the N2 in the following discussion.

Figure 5. Medial frontal N2 scalp topography and waveform at FCZ
While much of the behavioral and imaging evidence discussed above centers on flanker tasks, it is important to examine the go/nogo literature as well. Briefly, go/nogo tasks consist of two stimuli, A and B. One stimulus is identified as the “go” stimulus, requiring a motor response from the subject when it is presented. The other stimulus is thus the nogo stimulus; a response must be withheld to this stimulus. Stimuli are presented in rapid succession, and go stimuli are presented at much higher probabilities than nogo stimuli. This establishes a prepotent response bias that must be overcome when rare nogo stimuli are presented.

Gomez et al. (2007) provide a detailed examination of behavioral data which shows that the discrimination between go and nogo responses can be explained using the general accumulation models of decision making described previously. The withholding of an overt motor response to a nogo stimulus is still an implicit choice at an implicit decision boundary. Similarity between the cognitive processes required for a wide range of tasks involving cognitive control is supported by both imaging (Braver et al., 2001) and ERP studies (Nieuwenhuis et al., 2003).

Jodo and Kayama (1992) showed that when response time pressure was increased by shortening the deadline by which participants were required to respond to go stimuli in a go/nogo task, the amplitude of the N2 to nogo stimuli increased significantly. These findings are interpreted as evidence for an inhibitory process reflected by the magnitude of N2, which is enhanced when the demands for control are increased. Time pressure to respond forces a decision prior to full processing. Similar to the explanation put forth in an Eriksen flanker study by Gratton et al. (1988) discussed above, the introduction of
time pressure forces the brain to incur costs—in the form of inhibitory control—in an effort to avoid making a prepotent response.

In go/nogo tasks, the N2 has also been shown to persist when overt responses are not required of subjects (Pfeferbaum et al., 1985; Bruin and Wijers, 2002). In visual tasks requiring a button press upon presentation of a go stimulus, nogo stimuli invoked increased N2 amplitude 200-400ms post stimulus onset. In addition, when the same go and nogo stimuli were used, but subjects were required to count the number of go stimuli within a block instead of pressing a button upon go stimulus presentation, N2 amplitude was also increased for nogo stimulus presentation.

This suggests that modulations of N2 do not require an overt motor response, nor are the N2s elicited in response tasks solely a reflection of motor processes. The presentation of nogo stimuli in either an overt response or counting task lead to similar N2 increases as compared to go stimulus presentation. The modulation of N2 during counting tasks will be an important component of the second experiment described in chapter 3.

Low probability stimuli have been shown to elicit increased N2 in go/nogo tasks. Multiple studies (Bruin and Wijers, 2002; Eimer, 1993; Nieuwenhuis et al., 2003) have shown that decreased stimulus probability leads to an increased requirement for cognitive control, reflected in enhanced N2 amplitude. Much of this work has focused on manipulations in the frequency of nogo stimulus presentation (Bruin and Wijers, 2002; Eimer, 1993). In these studies, the enhanced N2 in the presence of a rare nogo stimulus
is thought to reflect the increase in control needed to overcome the prepotent go response that is facilitated by the high probability of go stimuli in the task.

Nieuwenhuis et al. (2003) observed probability-based N2 modulations of go stimuli as well. Modulation of N2 with rare nogo stimuli is consistent with an inhibition process; however, the modulation of N2 with rare go stimuli observed by Nieuwenhuis et al. (2003) is seen as support for a conflict monitoring process instead. Since the rare go stimulus elicits an enhanced N2 as compared to the frequent nogo stimulus, inhibition is insufficient to fully explain the underlying sensitivity.

Nieuwenhuis et al. (2003) interpret their findings as support for a conflict monitoring explanation for the cognitive processes underlying N2. They reason that an inhibition explanation would not predict any N2 modulations associated with the go stimulus. Since the go stimulus requires a response, there is no motor inhibition involved in processing. Further, the fact that rare go stimuli elicit a slightly larger N2 suggests that the probability of stimulus presentation influences the underlying cognitive processes.

This explanation is also supported by intracortical source localization results showing that the dipole responsible for the N2 to a rare go or nogo stimulus was located in the vicinity of the ACC within the MFC. While source localization alone is insufficient to determine a neural generator for an ERP component, this interpretation is directionally consistent with imaging evidence of increased ACC activation in a variety of tasks requiring cognitive control (Braver et al., 2001).

Additional evidence supporting a conflict monitoring origin for the medial frontal N2 is provided by the so called go/GO task (Donkers and van Boxtel, 2004). In this task,
subjects were presented with a typical go stimulus that required a normal motor response. In addition, they were occasionally presented with a separate GO stimulus that required a maximal force response. The differences in response strength between go and GO stimuli were quantified using displacement dynamosimeters, essentially squeeze balls, instead of a discrete button press. This allowed collection of an analog measure of response force magnitude and the ability to separate go from GO responses.

When subjects completed both a standard go/nogo task as well as a go/GO task, Donkers and van Boxtel (2004) found that both the nogo and GO stimuli elicited increased N2 when compared to the standard go (i.e. normal motor response) stimulus. In addition, there were no significant differences in N2 amplitude between the nogo and GO stimuli. This suggests that the key process in eliciting N2 relates to the choice between response alternatives, not to the inhibition of all motor activity upon nogo stimulus presentation.

These results are in line with the findings from counting go/nogo tasks discussed above (Pfefferbaum et al., 1985; Bruin and Weijers, 2002), as well as the theoretical explanation of go/nogo task information processing (Gomez et al., 2007) consistent with accumulation models of decision making. Together, this suggests that motor inhibition is an insufficient explanation of the cognitive processes indexed by N2.

In addition to evidence from go/nogo tasks, Eriksen flanker tasks have been shown to consistently elicit N2 modulations associated with conditions requiring cognitive control. As discussed above, Eriksen flanker tasks require cognitive control to
select between competing responses which are activated by the processing of both the flankers and central target stimuli.

Early work by Kopp et al., (1996) showed that in the presence of incompatible Eriksen flanker stimuli (i.e., < < > < < ), N2 was enhanced as compared to compatible (i.e., > > > > > ) or neutral (i.e., * * > * * ) flanker stimuli. This suggests that N2 indexes the processing of incompatibility between the flankers and the central target. Additional support for this interpretation is provided by the finding that reduction of the space between incompatible flankers and the central target leads to an enhanced N2. This is in line with the original Eriksen and Eriksen (1974) behavioral findings showing increased flanker effects on reaction time with crowded stimuli.

Other influential theories suggest that the processes underlying N2 are a reflection of conflict monitoring that occurs prior to a task response. They draw a connection between N2 and the error related negativity (ERN), a frontal midline ERP component associated with error processing that peaks within 100ms of an erroneous response (Gehring et al., 1990; Falkenstein et al., 1990).

Van Veen and Carter (2002) argue that impulsive, fast response errors are due to incomplete processing. Within the accumulation model of decision making described above, these errors lead to responses prior to threshold levels of activation of response alternatives being reached. In this case, the activation of conflict monitoring processes does not occur prior to response, but the continued processing of stimuli leads to sufficient activation immediately after an overt response, generating the ERN.
In contrast, when accurate responses to incompatible flanker stimuli are made, any conflict that arises during information processing must be adequately dealt with prior to an overt response. Van Veen and Carter (2002) argue that this pre-response conflict monitoring is reflected in the N2. The increase in N2 when flanker stimuli are incompatible (Kopp et al., 1996) reflects conflict monitoring that occurs early enough in time to be adequately processed. This adequate processing is theorized to allow conflict monitoring to activate compensatory processes and thus exert an influence on the response selection stage. This influence leads to an accurate, though delayed, response.

The predictions put forth by Van Veen and Carter (2002) have been formalized in a computational model by Yeung et al. (2004). This model is based on the same assumption, that ERN and N2 share a neural generator and index the same response conflict monitoring process. Conflict monitoring prior to a response is indexed by N2, while conflict monitoring following a response is indexed by the ERN.

This conflict monitoring view of N2 provides temporally sensitive evidence explaining why imaging results (Carter et al., 1998; Botvinick et al., 1999) have shown enhanced MFC activation for accurate responses to incompatible flanker stimuli. The argument is that sufficient conflict monitoring prior to response selection allows for identification of conflict and recruitment of compensatory behaviors to mitigate the influence of stimulus components leading to errors.

The computational model described by Yeung et al. (2004) is supported by empirical ERP findings. N2 is enhanced on correct incompatible flanker trials as compared to correct compatible trials and incorrect trials. Furthermore, source
localization shows a shared neural generator for both N2 and the ERN. The similarity in neural generators between N2 and ERN suggest that a common cognitive process is responsible for both ERP components. The Yeung et al. interpretation is attractive, in part because it fits squarely with the behavioral evidence found by Gratton et al. (1988, 1992) as well as early imaging work (Botvinick et al., 1999; Carter et al., 1998) showing that correct response trials that include response conflict lead to adaptations on subsequent trials.

The evidence described above suggests that conflict monitoring accounts do a good job of explaining the cognitive processes indexed by N2. Yet, in much the same way that Bush et al. (2000) describe the difficulty in arriving at a unified theory of ACC function, Folstein and van Petten (2008) describe the difficulty in unequivocally attributing N2 signals to either conflict monitoring or processing of novelty and mismatches between attended and expected stimuli.

As will be discussed below, stimulus uncertainty results showing increased N2 with increased target and non-target similarity refute theoretical explanations that center on perceptual mismatch or novelty as the cognitive processes underlying the N2 component. Nonetheless, the differences between motor and non-motor conflict are ripe for further exploration.

**Difficulties in Functional Separation within the MFC**

One outstanding issue in the current theoretical understanding of conflict monitoring is the inconsistent evidence for non-motor conflict activation of the medial frontal cortex. To date, the empirical description of Botvinick’s conflict monitoring
model has focused on response conflict. Neuroanatomical evidence suggests that the proximity and close links between the ACC and motor cortex (Paus, 2001, Nachev et al., 2008) mean the ACC is involved primarily with the detection of motor conflict.

In fact, the current model of conflict monitoring in the Eriksen flanker task (Botvinick et al., 2004) requires the simultaneous activation of multiple representations at the response level in order to activate the conflict monitoring module. Figure 6 illustrates this model for an Eriksen flanker task.

If both responses are simultaneously and sufficiently activated in the Response module, the Conflict Monitoring module is activated and subsequently influences the Attention module to bias processing among the three stimulus locations (left, central, right). The Botvinick model focuses primarily on the activation of the Conflict Monitoring module, leaving the mechanism of attentional bias used to resolve dual activation within the Response module unaddressed. The Yeung et al. (2004) theoretical explanation suggests that the influence of the Conflict Monitoring module upon the Attention module can occur within the same trial, while single cell evidence in monkeys (Stuphorn et al., 2000; Ito et al., 2003) suggests that the influence is first exerted on the subsequent trial. The influence of medial frontal activation on subsequent trials is also supported by human neuroimaging evidence (Kerns et al., 2004; Cohen and Ranganath, 2007).
Figure 6: Conflict monitoring theory.
The Botvinick model consists of four modules Input, Response, Conflict Monitoring, and Attention. Compatible stimuli are HHH or SSS and incompatible stimuli are SHS or HSH. In this model, activations of stimulus identity (H or S) at each of three stimulus locations (left, center, right) within the Input module lead to an activation of the appropriate response (H or S) in the Response module.
Image source: Botvinick et al. (2004)

There is a growing body of literature that suggests this current model requires amendment in order to accurately represent the processing of other forms of conflict. In addition to the N2 modulations seen in counting tasks, empirical evidence has shown that both higher order decision conflict and lower-order perceptual conflict influence the brain regions associated with conflict monitoring. These findings suggest that motor responses are not necessary for activation of the conflict monitoring circuit.

Multiple Levels of Conflict Processing

Evidence of non-motor conflict monitoring in the brain can be found in studies that elicit representational conflict at different levels within the cognitive processing hierarchy. As stated above, early work in the field leaves open the possibility than non-
motor representations might be processed by the same MFC brain regions (Botvinick et al., 2001; Carter et al., 1998; Carter et al., 1999). Recent evidence has shown that both the MFC and the dlPFC show differential activation depending on the source of conflict processed (Badre and Wagner, 2004; Nelson et al., 2003; Van Veen and Carter, 2005).

Taken together, this evidence suggests a more general conflict monitoring system, sensitive to more than response conflict.

Starting broadly, there is a large body of evidence showing that tasks involving different types of conflict elicit activations in the MFC and specifically the ACC. In an overt verb generation task, increased ACC activation was seen when verbs were generated for nouns without a prepotent verb association (Barch et al., 2000): For example “bell”, which could be associated with “hear” or “ring” versus “car” which would normally be associated more strongly with “drive.”

Additional evidence of ACC activation associated with decision conflict in addition to response conflict has been found. When the attractiveness of faces was judged, similarly normed face pairs elicited higher levels of ACC activity (Pochon et al., 2008). When subjects read narratives that did not form an integrated whole, increased ACC activity was also observed (Robertson et al., 2000). Decisions with ambiguous moral judgments have been shown to elicit increased activation in ACC (Greene et al., 2004). Taken together, these findings suggest an ACC role in decision processes prior to response selection in the information processing cascade. In each of the studies described, the emphasis of the task was on a complex decision, not on the overt physical response at a deadline.
Other evidence, however, has suggested that different types of conflict are processed in different brain regions. While response conflict is processed in the ACC, conflict elicited by the familiarity of a stimulus used in a different context is instead processed in the inferior frontal gyrus (IFG) (Nelson et al., 2003). In this task, subjects were presented with the same letter stimulus in different contexts, eliciting conflict not in the ACC, but in more lateral prefrontal brain regions. This role for the IFG is supported by other work suggesting this region plays a role in mediating semantic conflict (Thompson-Schill et al., 1999; Novick et al., 2005). In this interpretation, there is no support for the ACC processing conflict that arises earlier in the information processing stream than response selection.

Focusing on more controlled laboratory tasks, Braver et al. (2001) showed that ACC activation was elicited by infrequent responses regardless of the task paradigm. The fact that ACC activation was seen in various tasks that required different cognitive processes for successful performance suggests that the underlying conflict monitor is general, and activated primarily by the frequency of the required response.

*Stimulus Uncertainty*

While evidence of MFC activation with decision uncertainty strongly suggests that the ACC plays a role in processing conflict at abstract levels of representation, the findings when stimulus uncertainty is manipulated are less clear. It appears that in order to elicit ACC activation from stimulus uncertainty, tasks requiring discriminations of highly similar stimuli must be used. Evidence showing a lack of ACC sensitivity to non-response conflict focuses on tasks where the similarity among competing stimuli is
arbitrarily manipulated. In studies where a more psychophysical approach is taken to manipulate perceptual similarity, the results suggest non-response conflict may indeed be monitored by the ACC.

A number of studies have shown that the ACC is exclusively sensitive to response-related conflict. In a pair of studies that used fMRI (Van Veen et al., 2001) and ERP (Van Veen and Carter, 2002) methods, results indicated that the ACC responds only to response conflict. The task manipulated conflict at both the stimulus identification level and the response level by mapping different stimulus elements to either the same or different response fingers.

Central targets of “H” and “S” were mapped to the same response hand, while central targets of “T” were mapped to the opposite hand. From these response sets, three types of Eriksen flanker stimuli were created; compatible stimuli where flankers and central targets were identical (HHH, SSS, or TTT); stimuli where the flankers and the central target were mapped to responses from different hands (THT, STS, etc) which created response conflict; and stimuli were the flankers and the central target responses were mapped to the same hand but were perceptually distinct (HSH, SHS) which created stimulus identification conflict but did not create response conflict.

When only response conflict was manipulated (i.e., THT, STS, etc) both the fMRI (Van Veen et al., 2001) and ERP (Van Veen and Carter, 2002) studies showed increased activation. fMRI results showed brain activation with response conflict was localized to the ACC. ERP results showed that N2 was enhanced in the response conflict condition as compared to both the compatible and stimulus identification conflict conditions. Source
localization of the N2 component suggested it originated from the vicinity of the ACC. In contrast to these response conflict findings, when stimulus identification conflict alone was manipulated (i.e., HSH or SHS), no ACC activation was elicited in the fMRI (Van Veen et al, 2001) and no N2 modulation due to flanker incompatibility was seen (Van Veen and Carter, 2002). These results suggest that ACC contributes only to response-conflict monitoring, and is insensitive to stimulus identification conflict.

Van Veen and Carter (2002) interpret the lack of frontal central N2 modulation in the stimulus incompatibility condition as evidence that the ACC is only involved in conflict monitoring at the response selection stage or later. However, as the evidence below points out, psychophysically derived, just noticeable differences between pairs of visual or auditory stimuli have been shown to elicit N2 modulations. This suggests that the interpretation that N2 reflects only response conflict monitoring may be incorrect.

Other studies have shown that under certain circumstances, stimulus uncertainty alone is sufficient to elicit ACC activation. Milham et al. (2003) showed that in a Stroop task, infrequent stimuli that created stimulus uncertainty did in fact elicit increased ACC activation in a sub region near Brodmann area 9. This more rostral brain region is physically distant from the overlapping preSMA/ACC region discussed above. In addition, West et al. (2004) found that decreasing the frequency of stimuli that induced stimulus uncertainty in a counting Stroop task lead to an enhanced ERP component with a neural generator in the ACC. These findings, along with the work of Braver et al. (2001), strongly suggest that infrequent presentation is a requirement for MFC activation.
in the presence of non-response conflict. N2 evidence showing a sensitivity to stimulus probability (i.e., Bruin and Weijers, 2002) described above supports this finding.

Nieuwenhuis et al. (2004) found modulation of N2 only for aural overlap between targets and non-targets while increased visual overlap between target and non-target stimuli did not lead to a reduction in N2. The auditory overlap used (the similarity between “F” and “S” sounds) might have approached the level of the just noticeable difference, the overlapping visual stimuli used (the letters “F” and “T”) might still be too dissimilar to elicit perceptual conflict. The inconsistency of the N2 modulation across stimulus modalities underscores the danger of using arbitrary hard/easy discriminations.

A related study (Szmalec et al., 2008) used a just noticeable difference (JND) protocol to tailor the auditory stimuli to each participant individually. They found that increased perceptual overlap lead to similar influences on N2 as those observed by Nieuwenhuis et al. (2004). The requirement of a JND protocol to create increased overlap between targets and non targets suggests that a high degree of perceptual overlap is required in order to elicit stimulus uncertainty-driven changes in conflict monitoring.

Recent studies have focused on conflict elicited by stimulus uncertainty. If both targets and a subset of non-targets are made rare, an interesting decoupling of the N2/P3 complex occurs (Azizian et al., 2006). The largest N2 is elicited by rare non-targets that share perceptual overlap with rare targets when the frequency of both targets and non-targets are equal. This suggests that the conflict elicited by stimulus similarity is an additional component of the N2, since rare non-targets that do not share perceptual overlap with the target elicit equivalent or slightly smaller N2 than do rare targets.
Thus, an open question concerns the conditions under which stimulus uncertainty elicits MFC activation and N2 modulation. Current evidence is inconsistent, with some studies—especially those employing auditory stimulus presentation—showing N2 modulations, while others do not. Potentially, this inconsistency is related to the level of perceptual overlap required to activate the conflict monitoring circuit in the presence of stimulus uncertainty. Depending on how stimulus uncertainty is resolved, activation within the MFC may be related to within trial processing, or instead to higher level contextual processing. This contextual processing may result in probability matching based on the anticipated exposure to uncertainty. Additional conflict processing might occur is at the perceptual level, localized within the visual cortex. An influential theory in this literature, the biased competition model of attentional allocation, will be discussed in the following section.

**Biased Competition Model of Attentional Allocation**

As discussed above, cognitive control processes mitigate the influence of conflict through attentional resource allocation. One influential theory that describes a mechanism for this attentional resource allocation as well as how these attentional resources are allocated is the biased competition model developed by Desimone and Duncan (Desimone and Duncan, 1995, Desimone, 1998, Duncan, 1996).

In this model, competing visual stimuli vie for processing resources in the visual system. Specifically, the competition is for the response of a single neuron tuned to the competing stimuli. This competition among sensory representations is resolved either by
bottom-up sensory properties like the salience of a particular stimulus, or by top-down biasing of the visual system based on feedback from higher cognitive processes located in the frontal and parietal regions (Desimone and Duncan, 1995).

While it has been shown that this competition among sensory representations occurs within V1-V4 (Kastner et al., 2001) many theoretical explanations of these cognitive processes provide a link between these bottom-up competitions and top-down biasing from frontal brain regions. Given the link between MFC and conflict monitoring described above, it is prudent to explore a link between conflict monitoring theory and the biased competition model. It is likely that the cognitive processes responsible for the compensatory attentional allocations in response to the activation of the conflict monitoring system are the same processes described by the biased competition model. The use of stimulus uncertainty evoking stimuli in the current series of experiments provides a stronger link between the top-down frontal monitoring processes and the sensory processes that illustrate compensation. The biased completion model describes a mechanism for resolving sensory conflict.

*Neural Processes Instantiating Perceptual Bias*

Compelling evidence for a bottom-up conflict processing mechanism is the work of Kastner and Beck (for a review see Beck and Kastner, 2008) showing that the suppressive interactions that are the neural signature of competition in the visual cortex are most apparent at the level where sensory competition would occur. For example, with small stimuli, the suppressive interactions are seen in early visual cortical regions like V1, whereas for larger stimulus arrays, the suppressive effects are seen in higher
visual areas like V4. These results are in line with the extensive evidence of functional organization of the visual cortex. V1 neurons have much smaller receptive fields than do V4 neurons.

Thus, it stands to reason that smaller stimuli should elicit conflict earlier along the visual pathway. This suggests that the local competition occurs at the level of processing where the stimuli are competing for the representation of specific neurons. Given the increasing complexity of functional representation from V1 to V4, larger or more abstract stimulus features would be expected to elicit competition at higher levels of visual processing (Kastner and Ungerleider, 2001). Similar patterns have been observed for perceptual decision making in higher level cortical regions such as those responsible for face and house processing (Heekeren et al., 2004).

Another important neural signature of the interaction between top-down and bottom-up processing is the increase in baseline activity in visual cortex following exposure to competing stimuli. In much the same way that the compatibility sequence effects literature (Gratton et al., 1992; Egner et al., 2010) illustrates that trials following exposure to conflict showed enhanced cognitive control and decreased conflicting stimulus processing, the increase in baseline V1 activation when a difficult task is anticipated (Ress et al., 2000) suggests the top-down bias of predictive brain regions does indeed manifest itself in sensory processing areas of the brain.

These sensory activations are also in line with the general premise of the biased competition model. That is, when discriminations among alternatives are easy, little to no bias is required. However, when these discriminations are difficult, cognitive control
in the form of attentional focus is required to discern between alternatives. Expectations of difficult discriminations lead to top-down biasing of attention. Similar results showing increases in early visual ERP components like P1 in anticipation of conflict in Eriksen flanker tasks (Scerif et al., 2006; Correa et al., 2009) have also been found.

One difficulty in using early visual ERP evidence to directly support the biased competition model is that the anticipated changes in P1 and N1 could explain alternative models of attentional allocation as well. Enhanced P1 and N1 amplitudes in attentionally demanding conditions (Hillyard et al., 1998; Luck et al., 2000) do not differentiate between the biased competition model and the spotlight model of attention (Posner et al., 1980; Hillyard et al., 1998).

Thus, early attention ERPs are likely an insufficient tool to provide the evidence necessary to link between biased competition and conflict monitoring. Instead, looking at the time course of frontal activation in stimulus uncertainty and response conflict eliciting conditions may provide evidence in support of top-down modulations consistent with the biased competition model.

Expansion of Biased Competition Model

In an affordance that is similar to those made by Botvinick et al. (2001) for non-response conflict to influence MFC activation, Beck and Kastner (2008) state that while the majority of evidence to date on the top-down biasing of sensory processing has been produced in the realm of spatially directed attention, there is no theoretical reason why other forms of bias—working memory, emotional, effort related—should not show similar effects in terms of top-down modulation of stimulus-driven processing. As was
the case with conflict monitoring, this suggests an expansion of study, using related tasks to illustrate biased competition and top-down influence.

In fact, if targets and distractors are manipulated to be perceived as different (i.e., dissimilar X’s and O’s instead of similar Q’s and O’s), then the competition in visual cortex will be reduced (Beck and Kastner, 2005) due to the increased salience of the target. However, in the Q’s and O’s condition, where the target (Q) is much less salient, a strong top down bias is required to reduce the competition between the target and distracters.

It is important to note that these competitive interactions among targets and distracters in a cluttered visual field are believed to occur locally within the visual cortex (Beck and Kastner, 2005, 2008). As such, there is no requirement for frontal or parietal involvement to resolve these bottom-up competitions. Nonetheless, strong links between dLPFC and posterior cortical areas associated with decision making have been previously established (Heekeren et al., 2006; Rossi et al., 2009).

With increased target/ non-target similarity, more visual competition occurs and the need for top-down regulation increases. The question remains, where do these top-down signals originate? Given the theoretical architecture of conflict monitoring theory discussed above, there is an affordance for this type of visual conflict to be monitored. However, the question then becomes “how does the visual cortex know to resolve the conflict?” That is, what turns on the system?

Conflict related processing in the primate brain appears to occur following a motor response. Given the single cell activations associated with conflict monitoring
(Stuphorn et al., 2000; Ito et al., 2003), it is unlikely that neurons sensitive to the activation of competing representations fire early enough in the information processing cascade to influence visual perception during the same trial. The human ERP evidence suggests that response conflict monitoring may occur pre-response (Yeung et al., 2004; Appelbaum et al., in press), but to date, the majority of studies focused on visual perception have shown that prior response conflict (Scerif et al., 2006) or cues indicative of upcoming conflict (Correa et al., 2009) influence early perceptual processes only on subsequent and not the current trials.

Even if within trial top-down biasing cannot occur within the timescale necessary for visual categorization, the MFC might still be involved via strategic adjustments. A role for the MFC in strategic cognitive control has been suggested from human (Ridderinkhof et al., 2004) and primate (Kennerley et al., 2006) evidence. In these and other cases, MFC activation, and specifically ACC activation, has been associated with anticipatory processing (Luks et al., 2007; Correa et al., 2009; Scerif et al., 2006; Sohn et al., 2007) and learning (Mars et al., 2005; Rushworth et al., 2004). Thus, the biasing may take place at a more global level.

This question is further illuminated by recent work showing that lesions to the dIPFC in monkeys leads to an inability to flexibly reallocate attention as required by task changes. Rossi et al. (2009) have interrupted the top-down biasing signal by lesioning dIPFC. Strong connections both anatomically (Paus, 2001) and functionally (MacDonald et al., 2000; Kerns et al., 2004; Kerns, 2006; Carter et al., 2000) between the ACC and dIPFC, have previously been discussed.
Thus, part of the inability to perseverate reported in dlPFC-lesioned animals (Rossi et al., 2009) may be related to the absence of the top-down biasing signal originating in the MFC and conveyed by the dlPFC to the visual cortex. This evidence, in concert with the work showing that multiple sources of conflict activate regions of MFC and dlPFC, lead to the predictions of this dissertation.

Given the evidence of a relationship between visual cortex and dlPFC during decision making (Heekeren et al., 2006) and in the presence of conflict (Rossi et al., 2009) as well as the connections between dlPFC and MFC in conflict monitoring (MacDonald, 2000, Carter 2000), it is hypothesized that the MFC is responsible for the monitoring signal that recruits the dlPFC to send biasing signals to visual cortex to address conflicts as described by the biased competition model (Desimone and Duncan, 1995, Duncan 1996). If this is the case, it can be expected that the MFC is sensitive not only to response conflict, but also to stimulus uncertainty.

**Summary of Previous Findings**

Cognitive control is essential for performance of any task. However, the question remains, how does control originate and how is it implemented? Decision making theory centers on accumulation of adequate evidence in support of one or more alternatives. Single cell evidence of neurons sensitive to simultaneous activations of cognitive representations corroborates the theoretical model of decision making. Further, this conflict registers locally throughout the brain and impedes rapid, accurate decision making.
Cognitive control seeks to limit the conflict created by simultaneous activation of multiple alternative cognitive representations. In many cases, probabilistic estimations aid these processes and serve as heuristics to limit cognitive costs associated with invoking control. However, when predictions about potential sensory or response conflicts are not accurate, increased input from unwanted sources can result in performance costs.

Among these costs are the compatibility sequence effects caused by expectations of compatibility on a trial-to-trial basis. An invalid expectation of stimulus compatibility leads to a decrease in cognitive control, which increases the sensory input from incompatible flankers and leads to errors. The interplay between expectations and sensory processing has been recently modeled with a Bayesian compatibility bias calculation. This computation model is believed to be approximated in the brain by conflict monitoring in the MFC.

Conflict monitoring is theorized to be the first of a two-step cognitive control process. Conflicting representations activate control, which then mitigates the consequences of conflict through compensatory attentional allocations and resulting behavioral speed-accuracy tradeoffs. While the current literature has localized these conflict monitoring processes to the MFC, the time course in which they occur is still unclear. The current brain imaging literature suggests a wide overlap across both the ACC and preSMA for activations associated with conflict monitoring and error processing.
N2 has been shown to be sensitive to time pressure, stimulus probability and incompatibility. In addition, when ambiguity between alternative stimuli is increased, so is N2 across multiple modalities. These findings, along with source localization work suggesting an N2 source in the MFC, lead to the conclusion that N2 indexes the cognitive processes associated with cognitive control and conflict monitoring.

While conflict monitoring has rightfully assumed a prominent place as a theoretical explanation of cognitive control, questions remain. For example, if conflict monitoring occurs at the level of response representations, and if N2 is a scalp potential associated with these control processes, how can the presence of N2 modulations in the absence of overt responses as well as modulations sensitive to stimulus uncertainty be explained? It remains to be seen if representational conflict elicited early in information processing is resolved locally within the visual cortex, or if the conflict is propagated forward to the response selection stage and indexed by MFC activation.

Current theories of perceptual processing within the visual cortex focus on suppressive interactions between neuronal populations. However, a number of findings discussed above outline a wide-ranging cognitive control system that governs the biasing of these interactions. First, visual areas like V1 have been shown to be sensitive to anticipatory processes. These biases are likely to be instituted through mechanisms similar to those described in the Bayesian conflict model outlined above. In fact, recent evidence has shown that early attentional processes are sensitive to context effects associated with conflict monitoring. Taken together, these results suggest that top-down signals from the MFC might be used to aid in processing stimulus uncertainty.
**Rationale**

The novel experiments described below seek to further define the relationship between conflict monitoring and stimulus uncertainty in the human brain. Conflict monitoring is theorized to occur within a frontal brain circuit centered on the medial frontal cortex, and likely the ACC. Stimulus uncertainty is processed in the visual cortex, in a manner suggested by the biased competition model of visual attention. The question remains as to how these two systems interact to insure optimal performance in the processing of ambiguous visual input in the environment.

As discussed above, there is extensive evidence of a role for the medial frontal cortex in processing conflict that arises in tasks requiring cognitive control. While theoretical descriptions of conflict monitoring allow for the processing of conflict induced by stimulus uncertainty in addition to response conflict, the empirical evidence to date has been either inconsistent or not used well-controlled methods to quantify stimulus uncertainty. It is the aim of these experiments to determine if stimulus uncertainty is processed independently within the conflict monitoring circuit.

A series of three questions will be employed to characterize stimulus uncertainty processing in the conflict monitoring circuit. First, does the medial frontal conflict monitoring circuit differentially process conflict elicited by perceptually similar targets and non-targets in an Eriksen flanker task? If the medial frontal conflict monitoring circuit participates in within-trial processing of stimulus uncertainty, then it would be expected that perceptually similar stimuli would elicit an increased conflict monitoring response.
Second, is processing of stimulus uncertainty independent of response conflict processing? Prior evidence of N2 modulations in the absence of overt motor responses have employed stimuli that instantiate response conflict. Given the single cell and human imaging evidence of a dissociation between preSMA and ACC activation, it is predicted that stimulus uncertainty should elicit N2 modulations in the absence of an overt motor response requirement.

Finally, is the Bayesian compatibility bias calculation theoretically approximated by the conflict monitoring system sensitive to stimulus uncertainty in an Eriksen flanker task? Given the evidence that stimulus uncertainty is resolved locally in the visual cortex, it is possible that the elicited perceptual conflict is not processed by the MFC conflict monitoring circuit. However, this locally-resolved stimulus information may interact with top-down bias signals, and would thus require an updating of the compatibility expectations on subsequent trials. If compatibility bias is influenced by stimulus uncertainty in the flanker task, it would be expected that compatible, but uncertain, stimuli would elicit Gratton effects similar to those associated with exposure to prior incompatibility, as well as related MFC activation.

Predictions

Experiment 1

If the conflict monitoring system instantiated in the MFC is sensitive to conflict elicited by stimulus uncertainty in addition to response conflict, a main effect of stimulus uncertainty is predicted such that high stimulus overlap leads to increased N2 amplitude
across both levels of flanker stimulus compatibility. While some previous studies (Nieuwenhuis et al., 2004) have failed to show this predicted result, it is theorized that the lack of controlled manipulation of stimulus similarity might be the reason for these findings. When just noticeable differences between target and non-target tones have been used (Szmalc et al., 2008) N2 modulations have been seen. However, this study used auditory stimuli, and a similar procedure has not previously been used to examine conflict in the visual system. The present study would extend previous evidence of MFC processing of stimulus uncertainty using just noticeable differences into visual processing in addition to auditory processing. In addition, establishing a medial frontal sensitivity to non-response conflict is required to set the stage for the subsequent experiments.

Experiment 2

If stimulus uncertainty is processed by the same circuit as response conflict, similar N2 modulations should be seen for the conditions with and without response conflict. However, if the MFC conflict monitoring system is sensitive only to response conflict, the ERPs should show a decrease in N2 modulation when overt responses are not required. The findings of this experiment will help clarify how stimulus uncertainty is processed within the conflict monitoring circuit. A reduction of the N2 to the experimental manipulation would provide compelling evidence that response conflict is necessary to activate the MFC conflict monitoring circuit. Maintenance of the N2 without an overt response requirement would lend further credence to recent evidence of multiple levels of conflict processing in the MFC. In either case, the manipulation of
motor response requirements provides a method to clarify the independent contributions of each conflict source.

Experiment 3

If a compatible flanker stimulus with high stimulus uncertainty but low response conflict is presented, the increased attentional allocation required to adequately process the stimulus will lead to context effects on the subsequent trials that mirror those associated with classic incompatible flanker stimuli (which elicit high response conflict and low stimulus uncertainty). It is expected that since the compatible but uncertain Eriksen flanker stimuli will elicit conflict, either within the visual cortex or the MFC, these stimuli will also activate the compensatory attentional processes associated with the Gratton effect and the biased competition model. Thus, the stimulus uncertainty induced conflict monitoring will facilitate a faster reaction time on subsequent incompatible trials, as well as a decreased N2.
CHAPTER 2: Experiment One

Background

The first experiment was designed to determine if the MFC—as reflected in ERP components such as the N2—is involved in processing of stimulus uncertainty in addition to response conflict. While the theoretical models of conflict monitoring make allowances for the processing of non-response conflict by the MFC, empirical evidence to date has been inconsistent. Previous studies have shown that infrequent, perceptually conflicting stimuli (Milham et al., 2003; West et al., 2004; Azizian et al., 2006) can elicit MFC activation in the form of frontal-central ERP components like N2 (Azizan et al., 2006; Folstein and van Petten, 2008) and N450 (West et al., 2004).

If the conflict monitoring circuit instantiated in the MFC is sensitive to conflict elicited by stimulus uncertainty in addition to response conflict, a main effect of stimulus uncertainty is predicted such that high stimulus overlap leads to increased N2 amplitude across both levels of flanker stimulus compatibility. While previous studies (Nieuwenhuis et al., 2004) have failed to show this predicted result, it is theorized that the arbitrary nature of the easy versus difficult manipulation of stimulus similarity might be the cause. Recent attempts to psychophysically quantify the difference between easy and difficult to distinguish stimuli (Szmalalec et al., 2008) have illustrated N2 modulations, albeit only for auditory, not visual stimuli.
The present study provides additional evidence of MFC processing of stimulus uncertainty when controlled, quantifiable differences in stimulus identity are employed. These quantifiable differences are achieved by using a staircase procedure to create the difficult to discriminate stimuli. In addition, the present study increases the general applicability of previous auditory evidence (Szmalec et al., 2008) by using a visual paradigm. Finally, establishing an N2 sensitivity to stimulus uncertainty in addition to non-response conflict sets the stage for the subsequent experiments.

Methods

Participants

15 healthy participants (5 male, 10 female, average age 19.7 years (standard deviation 1.6 years)) from the undergraduate student population at George Mason University took part in the study. Participants were, right-handed, had normal or corrected to normal vision and no reported history of neurological illness. All participants were tested on the Rosenbaum Pocket Vision Screener and the Snellen eye chart and had visual acuity of at least 20/25. Informed consent was obtained from all participants, and each was granted partial course credit for their participation.

Apparatus and stimuli

All experimental instructions and stimuli were presented on a standard Windows desktop computer equipped with a 17” CRT monitor (1024 x 768 resolution, 75Hz refresh rate). Participants were seated 60cm from the computer screen. Experimental control was implemented using the E-Prime software package (Psychology Software
Tools Inc., Sharpsburg, PA). Responses were made with the first and fifth button of a horizontally-aligned 5 button response box.

Experimental stimuli consisted of modified Eriksen Flanker task (Eriksen and Eriksen, 1974) stimuli. All stimuli consisted of black lines presented on a white background. The entire stimulus—a central target and four flankers—subtended a visual angle of 2.5 degrees. A target line rotated clockwise or counterclockwise from vertical presented 0.5 degrees of visual angle above a central fixation dot on the CRT screen was flanked on either side by 2 flanker lines (4 total) with either equivalent (compatible) or opposite (incompatible) rotation from vertical. Task difficulty was modulated based on the degree of rotation of the target and flankers from vertical. Increased clockwise or counter clockwise rotation from vertical made stimulus discrimination easier. Examples of each of the four stimulus conditions are shown in figure 7.

![Figure 7: Experimental stimuli](image)

**Design and procedure**

Experiment one included two within-subjects variables: Task difficulty (easy, hard) as determined by the amount of stimulus rotation, and stimulus compatibility
(compatible, incompatible) as determined by the direction of rotation for the central
target and flankers in each stimulus. Participants were tasked with pressing the button
(left/right) that corresponded to the direction of the rotation of the target line in the center
of the flanker array. Targets rotated clockwise required a “right” button press, while
targets rotated counterclockwise required a “left” button press. Participants were
instructed to respond as fast as they could while maintaining high accuracy, equally
emphasizing speed and accuracy. Stimuli were presented for 200ms, with an additional
450ms before a response deadline. Inter-trial intervals were randomly jittered between
550ms and 1100ms.

Staircase procedure

Prior to the main experiment, participants completed an up-down staircase
procedure (Levitt, 1971) to determine the minimum amount of rotation from vertical for
flanker and target stimuli that would elicit accuracy above 70%. The procedure used E-
prime code modified from an example provided by Hariston and Maldjiam (2008). Each
staircase began with a rotation of 10 degrees from vertical (either clockwise or
counterclockwise) for both compatible and incompatible stimuli. The staircases
consisted of a 0.5 degree step for each reversal, two correct responses lead to the target
and flankers being rotated 0.5 degrees towards vertical, one incorrect response lead to
rotation 0.5 degrees towards horizontal. In order to determine the visual thresholds for
both compatible and incompatible stimuli rotated both clockwise and counterclockwise
from vertical, a total of 4 staircases (Compatible clockwise, Compatible
counterclockwise, Incompatible clockwise, Incompatible counterclockwise) were run concurrently.

Each staircase terminated after 12 reversals, with the values of the final 8 reversals averaged to calculate individual participants’ degrees of stimulus rotation for the hard task in the main experiment. For the 15 participants, average distance between clockwise and counterclockwise rotations was 5.77 degrees for the compatible hard stimuli and 9.27 degrees for the incompatible hard stimuli. The easy task consisted of stimuli with 40 degrees rotation for both compatible and incompatible stimuli. The staircase procedure lasted less than five minutes total, and participants were given short breaks every 90 seconds. Following the staircase procedure, participants rested for 20 minutes while the EEG cap was prepared for the main experiment.

**Main experiment**

The trials of the main experiment were split equally between easy and hard task blocks. The sequence of stimuli (compatible, incompatible) was completely randomized within each block. The main experiment included two practice blocks, one easy and one hard, of 60 trials each (30 compatible, 30 incompatible). Following practice, the experiment consisted of alternating blocks of easy and hard task, counterbalanced across participants. There were 8 blocks total (4 easy, 4 hard). Each block consisted of 240 stimuli (120 compatible, 120 incompatible) and participants were given short, self timed breaks every 90 seconds. In total, each participant responded to 480 trials of each stimulus type (compatible easy, incompatible easy, compatible hard, incompatible hard). The main experiment lasted approximately 1.25 hours, including self-timed breaks.
**EEG recording**

38 channels of EEG and EOG were recorded from the scalp by a 64 channel Ag-Ag/Cl sintered electrode cap (Compumedics Neuroscan, Charlotte, NC) during the main experiment only. The SCAN 4.3 software package (Compumedics Neuroscan, Charlotte, NC) was used to record and analyze EEG. Standard 10–20 electrode sites used were FPZ, FP1, FP2, FZ, F3, F4, F7, F8, FCZ, FC3, FC4, FT7, FT8, CZ, C3, C4, T7, T8, CPZ, CP3, CP4, TP7, TP8, PZ, P3, P4, P7, P8, POZ, PO3, PO4, PO7, PO8, OZ, O1, O2, M1 and M2. During EEG recording, the physical reference electrode was approximately 2 cm posterior to CZ. Following the recording session, EEG data were re-referenced to the average of M1 and M2 (left and right mastoid). Horizontal eye movements (HEOG) were monitored by placing two electrodes lateral to the left and right orbits. Vertical eye movements (VEOG) and eye blinks were measured by placing two electrodes 1.5 cm below and above the left eye. The EEG from each electrode site was digitized at 500 Hz and filtered with a band pass of 0.1–30 Hz. A 200-ms pre-stimulus epoch of EEG was used as the baseline, with the stimulus-locked epoch extending to 550ms post-stimulus onset. Eye movement artifacts were defined as a positive or negative amplitude deflection of greater than 75µV on the HEOG or VEOG channels. Any epoch with an eye movement artifact was discarded. Behavioral data (reaction time and accuracy of response) was merged with EEG data prior to further analysis.
Results

Behavioral and electrophysiological data were analyzed using repeated measures ANOVA with the factors stimulus compatibility (compatible vs. incompatible) and task difficulty (easy vs. hard). Trials with errors due to incorrect response or no response were discarded. For correct trials, responses within 150-650ms of stimulus onset were considered valid. All other trials were discarded.

Behavioral Results

Mean RT and accuracy data as a function of task difficulty and stimulus type are presented in table 1.

Table 1: Experiment 1 behavioral results

<table>
<thead>
<tr>
<th>Condition</th>
<th>Reaction Time (ms)</th>
<th>Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SE</td>
</tr>
<tr>
<td>Compatible Easy</td>
<td>342</td>
<td>8.9</td>
</tr>
<tr>
<td>Incompatible Easy</td>
<td>370</td>
<td>8.7</td>
</tr>
<tr>
<td>Compatible Hard</td>
<td>381</td>
<td>7.1</td>
</tr>
<tr>
<td>Incompatible Hard</td>
<td>383</td>
<td>8.1</td>
</tr>
</tbody>
</table>
A 2x2 repeated measures ANOVA with within subjects factors of task difficulty (easy, hard) and stimulus compatibility (compatible, incompatible) was performed for both mean RT and trial accuracy. Mean RT results showed the main effects of task difficulty $F(1,14)=35.82, p<0.001$ and stimulus compatibility $F(1,14)=54.67, p<0.001$ as well as their interaction $F(1,14)=60.60, p<0.001$. Planned comparisons showed that mean RT to incompatible easy stimuli were slower than to compatible easy stimuli, $t(14)=10.02, p<0.001$, while in the hard condition, there was no difference in mean RT between incompatible hard and compatible hard stimuli $t(14)=0.69, p=0.496$.

Accuracy results showed the main effect of task difficulty was significant $F(1,14)=7.71, p=0.015$, as was the task difficulty by stimulus compatibility interaction $F(1,14)=28.12, p<.001$, while the main effect of stimulus compatibility approached significance $F(1,14)=4.35, p=0.056$. Planned comparisons showed that in the easy condition, mean accuracy was higher for compatible easy than for incompatible easy stimuli $t(14)=6.44, p<.001$. However, in the hard task, the result was reversed. Incompatible hard stimuli elicited higher accuracy than did compatible stimuli $t(14)=2.93, p=0.011$. These results show that both response conflict and stimulus uncertainty instantiated conflict since they both slowed RT and reduced accuracy when compared to compatible easy stimulus performance.

**ERP results**

**Occipital Temporal N1**

Over occipital electrodes, inspection of the grand average, stimulus-locked waveforms (figure 8) showed a clear visual N1, a negative amplitude peak bilaterally
distributed with maximum negative amplitude between 140-180ms post-stimulus onset. The occipital temporal N1 was maximal at electrode PO7 across all experimental conditions, consistent with previous evidence showing larger visual N1 in the left hemisphere (Hopf et al., 2002; Vogel et al., 2000). For clarity, only amplitudes at PO7 will be reported, though results at PO8 were similar.

![Occipital Temporal N1 at Electrode P07](image)

**Figure 8:** Experiment 1 N1 at electrode P07
CE= compatible easy; IE= incompatible easy; CH= compatible hard; IH= incompatible hard
A 2x2 repeated measures ANOVA with within subjects factors of task difficulty (easy, hard) and stimulus type (compatible, incompatible) was performed for mean occipital temporal N1 amplitude from 140-180ms post stimulus onset. Results showed main effects of task difficulty (F(1,14)=21.47, p<0.001) and stimulus type (F(1, 14)=8.18, p=0.013). Stimuli in the easy task evoked larger amplitude N1s than stimuli in the hard task, and incompatible stimuli evoked larger occipital temporal N1 amplitude than the compatible stimuli.

Medial Frontal N2

Inspection of the scalp topographies (figure 9) and grand average waveforms over medial frontal electrodes showed a stimulus locked, negative voltage component peaking between 200-320ms post stimulus onset with a maximum negative deflection at electrode FCZ (figure 10). The N2 has a spatial location, peak latency and sensitivity to conflict-inducing stimuli consistent with an index of conflict monitoring.
Figure 9: Experiment 1 N2 scalp topographies
In addition, visual inspection of the grand average waveforms in figure 10 suggests that there are separate sub components of the medial frontal N2s modulated by stimuli in the different task difficulty conditions. The easy task shows an early N2 (eN2) with maximum negative amplitude between 200-250ms post stimulus onset, while the hard task shows a late N2 (lN2) with a maximum negative amplitude between 260-320ms post stimulus onset.
post stimulus onset. Since the peak latencies are different between these two subcomponents, statistical analysis was completed on the primary component within each window (eN2 200-250ms; lN2 260-320ms) as opposed to the overall 200-320ms window.

A 2x2x2 repeated measures ANOVA with within subjects factors of N2 subcomponent (eN2 200-250ms, lN2 260-320ms), task difficulty (easy, hard) and stimulus compatibility (compatible, incompatible) was performed for mean amplitude at medial frontal electrode FCZ. Results showed a significant N2 subcomponent by task difficulty interaction (F(1,14)=17.39, p=.001) as well as a significant task difficulty by stimulus compatibility interaction (F(1,14)=10.38, p=.006). Planned comparisons revealed that the N2 subcomponent by task difficulty interaction was driven by the amplitude difference between compatible easy eN2 and incompatible easy eN2 amplitude t(14)=2.28, p<.05 and the difference between compatible hard lN2 and incompatible hard lN2 amplitude t(14)=2.58, p<.05.

The task difficulty by stimulus compatibility interaction is driven by the difference in the maximal conflict inducing stimuli in the different tasks. In the easy task, incompatible stimuli elicit larger amplitude N2s than compatible stimuli in both the eN2 and lN2 subcomponent windows. Conversely, in the hard task compatible stimuli elicit larger lN2s than incompatible stimuli in the later subcomponent window.
Finally, the comparisons of interest, given the a priori hypothesis about the relationship between response conflict and stimulus uncertainty monitoring, are between the compatible easy (that is no conflict) and incompatible easy (response conflict) and compatible hard (stimulus uncertainty) stimuli respectively (figure 11). The compatible easy—incompatible easy comparison showed a significant difference in the eN2 amplitude $t(14)=2.28, p<.05$ and no difference in lN2 amplitude $t(14)=1.83, p>.05$, while the compatible easy—compatible hard comparison showed no difference in eN2 amplitude $t(14)=1.09, p>.05$ and a significant difference in the lN2 amplitude $t(14)=2.58, p<.05$.
Discussion

The first experiment compared the within-trial processing of response conflict and stimulus uncertainty in an Eriksen flanker task both behaviorally and using brain electrophysiological signals. Based on the theoretical description of the conflict monitoring sub processes of cognitive control, it was predicted that both types of conflict would modulate ERP components that index medial frontal conflict monitoring. That is, if the medial frontal conflict monitoring circuit participates in within-trial processing of perceptual conflict, then stimulus uncertainty should elicit an increased conflict monitoring response similar to that elicited by response conflict.

The results supported this hypothesis. Increases in both response conflict and stimulus uncertainty delayed reaction times and reduced accuracy when compared to non-conflict inducing stimuli. Occipital temporal, electrophysiological signals indexing early attentional modulations of stimulus discrimination processes were increased in the presence of response conflict as compared to stimulus uncertainty. Since occipital temporal N1 has been shown to index discrimination processes in the visual cortex (Hopf et al., 2002), the reduction in N1 amplitude in the hard task suggests that the difficulty modulation in the current experiment was successful in making stimulus discrimination more challenging. This occipital temporal N1 reduction is consistent with the findings of Lavie et al. (2005) showing that easily distinguished targets and distracters evoke larger early discrimination-related brain activations.

Later medial frontal signals associated with conflict monitoring showed a sensitivity to both response conflict and stimulus uncertainty, though this sensitivity
occurred at different times depending on the type of conflict induced by the stimulus. The observed temporal dissociation does not appear to be a modulation of the peak latency of a single cognitive process. Instead, the electrophysiological response to stimulus uncertainty and response conflict illustrate two dissociable sub processes of conflict monitoring. The eN2 is sensitive to response conflict while the IN2 is sensitive to stimulus uncertainty.

Previous empirical studies have not consistently shown modulations of conflict monitoring processes based on stimulus uncertainty. Arbitrary distinctions between high and low levels of stimulus uncertainty have previously been insufficient to evoke N2 modulations (Van Veen and Carter, 2002; Nieuwenhuis et al., 2004), though there are notable exceptions (Azizian et al., 2006). This lack of sensitivity to stimulus uncertainty is likely due to the arbitrarily defined difficult stimuli failing to instantiate sufficient conflict in the sensory processing system.

As an alternative to arbitrary easy/hard distinctions, the current experiment applied a psychophysical method to derive perceptually similar stimuli that remain discriminable at an above chance level. The use of the up-down staircase to derive hard stimuli for each participant individually allowed for instantiation of a sufficient level of stimulus uncertainty in the current task to evoke both behavioral and electrophysiological signals of conflict monitoring. These results corroborate the findings of Szmalec et al. (2008), who employed a different psychophysical method to derive perceptually similar stimuli in an auditory task paradigm. They found high levels of aural similarity can modulate N2 in an auditory go/no go task.
Additionally, previous studies showing N2 modulation related to stimulus uncertainty (Szmaléc et al., 2008; Azizian et al., 2006) were confounded by necessary manipulations of stimulus frequency in addition to stimulus uncertainty. One prominent review of the cognitive processes indexed by medial frontal N2 suggests that in addition to conflict monitoring, stimulus novelty can increase the amplitude of N2 (Folstein and van Petten, 2008).

Thus, an argument can be made that stimulus frequency manipulations played an additional role in the medial frontal N2 modulations reported in these previous studies. In the current study, however, both tasks were presented in equivalent blocks, and compatible and incompatible stimuli were presented with equal frequency. Thus, no portion of the eN2 and lN2 modulations reported can be attributed to infrequent stimuli. This removes an alternative explanatory process while the modulation of N2 remains.

The response conflict-induced modulation of the eN2 is consistent with a large body of previous literature on conflict monitoring (Kopp et al., 1996). It is somewhat surprising that the lN2 modulation associated with stimulus uncertainty occurs at a later latency. This is especially true given the central executive nature of conflict monitoring in theoretical descriptions of cognitive control (i.e., Botvinick et al., 2001). Based on the prior evidence of additive contributions of response conflict and stimulus uncertainty to N2 amplitude (Szmaléc et al., 2008; Azizian et al., 2006), the expectation would be for a single N2. However, the current data clearly show an interaction between the two identified sub components, eN2 and lN2, when processing response conflict and stimulus uncertainty respectively.
Both the behavioral and early stimulus processing data, however, show that conflict is indeed instantiated in the hard task condition due to stimulus uncertainty. Responses are significantly slower and less accurate in the hard condition, suggesting a delay in response execution when participants must process ambiguous stimuli. In addition, the occipital temporal N1, an index of sensory discrimination, is significantly reduced in the hard task. The combination of slow responses and reduced discrimination is consistent with an instantiation of stimulus uncertainty early in information processing. This corroborating evidence can be taken as support for the presence of conflict in the hard task. Given this conflict, and the interpretation of the observed subsequent increase in lN2 as an index of stimulus uncertainty related conflict monitoring is strengthened.

While these results show that response conflict and stimulus uncertainty elicit predictable behavioral and brain activation changes associated with both early attentional discrimination processing and later conflict monitoring, there is some difficulty in ascribing a similar source to the signals believed to index processing of response conflict and stimulus uncertainty. Both varieties of conflict slowed down the physical response to conflict-inducing stimuli. Additionally, both the response conflict-elicited eN2 and the stimulus uncertainty-elicited lN2 occurred within the broad time window (200-320ms post stimulus onset) associated with conflict monitoring processes and N2. However, the peak latencies of the two N2s occurred at different times. The response conflict eN2 reached a local minimum significantly earlier than did the stimulus uncertainty lN2. This temporal separation suggests the two medial frontal N2s index related, but discrete
processes; Related, in that they are both sensitive to conflict, discrete in that they are temporally dissociable and sensitive to separate varieties of conflict.

Given the ambiguities of these results, additional experiments were performed to further characterize the stimulus uncertainty induced IN2 as well as the sequential effects of prior exposure to either response conflict or stimulus uncertainty on subsequent processing of conflict-inducing stimuli. The following experiment was designed to isolate the conflict monitoring processes independent of an overt motor response requirement in the presence of stimulus uncertainty.
CHAPTER 3: Experiment Two

Background

Multiple studies have associated ACC activations with more strategic processing (Kennerley et al., 2006; Walton et al., 2004, Brown and Braver, 2005) and suggest a function beyond simply motor-related conflict processing. Additionally, the persistence of N2 modulations in the absence of overt motor responses seen in prior literature are consistent with an interpretation that there is a non-motor component to MFC processing indexed by the N2.

It remains to be seen if the preSMA and ACC differentially contribute to processing of motor conflict and non motor conflict. Results from experiment 1 as well as other studies (Szmalec et al., 2008) show that stimulus uncertainty can modulate sub components of N2, which is believed to index conflict monitoring processes. However, each of the previous studies has used a paradigm that required an overt motor response. Previous work has shown that N2s can be elicited in a counting go/no go task without an overt physical response (Pfefferbaum et al., 1985; Bruin and Wijers, 2002).

If stimulus uncertainty is processed by the same circuit as response conflict, similar N2 modulations should be seen for the conditions with and without response conflict. However, if the MFC conflict monitoring system is sensitive only to response
conflict, the ERPs should show a decrease or absence of N2 modulation when overt responses are not required.

A reduction of the stimulus uncertainty elicited lN2 in the absence of an overt motor response would provide compelling evidence that response conflict is necessary to activate the MFC conflict monitoring circuit. The manipulation of motor response requirements provides an additional method to clarify the independent contributions of response conflict and stimulus uncertainty. Given the single cell and human imaging evidence of a dissociation between preSMA and ACC activation, it is predicted that stimulus uncertainty should elicit MFC activation even in the absence of an overt motor response requirement.

In the predicted absence of any difference between response and no response lN2 for stimulus uncertainty, other ERP components are useful to characterize differences related to the presence or absence of a motor response. For example, modulations of early attentional components like P1 and N1 characterize the overall arousal or attention allocated to the task (Hillyard et al., 1998; Luck et al., 2000).

The absence of a time response deadline should reduce overall arousal and be reflected in the sensory ERPs. These changes should also be reflected in modulations of the P300, associated with stimulus categorization (Kok, 2001). Together, these complimentary ERP components should illustrate changes between the response and non response task versions in experiment 2 that may not be apparent based on the predicted insensitivity of lN2 to overt response requirements.
Method

Participants

19 healthy participants (7 male, 12 female, average age 22.8 years (standard deviation 9.1 years)) from the undergraduate student population at George Mason University took part in the study. Participants were right-handed, had normal or corrected to normal vision and no reported history of neurological illness. All participants were tested on the Rosenbaum Pocket Vision Screener and the Snellen eye chart and had visual acuity of at least 20/25. Informed consent was obtained from all participants, and each was granted partial course credit for their participation.

Apparatus and Stimuli

Apparatus and stimuli for experiment 2 were equivalent to those used in experiment 1. The same computer and response box were used for all three experiments, and experimental control was implemented using the E-Prime software package. Modified Eriksen Flanker Task stimuli with two flankers (4 total) on either side of a central target were again presented.

Design and Procedure

Experiment two included two within-subjects variables: Stimulus compatibility (compatible, incompatible) and response task (response, non-response). Unlike experiments one and three, experiment two employed only hard to differentiate stimuli, which induced stimulus uncertainty. The current experiment employs a method slightly modified from experiment one. Participants completed a staircase procedure to determine the minimum amount of stimulus rotation from vertical necessary for 80%
accurate performance in identification of compatible stimuli. This rotation value was then used to create the compatible and incompatible hard stimuli in the main experiment.

*Staircase Procedure*

Prior to the main experiment, participants completed an up-down staircase procedure (Levitt, 1971) similar to that described in experiment one. However, experiments two and three contained a minor change in staircase procedure. While participants in experiments two and three completed all four staircases as in experiment one, only the rotation values from the compatible stimulus presentations were calculated and used to create the stimuli for the main experiment.

This change was necessitated by the significant increase in accuracy and mean RT seen for the incompatible hard stimuli in experiment one. It is believed that the incompatible rotation value derived from the staircase is confounded by the diametrically opposed levels of response conflict and stimulus uncertainty throughout the procedure. Early in the staircase procedure, targets and flankers are rotated far from vertical, making discrimination easy, and reducing stimulus uncertainty while response conflict remains high. Later in the staircase procedure, when targets and flankers approach vertical, stimulus uncertainty is high, but response conflict is greatly reduced. Because response conflict is high early in the procedure for incompatible stimuli, any response errors early in the staircase procedure cannot be solely attributed to stimulus uncertainty. Instead, response conflict may be responsible for any incorrect response the participant makes, especially during the first few staircase reversals.
The incompatible stimulus rotation values derived from the staircase procedure are much greater (rotated further from vertical) than are the compatible stimulus rotation values in each of the experiments described in this dissertation. As the contribution of response conflict to this difference cannot be isolated given the above explanation, the rotation values derived from the compatible stimulus staircases were used for the main experiment in both experiments two and three. These compatible stimulus rotation values remain uninfluenced by response conflict (since there is no response conflict when processing a compatible flanker stimulus) and thus provide a cleaner measure of each participants’ stimulus differentiation ability as well as the influence of stimulus uncertainty on target discrimination in an Eriksen flanker task.

In experiment two, for the 19 participants, average distance between clockwise and counterclockwise rotations was 6.34 degrees for the compatible stimuli based on the described threshold adjustment, the same rotation values were used for the incompatible stimuli. Identical stimuli were used in both the response and non response tasks, as described in the next section. Following the staircase procedure, participants rested for 20 minutes with the EEG cap was prepared for the main experiment.

Main experiment

Experiment two included a manipulation of response requirements in an Eriksen flanker task. The main experiment included response and non-response tasks. For the response task, experimental timings were equivalent to experiment one. Response task blocks consisted of single flanker stimulus presentation which required a speeded button press corresponding to the orientation of the central flanker following each stimulus
presentation. The non-response task blocks presented a series of three compatible or incompatible flanker stimuli, counterbalanced between compatible and incompatible as well as between left and right rotated targets. Each of the three stimulus presentations was followed by a randomized 550-1100ms inter trial interval. Following the third presentation, participants had to make a non-speeded response indicating the number of left (or right) rotated targets presented within the previous three stimuli. Right or left target counting was counterbalanced across subjects. Figure 12 presents a schematic of the two response task conditions in experiment 2.

![Figure 12: Experiment 2 task schematic](image)

The main experiment included two practice blocks, one response task block and one non-response task block, of 56 trials each (28 compatible, 28 incompatible). Following practice, the experiment consisted of 48 task blocks, 24 response and 24 non-response blocks, presented in randomized order. Participants were given short, self timed
breaks every 90 seconds. In total, each participant was presented with 672 trials of each of the four stimulus types (compatible response, incompatible response, compatible non-response, incompatible non-response). The main experiment lasted approximately 1.5 hours, including self-timed breaks.

**EEG Recording**

38 channels of EEG and EOG were recorded from the scalp by a 64 channel Ag-Ag/Cl sintered electrode cap (Compumedics Neuroscan, Charlotte, NC) during the main experiment only. The SCAN 4.3 software package (Compumedics Neuroscan, Charlotte, NC) was used to record and analyze EEG. Standard 10–20 electrode sites used were FPZ, FP1, FP2, FZ, F3, F4, F7, F8, FCZ, FC3, FC4, FT7, FT8, CZ, C3, C4, T7, T8, CPZ, CP3, CP4, TP7, TP8, PZ, P3, P4, P7, P8, POZ, PO3, PO4, PO7, PO8, OZ, O1, O2, M1 and M2. During EEG recording, the physical reference electrode was approximately 2 cm posterior to CZ. Following the recording session, EEG data were re-referenced to the average of M1 and M2 (left and right mastoid). Horizontal eye movements (HEOG) were monitored by placing two electrodes lateral to the left and right orbits. Vertical eye movements (VEOG) and eye blinks were measured by placing two electrodes 1.5 cm below and above the left eye. The EEG from each electrode site was digitized at 500 Hz and filtered with a band pass of 0.1–30 Hz. A 200-ms pre-stimulus epoch of EEG was used as the baseline. Eye movement artifacts were defined as a positive or negative amplitude deflection of greater than 75μV on the HEOG or VEOG channels. Any epoch with an eye movement artifact detected was discarded. Behavioral data (reaction time and accuracy of response) was merged with EEG data prior to further analysis.
Results

Behavioral and electrophysiological data were analyzed using repeated measures ANOVA with the factors stimulus compatibility (compatible vs. incompatible) and task type (response vs. non-response). For both response types, trials with errors due to incorrect response were discarded. In the response blocks, correct responses within 150-650 ms of stimulus onset were considered valid. In the no response blocks, all correct responses were considered valid. All other trials were discarded.

Behavioral Results

Mean RT and accuracy data as a function of stimulus compatibility and task type are presented in table 2.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Reaction Time (ms)</th>
<th>Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SE</td>
</tr>
<tr>
<td>Compatible Response</td>
<td>385</td>
<td>8.1</td>
</tr>
<tr>
<td>Incompatible Response</td>
<td>396</td>
<td>7.8</td>
</tr>
<tr>
<td>Compatible Non-response</td>
<td>NA*</td>
<td></td>
</tr>
<tr>
<td>Incompatible Non-response</td>
<td>NA*</td>
<td></td>
</tr>
</tbody>
</table>

* speeded responses were not required for the non-response task
A 2x2 repeated measures ANOVA with within subjects factors of stimulus compatibility (compatible, incompatible) and response task type (response, non-response) was performed for trial accuracy. Accuracy results showed the main effect of task type was significant $F(1,18)= 13.78, p<0.005$, as was the stimulus compatibility by response task type interaction $F(1,18)= 17.98, p<.001$. However, this interaction was not analyzed further due to the fact that the two response tasks had different response criteria, and thus the interaction is not descriptive of any relevant cognitive differences between tasks.

A repeated measure ANOVA with a within subjects factor of stimulus compatibility (compatible, incompatible) was preformed for mean RT in the response version of the task. There were no individual stimulus responses in the non-response task, and thus no mean RT data was collected. Mean RT results showed a main effect of stimulus compatibility $F(1,18)=18.07, p<.001$, with compatible responses faster than incompatible responses.

**ERP results**

**ERP Component Selection**

Mean amplitude windows for the ERP components of interest—Occipital Temporal P1 and N1, IN2, and Parietal P3—were identified using inspection of the grand average waveforms for each condition.

**Occipital-Temporal P1 and N1 Window**

Over occipital electrodes, inspection of the grand average, stimulus-locked waveforms (figure 13) showed a clear visual P1 and N1. The P1 is a positive amplitude peak with maximum amplitude between 80-120ms. The N1 is a negative amplitude peak
bilateral distribution with maximum negative amplitude between 140-180ms post-stimulus onset.

The occipital temporal N1 was maximal at electrode PO7 across all experimental conditions, consistent with previous evidence showing larger visual N1 in the left hemisphere (Hopf et al., 2002; Vogel et al., 2000). For clarity, only amplitudes at PO7 will be reported, though results at PO8 were similar.

Late Medial Frontal N2 Window

Based on the findings of experiment 1, stimulus uncertainty was expected to modulate IN2 amplitude 260-320ms post-stimulus onset. Visual inspection of the scalp topographies (figure 14) and grand average waveforms at electrode FCZ (figure 15) support this expectation.

Parietal P3 Window

Over midline parietal electrodes, inspection of the grand average, stimulus-locked waveforms (figure 16) showed a clear parietal P3. The P3 is a positive amplitude peak with maximum amplitude between 400-500ms post-stimulus onset.

Sensory Processing: Occipital Temporal P1 and N1

Current stimulus elicited occipital temporal P1 and N1 grand averages are presented in figure 13.
Repeated-measures ANOVA results for mean P1 amplitude at electrode P07 show a main effect of task type ($F(1,18)= 4.679$, $p<.05$), with the non-response task eliciting smaller amplitude P1s than the response task for both compatible and incompatible stimuli.
Neither the main effect of stimulus compatibility (F(1,18)=1.729, p=.205) nor the interaction between task type and stimulus compatibility (F(1,18)=0.575, p=.458) reached significance. Similar results are seen for the repeated-measure ANOVA for mean N1 amplitude at electrode PO7; A significant main effect of task type (F(1,18)=6.521, p=0.02) was seen, with the non-response task eliciting smaller magnitude N1s than the response task for both compatible and incompatible stimuli. Additionally, there was an absence of a significant main effect of stimulus compatibility (F(1,18)=0.780, p=.389) or a task type by stimulus compatibility interaction (F(1,18)=0.485, p=0.495.

Conflict Monitoring: late medial frontal N2

Current stimulus-elicited lN2 grand averages are presented in figure 15. Since response conflict was not manipulated in the present experiment, mean amplitude differences in the eN2 range are not reported. Based on the experimental hypotheses related to stimulus uncertainty monitoring, only lN2 amplitudes are reported. Though, there were no differences among the results in the eN2 window (200-250ms) the IN2 window (260-320ms) and the entire N2 window (200-320ms).
Figure 14: Experiment 2 N2 scalp topographies
Figure 15: Experiment 2 In2 at electrode FCZ.
Blue = Compatible Response; Red = Incompatible Response;
Green = Compatible Non-response; Yellow = Incompatible Non-response
Repeated measures ANOVA results for ln2 mean amplitude at electrode FCZ show no significant main effects of task type (F(1,18)=0.690, \( p=0.417 \)), stimulus compatibility (F(1,18)=0.008, \( p=0.931 \)) or their interaction (F(1,18)=0.840, \( p=0.371 \)).

*Stimulus categorization: Parietal P3*

Current stimulus-elicited parietal P3 grand averages are presented in figure 16.
Repeated measure ANOVA results for the parietal P3 mean amplitude at electrode PZ show significant main effects of both task type \( (F(1,18)=26.64, p=.000) \) and stimulus compatibility \( (F(1,18)=12.78, p=.002) \) in the absence of any interaction between the two factors \( (F(1,18)=.005, p=.757) \).

**Discussion**

The goal of the present experiment was to further characterize the contributions of non-response processes to MFC conflict monitoring as indexed by the N2. Results showed the persistence of the IN2 in the absence of an overt motor response requirement when participants were presented with stimulus uncertainty. The persistence of the stimulus evoked IN2 in the absence of both response conflict and an overt motor response requirement suggests that the cognitive processes indexed by the N2 are not exclusively response-related.

Prior evidence indicates a persistence of N2 in a reduced form for non response task conditions. However, these previous studies all employed response conflict inducing stimuli. The current experiment uses a stimulus uncertainty inducing flanker stimulus and shows the persistence of IN2 in the non response task. Thus, the current results extend the previous findings into additional types of conflict processing, and illustrate a separation between stimulus uncertainty and response conflict processing.

While previous studies have shown a reduction in the N2 in the absence of an overt motor response on each trial, the current experiment showed no difference in the mean amplitude of the N2 for the response and non response tasks. It is difficult to draw
strong conclusions from such a null finding. However, the interpretation that the present null finding is consistent with a sensitivity of the MFC conflict monitoring circuit to stimulus uncertainty independent of response conflict is supported by the observed modulations in both early sensory as well as late categorization process indexing ERP components.

In the current experiment, the manipulation of task type (response, no response) is sufficient to elicit predictable modulations in both early sensory ERPs like occipital temporal P1 and N1 as well as late ERPs like parietal P3. In both cases, the non response task elicited smaller mean amplitudes from these ancillary ERP components. This is consistent with a lower level of overall processing.

In the case of occipital temporal P1 and N1, these reduced amplitudes observed in the non-response task are associated with lower levels of arousal and fewer attentional resources being directed to each stimulus presentation (Hillyard et al., 1998; Luck et al., 2000). Given the absence of a response deadline after each stimulus presentation in the non response task, it is unsurprising that the mean amplitudes of the early sensory ERPs are reduced. However, even in the presence of reduced early attention in the non response task, the within trial IN2 persists at the same mean amplitude in the 260-320ms post stimulus onset window as is seen in the response task. This suggests that the IN2 elicited by stimulus uncertainty is insensitive to the absence of a motor response requirement in the non response task.

Similar results are seen for the mean amplitude of parietal P3, an index of stimulus categorization (Kok, 2001). As expected, the non response task elicited a
significantly smaller P3, indicating a reduction in the degree of stimulus categorization processing. While the observed increase in negative amplitude between 300-400ms is likely due in part to the drastic reduction in the P3, the P3 for the response version of the task peaks around 450ms, well after the local minimum associated with the IN2 or the more general N2 described in previous literature. Therefore, it is unlikely that the observed IN2 in the non-response task is entirely an artifact of the reduced P3. The persistence of the IN2 in the absence of an overt motor response is indicative of a non-response activation indexed by the N2. In experiment 2, this activation is likely associated with the stimulus uncertainty induced by the psychophysically derived Eriksen flanker stimuli.

The persistence of N2 amplitude in the absence of an overt motor response seen in prior studies (Pfferbaum et al., 1985; Bruin and Weijers, 2004) and replicated in the non-response task reported above are consistent with the presence of a non-motor component to the MFC processing indexed by the N2. Further, the present results are consistent with the findings of experiment 1 of this dissertation, which showed a dissociable sensitivity to stimulus uncertainty in an Eriksen flanker task. Given the previous description of a strategic trial-to-trial role for MFC activation (Yu et al., 2009), experiment three seeks to further characterize the overlapping processes of response conflict and stimulus uncertainty monitoring beyond that which occurs within a single trial.
CHAPTER 4: Experiment Three

Background

When the brain is faced with conflicting information, its response is influenced not only by the current source of incompatibility, but also by the recent prior history of conflict. Such sequence effects have been observed in behavioral studies in flanker tasks (Gratton et al., 1992) as well as other cognitive control tasks (Egner et al., 2007; 2010) are well documented in the conflict monitoring literature. Prior exposure to conflict-inducing stimuli has a facilitative effect on the processing of current conflict-inducing stimuli, whereas prior exposure to non conflict-inducing stimuli leads to behavioral costs when processing current conflict-inducing stimuli. The facilitation of conflict processing is theorized to be due to modulations of the amount of cognitive control exerted in response to exposure to conflict during the prior trial. Prior exposure to conflict activates the conflict monitoring circuit, which leads to an increase in compensatory processes on subsequent trials, and a related reduction in the amount of conflict processed.

However, evidence shows that prior exposure to cross-task conflict does not show the same facilitative effects (Egner et al., 2007). That is, if two tasks are interleaved such that conflict-inducing stimuli from one task are presented prior to conflict-inducing stimuli from a second task—a Simon task stimulus followed by a Stroop task stimulus, for example—the processing of the first conflict-inducing stimulus does not facilitate
performance on the second, unrelated conflict-inducing stimulus. These results suggest that modulations in conflict-related cognitive control are task specific, as opposed to related sub processes of conflict monitoring and cognitive control.

Based on the widely accepted model of a central conflict monitor (Botvinick et al., 2001), the lack of cross task compatibility sequence effects appears at odds with a central executive conflict monitoring process in the MFC. Given the evidence of eN2 and related IN2 modulations following exposure to both response conflict and stimulus uncertainty respectively, one method of determining if these components index a common, underlying cognitive process is to examine the compatibility sequence effects across both the easy (i.e., response conflict inducing) and hard (i.e., stimulus uncertainty inducing) variants of the Eriksen flanker task.

If the two separate instantiations of conflict are monitored by a similar cognitive control process, facilitative effects are predicted for easy incompatible stimuli preceded by hard stimuli as well as hard stimuli preceded by easy incompatible stimuli. That is, similar compatibility sequence effects should be observed following the processing of both response conflict and stimulus uncertainty. However, if the two task variants tap separate conflict monitoring sub processes, there should be no interaction between prior exposure to stimulus uncertainty and subsequent processing of response conflict. Ideally, any facilitative effects will be reflected not only in behavioral performance, but also in electrophysiological signals associated with conflict monitoring, visual processing and attention on both the prior and current trials.
Method

Participants

18 healthy participants (8 male, 10 female, average age 24.7 years, (standard deviation 8.6 years)) from the undergraduate student population at George Mason University took part in the study. Participants were right-handed, had normal or corrected to normal vision and no reported history of neurological illness. All participants were tested on the Rosenbaum Pocket Vision Screener and the Snellen eye chart and had visual acuity of at least 20/25. Informed consent was obtained from all participants, and each was granted partial course credit for their participation.

Apparatus and Stimuli

Apparatus and stimuli for experiment three were similar to those used in experiments one and two. The same computer, monitor and response box were used for all three experiments, and experimental control was implemented using the E-Prime software package (Psychology Software Tools Inc., Sharpsburg, PA). Modified Eriksen Flanker Task stimuli subtending 2.5 degrees of visual angle, consisting of two flankers (4 total) on either side of a central target were again presented.

Design and Procedure

Experiment three included two within-subjects variables: Task difficulty (easy, hard) as determined by the amount of stimulus rotation, and stimulus compatibility (compatible, incompatible) as determined by the direction of rotation for the central target and flankers in each stimulus. In addition, to examine the compatibility sequence effects elicited by the different stimuli, the sequence of stimulus presentation was also
recorded. Therefore, two additional within-subjects variables were measured: Prior task
difficulty (prior easy, prior hard) and prior stimulus compatibility (prior compatible, prior
incompatible). The resulting 16 stimulus sequence combinations are presented in table 3.

<table>
<thead>
<tr>
<th>Prior Trial</th>
<th>Current Trial</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compatible Easy</td>
<td>Compatible Easy</td>
<td>CECE</td>
</tr>
<tr>
<td>Incompatible Easy</td>
<td>Compatible Easy</td>
<td>IECE</td>
</tr>
<tr>
<td>Compatible Hard</td>
<td>Compatible Easy</td>
<td>CHCE</td>
</tr>
<tr>
<td>Incompatible Hard</td>
<td>Compatible Easy</td>
<td>IHCE</td>
</tr>
<tr>
<td>Compatible Easy</td>
<td>Incompatible Easy</td>
<td>CEIE</td>
</tr>
<tr>
<td>Incompatible Easy</td>
<td>Incompatible Easy</td>
<td>IEIE</td>
</tr>
<tr>
<td>Compatible Hard</td>
<td>Incompatible Easy</td>
<td>CHIE</td>
</tr>
<tr>
<td>Incompatible Hard</td>
<td>Incompatible Easy</td>
<td>IHIE</td>
</tr>
<tr>
<td>Compatible Easy</td>
<td>Compatible Hard</td>
<td>CECH</td>
</tr>
<tr>
<td>Incompatible Easy</td>
<td>Compatible Hard</td>
<td>IECH</td>
</tr>
<tr>
<td>Compatible Hard</td>
<td>Compatible Hard</td>
<td>CHCH</td>
</tr>
<tr>
<td>Incompatible Hard</td>
<td>Compatible Hard</td>
<td>IHCH</td>
</tr>
<tr>
<td>Compatible Easy</td>
<td>Incompatible Hard</td>
<td>CEIH</td>
</tr>
<tr>
<td>Incompatible Easy</td>
<td>Incompatible Hard</td>
<td>IEIH</td>
</tr>
<tr>
<td>Compatible Hard</td>
<td>Incompatible Hard</td>
<td>CHIH</td>
</tr>
<tr>
<td>Incompatible Hard</td>
<td>Incompatible Hard</td>
<td>IHIH</td>
</tr>
</tbody>
</table>

Experimental procedures were similar to experiment one. Individual trial timings
were the same; 200ms stimulus presentation, 450ms response window, 550-1100ms inter
trial interval. The key difference in experiment three is the interleaving of the hard and
easy tasks. Instead of the blocked design of experiment one, experiment three used a
randomized sequence of compatible and incompatible easy stimuli and compatible and
incompatible hard stimuli. This randomized presentation allowed for the examination of compatibility sequence effects across both high and low response conflict and high and low stimulus uncertainty conditions simultaneously.

**Staircase procedure**

Prior to the main experiment, participants completed an up-down staircase procedure similar to experiments one and two. As was the case in experiment two, a threshold adjustment was employed for experiment three to determine the rotation for the hard incompatible stimuli. The staircase valued derived for the compatible stimuli were used for both the compatible and incompatible stimuli in the hard task of the main experiment. This was done to ensure that the rotation values reflected only participants’ ability to differentiation between left and right rotated stimuli, not response conflict induced by the incompatible stimuli. For a more detailed description, see the methods section of experiment two (Chapter 3). In experiment three, for the 15 participants, average distance between clockwise and counterclockwise rotations was 5.11 degrees for the compatible hard stimuli based on the described threshold adjustment, the same rotation values were used for the incompatible hard stimuli. The easy task consisted of stimuli with 40 degrees between clockwise and counterclockwise rotation for both compatible and incompatible stimuli. Following the staircase procedure, participants rested for 20 minutes with the EEG cap was prepared for the main experiment.

**Main experiment**

The trials of the main experiment were divided into 48 blocks of 56 trials each. The sequence of task difficulty (easy, hard) and stimulus compatibility (compatible,
incompatible) was randomized within each block, with 14 trials of each type (compatible easy (CE), incompatible easy (IE), compatible hard (CH), incompatible hard (IH)) presented within each block. The main experiment included one practice block of 56 trials. In total, each participant responded to 672 trials of each stimulus type. The main experiment lasted approximately 1.5 hours, including self-timed breaks.

**EEG recording**

38 channels of EEG and EOG were recorded from the scalp by a 64 channel Ag-Ag/Cl sintered electrode cap (Compumedics Neuroscan, Charlotte, NC) during the main experiment only. The SCAN 4.3 software package (Compumedics Neuroscan, Charlotte, NC) was used to record and analyze EEG. Standard 10–20 electrode sites used were FPZ, FP1, FP2, FZ, F3, F4, F7, F8, FCZ, FC3, FC4, FT7, FT8, CZ, C3, C4, T7, T8, CPZ, CP3, CP4, TP7, TP8, PZ, P3, P4, P7, P8, POZ, PO3, PO4, PO7, PO8, OZ, O1, O2, M1 and M2. During EEG recording, the physical reference electrode was approximately 2 cm posterior to CZ. Following the recording session, EEG data were re-referenced to the average of M1 and M2 (left and right mastoid). Horizontal eye movements (HEOG) were monitored by placing two electrodes lateral to the left and right orbits. Vertical eye movements (VEOG) and eye blinks were measured by placing two electrodes 1.5 cm below and above the left eye. The EEG from each electrode site was digitized at 500 Hz and filtered with a band pass of 0.1–30 Hz. A 200-ms pre-stimulus epoch of EEG was used as the baseline. Eye movement artifacts were defined as a positive or negative amplitude deflection of greater than 75µV on the HEOG or VEOG channels. Any epoch
with an eye movement artifact detected was discarded. Behavioral data (RT and accuracy of response) was merged with EEG data prior to further analysis.

**Results**

**Within Trial Results**

For within trial analysis, behavioral and electrophysiological data were analyzed using repeated measures ANOVA with the factors stimulus type (compatible vs. incompatible) and task difficulty (easy vs. hard). Trials with errors due to incorrect response or no response were discarded. For correct trials, responses within 150-650 ms of stimulus onset were considered valid. All other trials were discarded.

Behavioral and electrophysiological measures of within trial conflict processing were similar to those seen in experiment one and described earlier. Specifically, mean RT and error rate increased when either response conflict or stimulus uncertainty inducing stimuli were presented. However, based on the chance in staircase methodology in experiment three, the previously observed behavioral differences between compatible hard and incompatible hard stimuli were removed. Occipital temporal N1, an index of discrimination processing was enhanced for response conflict stimuli. Additionally, N2 showed the same sub component dissociation between eN2 response conflict activation and lN2 stimulus uncertainty activation. The scalp distributions of the eN2 (CE and IE) and lN2 (CH and IH) are presented in figure 17.
Figure 17: Experiment 3 N2 scalp topographies
Compatibility Sequence Effect Results

For compatibility sequence effect analysis, behavioral and electrophysiological data were analyzed using repeated measures ANOVA with the factors of prior trial compatibility (compatible, incompatible), prior trial difficulty (easy, hard), current trial compatibility (compatible, incompatible) and current trial difficulty (easy, hard). Based on the results of this omnibus test, interactions related to the experimental hypotheses were analyzed further.

Behavioral results

Current stimulus accuracy and mean RT for correct responses are presented in table 4.
Table 4: Experiment 3 behavioral results

<table>
<thead>
<tr>
<th>Stimulus Sequence</th>
<th>Reaction Time (ms)</th>
<th>Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SE</td>
</tr>
<tr>
<td>CECE</td>
<td>348</td>
<td>5.8</td>
</tr>
<tr>
<td>IECE</td>
<td>358</td>
<td>5.9</td>
</tr>
<tr>
<td>CHCE</td>
<td>351</td>
<td>5.7</td>
</tr>
<tr>
<td>IHCE</td>
<td>354</td>
<td>5.7</td>
</tr>
<tr>
<td>CEIE</td>
<td>390</td>
<td>7.0</td>
</tr>
<tr>
<td>IEIE</td>
<td>378</td>
<td>7.1</td>
</tr>
<tr>
<td>CHIE</td>
<td>390</td>
<td>7.1</td>
</tr>
<tr>
<td>IHIE</td>
<td>386</td>
<td>6.8</td>
</tr>
<tr>
<td>CECH</td>
<td>401</td>
<td>7.5</td>
</tr>
<tr>
<td>IECH</td>
<td>409</td>
<td>7.0</td>
</tr>
<tr>
<td>CHCH</td>
<td>397</td>
<td>7.1</td>
</tr>
<tr>
<td>IHCH</td>
<td>399</td>
<td>6.5</td>
</tr>
<tr>
<td>CEIH</td>
<td>407</td>
<td>6.7</td>
</tr>
<tr>
<td>IEIH</td>
<td>409</td>
<td>6.7</td>
</tr>
<tr>
<td>CHIH</td>
<td>403</td>
<td>6.3</td>
</tr>
<tr>
<td>IHIH</td>
<td>401</td>
<td>6.9</td>
</tr>
</tbody>
</table>

When the omnibus 2x2x2x2 repeated-measures ANOVA is run, accuracy results across the sequence conditions show main effects of prior task difficulty (F(1,17)=20.869, p<.001), prior stimulus compatibility (F(1,17)=9.936, p<.01) and current task difficulty (F(1,17)=81.057, p<.001). In addition, three first order interactions are observed; prior task difficulty by current task difficulty (F(1,17)=28.694, p<.001), prior stimulus compatibility by current stimulus compatibility (F(1,17)=28.318, p<.001), and current stimulus compatibility by current task difficulty (F(1,17)=56.494, p<.001). Two second order interactions were also observed; Prior task difficulty by prior stimulus compatibility
by current task difficulty (F(1,17)=8.392, p<.05), and prior task difficulty by prior stimulus compatibility by current stimulus compatibility (F(1,17)=19.701, p<.001).

Based on the experimental hypotheses predicting relationships between response conflict and stimulus uncertainty processing, the interaction of interest is the first order, prior task difficulty by current task difficulty interaction. In the current easy task, the easy-easy trial sequence leads to a significant prior stimulus compatibility by current stimulus compatibility interaction (F(1,17)=106.75, p<.001) which persists in the hard-easy trial sequence (F(1,17)=6.40, p<.05). Conversely, in the current hard task, the hard-hard trial sequence shows no prior stimulus compatibility by current stimulus compatibility interaction (F(1,17)=0.031, p>.05) while the easy-hard trial sequence shows a significant prior stimulus compatibility by current stimulus compatibility interaction (F(1,17)=13.59, p<.01). It is important to note that the observed easy-hard interaction is in the opposite direction than the easy-easy or hard-easy interactions. These relationships are illustrated in figure 18.
Mean RT for correct responses on the current trial show main effects of current task difficulty ($F(1,17)=51.547, p<.001$) and current stimulus compatibility ($F(1,17)=65.768, p<.001$). In accordance with the current trial accuracy results above, the same three first order interactions were observed for mean RT; prior task difficulty by current task difficulty ($F(1,17)=16.885, p<.005$), as well as prior stimulus compatibility by current stimulus compatibility ($F(1,17)=31.633, p<.001$), and current stimulus compatibility by current task difficulty ($F(1,17)=127.640, p<.001$). Additionally, two second order interactions were observed; Prior task difficulty by prior stimulus compatibility by current stimulus compatibility ($F(1,17)=9.343, p<.005$), and prior stimulus compatibility by current task difficulty by current stimulus compatibility ($F(1,17)=9.342, p<.01$).
Again, based on the experimental hypotheses, the interaction of interest is between prior task difficulty and current task difficulty. In the current easy task, the easy-easy trial sequence leads to a prior stimulus compatibility by current stimulus compatibility interaction $F(1,17)=106.75, p<.001$ which persists in the hard-easy trial sequence ($F(1,17)=10.86, p<.01$). In the current hard task, no prior stimulus compatibility by current stimulus compatibility interaction is seen in either the hard-hard task sequence ($F(1,17)=1.13, p>.05$) or the easy-hard task sequence ($F(1,17)=1.47, p>.05$). These relationships are illustrated in figure 19.

![Figure 19: Experiment 3 mean RT compatibility sequence effects](image)

*Event-Related Potential Results*

The behavioral compatibility sequence results presented above suggest that both response conflict and stimulus uncertainty can elicit conflict monitoring-related changes during subsequent easy trials. To strengthen this interpretation, it is important to
characterize not only behavioral changes, but also illustrate the neural mechanisms responsible for the observed changes. Using event related potential components associated with stimulus discrimination, it is possible to show such mechanisms.

**ERP Component Selection**

Mean amplitude windows for the ERP components of interest—occipital temporal N1, eN2, and IN2—were identified using inspection of the grand average waveforms for each condition.

**Occipital-Temporal N1 Window**

Over occipital electrodes, inspection of the grand average, stimulus-locked waveforms (figure 19) showed a clear visual N1, a negative amplitude peak bilaterally distributed with maximum negative amplitude between 140-180ms post-stimulus onset. The occipital temporal N1 was maximal at electrode PO7 across all experimental conditions, consistent with previous evidence showing larger visual N1 in the left hemisphere (Hopf et al., 2002; Vogel et al., 2000). For clarity, only amplitudes at PO7 will be reported, though results at PO8 were similar.

**Medial Frontal N2 Window**

Inspection of the grand average waveforms over medial frontal electrodes (figure 20) showed a stimulus locked, negative voltage component peaking between 200-320ms post stimulus onset with a maximum negative deflection at electrode FCZ. Visual inspection of the grand average waveforms in figure 20 suggests that there are separate sub components of the medial frontal N2 elicited by stimuli in the different task difficulties. The easy task shows an eN2 with maximum negative amplitude between
200-250ms post stimulus onset, while the hard task shows an IN2 with a maximum negative amplitude between 260-320ms post stimulus onset. Statistical analysis was completed on the each sub component window (eN2 200-250ms, IN2 260-320ms) as opposed to the overall 200-320ms window.

Occipital-Temporal N1 Results

Current stimulus elicited occipital temporal N1 at electrode P07 grand averages are presented in figure 20.
Figure 20: Experiment 3 N1 compatibility sequence effects at electrode P07
Within each figure, Blue= Prior CE; Red= Prior IE; Green= Prior CH; Yellow= Prior IH
Omnibus repeated-measure ANOVA results for mean N1 amplitude show main effects of prior stimulus compatibility (F(1,17)= 10.257, p<.01), current stimulus difficulty (F(1,17)=56.528, p<.001), and current stimulus compatibility (F(1,17)=16.513, p<.005). In addition, two first order interactions were observed; prior stimulus difficulty by current stimulus difficulty (F(1,17)=10.33, p<.01), and current stimulus compatibility by current stimulus difficulty (F(1,17)=15.75, p<.005).

Based on the experimental hypotheses predicting relationships between response conflict and stimulus uncertainty processing, the interaction of interest is the first order, prior difficulty by current difficulty interaction. Across the current easy task stimuli, a main effect of prior difficulty is seen (F(1,17)=14.671, p<.005), with the hard-easy sequence eliciting current stimulus N1s with larger negative amplitude. However, within each of the trial sequence combinations (easy-easy, hard-easy, easy-hard, hard-hard) no prior compatibility by current compatibility interactions are observed. A similar main effect of prior difficulty is not seen across the current hard task (F(1,17)=2.306, p>.05).

*Early Medial Frontal N2 Results*

Current stimulus-elicited eN2 grand averages are presented in figure 21.
Figure 21: Experiment 3 eN2 compatibility sequence effects at electrode FCZ
Within each figure, Blue = Prior CE; Red = Prior IE; Green = Prior CH; Yellow = Prior IH
Omnibus repeated-measures ANOVA results for mean eN2 amplitude at electrode FCZ show a main effect of current stimulus compatibility (F(1,17)=7.688, p<.05). In addition, a first order current stimulus compatibility by current stimulus difficulty interaction (F(1,17)=9.213, p<.01) and a second order prior stimulus compatibility by current stimulus compatibility by current stimulus difficulty interaction (F(1,17)=5.078, p<.05) are observed.

The observed current stimulus compatibility by current stimulus difficulty interaction is driven by an increase in negative amplitude for the eN2 for incompatible stimuli in the easy task condition. This result is in line with predictions that early N2 is an index of response conflict monitoring. Also of note is the absence of a prior task difficulty by current task difficulty interaction, which shows the eN2 is insensitive to modulations of prior or current trial stimulus uncertainty.

*Late Medial Frontal N2 Results*

Current stimulus-elicted lN2 grand averages are presented in figure 22. Omnibus repeated-measures ANOVA results for mean lN2 amplitude at electrode FCZ show main effects of current stimulus compatibility (F(1,17)= 5.237, p<.05) and current task difficulty (F(1,17)=14.437, p<.005). In addition, two first order interactions are observed; prior stimulus difficulty by current stimulus difficulty (F(1,17)=7.360, p<.05), and current stimulus compatibility by current stimulus difficulty (F(1,17)=9.857, p<.01). A single second order interaction is also observed; prior stimulus difficulty by prior stimulus compatibility by current stimulus difficulty (F(1,17)= 4.757, p<.05).
Based on the experimental hypotheses predicting relationships between response conflict and stimulus uncertainty processing, the interaction of interest is the first order, prior difficulty by current difficulty interaction. This interaction is driven by the presence of an effect of prior stimulus difficulty when processing current easy stimuli (F(1,17)=8.177, p<.05) that is absent when processing current hard stimuli (F(1,17)=0.671, p>.05). Within the current easy task, hard-easy sequence stimuli have a reduced (more positive) mean amplitude for lN2 compared to easy-easy sequence stimuli for both the compatible and incompatible easy stimuli.

Discussion

The third experiment compared both the within-trial and trial-to-trial processing of response conflict and stimulus uncertainty in an Eriksen flanker task using behavioral
and electrophysiological measures. The primary focus of the experiment was to characterize the compatibility sequence effects elicited by prior trial processing of response conflict or stimulus uncertainty on the current trial processing of conflict. Given the within trial sensitivity of the conflict monitoring circuit to both response conflict and stimulus uncertainty described in experiment one, it was predicted that prior exposure to either response conflict or stimulus uncertainty would facilitate the processing of current trial conflict. Experimental results were generally in line with these predictions, with some important caveats concerning the processing of the current task when it required a hard discrimination (high stimulus stimulus uncertainty).

Within trial conflict processing results replicated those of experiment one. However, the task difficulty conditions were not blocked in the current experiment. In experiment three, both response conflict and stimulus uncertainty elicited predictable behavioral and electrophysiological responses in line with the activation of a medial frontal conflict monitor. This suggests that the interleaved task design of experiment three did not fundamentally change the processing of response conflict or stimulus uncertainty. The persistence of the modulation of late medial frontal N2 to stimulus uncertainty also supports the interpretation of within trial processing described previously.

Based on the persistence of within trial effects, the analysis of experiment three included an examination of the modulation of compatibility sequence effects related to the difficulty and compatibility context instantiated by previous trial processing. Trial-to-trial results illustrate the limits of the similarities between response conflict and stimulus
uncertainty monitoring. Behavioral results from the easy-easy task sequence replicated the previously described (Gratton et al., 1992) compatibility sequence effects of prior exposure to response conflict inducing stimuli. Prior incompatible easy stimulus presentation lead to facilitation of the processing of current incompatible easy stimuli when compared to prior compatible easy stimulus presentation.

The current results showed that in addition to the expected compatibility sequence effects observed in the easy-easy task sequence, prior exposure to hard compatible stimuli also lead to behavioral facilitation in the processing of current incompatible easy stimuli. The error rate of current incompatible easy stimuli was lower when preceded by a compatible hard stimulus than when preceded by a compatible easy stimulus. Importantly, this decrease in error rate was not accompanied by an increase in mean RT, showing that the improved accuracy following stimulus uncertainty processing was not due to a speed accuracy tradeoff related to the generally slower responses to hard task stimuli.

This important finding illustrates a cross-task facilitation of conflict processing. The characterization of stimulus uncertainty and response conflict monitoring as different sub processes is validated by the difference in eN2 and lN2 modulation between the response conflict and stimulus uncertainty inducing stimuli (incompatible easy and compatible hard, respectively). As was discussed in experiment one and replicated in the within trial analysis of experiment three, dissociable N2 modulations are associated with the two types of conflict instantiated by the stimuli. This dissociation of eN2 and lN2 sensitivity is consistent with the presence of separate conflict monitoring sub processes.
Previous evidence (Egner et al., 2007) has shown a lack of facilitation across separate forms of conflict monitoring. However, based on the results of experiment three, prior processing of stimulus uncertainty shows behavioral signatures of the facilitation of current response conflict processing. This suggests that while the two types of conflict are monitored by separate sub processes, these processes do overlap to a degree. The similarity between the monitoring processes manifests itself in the compatibility sequence effects observed in the hard-easy task sequence.

While the currently described facilitative behavioral effects of prior stimulus uncertainty did not reach the level of prior exposure to response conflict, they were still significantly greater than with prior exposure to non-conflict inducing, compatible easy stimuli. As such, the hard-easy task sequence results of experiment three appear to be in conflict with the absence of cross-task facilitation previously reported.

The use of ERP results to aid in the interpretation of trial-to-trial behavioral results illustrates the value of using electrophysiology to better characterize the processes involved in conflict monitoring. Building on the within trial ERP results, it is important to look at the compatibility sequence effects seen in these same three ERP components; the occipital temporal N1, eN2 and lN2.

The stimulus differentiation processing indexed by occipital temporal N1 further defines the theoretical relationship between executive and sensory processing. Independent of prior trial difficulty, current easy stimuli elicit larger amplitude N1s than do current hard stimuli. This is consistent with evidence presented by Lavie et al. (2005) showing increases in stimulus differentiation with reduced visual load.
However, prior exposure to stimulus uncertainty leads to an increase in current easy trail N1 amplitude. The main effect of prior task difficulty on N1 amplitude suggests that prior exposure to stimulus uncertainty reduces stimulus load more than prior exposure to response conflict. This prior task difficulty effect on occipital temporal N1 is absent in current hard trials, perhaps due to the psychophysically derived hard stimuli approaching the capacity of the differentiation process for each participant. That is, in the hard task, current stimulus uncertainty is still too high to allow for full differentiation among targets and flankers, regardless of the previously instantiated context, facilitative or not.

The increased magnitude of easy trial occipital temporal N1 following exposure to stimulus uncertainty suggests that an increase in the N1-indexed attentional differentiation process is the compensatory processing elicited by the cognitive control circuit in response to prior exposure to stimulus uncertainty. This is in line with the two-part cognitive control process (Botvinick et al., 2001; 2004) which predicts the presence of both monitoring and compensatory attentional allocation sub processes.

In addition, the predictable increases in attentional allocation to stimulus discrimination following exposure to stimulus uncertainty are in line with the top-down control of visual attention characterized in the biased completion model (Desimone and Duncan, 1995; Kastner and Ungerleider, 2001). Finally, the observed N1 increase is consistent with previous work implicating momentary increases in attention as the neural mechanism responsible for behavioral compatibility sequence effects (Egner et al., 2010).

Thus, the occipital temporal N1 results described in experiment three provide empirical
evidence that is directionally consistent with theories of both biased competition in sensory processing and cognitive control.

In addition to the described modulations of occipital temporal N1-indexed stimulus differentiation, the current trial N2 results illustrate a dissociation based on prior trial conflict processing similar to that seen in the within trial results of experiments one and three.

These dissociations further strengthen the interpretation that stimulus uncertainty and response conflict are monitored by related, but separate conflict monitoring sub processes. Context mediated changes in N2 modulation are observed only for IN2, not e N2. The e N2 shows increased amplitude only in response to current easy incompatible stimuli. This effect is insensitive to prior stimulus uncertainty, and current hard stimuli show an eN2 amplitude similar to that of easy compatible stimuli. This suggests that, as is the case in within trial analysis, e N2 indexes response conflict monitoring processes.

It is surprising that the behavioral compatibility sequence effects seen in the easy-easy trial sequence are not reflected in the e N2 results. Potentially, the current experimental paradigm is insufficient to elicit modulations of current trial eN2. This is consistent with previous N2 literature, which to date has failed to characterize ERP modulations consistent with the observed behavioral (Gratton et al., 1992) or fMRI (Botvinick et al., 1999) compatibility sequence effects.

In contrast to the e N2, the l N2 is modulated by prior task difficulty, as evidenced by the observed prior stimulus difficulty by current stimulus difficulty interaction. For current easy trials, IN2 shows a reduced amplitude when preceded by a hard stimulus.
This prior task difficulty modulation is absent when processing current hard stimuli. The context-dependent dissociation in IN2 sensitivity further illustrates the differences between the cognitive processes indexed by eN2 and lN2 respectively. While the current paradigm does not elicit context dependent modulations of eN2, context dependent modulations of lN2 are clearly present.

Furthermore, these lN2 modulations are consistent with the predictions of conflict monitoring theory. The sequence of modulations of attentional and conflict monitoring indexing ERP components from the prior trial through the current trial illustrate a consistent predictable relationship between sensory and executive processing.

In the case of the hard-easy task sequence, the hard prior trial exhibits a reduced stimulus differentiation as well as an increased medial frontal activation modulated by stimulus uncertainty. The reduced stimulus differentiation is seen in the decreased amplitude of occipital temporal N1. The increased medial frontal activation is seen as an increase in lN2.

The context instantiated by the prior hard trial can be observed in modulations of the same cognitive processes in the subsequent easy trial in the hard-easy sequence. The subsequent easy trial exhibits an increase in stimulus differentiation and a decrease in stimulus uncertainty sensitivity. Occipital temporal N1 is increased following a hard trial and lN2 amplitude is decreased. Notably absent in this sequence is any modulation of response conflict monitoring, indexed by the eN2.

Taken together, this series of ERP modulations describes a sequence of changes in the conflict monitoring and compensatory attentional allocation processes described by
conflict monitoring theory and biased competition of attention. Specifically, the prior sensitivity to stimulus uncertainty elicits an compensatory increase in attentional allocation to stimulus differentiation that does not influence response conflict monitoring but reduces the subsequent stimulus uncertainty. This sequence ultimately facilitates behavioral performance without an apparent speed accuracy trade off.

Taken together, the relationship among prior trial conflict monitoring, current trial attentional processing, current trial conflict monitoring, and current trial behavioral performance begins to illustrate, using an electrophysiological measure, the mechanism responsible for the compatibility sequence effects described in both conflict monitoring theory and the biased competition model of attention.

While the current easy trial results reported correlate with the predictions of both conflict monitoring theory and biased competition model of attentional allocation, the results for current hard trial processing do not. In fact, the cross task behavioral compatibility sequence effects observed in the hard-easy task sequence were absent or reversed in the easy-hard sequence. Specifically, prior exposure to response conflict did not facilitate a decrease in error rate when processing current stimulus uncertainty. The behavioral data reported showed that prior response conflict inhibited accurate processing of subsequent stimulus uncertainty.

Also of note, none of the ERP components of interest showed any significant compatibility sequence effects in either the hard-hard or easy-hard task sequences. This suggests that the difficulty of stimulus discrimination may have overridden the observation of any predictable mechanistic compatibility sequence effects.
Characterization of the neural processes responsible for these differences is beyond the scope of the current experiment.

However, the lack of bi-directional compatibility sequence effects between response conflict and stimulus uncertainty monitoring can be interpreted as a further indication of the dissociation between these two processes. While prior stimulus uncertainty facilitates current response conflict processing, prior response conflict hinders current stimulus uncertainty processing. Based on the design of the current experiment, it is unclear if the observed reversal in compatibility sequence effects is due to the additional load of the hard task, or due to an unanticipated relationship between the response conflict and stimulus uncertainty monitoring sub processes of cognitive control.

In any case, the current findings are inconsistent with a unitary, executive conflict monitoring process in the medial frontal cortex. Instead, the executive conflict monitoring process may be constructed of related sub processes indexed by sub components of the N2 and likely localized within the MFC in much the same way as the compensatory attentional mechanism is constructed of related sub processes in the dLPFC (Badre and Wagner, 2004). The effect of conflict type order suggests that there are differences between the monitoring and compensatory processes associated with response conflict and stimulus uncertainty.

The results of experiment three illustrate a predictable relationship between executive and sensory processes in the easy-easy and hard-easy task sequences. That is, the top down influence of stimulus uncertainty on attentional modulation of differentiation. By manipulating stimulus uncertainty psychophysically, the current
method allows for modulation of a previously-described attentional mechanism (occipital temporal N1) that correlate with prior sensitivity in the IN2 to stimulus uncertainty.
CHAPTER 5: General Discussion

The three experiments of this dissertation were designed to explore the relationship between conflict instantiated at different levels of information processing and activation of the medial frontal cortex conflict monitoring circuit, as indexed by ERP components such as the frontal N2. The use of modified Eriksen flanker stimuli, psychophysically tuned to each participant individually, allowed for the controlled elicitation of both response conflict and stimulus uncertainty. By monitoring the behavioral and electrophysiological responses to both types of conflict during a given trial as well as on a trial-to-trial basis, the results illustrate a number of related conclusions which serve to better characterize both medial frontal cortex mediated conflict monitoring and visual cortex mediated attentional biasing.

The major findings of this dissertation concern the sensitivity of the conflict monitoring circuit to stimulus uncertainty in addition to response conflict. These two sources of conflict have related, but dissociable influences on conflict monitoring processes in the human brain. Current results illustrate the presence of two subcomponents within the broader medial frontal N2 time range—the early N2 (eN2) associated with response conflict, and the late N2 (lN2) associated with stimulus uncertainty.
The IN2 can be manipulated only through careful quantification of stimulus uncertainty. The psychophysically derived stimuli in the hard tasks of the three experiments of this dissertation allow for the instantiation of sufficient levels of stimulus uncertainty to independently modulate these cognitive processes. It is speculated that previous studies failed to establish an adequate level of stimulus uncertainty to elicit similar conflict monitoring modulations.

The persistence of the dissociable effects of response conflict and stimulus uncertainty on conflict monitoring processes both within and across individual trials establishes a link between the present results and previous evidence of compatibility sequence effects. These trial-to-trial effects not only tie the present results more closely with the existing conflict monitoring literature, they also suggest an extension of the current conflict monitoring theory to include stimulus uncertainty monitoring in addition to response conflict monitoring.

The reported results are consistent with the presence of multiple, related conflict monitoring sub processes. These sub processes are sensitive to conflict throughout the information processing cascade and influence sensory processing of conflict in subsequent trials via a top-down mechanism. Together, the reported results not only more fully characterize these two important theories in cognitive neuroscience—conflict monitoring and biased competition of attentional allocation—they also strongly connect the two via empirical evidence of a biologically plausible mechanism.

Results of the three experiments showed that the behavioral responses to both stimulus uncertainty and response conflict were similar. When presented with flanker
stimuli that induced either type of conflict, participants were less likely to respond accurately and slower to respond when they did respond accurately. Both sensory evoked ERP components like occipital temporal N1 and conflict monitor indexing components like IN2 showed a sensitivity to stimulus uncertainty. These results were consistent for both within trial and between trial processing, and modulations in conflict monitoring related components persisted in the absence of overt motor requirements.

The observed electrophysiological mechanism is consistent not only with theoretical models of attentional allocation and cognitive control, but also with recent computational models, and longstanding behavioral evidence of the consequences of prior conflict on subsequent performance. The results reported describe how stimulus uncertainty elicited from early visual processing and response conflict from later processing interact within the domain of cognitive control.

Within Trial Findings

Experiments one (Chapter 2) and two (Chapter 3) were designed to characterize attentional and conflict monitoring processes within a given trial. By comparing the cognitive processes related to response conflict and stimulus uncertainty monitoring in separate blocks, these experiments illustrated an important dissociation between the two types of conflict. In both tasks, the behavioral responses to conflict inducing stimuli—whether response conflict or stimulus uncertainty-inducing—showed predictable slowing and increased error rates for both types of conflict. In addition, the ERP components monitored showed modulations of the same component amplitudes for response conflict and stimulus uncertainty. However, in the case of medial frontal N2, these observed
modulations found indexes of two sub processes of conflict monitoring that were dissociable depending on the type of conflict processed.

Response conflict and stimulus uncertainty task performance were examined in experiment one (Chapter 2). Compared in separate task blocks, both response conflict and stimulus uncertainty inducing stimuli increased the amplitude of N2 when compared to non conflict inducing (i.e. easily distinguished compatible Eriksen flanker) stimuli. However, the time range post stimulus onset where these conflict related medial frontal N2 modulations occurred was different for the two types of conflict. Response conflict modulated the eN2 (200-250ms post stimulus onset), while stimulus uncertainty modulated the lN2 (260-320ms post stimulus onset).

The suggested dissociation in experiment one is further supported by the findings of experiment two (Chapter 3). In studies of response conflict processing, the interpretation of eN2 and lN2 modulations is confounded by the overlap between executive and motor processing areas. Experiment two was designed to address this confound.

In experiment two, the motor response component was removed from the processing of stimulus uncertainty. Results showed a persistence of the IN2 in the absence of an overt motor response requirement. These findings suggest that IN2 amplitude is not exclusively modulated by motor response related processes, and thus indexes an independent cognitive process sensitive to conflict earlier in information processing. This can be taken as additional evidence of a dissociation between response conflict and stimulus uncertainty processing in the MFC.
Between Trial Findings

The relationship between response conflict and stimulus uncertainty is strengthened by the findings of experiment three (Chapter 4). While the within trial results of experiments one and two suggest a dissociation between the processing of the different types of conflict studied, the trial-to-trial findings imply that the monitoring of the two types of conflict are in fact related sub processes of overall conflict monitoring. The strength of this relationship stems for the cross-task facilitation seen in trial-to-trial conflict processing.

The current processing of response conflict is facilitated by the prior processing of both response conflict and stimulus uncertainty. This finding is at odds with previous evidence showing no cross-task facilitation between different types of conflict (Egner et al., 2007). However, both the behavioral and electrophysiological results of experiment three show the presence of this facilitation. These findings thus suggest that stimulus uncertainty and response conflict, while dissociated within a given trial, do share some cognitive process overlap.

Functional Significance of medial frontal N2

The results of the three experiments of this dissertation begin to more fully describe the cognitive processes indexed by the medial frontal N2. While the N2 has previously been associated with general conflict monitoring (Yeung et al., 2004), the current evidence provides empirical proof that visual stimulus uncertainty does elicit modulations in this ERP component. To date, the majority of stimulus uncertainty
related modulation of N2 has been observed in auditory paradigms (Szmalec et al., 2008; Nieuwenhuis et al., 2004).

The current results establish an IN2 sensitivity to stimulus uncertainty (experiment one), show that this sensitivity is independent of an overt motor response requirement (experiment two), and show a facilitation between the processes responsible for stimulus uncertainty monitoring and response conflict monitoring (experiment three). These results are seen within a single trial in both blocked stimulus uncertainty and response conflict conditions and interleaved conditions, as well as on a trial to trial basis.

Floor Effects in Previous Stimulus Uncertainty Studies

The question remains as to why previous attempts to characterize visual stimulus uncertainty related N2 modulations have failed to show results consistent with those reported in this dissertation. In the visual modality, both go/nogo (Nieuwenhuis et al., 2004) and Eriksen flanker (Van Veen and Carter, 2001; 2002) paradigms have previously failed to report any N2 modulation associated with stimulus uncertainty. In each of these studies, the manipulation of stimulus uncertainty was arbitrary, possibly resulting in an insufficient level of stimulus uncertainty to activate the medial frontal cortex conflict monitoring circuit.

A number of factors lend support to the presence of floor effects in these prior studies. For example, the behavioral results of Van Veen and Carter (2002) show that their stimulus incompatibility condition (SI condition in A. of figure 22) elicited a mean RT intermediate between the no conflict (CO) and response conflict (RI) conditions. In addition, there was no difference between the accuracy of the no conflict and stimulus
incompatibility conditions. Compare these findings to those of experiment one, replicated in figure 23. In experiment one, the stimulus uncertainty inducing condition (CH) elicited mean RTs longer than the response conflict condition (IE), while accuracy for the stimulus uncertainty condition was greatly reduced when compared to both the response conflict and no conflict (CE) conditions.

Figure 23: Behavioral effects of stimulus uncertainty across studies
A. CO=no conflict; SI= stimulus incompatibility; RI= response incompatibility
B. CE= no conflict; CH= stimulus uncertainty; IE= response incompatibility
   Image source: Experiment one (Chapter 2)
The behavioral differences between Van Veen and Carter (2002) and experiment one suggest that a greater level of conflict is instantiated in the experiment one stimulus uncertainty condition than in Van Veen and Carter’s (2002) stimulus incompatibility condition. Given these behavioral differences, the increased magnitude of IN2 in the present experiments is consistent with an increase in the amount of stimulus uncertainty instantiated in the current paradigm.

The importance of establishing a sufficient level of stimulus uncertainty when examining conflict monitoring processes is underscored by evidence showing a non-linear relationship between the amount of stimulus uncertainty and the electrophysiological response indexed by IN2. For example, as discussed in chapter one, Szmalec et al. (2008) derived JNDs between auditory stimuli and showed that hard to discriminate stimuli elicited larger amplitude N2 than did easy to discriminate tone pairs. However, intermediate tone pairs—those half the distance between easy and hard to discriminate—showed no modulation of N2.

This suggests that there is a non-linear relationship between stimulus uncertainty and the degree of amplitude modulation of N2. That is, a criterion level of conflict is required to elicit any modulation. In light of the behavioral differences between Van Veen and Carter (2002) and experiment one, it is likely that the minimal level of conflict instantiated by the previous study was insufficient to exceed the required stimulus uncertainty criterion and lead to conflict monitoring circuit activation, as well as subsequent IN2 modulation.
Implications for theories of conflict monitoring

Within the N2 range (200-400ms post stimulus onset), the current results suggest sub processes index more than a single conflict monitoring process. The three experiments discussed in this dissertation combine to enhance the description of the cognitive processes indexed by the N2. Conflict monitoring theory (Botvinick et al., 2001; 2004) implicates activation of the MFC and electrophysiological evidence suggests the N2 indexes this MFC activation (Yeung et al., 2004). However, in both cases, the theoretical explanation centers on a broad, executive conflict monitor, that serves as a cognitive bottle neck where all conflict present in the brain is processed.

Contrast this interpretation with the compensatory mechanisms activated in response to the presence of conflict, and localized to the dIPFC (MacDonald et al., 2000; Kerns et al., 2004). Multiple dIPFC regions have been shown to be responsible for different types of compensatory processing (Badre and Wagner, 2004; Stephan et al., 2003). Functional specificity in the dIPFC may be reflected in the MFC as well. There is a connection between processing in the MFC and dIPFC, and there is clear functional specificity in the dIPFC. Thus, the interpretation of the results described above showing activation of related sub processes of conflict monitoring is consistent with evidence elsewhere in the human brain’s cognitive control circuit.

A number of brain imaging studies have reported evidence of separate conflict monitoring regions depending on the type of conflict instantiated by a given task. Stroop task findings (Van Veen and Carter, 2005) found semantic conflict activated more posterior regions of the ACC than did response conflict. These results suggest that
conflict monitoring, while occurring in many experimental contexts, may in fact be more widely distributed and partitioned within the medial frontal cortex. This interpretation is also consistent with the single cell findings of heterogeneous neuronal populations within the non human primate frontal cortex (Stuphorn et al., 2000; Bush et al., 2002). In addition, individual differences in the neuroanatomical partitioning of conflict monitoring sub processes may help explain the broad activation patterns seen across the MFC (Ridderinkhof, 2004; figure 1, Chapter 1) for both pre response conflict monitoring and post response outcome processing.

The dissociations between response conflict monitoring and stimulus uncertainty monitoring reported in the three experiments of this dissertation lend further support to these imaging and single cell results. By tapping the temporal sensitivity of event-related potentials, the current results illustrate a partitioning of the conflict monitoring process prior to response selection. Thus, within a single trial, the current results characterize when the different sub processes of conflict monitoring begin to diverge.

Though the evidence presented in the three experiments of this dissertation suggests a dissociation of conflict monitoring processes, there is still a clear relationship between the two sub processes. Trial-to-trial evidence from experiment three shows that prior exposure to both response conflict and stimulus uncertainty facilitates subsequent performance on response conflict inducing stimuli. This facilitation is an important finding, which leads to the interpretation that while the sub processes of response conflict and stimulus uncertainty monitoring are dissociable, conflict monitoring can still be seen as a unitary process made up of component sub processes. Thus, the theoretical
explanation posed by Botvinick et al. (2001) of cognitive control via a conflict monitoring process is accurate, if not fully descriptive of the neural events underlying it.

As discussed in previous chapters, prior evidence has shown that cross task conflict does not evoke behavioral compatibility sequence effects (Egner et al., 2007) similar to those reported by Gratton et al. (1992). A replication of these findings in experiment three would be interpreted as further support of the partitioning of conflict monitoring sub processes among tasks. However, the results of experiment three clearly show a facilitation of current response conflict processing following previous exposure to stimulus uncertainty. This finding suggests that stimulus uncertainty and response conflict are related cognitive processes.

That said, the extent of the similarity between stimulus uncertainty and response conflict monitoring has clear limits. For example, while the hard-easy task sequence showed facilitation of performance, the easy-hard task sequence did not. The absence of a bi-directional facilitation of conflict processing based on the variant of previously instantiated conflict is further support of a clear difference between stimulus uncertainty processing and response conflict processing. Given the results described in the current experiments as well the existing compatibility sequence effect literature, the trial-to-trial influence of previously processed conflict remains an area warranting further exploration.

In addition to the behavioral facilitation observed in the hard-easy task sequence of experiment three, the electrophysiological findings begin to characterize a biologically plausible, theoretically consistent mechanism for the facilitation of conflict processing. In addition to contributing to the further descriptive characterization of conflict...
monitoring theory discussed above, these electrophysiological results also provide empirical evidence of the biased completion model of attention allocation.

**Implications for the biased competition model of attention**

Given the evidence of a relationship between visual cortex and dIPFC during decision making (Heekeren et al., 2006) and in the presence of conflict (Rossi et al., 2009) as well as the connections between dIPFC and MFC in conflict monitoring (MacDonald, 2000, Carter 2000), it is hypothesized that the MFC is responsible for the monitoring signal that recruits the dIPFC to send biasing signals to visual cortex to address conflicts as described by the biased competition model (Desimone and Duncan, 1995, Duncan 1996). If this is the case, it can be expected that the MFC is sensitive not only to response conflict, but also to stimulus uncertainty.

The findings of each of the experiments of this dissertation, and specifically those of experiments one and three, show that the biased competition model is strengthened by a temporally plausible demonstration of prior conflict correlated with predictable effects on subsequent attentional allocation as well as reductions in the subsequent processing of conflict. In addition, ERP components associated with both sensory processing and cognitive control show correlated modulations consistent with the biased competition model, leading ultimately to behavioral facilitation.

Conflict monitoring theory (e.g., Botvinick et al., 2001; Kerns, 2004), strongly predicts that conflict, MFC activation, subsequent dIPFC activation, and control-related changes in performance should covary with one another. Thus, manipulations that result in
greater MFC activation should lead to greater dIPFC activation, which should lead to a
greater expression of control; conversely, manipulations that result in smaller MFC activation
should lead to smaller dIPFC activation, which should lead to a smaller expression of control.

The expression of control concerns attentional resource allocation. In the case of
response conflict, these are likely spatial attention processes used to filter out input from the
flankers. In the case of stimulus uncertainty, they are likely feature based processes used to
discriminate among stimulus alternatives.

Recent evidence has implicated attentional processes as the root cause of the
compatibility sequence effects previously described. A temporal sensitivity of behavioral
compatibility sequence effects to inter-trial interval (Egner et al., 2010) implicates
momentary increases in sensory processing mediated by attention as the prime driver of
conflict processing facilitation. While based on purely behavioral observation of reduced
compatibility sequence effects with increased inter-trial interval, these findings provide
an important empirical link between the component processes of cognitive control—
conflict monitoring and compensatory processing.

The momentary increase in attentional processes described by Egner et al. (2010)
is seen empirically in the occipital temporal N1 modulations reported in experiment three
of this dissertation. Specifically, in the hard-easy task sequence, reduced amplitude
occipital temporal N1 in response to stimulus uncertainty on the previous trial is
correlated with increased amplitude lN2 on the previous trial. This suggests a
relationship between the lack of stimulus differentiation indexed by the occipital
temporal N1 and a subsequent increase in stimulus uncertainty, indexed by the lN2.
After this insufficient stimulus differentiation on the prior trial, in the following easy trial compensatory attentional allocation leads to a reduction in conflict and a reduction in error rate without a concomitant increase in mean reaction time. The mechanism responsible for these current trial cognitive control modulations is as follows; an increased occipital temporal N1 is seen, followed by an increased eN2—elicited by the response conflict in the current trial—as well as a reduced lN2, likely correlated to the increased stimulus differentiation incurred based on the prior hard trial activation of the conflict monitoring circuit (lN2 on the prior trial) and compensatory attentional allocation (occipital temporal N1 on the current trial). Figure 24 illustrates this series of steps in the easy-easy and hard-easy trial sequences.

Figure 24: Sequence of electrophysiological responses to prior trial conflict
In this sequence of events, prior stimulus uncertainty elicits activation of the conflict monitoring circuit, which then activates compensatory processes that allocate attention on the subsequent trial, thereby reducing the incurred conflict on the current trial, improving performance. This sequence of cognitive processes is in line with both the conflict monitoring results discussed above, as well as with the proposed attentional mechanism implicated by Egner et al. (2010).

As such, the observed occipital temporal N1 modulations are coordinated with prior trial IN2 modulations, suggesting that previous exposure to stimulus uncertainty drives the subsequent attentional modulations associated with facilitated task performance. These results thus provide empirical proof of the top-down modulation of attention in the presence of previous conflict.

*Absence of Easy-Easy Task Sequence Findings*

While the data presented does provide additional evidence both supporting and clarifying the conflict monitoring theory of cognitive control, there are important limitations of the interpretation of these studies that must be noted. Among these limitations is the absence of electrophysiological evidence of trial-to-trial conflict monitoring in the easy-easy task sequence.

While the occipital temporal N1 indexes stimulus differentiation processes (Luck, 2000; Hopf, 2002) implicated in the processing of stimulus uncertainty, prior evidence implicates other sensory processes indexed by the sensory P1 for compatibility sequence effects related to response conflict processing (Scerif et al., 2006; Fedota et al., 2008). While these prior findings showing sensory P1 modulations following the processing of
response conflict in the easy task were not replicated in the current experimental design, they are still worth noting. A speculative reason for the lack of electrophysiologic evidence of top-down modulation in the processing of response conflict in experiment three may be the lack of sufficient response conflict context compared to prior examples.

In the Scerif et al. (2006) paradigm, three stimulus trials of the same conflict (three compatible or three incompatible) set the prior context, which was then supported or violated by the fourth compatible or incompatible trial. Both Scerif et al. (2006) and the replication by Fedota et al. (2008) showed that this three trial context was sufficient to elicit predictable modulations in early sensory P1 and N2 in addition to the behavioral signatures of compatibility sequence effects described by Gratton et al. (1992). Based on the absence of similar findings in the current one trial context setting paradigm, it is speculated that the previous evidence instantiated a stronger top down modulation, allowing for observation of ERP modulations.

The Yu et al. (2009) computational model and behavioral evidence in the current experiments (one and three) show behavioral signatures of these compatibility sequence effects. Perhaps the lack of sufficient context in the response conflict sequence (ie easy-easy stimulus sequence) does not allow for the observation of the corresponding attentional mechanism. This mechanism is, however, observed in the hard-easy sequence, perhaps due to the psychophysically tuned stimulus uncertainty eliciting stimuli.

The importance of the level of context set also has implications for more descriptive versions of the Yu et al., 2009 model. Their model relies on a Bayesian
adjustment of the Beta value on a trial to trial basis, but based on the strength of the context instantiated, the compatibility sequence effects may be seen in only the behavioral measures, or in behavioral and neural measure simultaneously with strongly supported context as is the case in the hard-easy task sequence of experiment three.

Alternative theoretical explanations

In addition to the evidence of stimulus uncertainty monitoring indexed by the N2 are the alternative explanations that are inconsistent with the results of these three experiments. Specifically, previous evidence has suggested that N2 indexes novelty processing (Folstein and Van Petten, 2008; Nieuwenhuis et al., 2003) and feature integration (Mayr et al., 2003; Hommel et al., 2004) as opposed to conflict monitoring. Aspects of the findings of the dissertation experiments preclude novelty and feature integration explanations when describing the cognitive processes responsible for the observed modulations of N2.

Stimulus Novelty Accounts of Medial Frontal N2

In the case of novelty processing, each of the three experiments described uses an equal number of stimulus presentations within each task condition. That is, equivalent compatible and incompatible stimuli in both the response conflict (easy) and stimulus uncertainty (hard) eliciting conditions. Thus, while the findings of prior go/nogo experiments showing N2 modulations in the presence of stimulus uncertainty (Szmalec et al, 2008; Azizian et al., 2006) can be at least partially explained by the relative novelty of the N2 amplitude increasing stimuli (Nieuwenhuis et al, 2003), that is not the case in the current three experiments.
A stimulus novelty explanation for increased medial frontal cortex activation would predict smaller N2s from the stimulus uncertainty inducing stimuli, as they are more similar and thus more difficult to tell apart than are the response conflict inducing stimuli. Milham et al. (2003) and West et al. (2004) showed that decreasing frequency of stimuli was needed for ACC activation with stimulus conflict. These findings, along with the work of Braver et al. (2001), strongly suggest that infrequent presentation is a requirement for MFC activation in the presence of non-response conflict. N2 evidence showing a sensitivity to stimulus probability (i.e., Bruin and Weijers, 2002) described above supports these stimulus uncertainty findings.

Explanations of N2 associated with stimulus novelty (Folestein and van Petten 2008; Daffner et al. 2000) are not appropriate in this case. The nature of the conflict in the hard task, which instantiates stimulus uncertainty, is based on the reduction of novelty, since the stimulus uncertainty inducing stimuli create stimulus uncertainty through their similarity, the opposite of novelty. The observation of a larger amplitude N2 in the compatible hard condition as compared to the compatible easy condition is diametrically opposed to the predictions of a stimulus novelty interpretation.

Results presented in this dissertation show that with equivalent frequency, N2 modulations can still be elicited. In the absence of stimulus novelty, the previously reported N2 modulations indexing cognitive processing of incompatibility among targets and flankers for Eriksen stimuli (Kopp, 1996) is likely to be responsible for the modulations seen in the stimulus uncertainty inducing (hard) task in experiments one, two and three.
As is the case with stimulus novelty as an alternative explanation of within trial N2 modulation, feature integration processing is an oft cited explanation for the compatibility sequence effects observed on a trial-to-trial basis. Briefly, the feature integration account of compatibility sequence effects argues that the behavioral (Gratton et al., 1992) facilitation of conflict processing and reduced MFC activation (Botvinick et al., 1999) observed following prior exposure to conflict is not related to cognitive control, but instead to the exact repetition of stimuli from trial to trial (Mayr, 2003; Hommel, 2004). That is, if participants are shown an exact replication of the previous stimulus, they will be better able to respond quickly and accurately due to stimulus priming within sensory processing, not medial frontal cortex mediated cognitive control.

Mayr (2003) showed that when exact repetition of stimuli in the incompatible-incompatible sequence is controlled for, the behavioral facilitation reported by Gratton et al., (1992) is absent. However, attempts to replicate this eradication of the compatibility sequence effect have been inconsistent with some evidence supporting feature integration (Nieuwenhuis et al., 2006) and other attempts failing to replicate Mayr’s original findings (Ullsperger, Blysma, Botvinick, 2005; Verbruggen et al., 2006).

Given these inconsistent replications in the literature, one argument to control for the issue of exact stimulus repletion on a trial-to-trial basis is to use large response sets with multiple distinct conflict inducing stimuli (Egner, 2007). However, large response sets can be problematic in neuroimaging experiments. In both ERP (Luck, 2005) and fMRI (Huettel, Song and McCarthy, 2004) paradigms, the low signal to noise ratio
necessitates many trial repetitions in order to collect usable data. Experiment duration becomes an issue when the large response sets proposed by Egner (2007) are combined with the required trial repetitions of neuroimaging. These facts make refuting the feature integration hypothesis more difficult in neuroimaging studies as opposed to behavioral studies.

However, experiment three uses different stimuli in the hard and easy conditions. Thus, differences in the compatibility sequence effects seen when switching between hard and easy flanker stimuli are not likely to be due to feature integration-based processes. Instead, the findings of experiment three point towards a conflict monitoring explanation for the compatibility sequence effects observed both behaviorally and electrophysiologically. This, along with the inconsistent replication of feature integration results suggests alternative, conflict-based explanations for the data reported.

Future Directions

The results of experiment one provide the basis for further exploration of the differences between response conflict monitoring and stimulus uncertainty monitoring processes indexed by the medial frontal N2. However, the trial to trial results of experiment three call into question the degree of overlap between the underlying conflict monitoring processes modulated by the two types of conflict.

One way to address the questions about the degree of overlap between the related subprocesses of conflict monitoring would be to employ fMRI. The results presented suggest a temporal and functional dissociation between different types of conflict
monitoring. It is anticipated that if participants completed similar in a magnet, the data would illustrate an anatomical difference in the locus of response conflict and stimulus uncertainty processing within the broader MFC. Coupled with the temporally sensitive data presented above, the additional spatial resolution of an imaging study would provide further evidence of a dissociation between response conflict processing and stimulus uncertainty processing.

Alternatively, using electrophysiological methods, one way to further characterize this overlap would be to combine the stimulus types by displaying easy to discriminate flankers along with hard to discriminate targets (figure 25).

![Figure 25: Potential combination flanker stimulus. Stimulus uncertainty target flanked by response conflict flankers](image)

The ERP modulations elicited by this combination of targets and flankers in comparison to the reported results of experiment one would allow for better characterization of the dissociation between response conflict monitoring and stimulus uncertainty monitoring. The results reported in this dissertation suggest that the two types of conflict are processed by temporally dissociable mechanisms. The proposed stimuli in figure 24 would induce both types of conflict within the same trial, which
would allow for further characterization of any interaction between the eN2 and lN2 indexed processes within the same stimulus trial.

Additionally, given the proposed importance of setting appropriate conflict context via prior stimulus presentation discussed above, modifications of the experiment three paradigm would likely be useful. Given the lack of electrophysiological evidence correlated with the behavioral compatibility sequence effects in the easy-easy task sequence, it is speculated that insufficient prior task response conflict context was created in experiment three. Previous studies using a three stimulus presentation context paradigm (Scerif et al., 2006; Fedota et al., 2008) did show response conflict based ERP component modulations consistent with the observed behavioral compatibility sequence effects.

Thus, increasing the number of context trials for both the response conflict and stimulus uncertainty conditions is predicted to elicit both those ERP modulations observed in the hard-easy task sequence of experiment three, as well as those previously observed in the easy-easy task sequence in prior studies (i.e. Scerif et al., 2006). Illustrating both mechanisms in the same participant group would aid in drawing comparisons between response conflict compatibility sequence effects, stimulus uncertainty compatibility sequence effects, and the observed cross-task compatibility sequence effects.
Final Conclusions

Cognitive control and attention allocation are among the most vital cognitive processes that occur in the brain. The three experiments of this dissertation more fully describe two major theoretical explanations of these processes, and provide a biologically plausible link between sensory processing and executive control. These findings have applications not only for the further characterization of cognitive control and attentional allocation processes, but also potentially in the identification and mitigation of dysfunction within both the executive control and attention allocation networks with the human brain.

The described link between conflict monitoring and the allocation of attention illustrates how strategic modulations in attention processes can be used to improve performance. Especially when stimuli are uncertain, enhanced selective attention plays an important role in optimal performance. Psychophysical quantification of stimulus uncertainty provides a condition where the attentional demands of the Eriksen flanker task are increased. In this condition and across multiple trials, correlations between conflict monitoring processes and attention related stimulus discrimination are observed.

Increased selective attention is needed to resolve the stimulus uncertainty and response conflict instantiated in the experiments of this dissertation. The reported results trace the cascade of cognitive processes responsible for this attentional allocation back to top-down influences from the MFC. These findings provide a biologically plausible mechanism linking frontal cognitive control processes to occipital modulations of attention. This trial-to-trial change is consistent with the theoretical explanations of both
conflict monitoring and biased completion, as well as a Bayesian computational model thought to formalize the mental calculations responsible for these findings.

The experiments described employ a straightforward methodological modification, the psychophysical quantification of stimulus uncertainty on an individual participant basis, to clarify some of the inconsistency in prior conflict monitoring literature concerning stimulus overlap. Using the Eriksen flanker paradigm, the three experiments replicate findings from other cognitive control paradigms and extend those findings into new modalities.

The results presented above suggest that the conflict monitoring process in the human brain is made up of dissociable but related sub processes. A similar partitioning of sub processes is seen in other cognitive control-related processes, such as the compensatory processing activated in response to conflict monitoring. Thus, the conclusions of this dissertation are supported by evidence elsewhere in the overall cognitive control circuit.

These results fit within the framework previously established that implicates MFC activation in higher-level decision tasks while expanding the empirical proof to lower-level cognitive processes as well as increasing the temporal resolution of the associated processing. Together, these results describe a conflict monitoring system that is truly executive—sensitive to competing cognitive representations at multiple levels of information processing—monitoring for both response conflict as well as conflict instantiated earlier in the information processing cascade.
REFERENCES


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