An Electrophysiological Investigation into the Role of Cognitive Control in the
Attentional Blink

by

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Dedication

This dissertation is dedicated to my family for all their love and support. It is especially dedicated to my husband, Brandon Keyes, the smartest person I know, who convinced me to pursue this challenge.

Abstract

Accuracy at reporting a second-target (T2) is reduced if it is presented within approximately 500 ms of the first target (T1) – an attentional blink (AB). Early models explained the AB in terms of attentional limitations creating a processing bottleneck such that T2 processing would be impaired while T1 processing was ongoing. Theoretical models of the AB have more recently been expanded to include the role of cognitive control. In this dissertation I propose that cognitive control, defined as the optimization of information processing in order to achieve goals, is maladapted to the dual-task conditions of the AB task in that cognitive control optimizes the T1 goal, due to its temporal proximity, at the cost of T2. I start with the concept that the role of cognitive control is to serve goals, and that how goals are conceived of and the degree of motivation associated with those goals will determine whether cognitive control will create the condition that cause the AB. This leads to the hypothesis that electrophysiological measures of cognitive control and the degree of attentional investment resulting from cognitive control modulate the AB and explain individual differences in the AB. In a series of four studies feedback-related N2 amplitude, (reflecting individual differences in the strength of cognitive control), and event-related and resting alpha frequency oscillatory activity (reflecting degree of attentional investment), are used to explain both intra- and inter-individual variability in performance on the AB task. Results supported the hypothesis that stronger cognitive control and greater attentional investment are associated with larger AB magnitudes. Attentional investment, as measured by alpha frequency oscillations, and cognitive control, as measured by the feedback-related N2, did not relate to each other as hypothesized. It is proposed that instead of a measure of attentional investment alone,

alpha frequency oscillatory activity actually reflects control over information processing over time, in other words the timing of attention. With this conceptualization, various aspects of cognitive control, either related to the management of goals (feedback-related N2) or the management of attention over time to meet goals, explain variability in the AB.

Keywords: dual-task costs, attention, electrophysiology, individual differences, cognitive control

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List of Abbreviations

AB – attentional blink

ACC – anterior cingulate cortex

EEG – electroencephalography

EOG – electro-oculogram

ERD – event-related desynchronization

ERN – error-related negativity

ERP – event-related potential

ERS – event-related synchronization

FRN – feedback-related negativity

RSVP – rapid serial visual presentation

T1 – first target

T2 – second target

VSTM – visual short term memory

WM – working memory

Chapter 1

General Introduction

When two targets are presented among distracters using rapid serial visual

presentation (RSVP), the ability to detect or identify the second target (T2) is reduced

when T2 is presented shortly after the first target (T1), relative to when T2 is presented at

longer lags following T1 (Raymond, Shapiro, & Arnell, 1992). So, when attending to

both targets, T2 performance increases as the presentation lag between T1 and T2

increases, and tends to reach single task target performance levels at lags longer than 500

ms; this effect on T2 performance as a function of lag is referred to as the attentional

blink (AB). The goal of this dissertation is to investigate the nature of the dual-task

attention costs that underlie the AB. Specifically, in a series of studies, various

electrophysiological measures of how cognitive resources are allocated are used to

determine both what predicts trial to trial performance in the AB paradigm, and to

account for variability among individuals in the magnitude of their AB, that is inter-

individual variability (individual differences) in dual-task attention costs.

This Introduction begins with a brief overview of the topic of selective attention,

the costs to performance that selectivity can incur in the pursuit of goals, and how the AB

paradigm assesses one aspect of those costs – temporal costs. This is followed by a brief

review of various theories of the AB, and a discussion of individual differences in AB

magnitude. The Introduction also provides an account of the general hypothesis that I test

in this dissertation, that cognitive control, maladapted to the particular dual-task context,

creates the conditions that cause the AB via inappropriate investment of attention.

Following this logic, measures of cognitive control and attentional investment should

both be associated with the AB and individual differences in the AB. Finally, in this

Introduction I explain how this hypothesis will be tested in a series of four separate

studies.

Selective Attention: Dual-Task Costs and Temporal Limitations

The selectivity of attention - that is, the allocation of information processing

resources to only a subset of the available information (Broadbent, 1958), serves the

optimal and efficient allocation of processing resources. Practically speaking, the optimal

allocation of processing resources means that relevant, but not irrelevant (i.e. distracting),

information is processed in order to influence behavior. The relevance of information is

defined by *a priori* goals. Thus, the selectivity of attention supports performing goal-

consistent behaviors.

Whereas optimization and efficiency are arguably a benefit of the selectivity of

attention, costs may also be observed. Specifically, it appears that although the selectivity

of attention may often result in the optimal allocation of processing resources, there are

exceptions. For example, should the relevant pieces of information exceed optimal

attentional capacity, some information could be prevented from influencing behavior and

one could behave in a manner not entirely consistent with goals. This is, essentially, the

circumstance created by a dual-task situation. In dual-task situations selective attention

must be optimized in the service of two goals. When the optimal configuration of

selective attention for one goal conflicts with the optimal configuration for another, it

may cost one the accomplishment of one or both goals.

For example, imagine that a critical processing resource is limited in such a way

as to optimally process only one unit of relevant information. If that critical process was

occupied when additional relevant information appears, then the processing of additionally relevant information could be delayed and/or impaired, such that it is prevented from influencing behavior in a goal-consistent manner, resulting in costs. However, should there be a sufficient temporal lag between the appearances of relevant information such that the limited processing resource was no longer occupied by the time the subsequent relevant information was encountered, then all relevant information could be accommodated and thus there would be no costs. In this manner there are temporal limitations caused by selective attention that can be revealed using paradigms such as the attentional blink (AB).

In the AB paradigm two target items are presented in a rapid serial presentation, embedded among task-irrelevant items, such that selective attention is required in order to identify or detect the target items from among the distractors. The presentation of the two targets is separated by a variable lag such that T2 follows a variable lag after the appearance of T1. The robust finding is that when T2 is presented following shorter lags (within ~500 milliseconds) after the appearance of T1, performance for identifying or detecting T2 is impaired (costs), but when presented following longer lags T2 performance is not impaired (Raymond et al., 1992). In contrast, typical T1 performance is uniformly high at all T1-T2 lags. Furthermore, T2 performance deficits at short lags are only observed when T1 requires attention; when T1 is presented, but can be ignored, T2 accuracy is equally high at all lags, ruling out perceptual explanations of the effect, and implicating attentional limitations (Raymond et al., 1992).

Therefore, the phenomenon of the AB demonstrates that when processing resources are limited in order to optimally process target information as demanded by one

task (i.e. T1) there are costs to performance on an additional subsequent task (i.e. T2).

Furthermore, it also suggests that costs to performance on a subsequent task (T2) are only

incurred for the duration that a previous task (T1) occupies the limited processing

resources. In this manner, one can use the AB as a tool to investigate the temporal cost of

selectively attending to a target.

The Attentional Blink

As outlined above, it appears that the demand for selective attention in order to

optimally process relevant information results in the conditions that cause the AB, as

opposed to, for example, limitations in the input of sensory information. This is supported

by evidence that when T1 does not require selective attention, for example if T1 is

present but no task accompanies T1, or when T1 is represented by a lack of information

in the form of a blank, the AB does not occur (Raymond et al., 1992; Shapiro, Raymond

& Arnell, 1994). Nor does it appear that the T2 accuracy cost at short lags is due to decay

in the quality of the T2 information given that the AB also occurs when T2 must simply

be detected and not identified (Raymond et al., 1992). Also, Shapiro et al. (1994) showed

that when T1 was present on only half of all trials and required a present/absent response

prior to the T2 response, an AB was observed when T1 was present, but not when T1 was

absent, even though the response requirements were the same in both tasks. Furthermore,

when the T2 task requires an immediate speeded response, response times are slower at

shorter lags indicating that the T2 costs can be reflected in slower response times as well

as errors (Jolicoeur & Dell'Acqua, 1998).

The mechanism that underlies the AB appears to be central, as opposed to

modality specific, given that the AB has been observed both when T1 and T2 were in the

same modality (visual, auditory, or tactile), as well as when T1 and T2 were presented in

different modalities from each other (Arnell & Jenkins, 2004; Arnell & Jolicoeur, 1999;

Arnell & Larson, 2002; Hein, Parr, & Duncan, 2006; Soto-Faraco, Spence, Fairbank,

Kingstone, Hillstrom, & Shapiro, 2002). The mechanism underlying the AB also appears

to be post-perceptual as both visual and semantic aspects of T2 appear to be processed

even when T2 cannot be reported at shorter lags. For example, T2 information can

produce semantic priming effects on subsequent responses even when "blinked" (i.e.

when T2 is not detected; Shapiro, Driver, Ward, & Sorensen, 1997) and blinked T2

shows an intact N400 response, which is thought to occur after semantic processing

(Vogel, Luck & Shapiro, 1998). In summary, it appears that the demands for selective

attention lead to the AB, not limitations of either sensory processing or memory

maintenance, and that the process limited by selective attention is both central and post-

perceptual. There are various theoretical accounts of the nature of this central, post-

perceptual attention-based process that produces the AB.

Theoretical Accounts of the Attentional Blink

The various theoretical accounts of the AB are similar in that there is thought to

be some limitation at a post-perceptual stage of processing caused by T1 that interrupts or

interferes with the processing of T2. One class of information processing models

proposes that processing is bottlenecked and that this processing bottleneck underlies the

AB. Specifically, in multi-stage bottleneck models of the AB (e.g. Chun & Potter, 1995;

Jolicoeur & Dell'Acqua, 1998) it is proposed that consolidating target information into

working memory is a serial process which creates a bottleneck in processing, preventing

subsequent information, including T2, from reaching the consolidation stage while T1 is

still being consolidated. This delays T2 at a vulnerable stage of processing, when the representation of T2 information is likely to decay, reducing the probability that T2 will be processed sufficiently for accurate report.

There is further specification regarding how the serial nature of encoding into working memory might be bottlenecked in Bowman and Wyble's simultaneous type, serial token model of the AB (or ST^2 theory; Bowman & Wyble, 2007). Types are representations of stimulus characteristics that are activated in parallel, tokens are the representation of an actual stimulus accessible to consciousness and are created via the binding of types with relevant episodic information (i.e. the "where" and the "when"). Tokens are what occupy visual short term memory (VSTM) or working memory (WM). The binding necessary to create tokens is a serial process, and furthermore the type representations will decay after a time. So, if the serial process of binding T1 into a token is occurring while T2 appears, the type representations relevant to T2 may then decay before the binding process becomes available (i.e. before T1 is tokenized).

More recent models of the AB, however, have shifted focus from information processing to the role of cognitive control in the AB (Di Lollo, Kawahara, Ghorashi, & Enns, 2005; Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen, Juvina, Schipper, Borst, & Martens, 2009). These models explore the role that the functions concerned with tracking and managing task goals play in creating the conditions that lead to the AB.

In the Temporary Loss of Control model (TLC; Di Lollo, Kawahara, Ghorashi, & Enns, 2005) it is suggested that an early input filter is configured initially for the efficient selection of the T1 stimulus, and that the configuration of the filter requires cognitive

control in a top-down manner. Once the T1 stimulus appears and processing of T1 begins, control of the filter is abandoned temporarily and the filter falls under bottom-up control, defaulting to the item that is presented immediately after T1. The input filter cannot be configured optimally for T2 until attention-demanding T1 processing has been completed. If T2 is presented before control of the filter is restored, this loss of cognitive control impairs selection of T2, resulting in the AB. Therefore, the TLC model implies that a lack of top-down cognitive control following T1 is responsible for the AB. Note, however, that in this model the cause of this lack of control which impairs T2 processing is due to the conditions that are initially created – that is, optimal configuration for the efficient processing of T1.

In the Boost-and-Bounce model (Olivers & Meeter, 2008) it is proposed that the presence of the T1 item elicits an excitatory "boost" for that item into working memory. That "boost", however, lasts long enough to also boost the distracter item that immediately follows T1 into working memory. Cognitive control then responds to the presence of this distracter with an inhibitory "bounce" that prevents subsequent items, including T2, from entering working memory. According to this model, an inability to prevent the inappropriate "bounce" seems to initiate the context necessary for an AB. Again, note that according to this model cognitive control is optimized in favor of processing T1, employing approaches that benefit the processing of T1 (i.e., the "boost" and the "bounce"), but which cost the processing of T2, the "bounce".

The hypothesis that some form of inhibitory cognitive control following T1 results in the AB is also seen in the Threaded Cognition model (Taatgen et al., 2009). It is suggested in the Threaded Cognition model that when T1 is encoded into memory in the

presence of distractors, a transient overexertion of cognitive control is applied to keep distractors from interfering (similar concept to the "bounce" from the Boost and Bounce model; Olivers & Meeter, 2008) and that this overly stringent control prevents detection of T2 at short lags. Taatgen et al. suggest that were this control function is not engaged, the probability of accurate T2 performance could be increased at short lags.

Olivers and Nieuwenhuis (2006) proposed an overinvestment hypothesis to account for the AB. Olivers and Nieuwenhuis (2005, 2006) observed that manipulations that diffused attention, or reduced attention to the RSVP stream, such as listening to music, thinking about a holiday, or performing an additional task simultaneously with AB trials, resulted in a reduction in the magnitude of the AB. In their overinvestment hypothesis they suggested that the AB results from an overinvestment of cognitive resources (i.e. attention). This overinvestment applies to all RSVP items, targets and distracters, allowing distractors enough cognitive resources to interfere with target representations. This interference has less impact on T1 representation due to its temporal priority. However, the overlap of T1 processing with T2 presentation at shorter lags makes T2's representation vulnerable to interference from the distractors and decreases the probability of accurate T2 performance. Manipulations, such as those employed by Olivers and Nieuwenhuis (2005, 2006), that reduce the capacity to overinvest attention during the AB task would then reduce the interference that threatens an already vulnerable T2 representation, increasing the probability of accurate T2 performance.

In all of these models some aspect of cognitive control is employed to the benefit of T1, whether it be to efficiently select it from amongst distractors (Di Lollo et al., 2005), to prevent distracting information from interfering with its representation in

memory (Olivers & Meeter, 2008; Taatgen et al., 2009), or to allocate as much attentional resources as possible to the RSVP items (Olivers & Nieuwenhuis, 2006). However, according to these models, this is an inappropriate strategy when the goal is to capture both T1 and T2, because an approach that is optimal for processing T1 could impede the processing of T2 should it arrive while processing is still configured optimally for T1.

Cognitive control models accommodate evidence showing that how the goals of the AB tasks are framed can influence the AB. For example, Nieuwenstein and Potter (2006) observed a typical AB when participants were asked to report the two red words from a short RSVP sentence, but showed that T2 performance was not impaired when participants were asked to report the whole RSVP sentence. Similarly, the AB does not occur when T1 and T2 are separated by another target item (i.e., a grouped sequence of three targets) but does occur when separated by a distractor as is typical (i.e., two separate targets; Di Lollo et al., 2005). In both these cases the goals of the AB task are reframed: Instead of two discrete goals, T1 followed by T2, they are part of a larger single goal, e.g. detect the series of targets or report a series of items. This reframing eliminates the AB, presumably due to the participant configuring cognitive control in order to accommodate a single (larger) goal rather than multiple conflicting goals.

Another way to characterize the role of cognitive control in the AB is that a subordinate goal, attending to the first target, usurps the super-ordinate goal of attending to both targets. The maintenance of multiple levels of goals from more abstract and/or temporally extended to more concrete and/or temporally proximate is a central factor of cognitive control attributed to prefrontal cortex (PFC) function (Badre, 2008). In the context of the AB, appropriate cognitive control would direct perceptual and cognitive

processes to maintain the temporally proximate goals of filtering perceptual information and eliminating interference from distractors without compromising the temporally extended goal of attending to both targets. In this case, PFC functioning should play a role in the AB, and this is reflected in neuro-cognitive accounts of the AB.

Based on a review of the literature on the neural correlates of the AB, Hommel et al. (2006) posits that both parietal and frontal components, which are suggested to work in concert, are involved in creating the conditions that lead to the AB. These fronto-parietal components are thought to exert their influence to optimize function for the temporally proximate goal of processing T1. However, this sub-ordinate goal usurps the super-ordinate goal of processing two temporally successive targets, T1 and T2. Maintaining multiple levels of goals requires that performance be monitored such that the allocation of cognitive resources can be adjusted. Monitoring and adjusting is a task largely attributed to the anterior cingulate cortex (ACC; Botvinick, Cohen, & Carter, 2004), a structure that Hommel et al. (2006) specifically mentions as responsible for the control over multiple tasks necessary when engaged in the AB task.

Cognitive Control and the Anterior Cingulate Cortex

The ACC receives information from the thalamus, giving it access to a great deal of sensory information, as well as from the PFC. It projects to various areas of the brain involved in motor functions, response selection, and affect, as well as having influence over autonomic and endocrine functions, among others (Devinsky, Morrell, & Vogt, 1995). Thus, the ACC appears perfectly situated to be the task manager of information processing in the pursuit of task goals.

According to Botvinick et al. (2004), the role of the ACC is to detect conflict due to competing response representations. In this sense, the ACC is not the source of cognitive control but rather functions to monitor how successful cognitive control is in its various forms, such as selective attention as managed by the pre-frontal cortex (PFC), and signal to appropriate areas of the cortex when adjustment is needed in order to achieve goals. This hypothesis is supported by evidence that the ACC is selectively active when conflict is present, as during an incongruent trial of a Stroop task. In contrast, the PFC is selectively active following instructions, resulting in a double-dissociation (MacDonald, Cohen, Stengar, & Carter, 2000). The authors proposed that the PFC activity following instructions represents the configuration of cognitive control, while the ACC activity represents a response to the presence of conflict. It was also observed that damage to the ACC does not impair some cognitive control functions (Fellows & Farah, 2005), which also supports the hypothesis that the ACC is not the seat of cognitive control but a necessary mechanism that provides feedback to control functions in order to maintain goal-relevant behavior. Given that the AB can be thought of as a failure to maintain multiple goals, or as the preferential ordering of goals according to temporal proximity by biasing cognitive control in favor of T1 over T2, ACC functions specifically seem a likely candidate for involvement in producing the AB.

In summary, the conditions that lead to the AB, that is selective attention in favor of T1, appear to be configured by cognitive control in the service of goals. The management of multiple goals is a function of the PFC (Badre, 2008), and the ACC, a critical structure in this frontal system, is thought to monitor and modify selective attention in order to insure that goals are met. Therefore, the functions of frontal

structures such as the ACC are good candidates for accounting not only for the AB, but also for individual differences in the AB.

Individual Differences Studies Implicating Cognitive Control in the AB

The AB is a reliable, trait-like, individual differences variable, showing good reliability over time and across tasks (Dale, Arnell, & Dux 2013; Dale & Arnell, 2013; McLaughlin, Shore & Klein, 2001). Recently, individual difference approaches to the study of the AB have shown that variability in AB magnitude among individuals is associated with variability in various measures associated with cognitive control and allocation of processing resources (i.e. the application of attention).

Recall that an important aspect of cognitive control is investing attention selectively to relevant information but not irrelevant information (Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). Dux and Marois (2008) reported that individual differences in the ability to ignore or inhibit irrelevant RSVP distractors predicted individual differences in AB magnitude such that more processing of irrelevant material was associated with larger ABs. A similar pattern was observed by Martens and Valchev (2009) who showed that greater ability to ignore or inhibit irrelevant visual displays presented beside the RSVP stream was associated with smaller AB magnitudes. Arnell and Stubitz (2010) extended these results by showing that an individual's ability to select only relevant information in a separate visual working memory task predicted their AB magnitude, such that the tendency to let irrelevant information into visual working memory predicted larger ABs. Similarly, individual scores on the Operation Span (OSPAN) task (Turner & Engle, 1989) predicted AB magnitudes such that individuals with higher OSPAN scores had smaller ABs, even when removing the contribution of

fluid intelligence (Arnell, Stokes, MacLean & Gicante, 2009, Colzato, Spape, Pannebakker & Hommel, 2007) and digit span (Arnell et al., 2009). The OSPAN task requires the participant to remember words presented with mathematical equations while the equations are being solved and stated aloud, thus providing a good measure of the ability to encode and maintain information in the face of competing information. Additionally, genetic markers of dopaminergic receptivity, associated with trait differences in the control of access of information to working memory, have also been shown to predict AB magnitude (Colzato et al., 2011).

Individual differences in state and trait positive and negative affect have also been associated with differences in the magnitude of the AB (MacLean & Arnell, 2010; MacLean, Arnell, & Busseri, 2010). MacLean et al. (2010) observed that naturally-occurring positive trait affect was associated with smaller AB magnitudes while naturally-occurring negative trait affect was associated with larger AB magnitudes. MacLean and Arnell (2010) also showed this same pattern with state affect and the AB such that state positive affect predicted smaller ABs and state negative affect predicted larger ABs. The finding of a reduction in the AB with positive affect has also been supported by Olivers and Nieuwenhuis (2006) who induced positive and negative affect using positively or negatively valenced pictures presented at the beginning of each RSVP trial. They observed that the magnitude of the AB was reduced when trials were preceded by a positively valenced picture compared to either a neutral or negatively valenced picture.

That affect can influence or predict the AB fits within the context of cognitive control models of the AB (Di Lollo et al., 2005; Olivers & Meeter, 2008; Olivers &

Nieuwenhuis, 2006; Taatgen et al., 2009), as both positive and negative affect have been shown to relate to differences in cognitive control. Positive affect has been shown to relate to increased cognitive flexibility (Dreisbach, 2006), and a broader more diffused attentional state (Fredrickson, 2001; Fredrickson & Branigan, 2005), while negative affect has been associated with a narrow and rigid attentional state (Ashby, Isen, & Turken, 1999). So, for example, consistent with the overinvestment hypothesis (Olivers & Nieuwenhuis, 2006) positive affect would be associated with a reduction in attentional investment due to its association with a broad, diffused attentional state, thus reducing the magnitude of the AB by increasing the probability that T2 would be correctly identified or detected. Negative affect would have the opposite effect, increasing investment with narrow, intense focus of attention, resulting in a larger AB. Another possibility is that positive affect might lead one to interpret the two AB goals (i.e. T1 and T2) more broadly as parts of one larger goal (i.e. detect two items rather than detect T1 then detect T2), and thus distribute attention more broadly throughout the RSVP. Negative affect, on the other hand, might lead one to focus more narrowly on the two AB tasks as separate goals, and thus attempt to apply attention more narrowly to targets only. When attention is applied more restrictively, as in the presence of negative affect, this would result in those limitations that cause the bottleneck, creating the AB. Instead, when attention is applied more broadly, that is less restrictively, the limitations that cause the bottleneck, and thus the AB, would not be enforced.

Personality traits have also been shown to predict the magnitude of the AB (MacLean & Arnell, 2010). Greater scores on extraversion and openness were both found to predict reduced AB magnitudes even over and above state affect. Further, neuroticism

predicted larger AB magnitudes, and there was a trend for conscientiousness to predict larger AB magnitudes. That personality can predict AB magnitude can also fit within the context of cognitive control models of the AB because, like affect, personality traits are associated with differences in cognitive control. Extraversion is associated with better control over working memory (Lieberman, 2000), while openness and conscientiousness have been associated with more or less cognitive flexibility respectively (Le Pine, Colquitt, & Erez, 2000). So, for example, according to the overinvestment hypothesis (Olivers & Nieuwenhuis, 2006) openness would be associated with greater cognitive flexibility, which, according to the hypothesis, reduces overinvestment and subsequently the magnitude of the AB. Thus, as was observed (MacLean & Arnell, 2010), openness should predict smaller AB magnitudes.

To summarize the points made thus far, currently, there are several cognitive control models of the AB, with all cognitive control models characterizing the AB as a result of maladaptive cognitive control processes. ACC functions are a likely candidate for the aspects of cognitive control that lead to an AB, and may predict individual differences in AB magnitude. There are currently a variety of behavioral performance measures that predict the AB and have been taken to reflect some aspect of cognitive control (e.g., OSPAN performance, WM filtering efficiency, N-back). Similarly, affective and personality traits related to individual differences in cognitive control and attentional investment can predict AB magnitude. This dissertation will extend this literature by using neural measures of cognitive control and investment to explain the AB and individual differences in the AB.

Summary and Hypothesis

As can be seen from the above discussion of cognitive control models of the AB, cognitive control is only vaguely defined in models of the AB, and what is meant exactly by cognitive control varies from model to model. However, common to all models is the idea that the application of cognitive control is inappropriate for detecting a subsequent target shortly following the first and that the AB results from this maladaptive (in terms of goals) cognitive control functioning. To clarify, while the optimization of information processing for proximal goals is an efficacious approach for many situations, in the particular conditions of the AB (i.e. a second target presented shortly after the first target) is maladaptive. This proposition is supported by evidence that individuals who invest more resources in the proximal goal, T1, have larger AB magnitudes (Shapiro, et al., 2006). I propose that ACC–driven performance monitoring and evaluation leads to adjustments of attention that are more or less effective for correct report of T1 and T2 in various tasks, situations, or for various individuals. Specifically, the logic of this dissertation is as follows: If the AB results, at least in part, from maladaptive cognitive control processes (Di Lollo et al., 2005; Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009), and the ACC is the area thought to be responsible for monitoring and adaptation of cognitive control (Botvinick et al., 2004; Cohen, Botvinick, & Carter, 2000), then individual differences in the functioning of the ACC may predict magnitude of the AB, as may individual differences in variables linked to ACC function. In particular, I propose to use a series of individual differences studies to examine the relationship between the AB and physiological variables linked to degree of attentional investment (alpha event-related desynchronization, or ERD, and resting power in the

electroencephalograph, or EEG) and ACC-driven performance modulation (the feedback-related negativity).

Study 1: ERN/FRN as Measure of ACC Driven Evaluation of Cognitive Control

The error-related negativity (ERN) is an event-related potential observed to be largest following errors in performance (Gehring, Goss, Coles, Meyer, & Donchin, 1993). The source of the ERN generator has been convincingly localized in the ACC in several different studies (e.g., Dehane, Posner, & Tucker, 1994; van Veen & Carter, 2002), and the ERN appears to be a reliable index of performance monitoring and appraisal as generated by the ACC. A related event-related potential (ERP) component, the feedback-related negativity (FRN), is observed following performance related feedback (Hajcak, Moser, Holroyd, & Simons, 2006; Yeung & Sanfey, 2004), and it has also been localized in the anterior cingulate cortex (Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997; Ruchsow, Grothe, Spitzer, & Kiefer, 2002). Similar to the ERN, it is suggested that the FRN reflects the evaluation of outcomes in the context of goals and motivations (Simons, 2010).

Following from cognitive control models of the AB, it is hypothesized that the AB is the result of mismanaged cognitive control functions. The evaluation of cognitive control functions and their subsequent adjustment in line with goals and motivations is thought to be performed by the ACC. Therefore, it is possible that ACC function would relate to the AB such that individuals with stronger performance monitoring would increase cognitive control, resulting in an increased AB. In Study 1 the ERN and the FRN were used as an index of individual differences in the evaluative function of the ACC. An Erikson flanker task was used to elicit an ERN while a time detection task adapted from

Hirsch and Inzlicht (2009) was used to elicit an FRN. The FRN was also measured following feedback on T2 performance of an AB task. I hypothesized that greater ERNs and FRNs (i.e. larger amplitude, more negative), indicative of stronger responses to errors, would be associated with an increased call for cognitive control and thus larger AB magnitudes.

Study 2: Alpha ERD as Measure of Attentional Overinvestment

It has been suggested that the inappropriate implementation of cognitive control could underlie the AB due to an overinvestment of attentional resources in RSVP items (Olivers & Nieuwenhuis, 2006). If so, then a measure of this investment should be associated with performance on the AB task.

Electrophysiological investigations of attentional investment relative to targets and distracters in an AB task support this hypothesis (Martens, Ellmallah, London & Johnson, 2006; Martens, Munneke, Smid & Johnson, 2006; Sessa, Luria, Verleger, & Dell'Acqua, 2007; Vogel & Luck, 2002; Vogel, Luck, & Shapiro, 1998). For example Martens et al. (2006b) showed that non-blinkers (individuals who reliably show no AB) had more discrete and significantly earlier P3's, an ERP thought by many to reflect WM updating (e.g. Donchin, 1981), to T1 and T2, indicating that their attentional investment in targets differed from individuals who demonstrated an AB. Non-blinkers also had significantly reduced attentional investment in distracters, as measured by activation during distracter-only RSVP trials, suggesting that they were also investing less attention in distracter items compared to individuals who demonstrated an AB (Martens et al., 2006b).

However, evidence that affective and personality traits, as well as individual differences in cognitive approach to distracting stimuli, are associated with magnitude of the AB suggests that cognitive control and attentional investment are not determined once the RSVP stream starts or within the 500 ms following T1. Instead, these results suggest that even before the RSVP task begins the degree of cognitive control or attentional investment with which an individual approaches the trial can influence the AB. This suggests that there may be a relationship between investment of attention before the RSVP trial, and T2 performance on that trial. Thus, it is possible that electrophysiological measures of attentional investment taken in advance of RSVP trials (i.e., in anticipation), and even in advance of an AB task (i.e., in readiness) could be associated with the AB.

Anticipatory attention has previously been measured using alpha band (8-12 Hz) event-related desynchronization (i.e., alpha ERD; Bastiaansen & Brunia, 2001; Bastiaansen, Böcker, & Brunia, 2002; Bastiaansen, Böcker, Brunia, de Munck, & Spekreijse, 2001; Capotosto, Babiloni, Romani, & Corbetta, 2009; Onoda, et al., 2007; Yamagashi, Goda, Callan, Anderson, & Kawato, 2005). There is evidence to suggest that alpha ERD captures anticipatory attentional investment in preparation for a relevant stimulus (Bastiaansen & Brunia, 2001; Bastiaansen et al., 2002; Brunia & van Boxtel, 2001). Therefore, it is possible that alpha ERD measured during the period preceding trials of an AB task could capture the level of anticipatory attentional investment that would be expected to influence the performance outcome of those trials.

The goal of Study 2 was to investigate attentional overinvestment as a result of maladaptive cognitive control, which underlies the AB as proposed by Olivers and Nieuwenhuis (2006). Alpha ERD was used as a measure of pretrial attentional

investment. It was hypothesized that alpha ERD before the RSVP stream would be greater when T2 was incorrectly reported at the short lag than when correctly reported at the short lag, providing evidence that increased attentional investment impaired T2 accuracy during the AB interval. This pattern, however, would not be expected when T2 was presented at a longer lag. Thus, attentional investment before the RSVP stream would predict which trials would and would not generate an AB.

Study 3: Resting Alpha as Measure of Attentional Overinvestment

Oscillatory activity in the alpha range of frequencies (8-12 Hz) is characteristically observed in the resting state. Specifically, alpha range oscillatory activity is increased during periods of rest with eyes closed compared to periods of rest with eyes open, an effect which is attributed to the desynchronizing effect of visual stimulation on the cortex (for a review of alpha oscillations see Niedermeyer, 1997). The presence of alpha in the waking state is thought to denote an "idling" or relatively unoccupied cortex (Pfurtscheller, et al., 1996), or alternatively, the presence of cortical inhibition (Klimesch, et al., 2006; Klimesch, et al., 2007). Indeed, levels of alpha are related to attentional investment such that when alpha is high, attention is not engaged and the cortex is "idling" (Brunia & van Boxtel, 2001; Pfurtscheller, et al., 1996) or inhibited (Klimesch et al., 2007).

The goal of Study 3 was, similar to Study 2, to investigate attentional overinvestment as a result of maladaptive cognitive control, which underlies the AB as proposed by Olivers and Nieuwenhuis (2006). Power in the alpha band while at rest was used to measure the degree of attentional investment while at rest. In contrast to alpha ERD, a measure of anticipatory attentional investment such that greater anticipatory

investment means more investment in the task, less attentional investment at rest is indicative of an 'idling' state, a state of readiness. Thus, while a larger AB was predicted to be associated with greater alpha ERD in Study 2 (i.e. greater investment in the task), in Study 3 I hypothesized that AB magnitude will be associated with less attentional investment at rest, indicative of a state of readiness to process information (i.e. invest attention). Specifically, since greater readiness to invest attention is associated with greater levels of alpha at rest, AB magnitude should correlate positively with alpha power at rest.

Concluding Summary

The selectivity of attention serves the optimal allocation of processing resources to goal-relevant information. In the dual-task situation of the AB, the typical configuration of processing resources for T1 impedes the processing of T2, while T1 occupies critical, but limited, processing resources (Raymond et al., 1992). Recent models of the AB have emphasized the importance of cognitive control (Di Lollo et al., 2005; Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). Thus, measures related to the goal-management functions of cognitive control (Botvinick et al., 2004) should be associated with the AB. Thus, in Study 1 (Chapter 2) I hypothesize that the ERN and FRN, both thought to be generated by the goal monitoring and management functions of the ACC, will predict individual differences in AB magnitude, such that stronger responses will be associated with larger ABs. One possible result of the maladaptive strategies enforced by cognitive control that creates the conditions for the AB is an inappropriate overinvestment of attention (Olivers & Nieuwenhuis, 2006). Thus in Study 2 (Chapter 3) I investigate whether measures of on-task investment

(anticipation), as measured by alpha ERD, explain variability in the AB. In Study 3 (Chapter 4) I investigate whether at-rest investment (readiness), as measured by alpha power at rest, can explain variability in the AB.

In an additional study, I collected measures of AB, FRN, alpha ERD, and resting EEG data from a new set of participants. This data was used to (1) attempt to replicate the findings of Studies 1-3, and (2) examine the inter-relations among the measures of cognitive control and attentional investment. Chapters 2-4 will each conclude with an additional section that includes the replication analyses using the data collected in the additional study. Chapter 5 will address the inter-relations among the predictors of Study 1-3 using the new sample for which all measures were recorded. Finally, the General Discussion section (Chapter 6) will contain an integrated discussion of the findings as a whole, and summarize what the present work can tell us about cognitive control and the AB.

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Preface to Chapter 2

Note that Chapter 2, excluding the replication study, is a published manuscript:

MacLean, M.H., & Arnell, K.M. (2013). Individual differences in electrophysiological
   responses to performance feedback predict AB magnitude. *Cognitive, Affective,
   and Behavioral Neuroscience, 13*, 270-283.

Chapter 2

Introduction

The Attentional Blink

When two to-be-attended targets are presented in a rapid serial visual presentation (RSVP) stream, accuracy for the second target (T2) is reduced when it is presented within 500 ms after the first target (T1), relative to longer T1-T2 separations (Raymond, Shapiro, & Arnell, 1992). This phenomenon has been named the attentional blink (AB; Raymond et al., 1992). The magnitude of the AB can be captured in the change in T2 accuracy as a function of the temporal separation or lag between T1 and T2, controlling for overall T2 accuracy (MacLean & Arnell, 2012). The AB has traditionally been interpreted as reflecting attentional limitations such that attentional processing of T1 interferes with and/or delays the allocation of attention to T2, if T2 is presented before T1 processing has been completed (e.g., Chun & Potter, 1995; Jolicoeur & Dell' Acqua, 1998; Shapiro, Arnell & Raymond, 1997). However, several recent models of the AB have implicated cognitive control as influential to the production of the AB.

The Attentional Blink and Cognitive Control

Several models of the AB such as the Temporary Loss of Control (TLC) model (Di Lollo, Kawahara, Ghorashi, & Enns, 2005), the Boost-and-Bounce model (Olivers & Meeter, 2008), the Threaded Cognition model (Taatgen, Juvina, Schipper, Borst, & Martens, 2009) and the Overinvestment hypothesis (Olivers & Nieuwenhuis, 2006) feature cognitive control as central to the presence of the AB. In several of these models strong cognitive control would predict an increased AB. For example, in the Boost-and-Bounce model (Olivers & Meeter, 2008) it is proposed that selection of the T1 item leads

to an excitatory 'boost' that carries-over to the distractor immediately trailing T1. In response to the incorrect boosting of the distractor into working memory, cognitive control is used to initiate an inhibitory 'bounce' that prevents subsequent items, including T2, from inclusion into working memory. Similarly, according to the Threaded Cognition model (Taatgen et al., 2009) selection of T1 initiates an overexertion of cognitive control (the 'memory function') which results in impaired T2 detection at short lags. Note that according to both the Boost-and-Bounce model and the Threaded Cognition model, better goal-driven cognitive control leads to larger ABs because greater cognitive control exerted to protect T1 identification comes at the cost of T2 identification at short lags. While it may seem counterintuitive that better cognitive control may predict larger ABs, there are data to suggest that somewhat distracted performance (due to the need to perform an additional task) may decrease the AB by reducing attentional investment in RSVP items (Olivers & Nieuwenhuis, 2005, 2006), and that the probability of failing to correctly identify T2 is predicted by investment in T1 (Shapiro et al., 2006). The Overinvestment hypothesis posits that the typical goal-dedicated, but inappropriate, overinvestment of attention (compared to what is needed) actually increases the AB by allowing distractors to gain enough attention to become effective competitors for limited working memory resources (Olivers & Nieuwenhuis, 2006).

In summary, various models of the AB propose that increased cognitive control, which may be advantageous for identifying single targets, is not advantageous in an AB task when two targets are presented closely in time, resulting in an AB.

Individual Differences in the AB and Investment

The AB is not observed in some individuals, so called "non-blinkers" (Martens, et al., 2006), and individuals differ reliably in the magnitude of their AB (Dale & Arnell, 2013; Dale, Dux & Arnell, 2013; McLaughlin, et al., 2001). The degree to which individuals rigidly try to perform the two tasks in order and try hard to perform well on both tasks could predict the magnitude of their AB via the degree of attentional investment and cognitive control. Individuals who invest more in the task and in T1 may have larger AB magnitudes.

In general, studies of the AB have shown that individual difference measures that reflect, or are associated with, a flexible, diffuse, and less invested processing style (e.g., Fredrickson & Branigan, 2005; Rowe, Hirsh, & Anderson, 2007) predict smaller AB magnitudes, and measures that reflect, or are associated with, a rigid, focused, and more invested processing style (e.g., Ashby, Isen, & Turken, 1999) predict larger AB magnitudes. Individual difference measures predicting a small AB include: high state and trait positive affect and low state and trait negative affect (MacLean & Arnell, 2010; MacLean, Arnell & Busseri, 2010), high scores on the personality factors of extraversion and openness to experience and low scores on the neuroticism factor (MacLean & Arnell, 2010), flexible and effective control of working memory (Arnell, Stokes, MacLean & Gicante, 2009; Arnell & Stubitz, 2010; Colzato, Spapé, Pannebakker & Hommel, 2007), and a tendency to see the global or big picture in a display (Dale & Arnell, 2010). The importance of attentional investment to the AB has also been shown more directly using pre-trial alpha event-related desynchrony (alpha ERD) which is a measure of attentional investment at the start of the RSVP trial. Alpha ERD was higher when T1 was correct

(versus incorrect), and when T2 was correct (versus incorrect) at longer lags. However, alpha ERD was lower when T2 was correct (versus incorrect) at shorter lags, suggesting that greater pre-trial investment of attention exacerbates the AB (MacLean & Arnell, 2011).

If individual differences in AB magnitude result, at least in part, from individual differences in cognitive control and attentional investment, then measures of an individual's investment in tasks and task relevant information and their concern with their performance may predict the magnitude of their AB.

ERP Indices of Investment in Task Performance

The error-related negativity (ERN) is a negative event-related potential (ERP) observed shortly following the commission of a response, and is observed to be largest (most negative) following errors in performance (Gehring, Goss, Coles, Meyer, & Donchin, 1993). In the same study, it was observed that indices of error detection (presumably the result of monitoring) and adjustment were related to the size of the ERN. In particular, the size of the ERN increased as the force used to make the erroneous response decreased, as the likelihood that performance on the following trial would be correct, and as the degree of response slowing on the following trial (when correct) increased. Therefore, the ERN appears to be a reliable index of performance monitoring and appraisal. ERN amplitude can be modulated by motivational factors such that a larger ERN has been observed when accuracy was emphasized (Gehring et al., 1993). ERN amplitude was also correlated with personality factors related to reward and punishment sensitivity (BIS/BAS) such that individuals high in the BIS factor tended to have larger ERN amplitudes (Boksem, Tops, Wester, Meijman, & Lorist, 2006). Neuroticism and

conscientiousness have also been found to predict the magnitude of the motivational

effect on ERN amplitude (Pailing & Segalowitz, 2004). Greater neuroticism was

associated with a greater increase in ERN amplitude as the incentive for accuracy

increased, while conscientiousness showed the opposite pattern. Thus the size of the ERN

appears to be related to investment in tasks and task performance.

The ERN is often measured as the difference in amplitude to the negative ERP

(which I will refer to as the medial-frontal negativity or MFN) that shortly follows the

commission of incorrect responses from that following the commission of correct

responses. This difference measure reflects the accuracy-related modulation of the MFN.

Should AB magnitude correlate positively with this difference measure that would

indicate that individuals who are more responsive to incorrect performance *relative* to

correct performance have larger AB magnitudes. Given that I do not hypothesize that the

relationship between the strength of response to performance and AB magnitude should

be confined to the accuracy-related modulation of the MFN, I hypothesize that greater

AB magnitude will be related to a stronger response to performance in general (i.e.

larger, more negative MFNs) regardless of accuracy.

ERN amplitude has good test-retest reliability, indicating that ERN amplitude is a

good candidate for a biological correlate of cognitive traits (Segalowitz, Santesso,

Murphy, Homan, Chantziantoniou, & Khan, 2010). The evidence that ERN amplitude

correlates with personality factors also suggests that ERN amplitude reflects a trait

(Boksem, Tops, Wester, Meijman, & Lorist, 2006; Pailing & Segalowitz, 2004). That

ERN amplitude differs according to motivational effects (Gehring et al., 1993), and can

differ according to subsequent behavioral adjustments (Rodriguez-Fornells et al., 2002),

suggests that ERN amplitude could reflect trait-like differences in the degree of investment in tasks and concern about performance (i.e. motivation).

In addition to examining responses to actual performance, as is reflected in the ERN, it is also possible to examine responses to information relevant to performance (i.e. feedback). The feedback-related negativity (FRN), is the second negative going deflection (N2) observed following feedback, and is typically observed to be larger following feedback that indicates that an error has occurred (Hajcak, Moser, Holroyd, & Simons, 2006; Yeung & Sanfey, 2004). Some evidence suggests that the FRN is the same component as the ERP referred to as the N2, which can be observed following rare or "odd-ball" stimuli (Holroyd, Pakzad-Vaezi, & Krigolson, 2008). Similar to the ERN, it is suggested that the FRN reflects the evaluation of outcomes in the context of goals and motivations (Simons, 2010).

The amplitude of the FRN has been shown to mirror the magnitude of the prediction error, as the likelihood of the negative outcome decreases (i.e., negative outcome not expected) the amplitude of the FRN to the negative outcome increases (Bellebaum & Daum, 2008). However, this modulation of FRN amplitude with magnitude of prediction error was only observed in individuals who learned strategies for maximizing reward in a learning task. Furthermore, FRN amplitude is smaller following mistakes when on a subsequent trial participants made the same erroneous response than when participants later made a different response (Cohen & Ranganath, 2007; van der Helden, Boksem, & Blom, 2010). Neuroticism also predicts FRN amplitude. Specifically, for individuals high in neuroticism the FRN following uninformative feedback was larger than that observed following negative feedback (Hirsch & Inzlicht, 2008).

Most often the FRN is measured as a difference measure obtained by subtracting the components evoked by correct/positive feedback from those evoked by incorrect/negative feedback. This difference measure reflects the valence-related modulation of the N2, specifically, that the amplitude of the N2 following feedback is more negative (larger) following incorrect/negative feedback than following correct/positive feedback. Should AB magnitude correlate positively with this difference measure that would indicate that individuals who are more responsive to incorrect feedback *relative* to correct feedback have larger AB magnitudes. Other individual differences have been related to differences in the valence-related modulation of response to feedback (e.g. Foti & Hajcak, 2009; Hirsch & Inzlicht, 2008).

However, the N2 following feedback stimuli is not modulated only by valence; for example it is also modulated by the magnitude or value of reward stimuli (Santesso et al., 2008) and the degree to which negative feedback is expected (Bellebaum & Daum, 2008; Hajcak et al., 2007). So, it appears that the N2 is a component sensitive *in general* to the performance-relevant information contained in feedback. The purpose of the difference measure is to eliminate the components commonly evoked by correct and incorrect feedback, for example those related to the frequency, expectedness, and value of the feedback, leaving only those related to the valence of the feedback (Holroyd & Krigolson, 2007). Given that I do not hypothesize that the relationship between the strength of response to feedback and AB magnitude should be confined to the valence-related modulation of the feedback-related N2, I hypothesize that greater AB magnitude will be related to a stronger response to feedback in general (i.e. larger, more negative N2s) regardless of valence. Other individual differences have been related to the general

response to feedback (i.e. a main effect, regardless of feedback valence; e.g. Tucker et al., 2003; Santesso, Dzyundzyak, & Segalowitz, 2011).

The amplitude of the N2 components to positive and negative feedback (which are used to measure the FRN) have good test-retest reliability, indicating that N2/FRN amplitude is also a good candidate for a biological correlate of cognitive traits (Segalowitz et al., 2010). The evidence that FRN amplitude correlates with a personality factor also suggests that FRN amplitude reflects a trait (Hirsch & Inzlicht, 2008). That FRN amplitude differs according to the magnitude of the prediction error (Bellebaum & Daum, 2008) and subsequent behavioral adjustments (Bellebaum & Daum, 2008; Cohen & Ranganath, 2007; van der Helden, Boksem, & Blom, 2009) suggests that FRN amplitude could, like ERN amplitude, reflect trait-like differences in the degree of investment in task and performance.

Although both the FRN and ERN are components thought to reflect performance monitoring functions, the FRN is generated when external signals regarding outcomes are necessary, whereas the ERN occurs when outcomes can be determined without external signals (i.e. are internally generated; Müller, Möller, Rodriguez-Fornells, Münte, 2005). Consistent with this distinction, there is evidence that the FRN and ERN are the result of different cortical generators (Gehring & Willoughby, 2002; Potts, Martin, Kamp, & Donchin, 2011). As our hypotheses concern performance monitoring in general we do not make specific hypotheses regarding whether the relationship between the AB and performance monitoring functions will differ according to whether performance outcomes were determined internally (ERN), or required external feedback (FRN).

The Present Study

It has been suggested that cognitive control and the overinvestment of attention contribute to the production of the AB (Di Lollo et al., 2005; Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). Thus, the degree to which individuals invest in task performance may predict individual differences in AB magnitude. The ERN (and its MFN components) and the FRN (and its N2 components) are thought to reflect the responses to performance outcomes in the context of expectations and goals. ERN amplitude has been shown to reflect the strength of incongruence between expected/correct behavior and the context of erroneous outcomes (e.g. Danielmeier et al., 2009; Rodriguez-Fornells et al., 2002). FRN amplitude has been shown to reflect the degree to which individuals adopt and enforce strategy (i.e. invest in tasks) to achieve goals (Bellebaum & Daum, 2008; van der Helden, Boksem, & Blom, 2009). Thus, I hypothesize that both MFN and feedback-N2 amplitude will predict AB magnitude such that greater MFN/N2 amplitude will be associated with larger AB magnitudes. It is also possible that the valence modulation of the MFN/N2 components (i.e. the "ERN" and "FRN") will also predict AB magnitudes, in which case we would hypothesize that larger valence modulations (larger ERNs/FRNs) would be associated with larger AB magnitudes.

In this study I will measure the response-locked ERN and its component MFNs generated by a Flanker task, and the stimulus-locked FRN and its component N2s generated by feedback in two separate tasks: an AB task and a time production task. The purpose of measuring the feedback-related N2 from two different tasks was to investigate whether any relationship between AB magnitude and feedback-related N2 amplitude was

confined to the context in which the AB is measured (i.e. the AB task), or extended to feedback in other contexts (i.e. tasks). If the relationship were confined to the AB task this would indicate that only state-dependant variability in feedback-related N2 to AB task-relevant information predicts AB magnitude. If the relationship between feedback-related N2 amplitude and AB magnitude extended to N2s measures in other tasks this would suggest that AB magnitude is predicted by variability in the trait-like response to performance-relevant information in general. I will correlate AB magnitude with the amplitude of the MFN and feedback-N2s to look for relationships between the AB and feedback responsivity. I will also correlate AB magnitude with the FRNs and ERN using incorrect minus correct trial difference measures as predictors, to see if any relationships between feedback responsivity and the AB are modulated by the valence of the feedback/accuracy of performance.

Methods

Participants. Participants were 71 Brock University undergraduate students (45 females; *M* age = 20 years), recruited through the Brock Psychology Department's online system for participant recruitment. Due to an error in the recording procedures, EEG data for the AB task was unavailable for four participants. Twenty-six participants did not complete the Flanker task which was the last task in the study.

Procedure. Participants completed first the AB task, then the time production task, and finally the flanker task. Stimuli were displayed and responses were logged using E-Prime, running on a desktop PC and presented on a CRT monitor (refresh rate of 60 Hz).

AB Task. The AB task consisted of four blocks of 60 RSVP trials; 120 were T2

absent trials (T1 only), and 120 contained both T1 and T2. When present, T2 was shown

3 items, or 252 ms, after T1 (lag 3) on half of the trials, or 8 items, or 672 ms, after T1

(lag 8) on the other half. All trial types were presented randomly within each block. T1

was always presented in white font as the 6th item in the stream. T1 was a single

uppercase letter randomly selected from among the letters M, N, P, R, S, T, U, V, W, Y,

and Z (11 AFC). T2 was the uppercase letter X always presented in black font. The

distracter items consisted of single uppercase letters always presented in black font,

randomly selected from the alphabet with the exclusion of the letters I, O, Q and X. No

letter was ever repeated sequentially. Each trial began with a fixation cross for 417 ms,

followed immediately by the RSVP stream. The RSVP stream consisted of 15 stimuli

with an SOA of 84 ms/item (no blank interstimulus interval) presented on a gray

background. At the end of each stream, participants indicated the identity of the white

letter (T1 response), and then reported whether an X was present or absent (T2 response).

Participants made unspeeded T1 and T2 responses using a keyboard in response to on-

screen prompts presented after each RSVP stream. Participants were instructed that the X

was not always present, but that when present it could appear at any point after the white

letter.

In an attempt to control the false alarm rate, participants were instructed to

indicate that an X was present only if they were "pretty sure" that they saw one.

Following the participant's T2 response was an ISI of 1,000 ms and then the presentation

of feedback for 167 ms. The feedback accurately indicated whether the participant's T2

response was correct ("Correct!" was presented following a hit or correct rejection) or

incorrect ("Incorrect!" was presented in following a miss or a false alarm). For 20

participants the feedback on both correct and incorrect trials was presented in black font.

For the other 51 participants "Correct" was presented in blue font and "Incorrect" was

presented in red font. There were no differences in group AB performance as a function

of black versus colored feedback, and feedback color did not influence any of the

relationships reported below, therefore data are collapsed across this variable. An ITI of

1,000 ms followed the feedback. Note that the use of a T2 detection task in this Study

was purposeful in order that there would be a degree of uncertainty with regards to

whether T2 performance was correct or not and thus the feedback would be meaningful.

Time Production Task. The time production task was adapted from Hirsch and

Inzlicht (2009) and consisted of four blocks of 42 trials. Each trial began with a fixation

cross (167 ms) followed by a 2,000 ms blank response period. Participants were

instructed to press the spacebar when they thought that 1 second had elapsed since the

fixation cross disappeared (i.e. that the screen had been blank). Following the 2,000 ms

blank response period, feedback was presented for 917 ms, followed by a blank ITI of

either 1,000, 1,500, or 2,000 ms. On 112 trials (2/3rd of trials) the feedback was

informative, indicating whether the participants estimate was correct ("+") or incorrect

("-"). On the remaining 56 trials (1/3rd of trials) feedback was uninformative regarding

performance ("?"). This feedback, correct, incorrect, and uninformative was presented in

white font for 20 participants, and in green ("+"), red ("-") or white ("?") for the other 51

participants. As with the AB task, feedback color did not influence time estimation

performance or any of the relationships reported below, so the data were collapsed across

this variable.

Participants were instructed before the task began regarding the meaning of the different feedback symbols. Initially an estimate was considered correct if it was presented within 100 ms of 1 second (i.e. 900-1,100 ms into the blank response period). Following a correct estimate this window decreased by 10 ms, and following an incorrect estimate the window increased by 10 ms. This procedure ensured that all participants received incorrect feedback on approximately the same number of trials (~1/2 of informative feedback trials, or 56 trials).

Flanker Task. The Flanker task consisted of four blocks of 160 trials. Each trial consisted of a Flanker stimulus presented for 117 ms followed by a 1,250 ms blank ITI before the flanker stimulus for the next trial. The Flanker stimuli consisted of a string of five uppercase letters (S's or H's) in black font presented on a gray background. On half of trials the Flanker stimulus was congruent, such that all five letters were the same. On the other half of trials the Flanker stimulus was incongruent, such that the letter in the middle of the string differed from the four flanking letters. Participants were asked to indicate what the middle letter of the letter string was by pressing either the s or h button on a keyboard. Participants were instructed to respond as quickly as possible while still being accurate. In order to increase errors, if the participant had less than 10% errors in a given block, a message was presented during the following break telling the participant that they were responding too slowly.

EEG Acquisition. EEG was recorded continuously using tin electrodes embedded in an Electro-Cap© (Electro-cap International Inc., Eaton, Ohio) from 29 sites distributed according to the 10–20 system, with an electrode placed anterior to Fz as ground. EEG was recorded using linked left and right earlobes as reference and was re-referenced to a

common average of the EEG. EEG data were acquired with Neuroscan acquisition

software (Compumedics USA, Charlotte, North Carolina), using a 32-channel NeuroScan

SynAmp. Data were sampled at a rate of 500 Hz. Electro-oculogram (EOG) recorded

horizontal eye movements using electrodes placed on the outer canthus of each eye, and

vertical eye movement and blinks using electrodes placed on the infra- and supra- orbital

regions of each eye. Impedance for both the EEG and EOG was maintained below 10 kΩ.

EEG was low-pass filtered offline at 30 Hz using a roll-off of 48 dB/oct.

FRN Analysis. For the EEG data from both the AB and time production tasks,

epochs were created beginning 200 ms before and 1,000 ms after the presentation of the

feedback. Epochs were baseline corrected using the 200 ms pre-feedback interval.

Epochs with amplitudes exceeding ± 50 µV were rejected. Epochs were then visually

examined and remaining epochs containing artifacts were also rejected. The remaining

epochs were then averaged. In the AB task, averages were created for each combination

of T2 condition (absent, lag 3, lag 8) and T2 response (correct or incorrect). Only epochs

where T1 was correct were included in these averages. In the time production task

averages were created according to the type of feedback presented: uninformative, correct

and incorrect.

Group averages were then created and examined for each task to isolate the site

where the feedback-related N2 was maximal and the time windows for defining two ERP

peaks: the positivity preceding the N2 (the P2) and the N2 used to isolate the FRN. For

both tasks ERPs were scored at FCZ where the N2 was largest. Based on visual

inspection of the average waves for each task, for purposes of scoring, the P2 was defined

as the most positive value between 160-230 ms in the AB task, and 150-250 ms in the

time production task. The N2 (AB) was defined as the most negative value between 230-300 ms in the AB task and the N2 (TIME) as the most negative value between 200-300 ms in the time production task.

ERN Analysis. Epochs were created beginning at 500 ms before and 250 ms after response. Epochs with amplitudes exceeding ± 50 µV were rejected. Epochs were then visually examined and remaining epochs containing artifacts were also rejected. Of the remaining trials only trials with RTs > 200 ms and < 1,000 ms were then averaged. Averages were created for each combination of congruency (congruent and incongruent) and performance (correct and incorrect). Group averages were then created and examined to identify the site where the performance-related medial frontal negativity (MFN) was maximal and the time windows for defining two ERP peaks: the positivity preceding the MFN (the PP) and the MFN. ERPs were scored at Cz where the MFN was largest. The PP was defined as the most positive value between -100 and -20 ms. The MFN was defined as the most negative value between -50 and 50 ms.

Results

AB Task Performance. The AB data from 13 participants were excluded due to either poor T1 performance (10 participants with < 40%) or poor T2 sensitivity at lag 8 (three participants with < 10% hits minus false alarms) on the AB task. Mean T1 accuracy for the remaining participants was 65.95% (SD = 14.13). Note that the unusually low T1 accuracy, which is still well above chance, is possibly due to the unintentional emphasis on T2 performance that the presence of T2 performance feedback introduced. T2 performance was conditionalized on T1 accuracy. Mean T2 hits at lag 3 was 47.81 (SD = 28.23), mean T2 hits at lag 8 was 81.74 (SD = 14.50), mean rate of false

alarms on T2 absent trials was 16.93 (*SD* = 12.10). Mean T2 performance (hits – false

alarms) at lag 3 was 21.09% (*SD* = 18.74). Mean T2 performance (hits-false alarms) at

lag 8 was 65.76% (*SD* = 18.14). A paired-samples t-test showed significantly lower T2

performance at lag 3 than lag 8 indicating the presence of an AB (*t* (41) = 18.71, *p* <

.001).

Individual AB magnitude was calculated as the residual difference measure. The

residual difference measure was calculated using the following steps: (1) T2 performance

on lag 3 trials was subtracted from T2 performance on lag 8 trials creating a difference

measure for each individual, (2) the difference measure was regressed onto T2

performance on lag 8 trials, (3) the standardized residual was saved, which represents the

variability in difference scores controlling for variability in baseline T2 performance (i.e.

T2 performance on lag 8 trials). *Note that larger/more positive values on the residual*

difference measure indicate larger AB magnitudes[1]. The residual measure of AB

magnitude was reliable as determined using split-half correlations (*r* = .58). A similar

residual variable (lag 8 T2 performance) was created for lag 8 T2 performance

controlling for lag 3 T2 performance. The residual measure of lag 8 T2 performance was

also reliable as determined using split-half correlations (*r* = .73).

Time Production Task Performance. The average time at which participants

indicated one second had passed was 980 ms (*SD* = 146) ranging from 348 to 1294 ms.

So, on average participants tended to prematurely indicate that 1 second had passed by 20

ms. The mean number of trials where participants received negative feedback was

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[1] The residual difference measure of AB magnitude is mathematically identical to the residual measure of lag 3 T2 performance (i.e. the residual of lag 3 T2 performance regressed on lag 8 T2 performance). I chose to use the residual of the difference measure as the measure of AB magnitude because it is more intuitive to think of larger values on the measure as indicating larger AB magnitudes, which is not the case with the residual of lag 3 T2 performance.

approximately 1/3 of the 168 trials ($M = 58$, $SD = 4$) with very little variability

(individual rates varied from 55 to 71).

   Flanker Task Performance. A paired-samples t-test indicated that accuracy on

congruent trials ($M = 85.53\%$, $SD = 10.15$) was significantly greater (t (35) = 5.75, $p <$

.001) than accuracy on incongruent trials ($M = 79.83\%$, $SD = 12.15$). Error-rates (both

congruent and incongruent trials) varied from 3-46% ($M = 17.58$, $SD = 10.81$). Reaction

time on correct congruent trials ($M = 472$, $SD = 53$) were significantly faster (t (35) =

8.92, $p < .001$) than reaction time on correct incongruent trials ($M = 493$, $SD = 53$). Thus,

the results display the typical Flanker effects.

   ERP Results. For the purposes of examining mean effects, the N2 and MFN were

estimated using a peak-to-peak measure of amplitude. N2 (for both the AB and time

production tasks) and MFN amplitude (for the Flanker task) were both calculated as the

difference in peak amplitude between the N2 or MFN and the P2 or PP that precedes it

(P2-N2; PP-MFN). Larger values on this peak-to-peak measure indicate larger N2/MFN

amplitudes.

   FRN (AB task). There was an N2 observed in the group averages and at the

individual level following the feedback in the AB task (see *Figure 2-1*). A paired-samples

t-test comparing the N2 (AB) peak-to-peak measure following correct and incorrect

feedback was significant (t (53) = 2.95, $p = .005$), indicating that the N2 (AB) was larger

following incorrect feedback ($M = 7.22$, $SD = 3.67$) than correct feedback ($M = 6.37$, SD

$= 3.14$)[2]. Note however, the sizeable N2 (AB) following correct feedback.

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[2] Note that the means for the peak-to-peak difference measures do not appear to correspond to the
difference between the means of the peaks in the grand averaged waveform (*Figures 2-1, 2-2, 2-3*). This is
because the peak-to-peak difference is calculated for each *individual*. Thus the average difference between
two points does not necessarily correspond to the difference between two averaged points.
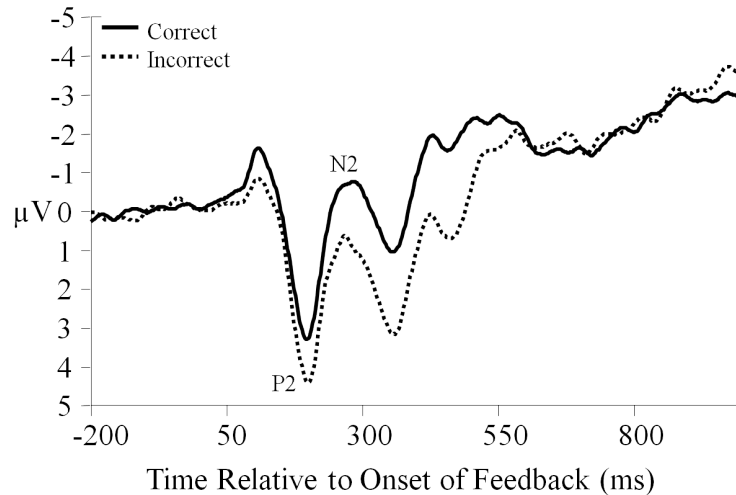
Figure 2-1. The group averaged waveform time-locked to the feedback in the AB task at FCZ. Both average waveform are averaged across lags and only include those trials where T1 performance was correct.

FRN (time production task). There was an N2 observed in the group averages

and at the individual level following the feedback in the time production task (see *Figure*

2-2). A repeated-measures ANOVA was performed with the factor of feedback type

(uninformative, correct, and incorrect) using the N2 (TIME) peak-to-peak measure. There

was a significant main effect of feedback type (F (2, 106) = 19.61, $p < .001$) such that the

N2 (TIME) was largest following incorrect feedback ($M = 5.76$, $SD = 3.36$), second

largest for uninformative feedback ($M = 4.87$, $SD = 3.06$), and smallest for correct

feedback ($M = 3.69$, $SD = 2.81$). Note again the sizeable N2 (TIME) following correct

feedback. Post-hoc paired t-tests revealed that all N2 (TIME) differences were significant

(incorrect – uninformative, uninformative-correct, incorrect-correct; all p's < .01).
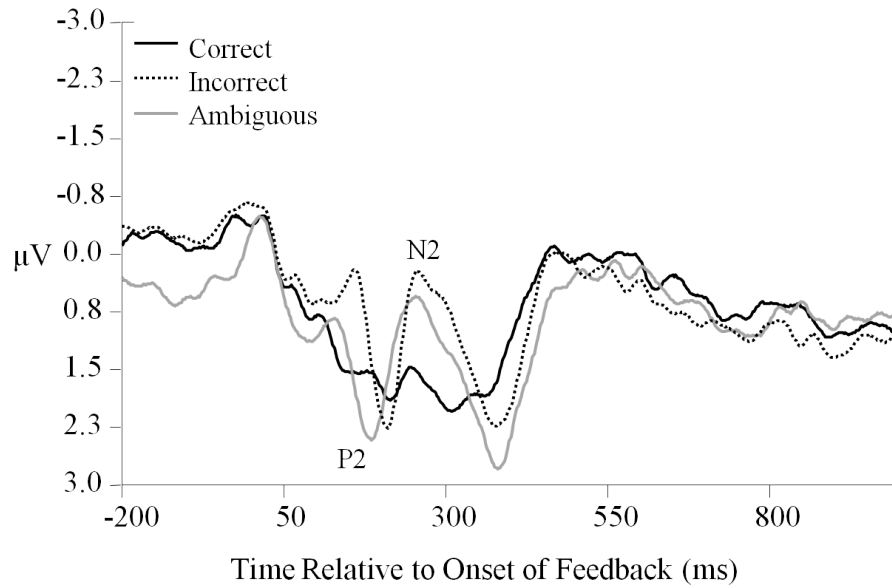
Figure 2-2. The group averaged waveform time-locked to the feedback in the time production task at FCZ.

ERN (flanker task). There was an apparent MFN observed in the group averages and at the individual level following both correct and incorrect responses in the flanker task (see *Figure 2-3*). A 2X2 repeated measures ANOVA was performed with the factors of flanker type (congruent and incongruent) and performance (correct and incorrect) using the MFN peak-to-peak measure. The only significant effect was a main effect of performance (F (1, 35) = 48.73, $p < .001$) such that the MFN was larger following incorrect responses ($M = 5.15$, $SE = .57$) than correct responses ($M = 1.67$, $SE = .25$). However, a sizeable MFN was still observed following correct responses.
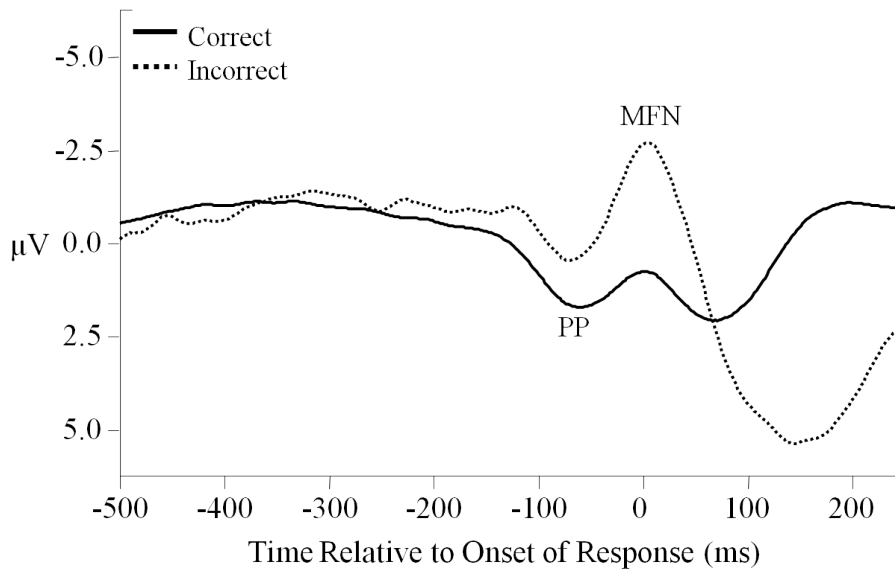
Figure 2-3. The group averaged waveform time-locked to response in the Flanker task at Cz.

Relationships between AB Performance and ERPs

For the purpose of examining individual differences in the N2 and MFN, new variables were created by saving the standardized residuals of the FRN or ERN peak scores controlling for the P2 or PP peak scores for each separate task (i.e. the residual variability in N2 or MFN peak score after removing the shared variance with P2 or PP peak score). All standardized residuals are the product of a linear regression with the ERP of interest (N2 or MFN) as the dependant variable and the preceding ERP (P2 or PP) as the predictor variable. These new residual variables were used for all correlations and regressions given that: 1) they are more reliable than the peak-to-peak difference measures used to examine mean effects, and 2) this method controls for individual differences in the peak used as the reference peak (e.g., the P2 or PP for the N2 and MFN). The reliability (internal consistency) for all residual ERP measures was extremely high as determined using split-half correlations: N2 (AB) $r = .91$; N2 (TIME) $r = .87$; MFN $r = .80$.

As discussed above, it was predicted that the N2 following feedback of all kinds, in other words the general strength of response to feedback, would relate to AB task performance. In order to test this possibility the N2 (AB) residual amplitude scores were averaged across all T2 types creating N2 (AB) residual amplitude scores for correct and incorrect feedback. The correct and incorrect N2s were also averaged across tasks (AB and TIME) to create overall N2 residual amplitudes scores for correct and incorrect feedback. It was also possible that relationship between individual differences in feedback responsivity and the AB would be modulated by the valence of the feedback. In order to examine this, I also created difference measures for the FRN in both the AB and time production tasks and the ERN in the Flanker task. The difference measures were calculated by subtracting the standardized residual measures of the FRN/ERN following correct feedback from that following incorrect feedback[3].

N2s and MFNs.

The N2s were highly positively correlated with each other across feedback types and within, between, and averaged across (overall) tasks (all p's < .001), and were somewhat positively related to the MFNs, but not significantly. T1 accuracy was not found to correlate with any of the ERPs (see Tables 2-1 & 2-2[4]). Lag 8 T2 performance correlated negatively with correct MFN amplitude (see Table 2-1) such that greater lag 8 T2 performance was associated with more negative (i.e., larger) MFN to correct performance. AB magnitude correlated negatively with all N2 amplitudes such that

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[3] This method for creating a difference measure eliminates the possibility of confounding latency effects with amplitude effects that the other method of subtracting entire waveforms prior to scoring the peak amplitude can create.

[4] The pattern of relationships remained when rate of negative feedback was controlled for in a multiple regression of lag 3 T2 performance.

greater AB magnitudes were associated with more negative (i.e., larger) N2s in both in

the AB task and in the time production task (see Table 2-2). Although these relationships

were only significant with the N2's to correct feedback (see *Figure 2-4a&b*) from each

task (incorrect *p*'s < .10), AB magnitude was significantly negatively correlated with both

overall N2s to correct and incorrect feedback (averaged across task; see *Figure 2-4c&d*).

As Pearson's *r* is sensitive to extreme scores, and it appears from the scatterplots in

Figure 2-4 that my sample does contain at least one extreme score, I also calculated

Spearman rank ordered correlations (rho). These analyses did not change the pattern or

significance of any of the relationships concerning the N2: AB magnitude and overall

correct N2 *r* = -.39 (*p* = .002), and overall incorrect N2 *r* = -.28 (*p* = .034). However, the

relationship between lag 8 T2 performance and correct MFN was not significant with the

Spearman rank ordered correlation (*r* = -.29, *p* = .068). Similarly, when the one extreme

standardized score (> 3) was removed all previously significant relationships remained

significant (all *p*'s < .05).

Table 2-1. Correlations between AB task performance and MFNs (Flanker task)

	Correct	Incorrect
T1 accuracy	.09	-.29
Lag 8 T2 performance	-.35*	-.17
AB magnitude	-.19	.09

*n = 39, *p* < .05 (two-tailed)*
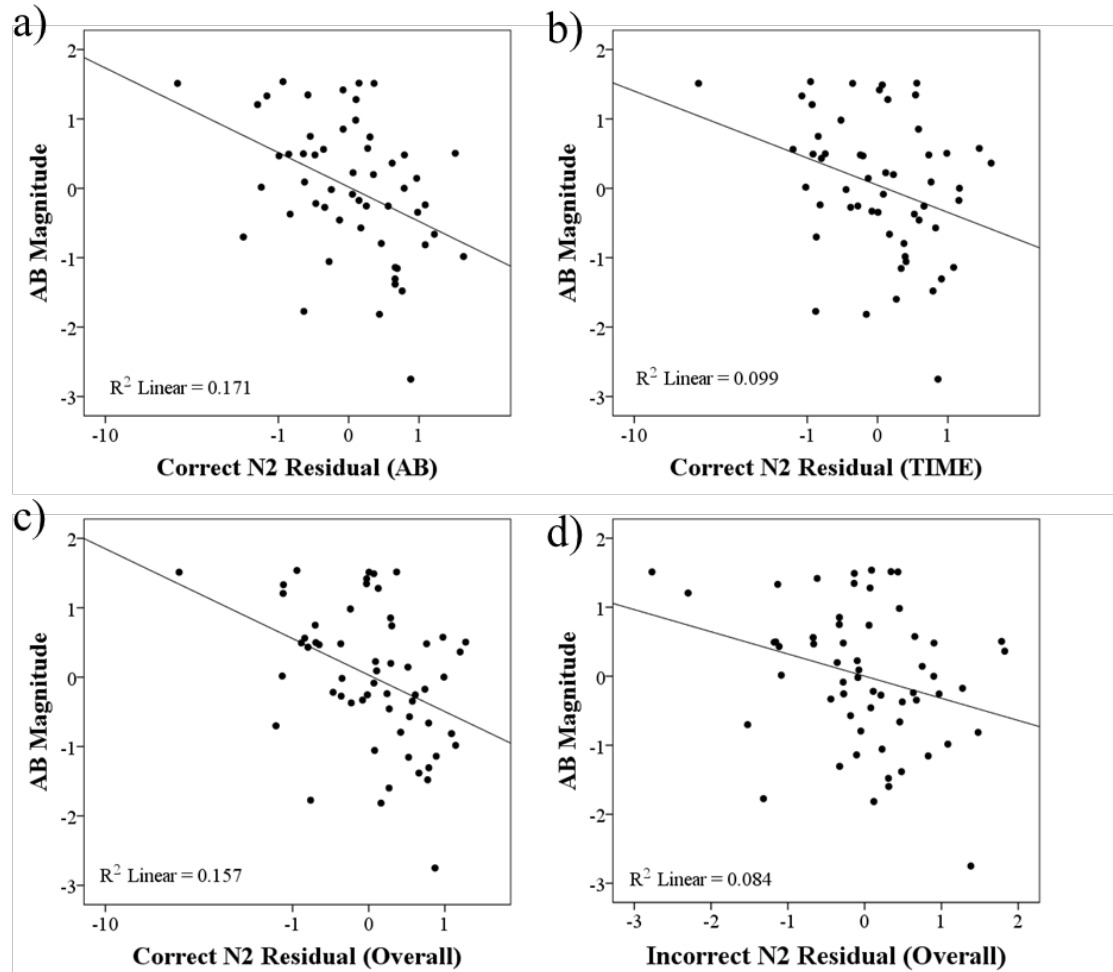
Figure 2-4 (a-d). Scatterplots depicting the correlations between AB magnitude and the correct N2 (AB) (a), correct N2 (TIME) (b), overall correct N2 (averaged across task) (c), and the overall incorrect N2 (averaged across task; d).

Table 2-2. Correlations between AB task performance and feedback N2s

	AB			TIME		OVERALL	
	Correct	Incorrect	Uninform.	Correct	Incorrect	Correct	Incorrect
T1 accuracy	-.10	-.14	-.09	.00	.08	-.07	-.06
Lag 8 T2 perform.	-.24	-.13	.07	-.06	-.07	-.16	-.12
AB magnitude	-.39**	-.26	-.17	-.31*	-.25	-.38**	-.29*

*n = 54, *p < .05, **p < .01 (two-tailed)*

The results of the correlations were also replicated when I examined mean

differences in AB magnitude based on a median split of participants according to N2

amplitude to correct feedback on each task (TIME and AB). In both cases individuals with more negative (i.e. larger) N2s to correct feedback had larger AB magnitudes (M (AB) = .37, M (TIME) = .33) than individuals with more positive (i.e. smaller) N2s to correct feedback (M (AB) = -.37, M (TIME) = -.27); AB t (52) = 3.01, p = .004, TIME t (52) = 2.31, p = .025.

Given that both overall N2 amplitudes (correct and incorrect) correlated significantly with AB magnitude, and also correlated significantly with each other, a simultaneous multiple regression was performed predicting AB magnitude with both overall N2s to determine whether the two overall N2 amplitudes explain the same variability in AB magnitude. The model was significant, R = .38, p = .013, and the overall correct N2 was nearly a significant unique predictor (semi-partial r = .25, p = .053) over and above overall incorrect N2, while overall incorrect N2 was not (semi-partial r = -.05, p = .694). The results of the multiple regression indicate that the correlation between overall incorrect N2 amplitude and AB magnitude is entirely due to shared variance between overall correct and incorrect N2 amplitude, but that the relationship between overall correct N2 amplitude AB magnitude can also be attributed to variance unique to overall correct N2 amplitude[5].

FRNs and ERNs (difference measures).

Correlations between AB performance measures and the FRN and ERN difference measures were performed to see if the relationship between individual differences in feedback responsivity and the AB was modulated by feedback valence. T1 accuracy was significantly correlated with the ERN difference measure, such that greater

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[5] When the same simultaneous regression was performed separately for each task the same pattern was observed, except that in the case of the N2s from the AB task the N2 to correct feedback reached significance as a unique predictor of lag 3 T2 performance (semi-partial r = .33, p = .007).

T1 accuracy was associated with a smaller accuracy-related ERP modulation (see Table

2-3). Lag 8 T2 performance was not found to correlate significantly with any of the FRN

or ERN measures. AB magnitude was also unrelated to the FRN or ERN measures, but

showed a trend towards positive correlations. This suggests that, if anything, smaller ABs

were somewhat associated with smaller accuracy- and valence-related modulations of the

ERPs (given that higher FRN/ERN values reflect larger FRN/ERNs), which would be

expected given the findings above that larger N2s to correct feedback were especially

strong predictors of greater ABs.

Table 2-3. Correlations between AB task performance and FRNs and ERN

	FRN (AB)	FRN (TIME)	ERN
T1 accuracy	-.07	.09	-.34[*]
Lag 8 T2 performance	.19	-.02	.17
AB magnitude	.25[#]	.08	.22

*n = 54, #p < .10 *p < .05*

Trial-to-Trial FRN (AB)

It is possible that the relationship between AB magnitude and overall N2 (AB)

magnitude could result from a large N2 on trial *n* leading to a short-term increase in

investment that might be expected to increase long lag T2 accuracy, but impair short lag

T2 accuracy on trial *n+1*. To test this, N2s in the AB task were averaged according to T2

performance on the subsequent trial (correct or incorrect) as well as T2 condition on the

subsequent trial (lag 3, lag 8 and absent); e.g. N2 (on trial *n*) where T2 performance was

incorrect on the subsequent (*n+1*) trial where T2 was presented at lag 3. This yielded six

different averages for each individual. Based on visual inspection of the group average

waveforms at site FCZ, the P2 was defined as the most positive peak between 160-200

ms, and the N2 was defined as the most negative peak between 200-260 ms.

A 3X2 repeated measures ANOVA was performed with the factors of T2 type on the subsequent trial (absent, lag 3, and lag 8) and T2 performance on the subsequent trial (correct and incorrect) using the N2 (AB) peak-to-peak measure as the dependent variable[6]. Note that two individuals were missing data for lag 8 incorrect averages (i.e. they had no artifact-free epochs for lag-8 incorrect). There was a significant main effect of T2 performance (F (1, 48) = 22.88, $p < .001$) such that the N2 was larger preceding trials where T2 performance was incorrect ($M = 7.88$, $SE = .52$) than preceding trials where T2 performance was correct ($M = 6.14$, $SE = .39$). There was also a significant interaction between T2 performance and T2 type (F (2, 96) = 12.01, $p < .001$; see *Figure 2-5*). In contrast to the possibility described above, the nature of this interaction was such that the N2 was larger preceding trials where T2 performance was incorrect than preceding trials where T2 performance was correct only for trials where T2 was presented at lag 8 (t (48) = 5.09, $p < .001$) or when T2 was absent (t (50) = 5.33, $p < .001$). There was no difference in the preceding trial's N2 for correct and incorrect T2 trials when T2 was presented at lag 3 (t (51) =-.36, $p = .721$).

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[6] The type of feedback that produced the N2 did not interact with any of these effects.
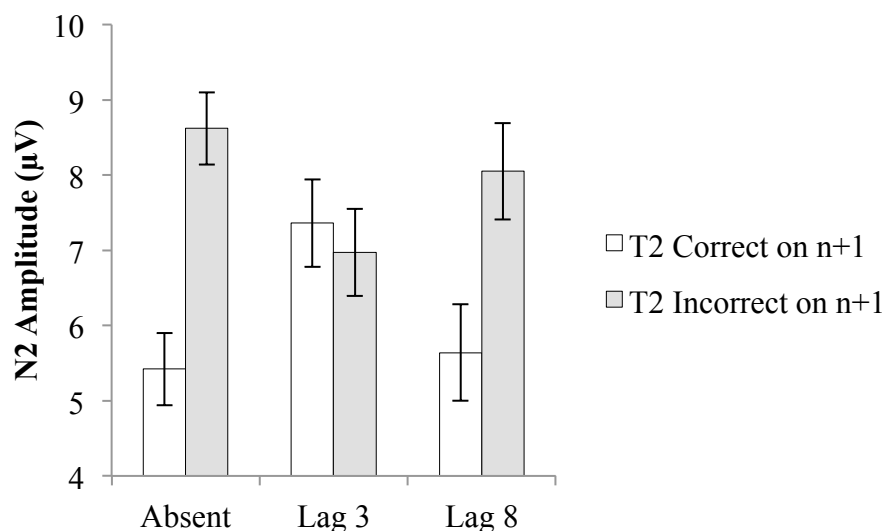
Figure 2-5. Mean N2 (AB) peak-to-peak amplitude on current trial (n) according to T2 type and T2 performance on the subsequent trial (n+1). Error bars represent the standard error of the mean difference (incorrect – correct).

General Discussion

The purpose of this investigation was to determine whether response to performance and feedback on performance, as measured by the N2 and MFN, would predict AB magnitude, and whether this relationship was modulated by whether feedback/performance was correct or incorrect. It has been proposed in several models that cognitive control and overinvestment contributes to the production of the AB (Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). Based on these models, and findings showing that individual differences that reflect, or are associated with, a flexible, diffuse, and less invested processing style predict smaller AB magnitudes (e.g., Dale & Arnell, 2010; MacLean & Arnell, 2010; MacLean et al., 2010), I hypothesized that greater AB magnitude should be associated with larger amplitude feedback-related N2 and performance-related MFN amplitudes. Specifically, individuals who demonstrate strong responses to performance and feedback may be more invested in the task and concerned with their performance and thus were hypothesized to have larger

AB magnitudes than individuals with weaker responses and weaker cognitive control/less investment.

Our hypothesis regarding feedback was supported. Feedback-related N2 amplitude (correct and incorrect), but not the valence-related modulation (difference measure), was found to predict AB magnitude significantly, such that, overall, larger N2 amplitudes to feedback were associated with larger AB magnitudes. This relationship was observed using N2 amplitudes measured following correct and incorrect feedback in two different tasks, an AB task and a time production task. Thus, individual differences in AB magnitude were predicted by individual differences in overall N2 amplitude from the same AB task and in a completely unrelated time production task. N2 amplitude, either to correct or incorrect feedback, did not predict T1 accuracy, or long lag T2 performance, so the relationship between N2 amplitude and AB task performance was confined to the T2 performance at short lags (i.e., during the AB interval). The FRN difference measure did not predict AB magnitude, suggesting that it was not the differential response to negative feedback that drove the relationship, but the response to feedback regardless of valence. However, response to feedback on correct trials was the most reliable predictor of AB magnitude, and the results of a simultaneous regression suggested that the relationship between AB magnitude and the N2 to correct feedback was due in part to unique variance unrelated to the N2 to incorrect feedback.

There is evidence that the valence modulation of the N2 (i.e. the FRN), is in fact due to a reduction in the negativity of the N2 caused by a positive-going component related to the processing of positive feedback (Holroyd et al., 2008). In other words, the valence effect of the N2 that is typically treated as the FRN is due to variability in the N2

to positive feedback and not variability in the N2 to negative feedback, which the authors classify as a "common" N2. In this case, it is possible that the more reliable and unique relationship I observed between the N2 to correct feedback and AB magnitude is due to individual differences in that positive-going component that reduces the size of the N2 to correct feedback as compared to the N2 to incorrect feedback. Other individual differences have also been found to be uniquely related to the processing of positive feedback, for example, problem gamblers were found to have a smaller N2 to positive feedback than controls, but not to differ in response to negative feedback (Hewig et al., 2010). I speculate that that majority of participants might be expected to be at least modestly responsive to negative feedback, but that only those participants who were truly high performance monitors would be expected to show a large response to positive external feedback.

Neither the ERN nor the MFN component correlated with AB magnitude. However, T1 performance was related to the performance-related modulation (difference measure) of the ERN such that larger differences were associated with better T1 performance and overall MFN amplitude did predict long lag T2 performance such that larger ERN amplitudes were associated with better long lag T2 performance. Thus greater responsivity to internal feedback cues does not appear to predict the AB, but may be associated with superior target performance outside of the AB.

Individual differences in electrophysiological responses to performance feedback were associated with individual differences in AB magnitude, as hypothesized. This could happen in at least two ways. One of these ways is more trait-like such that some individuals approach each experiment more or less invested than other individuals in

terms of assigning strict cognitive control to the task at hand. This would suggest that some individuals should invest more than others throughout the task, regardless of the feedback on any given trial. Another possibility is that some individuals are more responsive to feedback on a trial-to-trial basis and that feedback-related N2 amplitude predicts individual differences in AB magnitude through trial-to-trial changes in cognitive control. For example, larger N2s following feedback on a given trial may increase cognitive control momentarily, and thus increase the probability that T2 performance will be incorrect on the following trial if T2 is presented at a short lag following T1 (i.e. increase probability of an AB). However, my trial-based analysis did not support this possibility. Instead, the feedback-related N2 was larger preceding trials where T2 performance was incorrect than those where T2 performance was correct and only when T2 was presented at the long lag, or when T2 was absent. There was no difference in the feedback-related N2 preceding trials where T2 was presented at the short lag. This suggests that the feedback-related N2 amplitude does not predict individual differences in AB magnitude through trial-to-trial changes in cognitive control which increase the probability of the AB. Indirectly then, this provides some support for the idea that feedback-related N2 amplitudes may predict individual differences in AB magnitude due to general trait-like differences in responses to feedback and the rigid enforcement of cognitive control across the entire task.

The trial-based analysis did reveal that trial-to-trial changes in feedback-related N2 amplitude did result in differences in T2 performance when T2 was presented at long lags and when T2 was absent. So, it appears that trial-to-trial changes in feedback-related N2 amplitude were associated with differences in sensitivity at T2 detection when T2 was

not affected by proximity to T1. Specifically, larger feedback-related N2s were associated with a reduction of T2 detection sensitivity (lower probability of hits, higher probability of false alarms) outside of the critical AB period. This is contrary to the evidence that larger FRNs are associated with improvements in performance, presumably via strengthening of cognitive control (Cohen & Ranganath, 2007; van der Helden, Boksem, & Blom, 2010). It is not clear why trial-to-trial responses to feedback influence T2 performance outside of the AB interval, whereas individual-to-individual differences predict AB magnitude. However, this finding does underscore that these AB conditions can produce dissociable effects.

We made similar hypotheses about the relationship of the AB with both the N2 and MFN, however only my hypothesis concerning the N2 and the AB were supported by the results. I do not wish to make the claim that this is necessarily evidence of dissociation. However, there are differences between the FRN and ERN that allow me to make suggestions about the nature of the relationship between the electrophysiological responses to internal and external sources of feedback. The FRN is generated by externally generated representations of goal failures while the ERN is generated by internally generated representations of goal failures (Heldmann, Rüsseler, & Münte, 2008; Holroyd & Coles, 2002; Nieuwenhuis, Nielen, Mol, Hacjak, & Veltman, 2005; Nieuwenhuis et al., 2005). So, when one is able to determine the outcome of performance, feedback is redundant, and an ERN is generated (Müller, Möller, Rodriguez-Fornells, Münte, 2005).

The FRN and ERN also tend to have different scalp topographies (e.g. Gentsch, Ullsperger, & Ullsperger, 2009; Müller et al., 2005), as they did in this study, suggesting

that there are at least some neural generators not shared between the two components (Gehring & Willoughby, 2002; Potts, Martin, Kamp, & Donchin, 2011). Indeed, in my study I found that the ERN and FRNs were not correlated and the N2 and MFN were only modestly correlated. Unique neural generators of the FRN, e.g. the posterior cingulate, are involved in the evaluation of these external signals, for example evaluation of a reward as indicated by an external stimulus (Müller et al., 2005; Nieuwenhuis et al., 2005). So, it is possible that individual differences in performance monitoring functions unique to the evaluation of externally-generated information regarding outcomes are what predict AB magnitude, rather than investment in tasks in general or performance monitoring more generally.

It is, of course, also possible that the failure to find a relationship between MFN amplitude and AB magnitude is due to, for example, measurement error. However, both AB task performance measures and ERP amplitudes demonstrated either high or very high reliability (internal consistency) as measured by split-half (even vs. odd trials/epochs) correlations. This is consistent with previous evidence that these measures, both the AB (Dale & Arnell, 2013; Dale et al., 2013) and FRN/ERN amplitude (Segalowitz et al., 2010) have good test-retest reliability and can all be considered trait-like measures. The two measures of N2 amplitude in my study, from completely different tasks, also correlated positively and very strongly with each other, and both correlated positively with the MFN. So, my measures were all likely reliable measures of individual differences associated with AB magnitude and responses both to performance and feedback. Therefore, the failure to find a relationship between individual differences in MFN amplitude and individual differences in AB magnitude is not due to poor,

unreliable, measures of those individual differences. It is possible, however, that there are task-based differences in the ERN that mean that my ERN task, the Flanker task where speed was encouraged, does not capture the kind of variability in performance monitoring that might correlate with AB magnitude.

Additionally, the MFN/ERN was measured during the Flanker task which was the last task that participants completed. This task was also separated from the AB task, used to capture AB magnitude, by the time production task. Thus, it is also possible that the distance between the two measures and the presence of an intervening task may have affected the relationship between the MFN/ERN and AB magnitude.

Implications for Models of the AB

Our results are consistent with models of the AB, such as the Boost-and-Bounce model (Olivers & Meeter, 2008) and the Threaded Cognition model (Taatgen et al., 2009), that posit cognitive control as contributing to the production of the AB. My results extend these models by suggesting that individual differences in cognitive control can underlie individual differences in the AB, such that those individuals who generally produce larger electrophysiological responses to performance feedback would generally enforce cognitive control more strongly and thus have larger AB magnitudes. For example, in the Boost-and-Bounce model of the AB, a "boost" of attention initiated in response to T1 is followed by a cognitive control function, the "bounce", initiated by the presence of the distracter trailing T1 (Olivers & Meeter, 2008). This "bounce" prevents T2 from entering working memory when presented shortly after T1 during the "bounce". The Threaded Cognition model (Taatgen et al., 2009) features a similar cognitive control function, a "memory function", which impairs T2 detection. My results suggest that

individuals may differ with respect to the strength of this bounce response or memory function, such that some individuals exhibit greater cognitive control than others in response to external stimuli that suggest that performance goals are at risk – namely feedback and the presence of the distractor that trails T1. In this manner, individuals who more strongly enforce this "bounce"/"memory function" will be more likely to miss T2 at short lags and thus have larger AB magnitudes than individuals who are weaker enforcers.

The observed relationships between electrophysiological responses to external performance feedback and the AB is also consistent with previous individual differences studies showing that a flexible, diffuse, and less invested processing style predicts smaller AB magnitudes using measures such as global/local bias (Dale & Arnell, 2010), personality (MacLean & Arnell, 2010), and affect (MacLean et al., 2010), and with previous findings showing that performing a simultaneous additional task such as detecting yells in music attenuates the AB (Olivers & Nieuwenhuis, 2005). Diffuse and less invested processing (either via individual differences or task manipulations) may work to decrease performance monitoring and its call for heightened cognitive control. In this manner the present results are also consistent with the Overinvestment hypothesis of Olivers and Nieuwenhuis (2005, 2006) which proposes that the AB results from an overinvestment of unnecessary attentional resources in the RSVP items, including the distracters such that they strongly compete with target items.

In conclusion, I observed that electrophysiological responses to external performance feedback predicted individual differences in AB magnitude such that greater feedback-related N2 amplitude from the same AB task, and an unrelated time-production

task, were associated with larger AB magnitudes, regardless of the valence of the feedback. These results support cognitive control models of the AB, such that greater feedback responsivity is associated with the stronger enforcement of cognitive control which leads to larger AB magnitudes. However, I did not find the hypothesized relationship with electrophysiological responses to internally generated feedback (e.g., the MFN/ERN). I suggest that it is possible, then, that it is variability in the evaluation of externally-generated information regarding outcomes which predicts individual differences in the AB.

Replication Study

As part of a separate study, feedback-N2s were collected from an additional 43 participants. MFNs/ERNs were not collected as they did not predict AB magnitude above. With this additional sample I will (1) attempt to replicate the effects observed in the original study (Study 1, as described above) in the additional sample, (2) examine differences in the data from the sample in the original study and the additional sample, and finally (3) examine the effects observed in the original study with increased power by combining the original and the additional sample into one, composite sample.

Note that 20 of the participants in the original sample from Study 1 (reported above), were the first 20 participants in the study from which the replication sample was taken. However, the additional sample analyzed below does not include these 20 participants.

Methods

Participants

Participants were 43 Brock University undergraduate students (40 females; *M* age = 19 years), recruited through the Brock Psychology Department's online system for participant recruitment. Due to attrition 6 participants did not complete the time production task.

Procedure

Participants completed two resting EEG recordings (approximately 10 minutes) before completing two AB tasks, first the AB detection task (approximately 30 minutes), followed by another AB task requiring 10 alternative-forced choice (AFC) as the T2 task (approximately 1.5 hours), and finally the time production task (approximately 15 minutes). Note that unlike in the original Study 1, there was no Flanker task. The resting EEG recordings and the AB AFC task are not relevant to the current investigation and will not be discussed in this section.

Tasks

The AB detection task and time production task were identical to those used in Study 1 except that all participants received all forms of feedback in white font.

EEG Acquisition and Analysis

EEG acquisition, estimation, and analysis procedures were identical to those employed in Study 1.

Behavioral Results

AB Task Performance

The AB data from 19 participants were excluded due to either poor T1 performance (10 participants with < 40%) or poor T2 sensitivity at lag 8 (three participants with < 10% hits minus false alarms) on the AB task. Mean T1 accuracy for the remaining participants was 58.25% ($SD = 10.56$). Note the unusually low T1 accuracy here, as in Study 1. T2 performance was conditionalized on T1 accuracy. Mean T2 hits at lag 3 was 43.43% ($SD = 20.78$), mean T2 hits at lag 8 was 89.74% ($SD = 8.86$), mean rate of false alarms on T2 absent trials was 14.58 % ($SD = 7.52$). Mean T2 performance (hits – false alarms) at lag 3 was 28.42 ($SD = 19.55$). Mean T2 performance (hits-false alarms) at lag 8 was 75.08 ($SD = 10.32$). A paired-samples t-test showed significantly lower T2 performance at lag 3 than lag 8 indicating the presence of an AB (t (23) = 13.68, $p <$ .001). Individual AB magnitude was calculated as the residual difference measure as above. The mean difference score was 46.67% ($SD = 16.71$).

The residual measure of AB magnitude was reliable as determined using split-half correlations ($r = .55$). A similar residual variable (lag 8 T2 performance) was created for lag 8 T2 performance controlling for lag 3 T2 performance. The residual measure of lag 8 T2 performance was not as reliable as the AB magnitude measure as determined using split-half correlations ($r = .26$).

T1 accuracy in the additional sample was significantly lower than in the original sample from Study 1 (t (80) = 2.39, $p = .019$), while T2 performance at lag 8 was significantly higher in the additional sample than in the original sample from Study 1 (t (80) = 2.36, $p = .021$; see Table 2-4). However, neither T2 performance at lag 3, rate of

false alarms, or AB magnitude difference scores were significantly different from the

original sample in Study 1 (all p's > .11).

Table 2-4 Comparison of AB task performance between the additional and original samples

	Additional Sample M (SD)		Original Sample M (SD)
T1 Performance	58.25 (10.56)	<	65.93 (14.16)
Lag 3 T2 Performance	28.42 (19.55)	=	21.09 (18.74)
Lag 8 T2 Performance	75.08 (10.32)	>	65.76 (18.14)
AB Magnitude	46.67 (16.71)	=	44.67 (18.19)
False Alarms	14.58 (7.52)	=	16.93 (12.10)

Time Production Task Performance

The average time at which participants indicated one second had passed was 978

ms (SD = 126) ranging from 741 to 1318 ms. So, on average participants tended to

prematurely indicate that 1 second had passed by 22 ms, which was very similar to the

980 ms mean time estimation performance from Study 1.

<div align="center">ERP Results</div>

FRN (AB task)

There was an N2 observed in the group averages and at the individual level

following the feedback in the AB task (see *Figure 2-6*). A paired-samples t-test

comparing the N2 (AB) peak-to-peak measure following correct and incorrect feedback

was not significant (t (23) = .09, p = .933), indicating that the N2s (AB) were statistical

equivalent following incorrect feedback (M = 5.98, SD = 2.79) and correct feedback (M =

5.95, SD = 2.16). This does not replicate the finding in Study 1 that the N2 (AB)

following incorrect feedback was significantly larger than that following correct

feedback. However, neither the N2 (AB) following incorrect feedback (t (76) = 1.48, p =

.143) or the N2 (AB) following correct feedback (t (76) = .59, p = .558) differed

significantly between the additional and original samples (independent samples t-tests).

When the additional sample was combined with the original sample from Study 1, the

paired-samples t-test indicated that in the combined sample the N2 (AB) following

incorrect feedback ($M = 6.84$, $SD = 3.45$) was significantly larger than the N2 (AB)

following correct feedback ($M = 6.24$, $SD = 2.86$; $t(77) = 2.69$, $p = .009$).
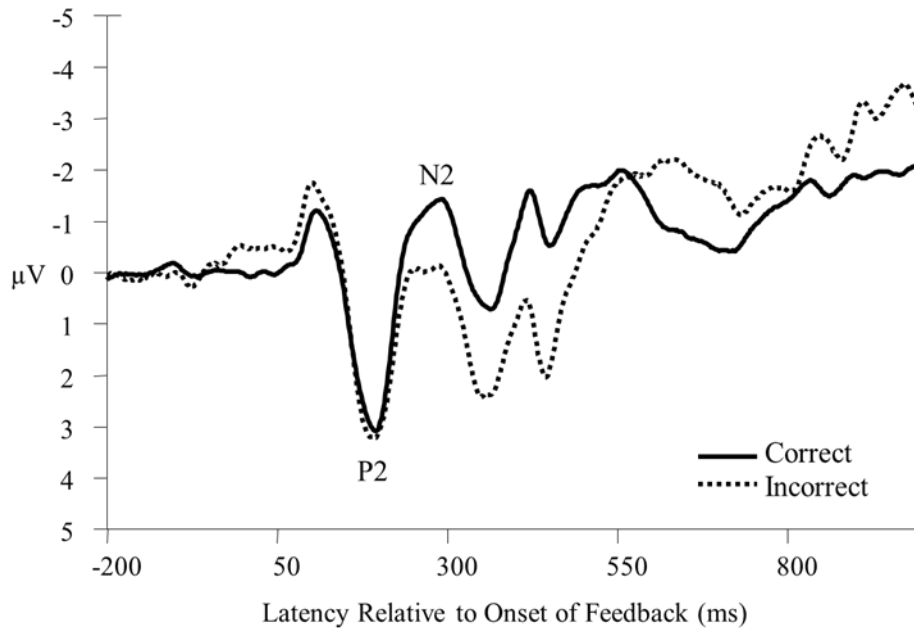
Figure 2-6. The group averaged waveform time-locked to the feedback in the AB task at FCZ. Both average waveform are averaged across lags and only include those trials where T1 performance was correct.

FRN (time production task)

There was an N2 observed in the group averages and at the individual level

following the feedback in the time production task (see *Figure 2-7)*. A repeated-measures

ANOVA was performed with the factor of feedback type (uninformative, correct, and

incorrect) using the N2 (TIME) peak-to-peak measure. There was a significant main

effect of feedback type ($F(2, 42) = 3.24$, $p = .049$) such that the N2 (TIME) was largest

following correct feedback ($M = 3.04$, $SD = 1.28$), second largest for uninformative

feedback ($M = 2.63$, $SD = 1.29$), and smallest for incorrect feedback ($M = 2.45$, $SD = $ 1.23). Post-hoc paired t-tests revealed that only the comparisons of the N2 (TIME) following correct feedback and that following incorrect feedback differences was significant ($p = .047$). This pattern does not match that observed in Study 1, where the N2 (TIME) was largest following incorrect feedback and smallest following correct feedback). It is possible that the reduction in target accuracy in the additional sample indicates that the participants in the additional sample found the task difficult, and thus perhaps correct feedback was novel as compared to incorrect feedback, leading to a stronger response. Expectations of feedback induced via probability of negative feedback have previously been shown to influence the size of the FRN, such that FRN difference was larger when the likelihood of negative feedback was greater, (i.e. was expected; Bellebaum & Daum, 2008).

Independent samples t-tests comparing N2 (TIME) amplitudes following correct, incorrect and uninformative feedback between the additional and the original samples were performed. Those t-tests indicated that the N2 (TIME) was significantly reduced in the additional sample when following incorrect ($t (74) = 4.49$, $p < .001$) and uninformative feedback ($t (74) = 3.30$, $p = .001$) as compared to the original sample from Study 1, but that the N2 (TIME) following correct feedback was not significantly different ($t (74) = 1.04$, $p = .303$).

When the additional and original samples are combined the pattern in Study 1 (incorrect > uninformative > correct, all comparisons significant, $p < .05$) is replicated in this combined sample.
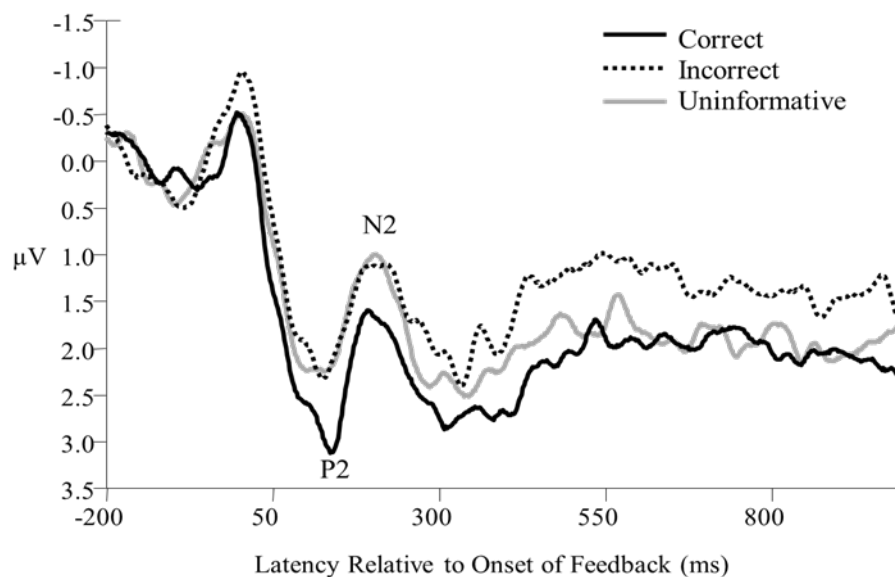
Figure 2-7. The group averaged waveform time-locked to the feedback in the time production task at FCZ.

Relationships between AB Performance and ERPs

The reliability (internal consistency) for all residual ERP measures was high as determined using split-half correlations: N2 (AB) r = .80; N2 (TIME) r = .49. As with the original sample in Study 1, N2s in the additional sample were highly positively correlated with each other across feedback types and within, between, and averaged across (overall) tasks, although not always significantly, likely due to the small size of the additional sample (all r's > .30). This possibility is supported by the finding that when the additional and original samples are combined all correlations are highly positive and significant (all p's < .001) in the combined sample.

T1 accuracy was not found to correlate with any of the ERPs in either the additional or the combined sample again replicating the results observed in Study 1 (see Tables 2-5[7]).

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[7] The pattern of relationships remained when rate of negative feedback was controlled for in a multiple regression of lag 3 T2 performance.

Table 2-5. Correlations between AB task performance and feedback N2s

	AB			TIME		OVERALL	
	Correct	Incorrect	Uninform.	Correct	Incorrect	Correct	Incorrect
T1 accuracy	-.05	.01	.04	-.07	.24	-.07	.15
Lag 8 T2 perform.	-.51*	-.53**	-.29	-.62**	-.13	-.62**	-.43*
AB magnitude	-.49*	-.38	-.11	-.52*	-.02	-.56**	-.26

n = 22-24, *p < .05, **p < .01 (two-tailed)

The pattern of relationships between AB magnitude and the N2s was identical to that observed in Study 1. AB magnitude correlated negatively with all N2 amplitudes such that greater AB magnitude was associated with more negative (i.e. larger) N2s in both in the AB task and in the time production task (see Table 2-5). These relationships were only significant with the N2's to correct feedback (see *Figure 2-8a&b*) from each task, and AB magnitude was significantly negatively correlated with the overall N2s to correct but not incorrect feedback (averaged across task; see *Figure 2-8c*). In the combined sample AB magnitude is significantly negatively correlated with all N2s except the N2 to uninformative feedback from the time production task (all *p's* < .05).

In order to determine whether the variability in AB magnitude predicted by N2 amplitude is common to the N2s from each task, or whether N2s from a particular task uniquely predict AB magnitude, a hierarchical regression was performed predicting AB magnitude with the N2s from the time production task on the first step and the N2s from the AB task on the second step. None of the predictors were significant unique predictors of AB magnitude (see Appendix C.2), indicating that the variability in AB magnitude predicted by N2 amplitude is common across the two tasks.
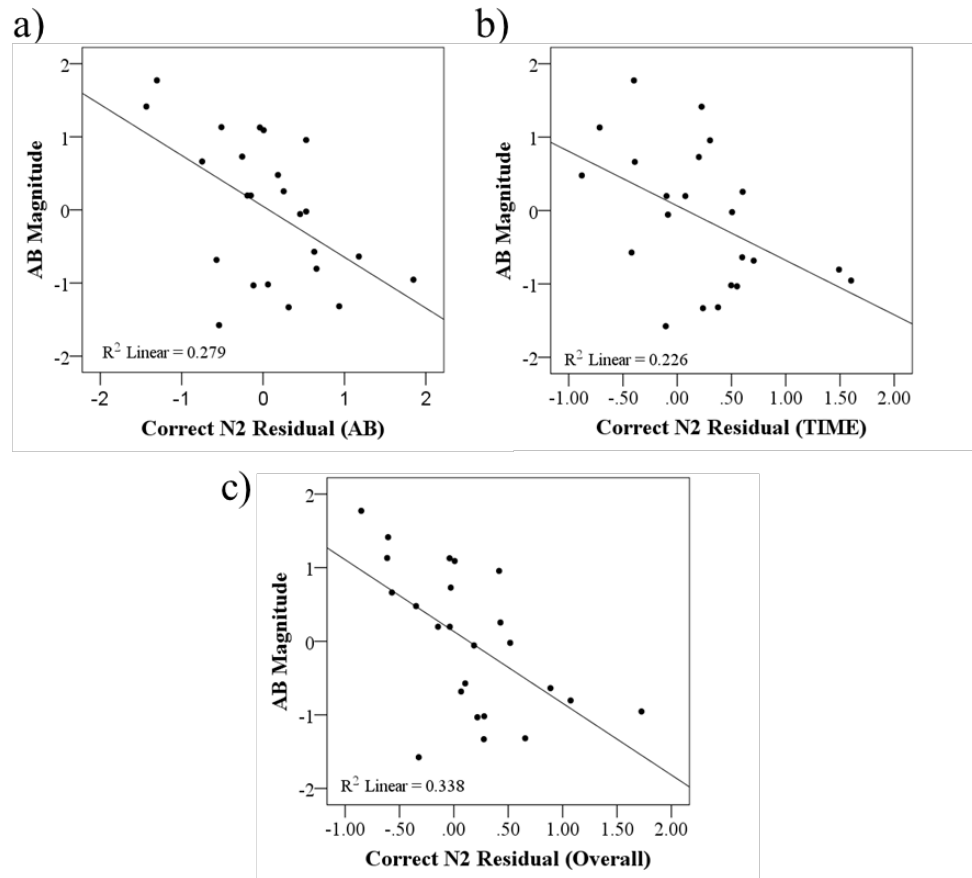
Figure 2-8 (a-c). Scatterplots depicting the correlations between AB magnitude and the correct N2 (AB) (a), correct N2 (TIME) (b), and overall correct N2 (averaged across task) (c)

Unlike in Study 1, however, T2 performance at lag 8 was significantly negatively correlated with all N2 amplitudes as well, such that higher T2 performance on lag 8 trials was associated with more negative (i.e. larger) N2's in both the AB task and in the time production task (see Table 2-5). Although these relationships were not significant with the N2s to incorrect or uninformative feedback on the time production task, T2 performance at lag 8 was significantly negatively correlated with the N2s to both correct and incorrect feedback overall. In the combined sample T2 performance at lag 8 was still significantly negatively correlated with the N2 to correct feedback on the AB task and the N2 to correct feedback overall.

As was the case in Study 1, the results of the correlations were also replicated when I examined mean differences in AB magnitude based on a median split of participants according to N2 amplitude to correct feedback overall. Individuals with more negative (i.e. larger) N2s to correct feedback had larger AB magnitudes ($M = .42$, SD = 1.04) than individuals with more positive (i.e. smaller) N2s to correct feedback ($M = -.42$, SD = .74); $t(22) = 2.28$, $p = .032$. The same results were observed in the combined sample ($t(76) = 3.52$, $p = .001$).

For replication purposes, a simultaneous multiple regression was performed on AB magnitude with both overall N2s as predictors despite the finding that AB magnitude was not significantly correlated with the N2 to incorrect feedback overall. The model was significant, $R = .62$, $p = .006$, and the overall correct N2 was a significant unique predictor (semi-partial $r = -.56$, $p = .004$) over and above overall incorrect N2, while overall incorrect N2 was not (semi-partial $r = .26$, $p = .149$). The results of the multiple regression indicate that the relationship between overall correct N2 amplitude and AB magnitude is entirely unique from N2 amplitude to incorrect feedback. The same results were observed in the combined sample.

Correlations between AB Performance Measures and FRNs (difference measures)

As found in Study 1, the FRN difference measure from the AB task was not significantly correlated with any of the AB performance measures (see Table 2-6). However, T2 accuracy at lag 8 and AB magnitude were both significantly correlated with the FRN (TIME) difference measure, such that greater T2 at lag 8 accuracy and larger AB magnitudes were associated with a greater feedback valence-related ERP modulation

(see Table 2-6). In the combined sample only the positive correlation between the FRN

(AB) and AB magnitude was significant ($r = .25$, $p = .025$; all other p's > .210).

Table 2-6. Correlations between AB task performance and FRNs and ERN

	FRN (AB)	FRN (TIME)
T1 accuracy	.13	.32
Lag 8 T2 performance	-.06	.49*
AB magnitude	.22	.51*

n = 22-24, *p < .05 (two-tailed)

Because the FRN (TIME) is a difference measure it is difficult to say whether the

relationship between AB magnitude and the FRN (TIME) is due to variability in the

difference or variability in one of the constituents of the difference in particular (i.e. the

N2 to either correct or incorrect feedback could be driving the relationship). Upon further

investigation the correlation between AB magnitude and the FRN difference measure

appears to be mainly due to variability in N2 amplitude to correct feedback rather than

the modulation of N2 amplitude by valence per se. This is indicated by two simultaneous

multiple regressions of AB magnitude, with the FRN (TIME) difference measure as a

predictor in both, accompanied by the N2 (TIME) to correct feedback in one regression

and the N2 (TIME) to incorrect feedback in the other. While the FRN difference measure

is a significant predictor of AB magnitude over and above the N2 (TIME) to incorrect

feedback ($p = .004$), it is no longer a significant predictor of AB magnitude over and

above the N2 (TIME) to correct feedback ($p = .130$).

Discussion

In Study 1 I found that N2 amplitude to feedback predicted AB magnitude such

that individuals with larger N2's had larger AB magnitudes, this effect was stronger and

more consistent with the N2 to correct feedback. I conceptualized greater N2 amplitudes

following feedback as greater attentional investment in a task/performance-relevant stimulus, thus greater attentional investment in task/performance-relevant information was associated with a larger AB.

As part of another study I collected N2 amplitudes and AB performance data from an additional sample of participants. While the pattern of mean N2 amplitudes among feedback types differed somewhat in the additional sample, overall the correlations between AB performance and N2 amplitudes reported in Study 1 were replicated in the additional sample, and furthermore in a combined sample (original sample from Study 1 combined with additional sample). So, again in another sample, and in a large sample with greater power, I found that N2 amplitudes predicted AB magnitudes, indicating that individuals who invested more in task/performance-related stimuli had larger AB magnitudes. This appears to be a robust and reliable relationship.

There were, however, correlations found in the additional sample that were not present in the original sample of Study 1. Specifically, T2 performance at lag 8 was found to correlate significantly with feedback-related N2 amplitude, such that larger feedback-related N2 amplitudes were associated with better T2 performance at the longer lag. This indicates that while stronger responses to feedback were associated with larger AB magnitudes (i.e. larger T2 deficits at a shorter lag relative to a longer lag), they were also associated with better T2 performance at a longer lag (i.e. better baseline T2 performance). This fits with my interpretation of larger feedback-related N2 amplitudes being representative of stronger cognitive control. Stronger cognitive control would be expected to lead to better target performance, except in the particular conditions that create the AB.

Additionally, AB magnitude was found to correlate with the FRN difference measure such that larger FRNs were associated with larger AB magnitudes, although only that derived from the time production task. As before, any relationships between the FRN difference measures and the AB appeared to result from relationships with feedback on correct trials. In summary, the results of the replication study match those found in Study 1, and support the conclusions of Study 1 that stronger cognitive control, as indicated by stronger responses to goal-relevant information (i.e. feedback) is associated with larger AB magnitudes.

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Preface to Chapter 3

Note that Chapter 3, excluding the replication study, is a published manuscript:

MacLean, M.H. & Arnell, K.M. (2011). Greater attentional blink magnitude is associated
with higher levels of anticipatory attention as measured by alpha event-related
desynchronization (ERD). *Brain Research,* 1387, 99-107.

Chapter 3

Introduction

When two to-be-attended targets are presented in a rapid serial visual presentation (RSVP) stream, accuracy for the second target (T2) is reduced when it is presented within 500 ms of the first target (T1), relative to longer T1-T2 separations — a phenomenon known as the attentional blink (AB; Raymond et al., 1992). The AB has been interpreted as reflecting attentional limitations such that attentional processing of T1 interferes with and/or delays the allocation of attention to T2 if T2 is presented before T1 processing has been completed (Shapiro et al., 1997).

Models of the AB

Traditional models of the AB tend to characterize the AB in terms of bottlenecks on information processing (e.g., Chun & Potter, 1995; Jolicoeur, 1998). For example, in the two-stage model of the AB (Chun & Potter, 1995) it is proposed that there are two stages to target processing. At the first stage, multiple stimuli can be processed in parallel and temporary fragile representations of the stimuli are created. In the second stage of processing the fragile and temporary representations are encoding into more durable working memory representations that can be used for later report. Stage two processing is time and attention demanding such that a bottleneck is created at stage two processing if T2 is presented while T1 is still undergoing stage two processing, or if RSVP distractors are currently competing for stage two processing resources. Until that bottleneck is resolved the encoding of any subsequent targets is delayed leaving their perceptual representations vulnerable to decay and reducing the probability that they will be

accurately reported. Thus, any unnecessary investment of stage 2 processing resources in T1 would be expected to exacerbate the AB.

More recently, there have been models of the AB suggesting that some feature of cognitive control is responsible for the pattern of attentional investment that results in the failure to accurately report T2 at short target separations. For example, in the Temporary Loss of Control model (TLC; Di Lollo et al., 2005) it is suggested that cognitive control initially optimizes an input filter in favor of T1. When attention is needed to process the T1 stimulus, less attention is available to control the input filter and the filter falls under bottom-up control. If T2 is presented before cognitive control of the input filter is restored, this loss of cognitive control impairs selection of T2, resulting in the AB. Therefore, the TLC model implies that a lack of top-down cognitive control following T1 is responsible for the AB.

In the Boost-and-Bounce model (Olivers & Meeter, 2008) it is proposed that the T1 item elicits an excitatory "boost" that last long enough to also boost the distracter item that immediately follows T1 into working memory. Cognitive control then responds to the presence of this distracter with an inhibitory "bounce" that prevents subsequent items, including T2, from entering working memory. According to this model, poor cognitive control over the "bounce" response (i.e., an inability to prevent the "bounce") seems to initiate the context necessary for an AB.

The Threaded Cognition model (Taatgen et al., 2009) also suggests that a memory function initiated by T1 prevents the further detection of targets. Taatgen et al. characterize this memory function as an overexertion of control, and suggest that when

this control function is not engaged, the probability of accurate T2 performance is increased.

In their Overinvestment Hypothesis, Olivers and Nieuwenhuis (2005, 2006) propose that the AB results from the unrestrained investment of attentional resources extending to all RSVP items such that distractors become effective competitors for entrance into working memory. When T2 appears soon after T1 it is particularly vulnerable to this interference given the additional attention required for encoding T1, resulting in the AB. However, Olivers and Nieuwenhuis suggest that if investment of attention was reduced to a level just sufficient to encode the targets, then interference would be reduced and the probability of accurate T2 performance would increase, particularly at short target separations.

In all of the above models, limited attentional resources and inappropriate application of attention underlie the AB. Cognitive control models further suggest that this is a result of maladaptive management of attentional resources by top-down cognitive control. If more or less adaptive cognitive control and the resultant investment of attentional resources could influence the magnitude of the AB, then that would imply that the AB does not reflect a fundamental attentional processing limitation. Instead, the AB would be conceptualized as resulting from a particular attentional style, such that its magnitude is influenced by the degree of investment of attentional resources with which an individual approaches the RSVP task.

Recent evidence where researchers have manipulated or measured the level of cognitive control and/or attentional investment supports this conceptualization of the AB - specifically the possibility that overly stringent cognitive control and inappropriate

attentional investment contribute to the AB. For example, when participants engaged in concurrent task such as detecting yells in music or performing a match to sample task Olivers & Nieuwenhuis (2005, 2006)[8] found that the AB was reduced relative to control conditions when participants performed only the AB task. Similarly, the AB has been reduced when task instructions emphasized a more passive target search strategy where you let the targets jump out at you (Olivers & Nieuwenhuis, 2005), and when AB task instructions emphasized reporting the two targets as a combination or pair (Ferlazzo et al., 2007). Olivers & Nieuwenhuis (2006) also observed a reduced AB when participants were exposed to positive affective pictures, relative to negative or neutral pictures. This result has implications for models of the AB given that positive affect is associated with an open and flexible cognitive processing style and diffused attention (e.g., Fredrickson, 2001) while negative affect is associated with heightened focusing of attention (e.g., Kramer et al., 1990).

Individual differences in trait affect (MacLean et al., 2010) and state affect (MacLean & Arnell, 2010) have been shown to predict AB magnitude such that greater positive affect is associated with reduced AB magnitudes and greater negative affect is associated with increased AB magnitudes. Personality dimensions related to attentional investment and focus have also been shown to predict the magnitude of the AB such that higher scores on extraversion and openness to experience predicted smaller AB magnitudes, and higher scores on neuroticism predicted larger AB magnitudes (MacLean & Arnell, 2010). Individual differences in the degree of global versus local processing also predict AB magnitude, such that an individual's tendency to focus on the local

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[8] Olivers and Nieuwenhuis indicated that the effect of music played concurrently with the RSVP stream on AB magnitude could not be consistently replicated (Olivers & Nieuwenhuis, 2006, footnote 1).

information as opposed to seeing the global overall picture was positively associated with

larger AB magnitudes (Dale & Arnell, 2010). Individual differences in the ability to

effectively inhibit or ignore RSVP distractors has been shown to relate to the AB such

that greater inhibition of irrelevant RSVP distractors was associated with smaller AB

magnitudes (Dux & Marois, 2008). Similarly, individual's ability to ignore irrelevant

visual material presented beside the RSVP stream (Martens & Valchev, 2009), or in a

separate visual working memory task (Arnell & Stubitz, 2010) was negatively related to

AB magnitude, such that greater ability to ignore the irrelevant material predicted smaller

AB magnitudes. Notice that in each of these studies inappropriate allocation of

attentional resources is associated with larger ABs.

     While the above evidence supports models of the AB discussed previously, which

propose that maladaptive applications of cognitive control and inappropriate attentional

investment contribute to the AB, it also suggests that cognitive control and attentional

investment are not determined once the RSVP stream starts or within the 500 ms

following T1. Instead, these results suggest that even before the RSVP task begins the

degree of cognitive control or attentional investment with which an individual approaches

the trial can influence the AB. This suggests that there may be a relationship between

readiness to invest attention before the RSVP trial, and T2 performance on that trial.

Electrophysiological Measurement of Attentional Investment

     Attentional investment during the RSVP stream has often been measured using

event-related brain potentials (ERPs). Electrophysiological investigations of the role of

the AB have focused on attentional investment only during the RSVP task, either relative

to a target (Martens et al., 2006a; Martens et al., 2006b; Sessa et al., 2007; Vogel & Luck,

2002; Vogel et al., 1998) or distracters (Martens, et al., 2006b). Vogel et al. (1998) and

Sessa et al. (2007) both demonstrated that the P3 component, (an ERP component

thought by many to be related to updating working memory; e.g., Donchin, 1981), is

absent following T2 at shorter target separations, suggesting that T2 fails to enter

working memory when presented at shorter T1-T2 intervals. Martens et al. (2006a)

showed that a low probability T1 resulted in a larger P3 component and a larger AB

compared to a high probability T1 target, a finding they attributed to the greater

attentional investment required for improbable T1s. Martens et al. (2006b) were also able

to show that non-blinkers (individuals who reliably show no AB) had more discrete and

significantly earlier P3's to T1 and T2, indicating that their attentional investment in

targets differed from individuals who demonstrate an AB. Non-blinkers also had

significantly reduced attentional investment in distracters, as measured by activation

during distracter-only RSVP trials, suggesting that they were also investing less attention

in distracter items compared to individuals who demonstrate an AB (Martens et al.,

2006b).

The electrophysiological evidence reviewed above supports models of the AB that

propose that an inappropriate investment of attentional resources underlies the AB in that

greater ABs were associated with greater P3s to T1 and greater activation to RSVP

distracters. However, the electrophysiological measures used (the P3 and distracter

activation) were confined to measuring the attentional investment that occurs during the

RSVP trial. The goal of this study is to investigate whether the degree of anticipatory

attentional investment with which an individual approaches an AB trial influences the

behavioral outcome on that trial.

Alpha Desynchronization and Anticipatory Attention

Anticipatory attention has been captured by examining event-related changes in alpha frequency (~8-12 Hz) oscillations present in cortical electrophysiological activity (Bastiaansen & Brunia, 2001; Bastiaansen et al., 2002; Bastiaansen et al., 2001; Capotosto et al., 2009; Onoda, et al., 2007; Yamagashi et al., 2005). Alpha frequency oscillations are frequently observed over widespread cortical areas and are thought to be generated by thalamo-cortical connections as well as cortico-cortical communication (Lopes da Silva, 1991; Steriade et al., 1990). Specifically, during a particular thalamic state afferent stimulus information is prevented from proceeding to the cortex and this thalamic state results in synchronized cortical activity in the alpha range and an increase in alpha power. When afferent information is then allowed to reach the cortex, synchronization is disrupted, in other words, a desynchronization in the alpha range and a reduction in alpha power results (Lopes da Silva, 1991; Steriade et al., 1990).

Lopes da Silva (1991) suggests that the different thalamic states could represent a gating function very similar to an early attentional filter which controls the flow of specific information to the cortex as well as between cortical areas, and that the function of this gating system is reflected in changes to alpha frequency oscillations of the cortex. Brunia and van Boxtel (2001) suggest that anticipatory attention is initiated by top-down influences generated by cortical areas that control attention via the thalamus, such that the flow of information to the cortex is regulated in order to facilitate the processing of an upcoming relevant stimulus. These authors propose that the top-down influence is directed at the reticular nucleus which is thought to control the thalamic states which result in the synchronization and desynchronization of alpha frequency oscillations of the

cortex (Lopes da Silva, 1991; Steriade et al., 1990). Therefore, event-related desynchronization (ERD) in alpha frequency oscillations could be considered an index of anticipatory attention facilitating, in a top-down manner, the flow of information from the thalamus to the cortex. If this assumption is true, then alpha ERD should be observed prior to a to-be-attended stimulus when that stimulus can be anticipated and greater alpha ERD should reflect greater anticipation.

In support of the idea of alpha ERD as a measure of anticipatory attentional investment, alpha ERD has been observed following a cue to shift attention toward the location of an upcoming target, and found to persist until the target appeared (Yamagashi et al., 2005). Alpha ERD has also been observed prior to a visual stimulus providing feedback on performance of a time-estimation task (Bastiaansen & Brunia, 2001; Bastiaansen et al., 2002; Bastiaansen et al., 2001). Furthermore, interrupting alpha ERD prior to a relevant stimulus, using trans-cranial magnetic stimulation on fronto-parietal areas of the attention network, impaired target performance in a spatial attention task (Capotosto et al., 2009). Alpha ERD has also been shown to be larger following a cue indicating that the upcoming affective stimulus was negatively-valenced, compared to when the cue indicated it was positively-valenced (Onoda et al., 2007). These authors interpreted this as evidence that cortical sensory-perceptual areas were activated via top-down control in anticipation of a relevant stimulus, in order to facilitate the processing of that stimulus.

The Present Study

It has been suggested that the assignment of limited attentional resources to T1 and distracters (Chun & Potter, 1995), an overexertion of cognitive control (Olivers &

Meeter, 2008; Taatgen et al., 2009), and/or a general overinvestment of attention (Olivers & Nieuwenhuis, 2006) underlies the AB. These models have been supported in that: (1) manipulations meant to reduce the degree of attentional investment have been shown to reduce the AB (e.g., Olivers & Nieuwenhuis, 2005, 2006), (2) affective and personality traits associated with a open and flexible cognitive control and/or reduced attentional investment predict smaller ABs (MacLean & Arnell, 2010; MacLean et al., 2010), and (3) an individual inability to avoid processing irrelevant information predicts larger ABs (Arnell & Stubitz, 2010; Dux & Marois, 2008; Martens & Valchev, 2009).

The goal of this study is to test the hypothesis that an inappropriate investment of attention just prior to the onset of the RSVP stream contributes to the production of the AB. This was done by investigating whether the behavioral outcome of trials is related to the degree of anticipatory attentional investment with which an individual approaches an AB trial. To answer this question, I measured the level of anticipatory attentional investment prior to the RSVP stream, using alpha ERD as a more direct measure of anticipatory attention. If greater levels of anticipatory attentional investment are associated with a reduction in the probability of accurate T2 performance at shorter T1-T2 intervals, then alpha ERD should be greater prior to the RSVP stream on trials when T2 performance was incorrect than when T2 performance was correct at shorter T1-T2 intervals. I would not expect, however, that greater alpha ERD prior to the RSVP stream should be associated similarly with poor T2 performance at longer T1-T2 intervals. At shorter T1-T2 intervals over-investment of anticipatory attention would be expected to increase attention to T1, and this would leave less attention for a closely trailing T2. However, at longer T1-T2 intervals, T1 processing would likely be complete by the time

T2 was presented, and thus overinvestment would not have a detrimental effect on T2 processing.

When T2 is presented at longer T1-T2 intervals (more than 500 ms after T1) T2 performance resembles single target performance (Raymond et al., 1992). Greater attentional investment would generally be expected to improve target performance, except in the particular circumstances in which an AB is found to occur as discussed above. So, when those circumstances are absent, as is the case for T1 processing or when T2 is presented following longer T1-T2 intervals, greater levels of anticipatory attentional investment as indicated by greater alpha ERD should be associated with better target performance. This hypothesis assumes that attentional investment beyond some minimum threshold required for accurate identification of a target stimulus could still increase target performance.

Methods

Participants

The participants were 30 Brock University undergraduate students, recruited through the Brock Psychology Department's online system for participant recruitment. Participants (17 female, 11 male, 2 undeclared) varied from 18 to 28 years of age with a mean age of 20 years ($SD = 2.33$). All participants reported speaking English as their first language. None of the participants reported any perceptual or cognitive impairment. The data from one participant were excluded due to close to chance performance on the RSVP task (first target accuracy was 53%, and second target accuracy was 13%), and the data from another participant were excluded due to an error in the EEG recording.

Data from participants who had less than five remaining epochs in any one condition in either the T1 or T2 analysis were excluded from the analysis, resulting in the exclusion of six participants' data in the T2 analysis[9]. Data from participants demonstrating an event-related synchronization (negative ERD values) in any one condition in either the T1 or T2 analysis were also excluded from the analysis. This resulted in the removal of data from 6 participants for the T1 analysis and 1 participant for the T2 analysis. This removal was necessary given that it is inappropriate to consider negative ERD values (indicating an event-related synchronization, a different phenomenon) as relatively lower ERD values.

AB Task

The AB task consisted of five blocks of 140 RSVP trials. Of the 700 total trials, 100 were no-target trials, and 600 were dual-target (T1 and T2) trials. On half of the dual-target trials T2 was presented 3 items, or 351 ms after T1 (lag 3) and on the other half T2 was presented 8 items, or 936 ms after T1 (lag 8). T1 was always the 6th item in the stream. On 80% of trials at each lag T1 was a string of five repeated uppercase letters (e.g., BBBBB) chosen randomly from the letter set B, C, D, E, F, N, P, S, U, X, or Z. On the remaining 20% of trials T1 was a string of five repeated lowercase letters (e.g., bbbbb) chosen randomly from the same letter set. All trial types were presented randomly within each block. Each trial began with a fixation cross (500 ms), followed by a fore-period of 2 seconds before the onset of the RSVP stream. The T1 probability manipulation and the distracter-only trials were included for the purposes of a separate

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[9] When data from these six participants were included, the interaction of laterality (central, left, right) X frontal vs. parietal in the T2 analysis was no longer significant. The significance of all other effects from the entire set of analyses did not change with the addition of these six participants' data. Also, the results of all T2 analyses were the same when the data from the participant with negative ERD values were included.

study. The RSVP stream consisted of 18 alphanumeric stimuli with an SOA of 117 ms per item. T1 was presented in white font on a gray background. T2 was one of 10 different color words (e.g., "GREEN"), and appeared in black uppercase letters. The distracter items consisted of non-color, affectively neutral words also presented in black uppercase letters. At the end of each stream, participants indicated whether the white letter string was in upper- or lower-case letters, and then reported which color word was presented as T2. Participants were told that some of the trials would contain no targets, and on these trials they should simply press the spacebar to initiate the next trial. Participants made their T1 and T2 responses sequentially in an unspeeded manner using specified keys on the keyboard. Stimulus presentation and participant responses were controlled using E-Prime software (Schneider et al., 2002).

EEG Acquisition

EEG was recorded continuously during the RSVP blocks using tin electrodes embedded in an Electro-cap© (Electro-cap International Inc., Eaton, Ohio) from 60 scalp sites distributed according to the 10-20 system, with an electrode placed anterior to Fz as ground. EEG was recorded using linked left and right earlobes as reference and was re-referenced to a common average. EEG data were acquired with Neuroscan acquisition software (Compumedics USA, Charlotte, North Carolina) running on a Sony VAIO Pentium 4 desktop PC, and using two 32-channel NeuroScan SynAmps. Data were sampled at a rate of 500 Hz. Electro-oculogram (EOG) recorded horizontal eye movements using electrodes placed on the outer canthus of each eye, and vertical eye movement and blinks using electrodes placed on the infra- and supra- orbital regions of each eye. Impedance for both the EEG and EOG was maintained below 10 kΩ.

EEG Analysis

Using Neuroscan software, EEG data was corrected for electro-oculogram activity. The software uses an algorithm that calculates the amount of covariation between each EEG channel and a vertical EOG channel and removes the EOG from each EEG electrode on a sweep-by-sweep, point-by-point basis to the degree that the EEG and EOG covary. An epoch was created for each trial that spanned 2750 ms. Each epoch began 250 ms before the onset of the fixation cross and ended just before the onset of the RSVP stream. Thus the epoch encompassed the 250 ms baseline period before the onset of the fixation cross, the 500 ms fixation duration, and the 2000 ms blank foreperiod prior to the onset of the RSVP stream. Epochs were rejected if they contained activity exceeding ±75 µV in any channel except linked–ear reference and EOG electrodes. Each accepted epoch was subsequently visually inspected for the presence of artifacts, and rejected if any were found.

Alpha ERD Computation

Alpha ERD is traditionally examined by dividing alpha frequencies into a lower bandwidth (8-10 Hz) and a higher bandwidth (10-12 Hz) as lower and higher frequencies of alpha have been shown to have dissociable effects (Klimesch et al., 1992). Klimesch et al. proposed that ERD in the lower alpha range is representative of general alertness, and input/output processes, while ERD in the higher alpha range represents task specific, computational processes, such as stimulus identification and controlled processing that are more closely related to selective attention. In the present study I was interested in examining anticipatory attention modulated by the top-down cognitive control processes discussed in cognitive control models of the AB (Di Lollo et al., 2005; Olivers & Meeter,

2008; Taatgen et al, 2009). Therefore, it was appropriate to confine my analyses of alpha

ERD to the higher range of alpha frequencies, which are thought to represent such

controlled cognitive processes.

In order to determine alpha ERD, epochs were bandpass filtered with a low-pass

of 12 Hz and a high-pass of 10 Hz at 48 dB/oct. The amplitude of the filtered EEG was

then squared to provide an estimate of power. The power estimate was then collapsed

across 125 ms intervals by averaging the power within that interval in order to yield a

more reliable estimate. ERD was then computed as the percent difference in power at

each 125 ms interval and the baseline period, where baseline was defined as the average

alpha power during the 250 ms preceding the fixation stimulus. For the T1 analysis

epochs were averaged according to T1 accuracy (correct or incorrect), resulting in two

averages for each participant. For the T2 analysis epochs where T1 was correct were then

averaged according to two factors: lag (3 or 8) and T2 accuracy (correct or incorrect),

resulting in four averages for each participant. Anticipatory alpha ERD was then

computed as the mean alpha ERD during the 2,000 ms foreperiod by averaging across the

sixteen 125 ms intervals that comprised the foreperiod.

The mean number of epochs per participant for the T1 correct average was 597.18

(SD = 96.49, range from 314-689), T1 incorrect average was 103.07 (SD = 97.89, range

from 11-398)[10]. The mean number of epochs per participant for the lag 3/T2 correct

average was 57.67 (SD = 29.85; range from 25-112); lag 3/T2 incorrect was 34.90 (SD =

15.95; range from 7 to 65); lag 8/T2 correct was 99.10 (SD = 28.43; range from 44 to

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[10] One may be concerned that the T1 results are an artifact of the large disparity in number of epochs for correct and incorrect trials. However, the same pattern of results was obtained when T1 correct averages were calculated based on a random sample of T1 correct epochs equal to the number of incorrect T1 epochs, with the exception that the interaction of T1 performance outcome with frontal vs. parietal (F (1, 21) = 4.09, p = .057) just missed the traditional level of significance.

158); and for lag 8/T2 incorrect was 11.52 (SD = 5.73; range from 6 to 26).The relatively

low number of epochs for lag 8/T2 incorrect averages is due to the normally high

accuracy of T2 at the long lag that is meant to reflect baseline T2 accuracy without a

dual-task deficit.

Alpha ERD was analyzed across six ROIs. Those ROIs were defined as fronto-

central (sites FZ, CZA, F1, F2), left frontal (sites F3, F5, C3A, C5A), right frontal (sites

F4, F6, C4A, C6A), parieto-central (sites PZ, PZA, P1, P2), left parietal (sites P3, P5,

P3P), and right parietal (sites P4, P6, P4P). Mean alpha ERD values for the foreperiod

were averaged across the sites specified for each ROI.

Results

AB Task Performance

Mean T1 accuracy was 90.67% (*SD* = 8.34), and ranged from 70% to 98% for

individual participants. T2 accuracy was calculated for T1 correct trials only. Mean T2

accuracy at lag 3 was 66.67% (*SD* = 15.22), while mean accuracy at lag 8 was 89.33%

(*SD* = 5.54). A paired-samples t-test between T2 accuracy at lag 8 and 3 was significant (*t*

(20) = 8.15, *p* < .001), such that mean T2 accuracy increased from lag 3 to lag 8,

indicating the presence of an AB.

Alpha ERD Results

A widespread, sustained anticipatory alpha ERD was observed during the

foreperiod with a mean of 37.85% across all regions of interest (regardless of condition).

Figure 3-1 depicts the group average alpha ERD during the foreperiod for each factorial

combination of lag (black lines for lag 3 and gray lines for lag 8) and T2 accuracy (solid

lines for T2 correct and dashed lines for T2 incorrect) at site CZA. Figure 3-2 shows the

topographical distribution of anticipatory alpha ERD averaged across participants.
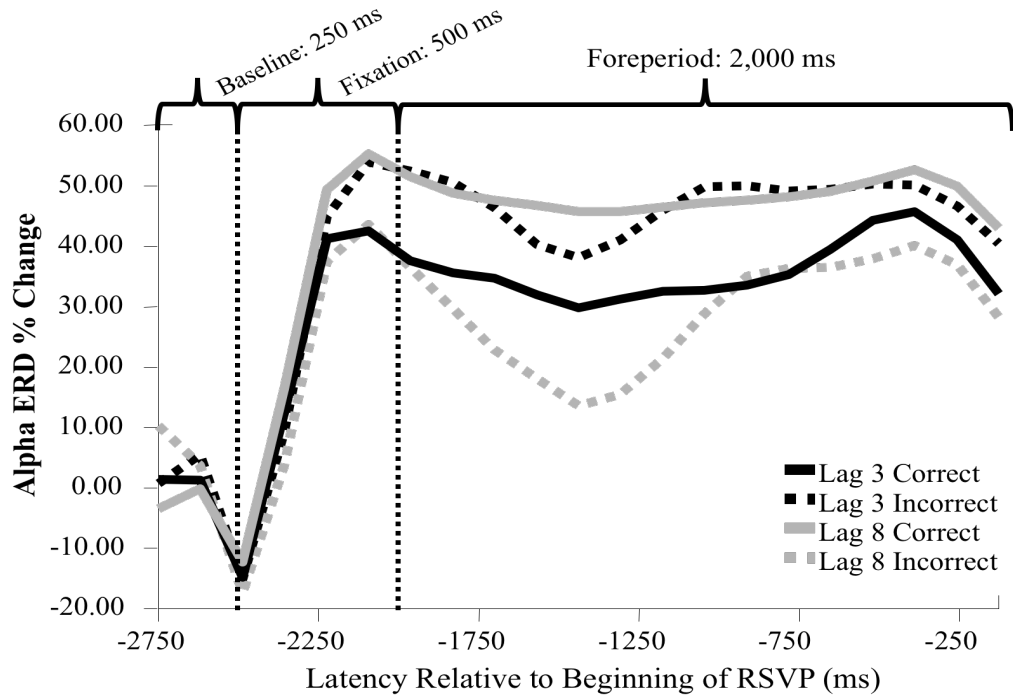
Figure 3-1. Group average alpha ERD during the foreperiod for each factorial
combination of lag (black lines for lag 3 and gray lines for lag 8) and T2 accuracy (solid
lines for T2 correct and dashed lines for T2 incorrect) at site CZA
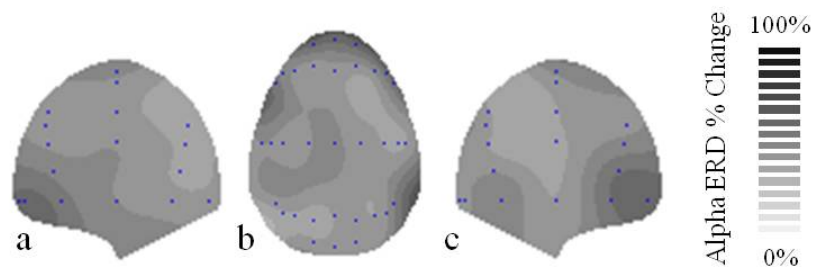
Figure 3-2. Topographical distribution of group average alpha ERD, averaged across all
conditions and over the entire foreperiod: (a) view from left, (b) view from top, (c) view
from right

T2 Analysis. A 3 X 2 X 2 X 2 repeated-measures ANOVA was conducted to

examine the effects of ROI, lag (3 and 8), and T2 performance (correct and incorrect)

associated with anticipatory alpha ERD. ROI was divided into two separate factors: laterality (central, left, right) and frontal vs. parietal[11]. All effects were examined for violations of the sphericity assumption and Greenhouse-Geisser corrected values are reported where violations were present. The main effect of frontal vs. parietal was significant (F (1, 20) = 11.67, p = .003) such that anticipatory alpha ERD was greater at frontal sites than at parietal sites. The interaction of laterality (central, left, right) X frontal vs. parietal was significant (F (2, 40) = 7.12, p = .002), such that at frontal sites anticipatory alpha ERD was greatest at right lateralized sites and at parietal sites anticipatory alpha ERD was greatest centrally. The interaction of lag X T2 accuracy was also significant (F (1, 26) = 6.63, p = .018)[12]. *Figure 3-3* depicts the group average alpha ERD during the foreperiod for each factorial combination of lag and T2 accuracy averaged across ROI. The interaction of lag and T2 accuracy did not enter into any higher-order interactions with ROI variables, nor did lag or accuracy separately (all p's > .23). No other effects reached significance (all p's > .09).

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[11] Additionally, I examined alpha ERD at an occipital ROI (average of O1, OZ, and O2). A 2 (lag) X 2 (T2 correct/incorrect) repeated measures ANOVA on alpha ERD at the occipital ROI showed a significant interaction of lag and T2 accuracy (F (1, 20) = 4.47, p = .047) with the same pattern of means as that observed across ROIs in the original analysis.

[12] As alpha ERD is calculated as percent change in alpha relative to the baseline, it is possible that the interaction effect of lag and T2 performance associated with alpha ERD is due to pre-existing differences in alpha power during the baseline period. In order to test this possibility I performed the same 3 X 2 X 2 X 2 repeated-measures ANOVA with baseline alpha power as a covariate. The pattern and significance of the results from this analysis did not differ from the analysis without baseline alpha power as a covariate. Also, see Figure 1 for an indication that the baseline ERD levels were very similar across the four conditions.
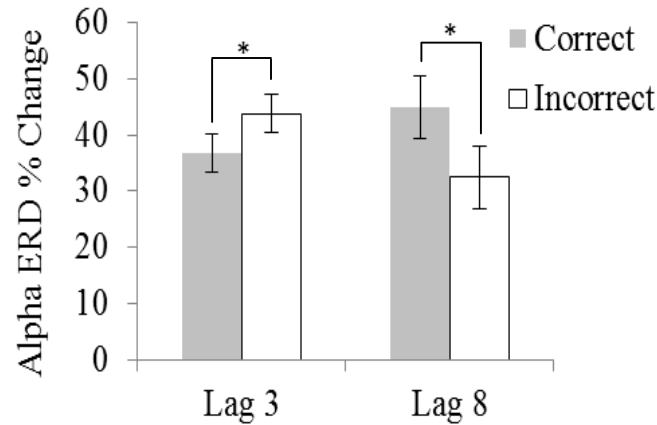
Figure 3-3. Mean alpha ERD during the RSVP foreperiod as a function of lag and T2 accuracy, averaged across ROI. Error bars represent standard error on the difference between correct trials and incorrect trials at each lag (* indicates $p < .05$ for the comparison indicated).

In order to further explore the significant interaction between lag and accuracy I examined the difference in anticipatory alpha ERD between T2 correct and incorrect trials separately for each lag. This would allow me to address my hypothesis that greater anticipatory attentional investment would be associated with incorrect T2 performance at lag 3, but that greater anticipatory alpha ERD would be associated with correct T2 performance at lag 8. Paired t-tests revealed that anticipatory alpha ERD was greater on T2 incorrect trials ($M = 42.27$, $SD = 25.72$) than T2 correct trials ($M = 35.17$, $SD = 28.06$) when T2 was presented at lag 3 ($t (20) = 2.08$, $p = .05$). This pattern of results was reversed when T2 was presented at lag 8, such that anticipatory alpha ERD was greater on T2 correct trials ($M = 43.47$, $SD = 21.42$) than T2 incorrect trials ($M = 30.50$, $SD =$

33.19) when T2 was presented at lag 8 (t (20) = 2.31, p = .031)[13]. It is also worth noting

that while the comparison of alpha ERD on T2 incorrect trials at lags 3 and 8 was not

significant (t (20) = 1.43, p = .168), the comparison of alpha ERD on T2 correct trials at

lags 3 and 8 was (t (20) = 2.40, p = .026).

T1 Analysis. A 3 X 2 X 2 repeated-measures ANOVA was conducted to examine

the effects of ROI and T1 performance (correct and incorrect) associated with

anticipatory alpha ERD. ROI was again divided into two separate factors: laterality

(central, left, right) and frontal vs. parietal. No main effects were significant (all p's >

.15). The interaction of T1 performance and frontal vs. parietal was significant (F (1, 20)

= 7.56, p = .012). In order to further investigate this interaction, two separate paired

samples t-tests were conducted comparing mean alpha ERD on T1 correct trials with

mean alpha ERD on T1 incorrect trials for frontal and parietal ROIs (collapsed across

laterality). The effect of T1 performance was significant (t (20) = 2.13, p = .045) at

frontal ROIs such that anticipatory alpha ERD was greater on trials where T1 was correct

(M = 51.24, SD = 16.16) than on trials where T1 was incorrect (M = 45.03, SD = 21.89).

In contrast, at parietal ROIs there was no significant difference in anticipatory alpha ERD

for correct (M = 45.73, SD = 18.71) and incorrect (M = 45.82, SD = 21.08) T1 trials, (t

(20) = -.026, p = .980).

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[13] As another test of whether greater anticipatory alpha ERD is associated with the AB, I calculated each
participant's overall anticipatory alpha ERD value (ERD averaged across all possible conditions and ROIs)
and each participant's AB magnitude, and examined the correlation between them. It was expected that
those individuals with greater anticipatory alpha ERD would show larger ABs (i.e., that there would be a
positive correlation between alpha ERD and AB magnitude). AB magnitude was calculated as the residual
difference measure. The residual difference measure was calculated using the following steps: (1) T2
performance on lag 3 trials was subtracted from T2 performance on lag 8 trials creating a difference
measure for each individual, (2) T2 performance on lag 8 trials was regressed on the difference measure,
(3) the standardized residual was saved, it represents the variability in difference scores controlling for
variability in baseline T2 performance (i.e. T2 performance on lag 8 trials). *Note that larger/more positive
values on the residual difference measure indicate larger AB magnitudes*. Overall anticipatory alpha ERD
was positively correlated with AB magnitude but not significantly (r = .26, p = .252).

Discussion

The purpose of this study was to investigate whether the level of attention invested in anticipation of the AB task differed with target accuracy according to hypotheses derived from various models of the AB. Specifically, those models suggest that an overexertion of cognitive control and/or an inappropriate investment of attention underlies the AB (Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). I measured the level of anticipatory attentional investment by examining alpha ERD during a 2 second foreperiod following a cue that the RSVP stream was to begin.

I hypothesized that anticipatory alpha ERD would be larger on trials where an AB was present (incorrect T2 performance at short lag) than when no AB was present (correct T2 performance at short lag). In line with my hypothesis, I found that anticipatory alpha ERD was larger on short lag trials with incorrect T2 performance than on short lag trials with correct T2 performance. I also hypothesized that anticipatory alpha ERD on T2 incorrect trials would be smaller than or equal to alpha ERD on T2 correct trials at the long lag, as greater attention would not be costly to T2 accuracy once T1 processing had been completed. As predicted, anticipatory alpha ERD was greater on long lag trials with correct T2 performance than on long lag trials with incorrect T2 performance. This lag X T2 accuracy pattern was not specific to certain ROIs, but was observed across the scalp. Finally, I hypothesized that anticipatory alpha ERD on T1 incorrect trials would be smaller than or equal to anticipatory alpha ERD on T1 correct trials, as greater attention should benefit T1 accuracy. As predicted, greater anticipatory alpha ERD was observed on T1 correct trials than on T1 incorrect trials.

My results suggest that the AB (impaired T2 performance at shorter but not at longer lags) is associated with greater attentional investment, as measured by alpha ERD and that this attentional investment is prepared in anticipation before each RSVP trial. However, my results cannot identify what patterns of investment during the RSVP stream may contribute to the AB. Furthermore, my results suggest that while greater attentional investment may cause low T2 accuracy at the short lag, when T2 is presented at the long lag, or in the case of T1, greater attentional investment results in higher target accuracy. So it is possible that, while overinvesting attention (i.e. applying as many perceptual or cognitive resources as possible) is a good strategy when interference from a preceding target is absent, when time restraints require greater control over the allocation of limited resources to multiple targets overinvesting attention impairs performance. The finding that greater attention improves T1 and long lag accuracy may also explain why participants persist in overinvesting attention despite poor T2 accuracy at the short lag.

In this study it was found that different levels of the same measure - anticipatory attentional investment as measured by alpha ERD - were associated with opposite behavioral outcomes at long and short lags (i.e., greater pre-trial ERD was associated with impaired T2 accuracy at short lags, but improved T2 accuracy at long lags). This finding is in line with the relationship observed between trait affect and the AB (MacLean et al., 2010). MacLean et al. found that while greater affect valence (positive affect > negative affect; which has been linked to an open and less focused attentional style) was associated with better T2 performance at the shorter lags it was also associated with worse T2 performance at the long lag. In line with my conclusions here, MacLean et al. proposed that a more focused or invested attentional state is harmful to T2

performance at the short lag as it increases interference from T1 and its surrounding distracters, but that at longer lags where the interference from T1 and its surrounding distracters is absent a more focused or invested attentional state results in better target selection.

The significant and sustained alpha ERD I observed during the 2-second foreperiod following a cue indicating that the RSVP stream was to begin is consistent with considering alpha ERD as an index of anticipatory attention. While there was no significant interaction of either ROI factor with lag, T2 accuracy, or the lag X T2 accuracy interaction, there was a significant laterality by frontal/parietal interaction where alpha ERD was larger at frontal sites, where it was right-lateralized, than parietal sites, where it was centralized. Typically alpha ERD is examined over parieto-occipital sites (Bastiaansen & Brunia, 2001; Bastiaansen et al., 2002; Bastiaansen et al., 2001; Capotosto et al., 2009; Yamagashi et al., 2005). The rationale for examining alpha ERD effects at parieto-occipital sites is due to their position over the visual cortex. In the context of the attentional gating hypothesis this makes sense as changes in alpha power in the cortex represent changes in the flow of stimulus information from the thalamus to the cortex, specifically to primary sensory areas (Lopes da Silva, 1991; Steriade et al., 1990).

However, similar to my findings, Onoda et al. (2007) observed effects on alpha ERD over both occipital and right frontal areas using MEG. They proposed that the alpha ERD observed over right frontal areas represented top-down control of anticipatory attention by areas in the right frontal cortex, which may be part of a distributed frontal attention network, modulating activity in the visual cortex via the thalamus. If that were the case it would suggest that the larger frontal alpha ERD I observed is representative of

sustained top-down control over early perceptual processes. This would also be consistent with a variety of neurophysiological studies (e.g., Marcantoni et al., 2003; Marois et al., 2000; Martens et al., 2006b) showing that the AB is associated with activation in sites such as prefrontal cortex and lateral frontal cortex that are thought to be involved in executive control of attention (e.g., Posner & Dehaene, 1994).

In conclusion, using an electrophysiological index of anticipatory attention (alpha ERD), I found that anticipatory attentional investment was associated with both T1 and T2 performance outcomes during the AB task. Greater levels of anticipatory attentional investment were associated with, worse T2 performance at shorter T1-T2 separations, better T2 performance at longer separations, and better T1 performance. However, anticipatory attentional investment as measured by alpha ERD did not predict individual differences in AB magnitude. This suggests that although the level of anticipatory attentional investment may change with fluctuations in T2 performance within an individual, their average level of anticipatory attentional investment does not vary with the size of their AB deficit (i.e. the proportion of trials where T2 performance was correct/incorrect at short/long lags). These results provide support for various models of the AB which propose that maladaptive cognitive control or inappropriate investment of attention underlies the AB.

Replication Study

As part of a separate study, alpha ERD data was collected from an additional 63 participants. With this additional sample I will (1) attempt to replicate the effects observed in the original study (i.e. Study 2, as described above) in the additional sample, (2) examine any differences in the data from the sample in the original study and the

additional sample, and finally (3) examine the effects observed in the original study with increased power by combining the original and the additional sample into one, composite sample.

Methods

Participants

Participants were 63 Brock University undergraduate students (57 females; *M* age = 20 years), recruited through the Brock Psychology Department's online system for participant recruitment. Complete data were only available for the 58 participants who finished the tasks. The data for a further12 participants were excluded due to either poor T1 performance (< 70%) or poor T2 performance at lag 8 (< 50%). Data for an additional six participants was removed as they displayed event-related synchronization (ERS; negative ERD values). The final sample included in the following analyses was comprised of 40 participants.

Procedure

The AB task, the procedures, and the EEG acquisition and analyses were identical to those above with the exception that participants completed a resting EEG procedure and an AB detection task prior to the AB AFC task used above. They also completed a time production task after the AB AFC task as part of a larger study (not examined here).

Replication Results

AB Task Performance

Mean T1 accuracy for the remaining participants was 86.23% (*SD* = 8.98). Mean T2 accuracy at lag 3 was 58.98% (*SD* = 18.22). Mean T2 accuracy at lag 8 was 86.85% (*SD* = 9.97). A paired-samples t-test showed significantly lower T2 accuracy at lag 3 than

lag 8 indicating the presence of an AB (t (39) = 12.87, $p < .001$). There was no significant

difference for any of the AB performance measures between the original and the

additional sample, although accuracy was lower in general and AB magnitude was larger

(see Table 3-1).

Table 3-1 Comparisons with AB performance from Study 2

	Additional		Original	
	M		M	t
T1	86.23	=	90.67	1.88
Lag 8 T2 Accuracy	86.85	=	89.33	1.06
Lag 3 T2 Accuracy	58.98	=	66.67	1.65
AB Magnitude (Lag 8 – Lag 3)	27.88	=	22.67	1.44

*$p < .05$

Alpha ERD Results

A widespread, sustained anticipatory alpha ERD was observed during the

foreperiod with a mean of 33.67% across all regions of interest (regardless of condition).

Figure 3-4 depicts the group average alpha ERD during the foreperiod for each factorial

combination of lag (black lines for lag 3 and gray lines for lag 8) and T2 accuracy (solid

lines for T2 correct and dashed lines for T2 incorrect) at site CZA. *Figure 3-5* shows the

topographical distribution of anticipatory alpha ERD averaged across participants. Mean

anticipatory alpha ERD in the additional sample was not significantly different from that

observed in the original sample (t (59) = .82, $p = .413$).
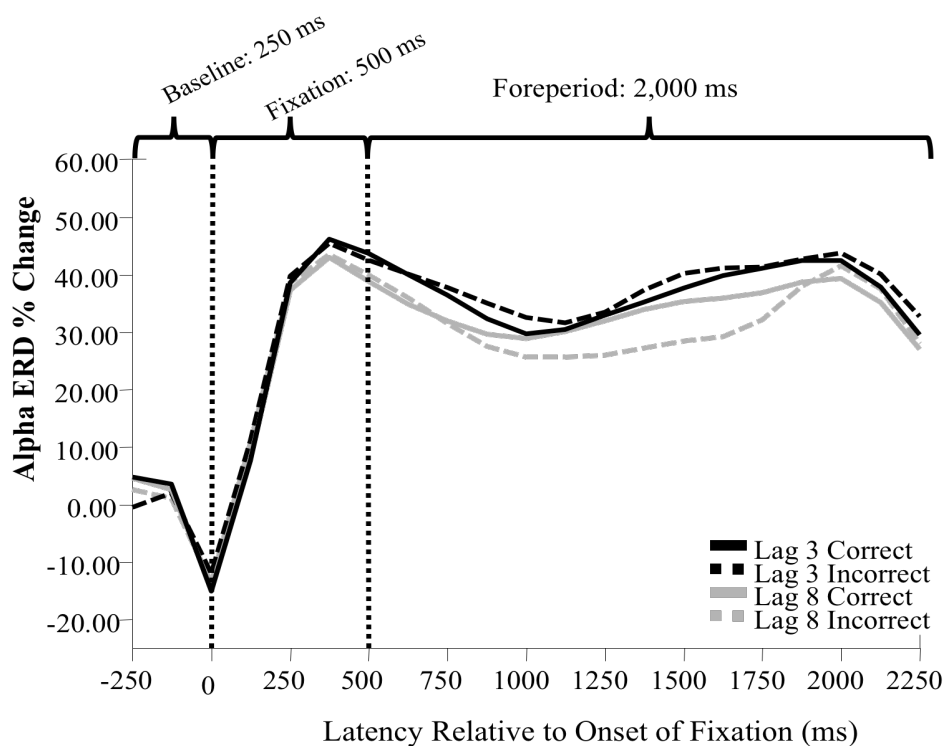
Figure 3-4. Group average alpha ERD during the foreperiod for each factorial combination of lag (black lines for lag 3 and gray lines for lag 8) and T2 accuracy (solid lines for T2 correct and dashed lines for T2 incorrect) at site CZA
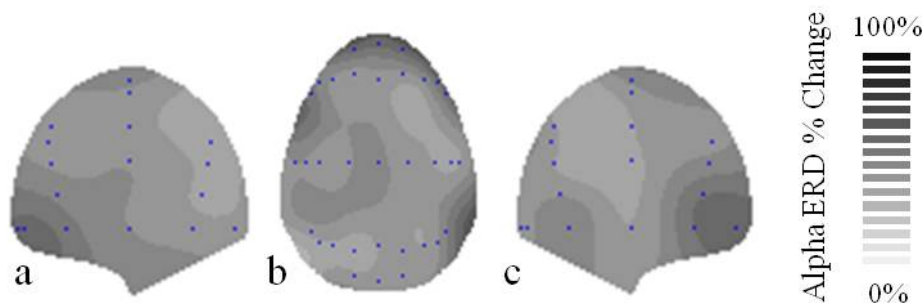
Figure 3-5. Topographical distribution of group average alpha ERD, averaged across all conditions and over the entire foreperiod: (a) view from left, (b) view from top, (c) view from right

T2 Analysis. A 3 X 2 X 2 X 2 repeated-measures ANOVA was conducted to

examine the effects of ROI, lag (3 and 8), and T2 performance (correct and incorrect)

associated with anticipatory alpha ERD. ROI was divided into two separate factors:

laterality (central, left, right) and frontal vs. parietal. All effects were examined for violations of the sphericity assumption and Greenhouse-Geisser corrected values are reported where violations were present. The main effect of frontal vs. parietal was significant (F (1, 39) = 29.89, p < .001) such that anticipatory alpha ERD was greater at frontal sites than at parietal sites. The interaction of laterality X frontal vs. parietal was significant (F (2, 78) = 19.66, p < .001), such that at frontal sites anticipatory alpha ERD was greatest at left lateralized sites and at parietal sites anticipatory alpha ERD was greatest centrally. The topographies observed in the additional sample are different from those in the original sample where alpha ERD was greatest at *right* lateralized sites frontally.

Unlike in the original sample the interaction of lag X T2 accuracy was not significant (F (1, 39) = 1.36, p = .250). However, also unlike in the original sample the interaction of lag X T2 accuracy entered into a significant higher order interaction with both frontal vs. parietal and laterality (F (2, 78) = 3.64, p = .031). No other effects reached significance (all p's > .09).

In order to explore this four-way interaction I performed separate 2 X 2 repeated measures ANOVAs of anticipatory alpha ERD with lag and T2 accuracy as factors for each ROI separately. ROIs were defined as the factorial combinations of frontal vs. parietal and laterality (left, center, right), resulting in six ROIs. The lag X T2 accuracy interaction was only significant at the right frontal ROI (F (1, 39) = 4.81, p = .034; see *Figure 3-6)*. This interaction was such that alpha ERD was significantly larger on T2 incorrect trials (M = 45.33, SD = 2.41) than T2 correct trials (M = 39.49, SD = 2.92) when T2 was presented at lag 3 (t (39) = 2.33, p = .025), but did not differ between T2

correct trials ($M = 39.82$, $SD = 2.60$) and T2 incorrect trials ($M = 35.93$, $SD = 4.25$) when T2 was presented at lag 8 ($t(39) = 1.05$, $p = .300$). It is also worth noting that the comparison of alpha ERD on T2 incorrect trials at lags 3 and 8 was not significant ($t(39) = 1.23$, $p = .227$), and neither was the comparison of alpha ERD on T2 correct trials ($t(39) = .24$, $p = .812$).
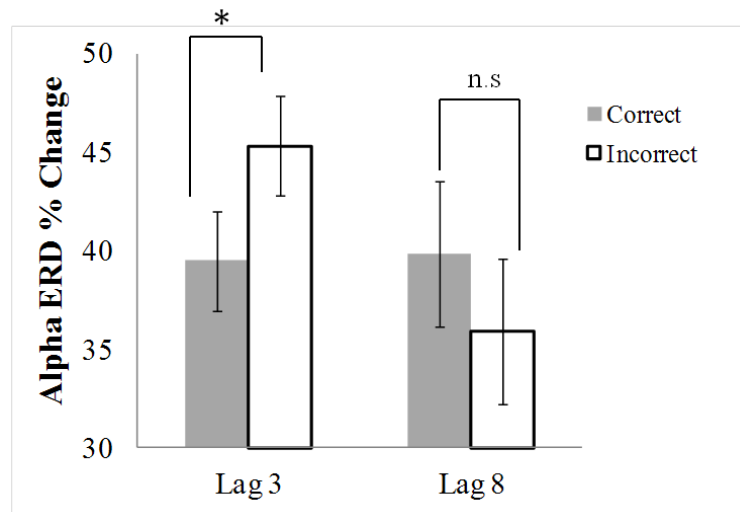
Figure 3-6. Mean alpha ERD during the RSVP foreperiod as a function of lag and T2 accuracy, at the right frontal ROI. Error bars represent standard error on the difference between correct trials and incorrect trials at each lag (* indicates $p < .05$ for the comparison indicated).

T1 Analysis. A 3 X 2 X 2 repeated-measures ANOVA was conducted to examine the effects of ROI and T1 performance (correct and incorrect) associated with anticipatory alpha ERD. ROI was again divided into two separate factors: laterality (central, left, right) and frontal vs. parietal. Both main effects of ROI were significant, such that alpha ERD was greater at frontal ROIs than at parietal ROIs ($F(1, 39) = 19.63$, $p < .001$), and alpha ERD was greater a left and central ROIs than right ROIs ($F(2, 78) = 3.37$, $p = .039$). The interaction of the ROI factors was also significant such that alpha

ERD was greater centrally at parietal ROIs and greater laterally (left and right) at frontal ROIs (F (2, 78) = 14.88, $p <$ .001).

The interaction of T1 performance and frontal vs. parietal, which was significant in Study 2, only approached statistical significance in the additional sample (F (1, 39) = 2.86, p =.099). In order to further investigate this interaction, two separate paired samples t-tests were conducted comparing mean alpha ERD on T1 correct trials with mean alpha ERD on T1 incorrect trials for frontal and parietal ROIs (collapsed across laterality). The effect of T1 performance was not significant (t (39) = 1.42, p = .164) at frontal ROIs or at parietal ROIs (t (39) = .46, p = .645). However, while not statistically significant, alpha ERD was larger preceding T1 correct than T2 incorrect trials at both frontal and parietal ROIs, and this difference was larger at frontal ROIs.

Combining the Samples

When the original and the additional samples were combined into a composite sample the 3 X 2 X 2 X 2 repeated-measures ANOVA yields a pattern of results identical to that observed with the original sample alone, including a significant interaction of lag X T2 accuracy (F (1, 60) = 7.11, p = .01), with no higher order interactions. In the combined sample anticipatory alpha ERD was significantly larger on incorrect than correct trials where T2 was presented at lag 3 (t (60) = 2.32, p = .024), and that while anticipatory alpha ERD was larger on correct than incorrect trials where T2 was presented at lag 8, the difference fell short of significance (t (60) = 1.88, p = .065). The T1 results observed in the original sample were replicated in the overall sample, with a significant interaction of frontal vs. parietal and T1 performance (F (1, 60) = 6.63, p =

.013), such that alpha ERD is greater preceding T1 correct trials than T1 incorrect trials but this difference is only significant at frontal sites.

So, while the key T2 finding showing that pretrial alpha ERD is greater on incorrect trials than correct trials at lag 3, but greater on correct trials than incorrect trials at lag 8 was replicated only at right frontal sites in the replication sample, the pattern of anticipatory alpha ERD observed in the original sample was replicated overall when the samples were combined. The results showing greater pretrial alpha ERD on T1 correct trials, than on T1 incorrect trials was observed in both samples, and overall.

Associations between Individual Differences in Alpha ERD and AB Magnitude

In the original sample a non-significant positive correlation ($r = .26$, $p = .252$) was observed between individuals' levels of overall anticipatory alpha ERD and individuals' AB magnitude, suggesting that individuals with greater pretrial ERD may also show larger ABs. The lack of statistical significance for that correlation was possibly due to low power ($n = 21$). However, that positive correlation was not replicated in either the additional sample ($r = -.07$, $p = .690$) with an n of 40, or in the combined sample ($r = .05$, $p = .685$) with an n of 61 (see Appendices B.3 and B.4).

Additional Analyses: Reference Interval and Fore-Period Alpha, and Absolute Alpha ERD

Why was the replication of the T2 pattern of ERD results restricted to right frontal sites in the replication study, but appeared at all sites in the main study (even though ERD was largest at right frontal sites)? Anticipatory alpha ERD is typically calculated as a percent difference in alpha power. The percent decrease is used as a means to control for individual differences in alpha power in the reference interval (i.e. baseline alpha

power). For example, a decrease in alpha power from the reference interval (pre-fixation)

to the fore-period of 2 μV^2 is relatively little reduction in alpha when alpha power in the

reference interval was 20 μV^2 (10% change) as compared to 4 μV^2 (50% change). The

percent decrease is the measure of anticipatory alpha ERD used in both the original study

and in the replication study (i.e. reference interval alpha power – foreperiod alpha power)

/ reference interval alpha power * 100. In order to further examine the differences

between the two studies I measured alpha power in the reference interval and in the

foreperiod interval, as well as the absolute difference in alpha power between the

reference and foreperiod intervals.

Specifically, while the percent difference measure (i.e. alpha ERD) did not differ

between the samples, it is possible that baseline alpha power in the reference interval and

absolute difference in alpha power (reference interval alpha power – foreperiod alpha

power) differed between the original and the additional sample. If the variability in the

baseline alpha and in absolute difference in alpha is reduced in the additional sample it

may account for why the significant interaction of ERD values as a function lag and T2

correct/incorrect was significant across all sites in the original sample, and only at right

frontal sites in the replication sample.

In order to measure alpha power, epochs were band pass filtered with a low pass

of 12 Hz and a high pass of 10 Hz at 48 dB/oct. The amplitude of the filtered EEG was

then squared to provide an estimate of power. The power estimate was then collapsed

across 125 ms intervals by averaging the power within that interval in order to yield a

more reliable estimate. Baseline alpha power was defined as the average alpha power

during the 250 ms preceding the fixation stimulus; foreperiod alpha power was defined as

the average alpha power during the 2,000 ms following the fixation stimulus. The absolute difference in alpha power was calculated as reference interval alpha power – foreperiod alpha power.

Variance in both baseline and foreperiod alpha power, and in the absolute difference in alpha power was significantly less in the additional sample than in the original sample as indicated by Levene's test for equality of variances (all p's < .01). In addition to less variance in the additional sample, mean alpha power was also significantly less in the baseline (t (29.36) = 7.50, p < .001), and in the fore-period (t (26.65) = 4.45, p < .001) as compared to the original sample. The absolute difference in alpha power was also significantly less in the additional sample as compared to the original sample (t (27.54) = 7.18, p < .001). It is possible that the low mean baseline alpha power in the additional sample created a floor effect, reducing variance as well as the absolute amount of alpha reduction possible, thereby restricting the replication of the T2 pattern of ERD results to right frontal sites in the replication study – an area that where alpha ERD has been previously shown to be maximal in Study 2, and an area linked to top-down control of anticipatory attention by areas in the right frontal cortex (Onada et al, 2007). This possible floor effect may also account for the inability to replicate the positive correlation between individual levels of alpha ERD and individual differences in AB magnitude.

Discussion

In Study 2 it was found that alpha ERD was greater preceding "AB"[14] trials (i.e., trials for which T2 was incorrect when presented at a short lag) than "no AB" trials (i.e., trials for which T2 was correct when presented at a short lag), but that alpha ERD was greater preceding T2 correct trials than incorrect trials when presented at a long lag. Alpha ERD was also greater on T1 correct trials than on T1 incorrect trials. A positive, although not significant, correlation between alpha ERD and AB magnitude was also reported. I conceptualized greater alpha ERD as indicative of greater anticipatory, preparatory investment of attention, thus high levels of anticipatory attentional investment were associated with the AB.

As part of another study I collected alpha ERD data from an additional 63 participants. The topographical distribution and distribution of AB performance observed in the additional sample generally replicated those observed in Study 2. However, the interaction of lag (short or long) and T2 performance (correct and incorrect) was observed only at right frontal sites. When the additional sample was combined with the original sample the lag X T2 performance interaction was significant across ROIs, and the pattern matched that reported in the original sample from Study 2.

The correlation between alpha ERD and AB magnitude reported in Study 2, however, was not observed in the additional sample or in the combined samples. It was found that mean alpha and the variance in alpha was significantly reduced in the additional sample as compared to the original sample from Study 2. This suggests that a

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[14] The terms "AB" and "no AB" trials are a short hand for those trials where T2 performance was either incorrect or correct at shorter lags. The AB itself represents a pattern where T2 performance is impaired at shorter but not longer lags. The pattern of Study 2, where alpha ERD is larger for T2 incorrect at shorter lags but larger for T2 correct at longer lags supports our assumption that the AB is associated with greater alpha ERD.

floor effect may be responsible for the failure to find the significant lag X T2 accuracy

interaction across ROIs, and the failure to replicate the correlation between alpha ERD

and AB magnitude.

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Preface to Chapter 4

Note that Chapter 4, excluding the replication study, is a published manuscript:

MacLean, M.H., Arnell, K.M., & Cote, K.A. (2012). Resting EEG in alpha and beta bands predicts individual differences in attentional blink magnitude. *Brain and Cognition*, 78, 218-229.

Chapter 4

Introduction

Attention allows for the selective processing of information. This selectivity can help to ensure that relevant information is available to influence behavior. However, attention is a finite resource, and there are limits on how much information one can attend to at any one time. When the relevant information available exceeds the available attentional resources, the probability that relevant information will be missed increases. The attentional blink (AB) is a deficit in performance attributed to such attentional limitations (Raymond, et al. 1992).

The Attentional Blink and Attentional Limitations

When the second of two to-be-attended targets is presented within half a second of the first target (T1) in a rapid serial visual presentation (RSVP) stream, report accuracy for the second target (T2) is impaired compared to when T2 is presented after a longer interval following T1 (Broadbent & Broadbent, 1987; Raymond, et al. 1992). This T1-T2 lag dependant dual-task effect is referred to as the attentional blink (AB; Raymond et al., 1992).

Generally, the AB has been attributed to a lack of sufficient attentional resources necessary for multiple targets to be appropriately processed into awareness. For some authors, this has been instantiated in terms of bottleneck information processing models. For example, bottleneck models of the AB (e.g., Chun & Potter, 1995; Jolicoeur, 1998) propose that encoding the temporary representation of T1 into working memory consumes resources to the extent that any subsequent encoding of relevant information, such as T2, would need to be suspended until encoding of T1 was complete. Suspending the encoding of the fragile, temporary representation of T2 when it's processing overlaps

with that of T1 is thought to reduce the fidelity of the T2 representation, leading to decreased report accuracy for T2 at short lags.

For other authors, a shortage of attentional resources has been instantiated in terms of limitations on cognitive control functions. For example, in the temporary loss of control model (Di Lollo, et al., 2005) top-down input filters control the efficient selection of task-relevant targets. However, because encoding T1 into working memory requires limited attentional resources, these resources are not available to exert top-down control over the input filter, and the filter falls under bottom-up control while T1 is being encoded. If T2 is presented before attention is again free to control this filter, then T2 selection is impaired reducing the likelihood of accurate T2 report at short lags.

In the context of these accounts it appears that the AB is an unavoidable consequence of the way in which selective information processing is necessarily carried out. However, the AB is not observed in some individuals, so called "non-blinkers" (Martens, et al., 2006), and individuals differ reliably in the magnitude of their AB (i.e. the slope of the lag dependant effect on T2 performance; McLaughlin, et al., 2001). This suggests that individuals may differ in their speed and efficiency of information processing and/or their approach to selective information processing (i.e. their attentional approach), which then influences the magnitude of their AB.

Measures of fluid intelligence (Arnell, et al., 2010; Colzato, et al., 2007), information processing speed (Arnell, et al., 2006), and working memory capacity (Arnell et al., 2010; Arnell & Stubitz, 2010) do not appear to predict individual differences in the AB. This suggests that it is not the quality or amount of cognitive resources that determines the magnitude of an individual's AB (Arnell et al., 2006).

There is, however, evidence that the attentional approach that an individual adopts, for example whether attention is generally diffused or focused (Dale & Arnell, 2010), or whether attention tends to be more or less invested in irrelevant information (Arnell & Stubitz, 2010; Dux & Marois, 2008; Martens & Valchev, 2009), predicts individual differences in AB magnitude. These results show that individuals who focus less on irrelevant information and have a diffuse, global processing style produce smaller ABs. Dispositional factors like trait affect (MacLean, et al., 2010) and personality (MacLean & Arnell, 2010) also predict AB magnitude. Individuals high in traits such as positive affect, openness to experience and extraversion (traits that have been linked to a diffuse processing style – e.g., Fredrickson & Branigan, 2005) show smaller ABs, while individuals high in negative affect and neuroticism (traits linked to a focused attentional style – e.g., Kramer et al., 1990) show larger ABs. So, it appears that how an individual tends to employ their cognitive resources is relevant to the magnitude of their AB, rather than the amount of resources they possess.

Attentional Approach and the AB

Recently, MacLean and Arnell (2011) operationalized attentional resource deployment in the AB task by measuring the amount of anticipatory attentional investment prior to each RSVP trial using alpha event-related desynchronization (ERD). Alpha ERD refers to a decrease in alpha power from baseline following an event; in the case of MacLean and Arnell the event was a cue that the RSVP was to begin shortly. Greater alpha ERD immediately before an RSVP trial began was shown to be beneficial for T1 performance and T2 performance at long T1-T2 lags. In contrast, alpha ERD was greater preceding short T1-T2 lag trials where T2 performance was subsequently

incorrect (an AB trial) than on those trials where T2 performance was subsequently correct (a no-AB trial), providing evidence that greater investment of attentional resources in advance of the RSVP trial, is associated with the AB (although not with individual differences in the magnitude of the AB).

In summary, measuring attentional approach tendencies between individuals using cognitive task performance and self-report questionnaires, and using EEG to measure trial-to-trial changes in state attentional investment within individuals has provided evidence that individual differences in attentional approach and attentional investment are relevant to understanding the AB and individual differences in the AB.

Personality and trait affect measures index individual differences in general tendencies over relatively long epochs of time, and alpha ERD was used to measure intra-individual states of anticipatory attentional investment several seconds before the first target appears. Thus, even before an RSVP stream begins, individuals appear to approach the trial in characteristic ways that influence performance outcomes. Therefore, it is possible that individual differences in attentional investment at rest, when not engaged in the primary goal-directed task of interest (i.e. the AB task), may predict individual differences in the AB. Enduring dispositions in attentional approach could be reflected in individual differences in attentional investment at rest. Individual differences in attentional investment at rest could be indicative of the level of investment that is likely to occur during the AB task. In the current study I examined electrophysiological measures of individual differences in attentional investment at rest for the purpose of predicting individual differences in the AB.

Electrophysiological Measures of Attention at Rest

Oscillatory activity in the alpha range of frequencies (8-12 Hz) is characteristically observed in the resting state. Specifically, alpha range oscillatory activity is increased during periods of rest with eyes closed compared to periods of rest with eyes open, an effect which is attributed to the desynchronizing effect of visual stimulation on the cortex (for a review of alpha oscillations see Niedermeyer, 1997). The presence of alpha in the waking state is thought to denote an "idling" or unoccupied cortex (Pfurtscheller, et al., 1996), or alternatively, the presence of cortical inhibition (Klimesch, et al., 2006; Klimesch, et al., 2007). In support of these conceptualizations, investigations of alpha oscillations at rest with concurrent fMRI imaging have observed a negative correlation between alpha and metabolic activity such that when alpha is present metabolic activity is reduced in the cortex (Goldman, et al., 2002; Laufs et al. 2003a, 2003b, 2006; Moosman et al., 2003). Alpha is also seen to decrease in response to a signal that an attention-demanding event is soon to occur (Brunia & van Boxtel, 2001), a phenomenon referred to as alpha event-related desynchronization (ERD) previously discussed here in relation to attentional investment and the AB. The desynchronization of alpha in response to a warning stimulus is thought to represent an engagement of attention (Brunia & van Boxtel, 2001) and/or a release of inhibition (Klimesch et al., 2006).

However, there is evidence to suggest that alpha desynchronization is not sufficient to indicate an increase in attentional investment at rest. Laufs et al. (2003b; 2006) suggest that the presence of alpha, more specifically alpha at rest, is indicative of an idling cortex and represents a baseline state. However, reduction of alpha at rest can

accompany two different kinds of fluctuations in attention. Alpha reductions may accompany an increase in vigilance, which increases their attention-demanding cognitive processes – a state that is accompanied by increased beta oscillations and decreased theta oscillations. Alpha reductions may also accompany an increase in drowsiness and a decrease in vigilance – a pattern associated with increased theta oscillations and decreased beta oscillations. Indeed, when alpha reduction was observed to correlate with increases in metabolic activity in frontal-parietal cortical areas thought to compose the attention network, faster oscillations in the beta range were increased while slower oscillations in the theta range were reduced (Laufs et al., 2006). This pattern, which Laufs et al. call a state of high vigilance, appears to represent an increase in attentional investment at rest such that the cortex is engaged in information processing and various mental activities could be occurring. However, when alpha reduction was seen to correlate with increases in metabolic activity in occipital and parietal areas of the cortex, beta was reduced and theta increased, indicating a state of drowsiness, or low vigilance.

In summary, levels of alpha are related to attentional investment such that when alpha is high, attention is not engaged and the cortex is "idling" (Brunia & van Boxtel, 2001; Pfurtscheller, et al., 1996) or inhibited (Klimesch et al., 2007). The relationship between reduced alpha and attention at rest depends on the cortical areas that are activated during alpha desynchronization, which is indicated by levels of oscillatory activity in frequency bands that neighbor alpha (Laufs et al., 2006). Attentional investment at rest is accompanied by high beta and low alpha, while low alpha and high theta is indicative of drowsiness not attentional investment. Thus, by measuring the relative contribution of EEG oscillations in alpha and beta bands, one can estimate the

degree of attentional investment of an individual at rest. In the current study I aimed to use individual differences in levels of oscillatory activity in the alpha, beta, and theta ranges of EEG frequencies to measure attentional investment at rest for the purpose of predicting individual differences in the AB[15].[1]

Hypotheses

Previously, I discussed evidence from various investigations indicating that increased attentional investment is associated with a larger AB. This might lead one to hypothesize that greater attentional investment at rest, operationalized in this study as more beta, less theta and less alpha power, should correlate positively with AB magnitude. However, there is additional evidence to suggest that the attentional investment at rest is not necessarily positively correlated with attentional investment during a goal-directed task as measured by alpha frequency oscillations. The relationship between attentional investment and the AB, we suggest, depends on the conditions under which attention is invested. That is, when attention is invested during a goal-directed task this would be indicative of an appropriate and controlled application of attention, however, when attention is invested at rest, in the absence of a goal-directed task it is less controlled (i.e. without purpose).

As noted above, within participants, greater alpha ERD during the RSVP foreperiod predicted poor T2 performance at lags during the AB interval but better T2 performance at lags outside of the AB interval as well as better T1 performance

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[15] Other investigators have examined oscillatory EEG in the context of the AB (for a recent review see Hanslmayr, et al., 2011). However, these other investigations have examined oscillatory activity that accompanies the RSVP stream. Except for MacLean & Arnell (2011), this investigation is the first to examine the relevance of oscillatory activity outside of RSVP to the AB, and the only investigation examining the relationship of oscillatory activity to individual differences in AB magnitude.

(MacLean & Arnell, 2011). MacLean and Arnell (2011) also reported a non-significant trend ($r = .27$) such that individuals with greater overall alpha ERD showed greater ABs.

Alpha power has been shown to predict cognitive performance on other tasks in specific ways. Several studies have shown that good performance on memory tasks is related to higher resting alpha power (Klimesch, et al., 1999), and larger alpha ERD is associated with better performance on a task requiring semantic search (Doppelmayr, et al., 2005). For perceptual tasks, good performance is related to low alpha immediately preceding the stimulus (Ergenoglu, et al., 2004; Hanslmayr, et al., 2005) – a pattern that matches the results observed by MacLean and Arnell (2011) such that greater alpha ERD (i.e. lower pre-stimulus alpha) was associated with better T1 performance and better T2 performance at longer lags outside of the AB interval. Klimesch et al. (2007) suggest that this is because performance on perceptual tasks is enhanced if the cortex is already activated. The MacLean and Arnell (2011) findings suggest that the alpha levels that benefit T1 performance and T2 performance at longer lags, which also apparently benefit perceptual performance more generally (Ergenoglu, et al., 2004), are costly for T2 performance at shorter lags during the AB interval.

Furthermore, the effects of induced affective state on perceptual performance were also found to be associated with differences in pre-stimulus alpha, such that negative mood was associated both with better perceptual performance and lower pre-stimulus alpha (Kuhbandner et al., 2009). Note that induced negative affective state has also been shown to influence AB performance, although with mixed results (Jefferies et al., 2008; Olivers & Nieuwenhuis, 2006). Finally, several studies have shown a positive relationship between alpha ERD and resting alpha power (tonic), in that greater

desynchronization is seen in response to greater alpha power (see Doppelmayr, et al., 1998; Klimesch, 1999 for reviews).

Therefore, based on the within participant findings of MacLean and Arnell (2011), that greater alpha ERD is associated with poor T2 performance at short lags but better T2 performance at long lags and better T1 performance, and the positive relationship between resting alpha and alpha ERD (e.g., Klimesch, 1999), I hypothesized that individual differences in resting alpha power would predict AB magnitude in that lower resting alpha would be associated with better T2 accuracy at short lags, and therefore smaller AB magnitudes. Low resting alpha power would be expected to be accompanied by either high resting beta or high resting theta. I hypothesized that high resting beta, if accompanied by low resting alpha (indicating a state of attentional investment), would predict improved AB performance. However, high resting theta, if accompanied by low resting alpha (reflecting greater drowsiness), should not correlate with AB performance (Laufs et al., 2006). I examined these hypotheses in two independent investigations.

Methods: Experiment 1

Participants

The participants were 30 Brock University undergraduate students, recruited through the Brock Psychology Department's online system for participant recruitment. The data from two participants were excluded due to close to chance performance on the RSVP task (T1 accuracy was 57%) in one case and in the other case outlying T2 accuracy at lag 8 (more than three SD below the mean). The data from another participant were excluded due to an error in the EEG recording.

AB Task

The AB task consisted of five blocks of 140 RSVP trials. Of the 700 total trials, 100 were no-target trials, and 600 were dual target (T1 and T2) trials. On half of the dual-target trials, T2 was presented 3 items, or 351 ms after T1 (lag 3), and on the other half, T2 was presented 8 items, or 936 ms after T1 (lag 8). T1 was always presented in white font as the 6th item in the stream. On 80% of trials at each lag, T1 was a string of five repeated uppercase letters (e.g., BBBBB) chosen randomly from the letter set B, C, D, E, F, N, P, S, U, X, or Z. On the remaining 20% of trials, T1 was a string of five repeated lowercase letters (e.g., bbbbb) chosen randomly from the same letter set. T2 was one of 10 different color words (e.g., "GREEN"), and appeared in black uppercase letters. The distracter items consisted of affectively neutral words that were unrelated to colors. Distractors were also presented in black uppercase letters. All trial types were presented randomly within each block. The T1 probability manipulation and the distracter-only trials were included for the purposes of a separate study.

Each trial began with a fixation cross (500 ms), followed by a foreperiod of 2 seconds before the onset of the RSVP stream. The RSVP stream consisted of 18 alphanumeric stimuli with an SOA of 117 ms per item presented on a gray background. At the end of each stream, participants indicated whether the white letter string was in upper- or lower-case letters, and then reported which color word was presented as T2. Participants were told that some of the trials would contain no targets, and on these trials they should simply press the spacebar to initiate the next trial. Participants made their T1 and T2 responses sequentially in an unspeeded manner using specified keys on the

keyboard. Stimulus presentation and participant responses were controlled using E-Prime software (Schneider et al., 2002).

Resting EEG Procedure

Five blocks of resting EEG were recorded, one prior to each of the five blocks of the AB task. Each block of recording consisted of three 30 second segments of eyes closed alternating with three 30 second segments of eyes open, with 1 second between each segment. Instructions would appear before each segment to indicate whether the participant should keep their eyes open or closed and a tone indicated the beginning and end of each segment. Participants were instructed to sit quietly without moving. Participants were monitored during recording to ensure they did not fall asleep. Only resting EEG from the eyes closed segments were further analyzed.

Resting EEG Acquisition

EEG was recorded continuously using tin electrodes embedded in an Electro-Cap© (Electro-cap International Inc., Eaton, Ohio) from 60 scalp sites distributed according to the 10–20 system, with an electrode placed anterior to Fz as ground. EEG was recorded using linked left and right earlobes as reference and was re-referenced to a common average of the EEG. EEG data were acquired with Neuroscan acquisition software (Compumedics USA, Charlotte, North Carolina), and using two 32-channel NeuroScan SynAmps. Data were sampled at a rate of 500 Hz. Electro-oculogram (EOG) recorded horizontal eye movements using electrodes placed on the outer canthus of each eye, and vertical eye movement and blinks using electrodes placed on the infra- and supra- orbital regions of each eye. Impedance for both the EEG and EOG was maintained

below 10 kΩ. EEG was band-pass filtered offline with a high pass of .01 Hz, a low pass of 57 Hz and a roll-off of 48 dB/oct.

Resting EEG Analysis

Each 30-second section of eyes closed resting EEG was visually inspected for artifact. EEG containing artifact was isolated and excluded from further analysis. Each section was then further segmented into 2-second epochs with 75% overlap. Epochs were spline fit to 512 points. These epochs were then submitted to a fast Fourier transform (FFT) using a Hanning window yielding absolute power values within pre-defined bands. Alpha was defined as the average power in the 8-12 Hz range, beta was defined at the average power in the 15-35 Hz range and theta was defined as the average power in the 4-8 Hz range. FFT power values were log transformed prior to statistical analysis.

Four topographic regions of interest (ROIs) were created by averaging power values across several electrode sites: frontal (F3, F4, Fz), central (C3, C4, Cz), parietal (P3, P4, Pz) and occipital (O1, O2, Oz). While the correlations Laufs et al. (2003) reported were based on power measured at occipital electrode sites (O1 & O2), they did note that these correlations were observed at the majority of electrode sites. I did not hypothesize that ROI would interact with the hypothesized correlations.

Results: Experiment 1

AB Task Performance

Mean T1 accuracy was 90.52% (SD = 7.84, range 70% to 98%). T2 accuracy was conditionalized on T1 performance. Mean T2 accuracy at lag 3 was 66.89% (SD = 16.65, range 22% to 95%). Mean T2 accuracy at lag 8 was 89.96% (SD = 5.45, range 78% to 97%). A paired-samples t-test indicated significantly lower T2 accuracy at lag 3 than lag

8 indicating the presence of an AB (t (26) = 8.26, $p < .001$). Individual AB magnitude was calculated as the difference measure (T2 performance on lag 3 trials was subtracted from T2 performance on lag 8 trials). Mean magnitude (difference) was 23.07% (SD = 14.52, range 1% to 67%).[16]

Spectral EEG

A 3 X 4 repeated measures ANOVA performed on power values, with frequency range (theta, alpha, and beta) and ROI (frontal, central, parietal, and occipital) as factors, indicated a significant main effect of frequency band (F (2, 50) = 230.96, $p < .001$) such that power was greatest in alpha band, and least in the beta frequency band, as well as a significant main effect of ROI (F (3, 75) = 37.99, $p < .001$) and a significant interaction of frequency band and ROI (F (6, 150) = 45.87, $p < .001$) such that that power decreased from occipital to frontal ROIs in the alpha and beta band but was similar across ROI for the theta band.

Absolute power in the different frequency bands for each ROI (e.g. the correlation between alpha at the frontal ROI with beta at the frontal ROI, etc.) was significantly positively correlated (r's ranged from .47 to .73), indicating that some individuals had higher power values than others across all frequency bands. In order to account for this inter-individual variability in overall absolute power, which may obscure individual differences in the unique and relative contributions of theta, alpha, and beta necessary to address my hypotheses, I regressed power in each frequency band on power in the other two frequency bands for each ROI using three simultaneous multiple regressions. I then saved each of the residuals. This yielded a residualized measure of power for each

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[16] The results of Experiment 1 are identical when the residual difference measure used in Study 1 was correlated with resting EEG (see Appendix B.5).

frequency band (i.e. the unique variability in absolute theta, alpha, and beta power for each individual independent of overall absolute power level in the entire band from 4-35 Hz). This procedure allows removal of the inter-individual variability due to overall absolute power shared between the three different frequency bands (i.e. that some individuals have higher overall absolute power values than others), leaving the unique variability in absolute power in each of the frequency bands[17].

Residual power values in alpha and beta within the same ROI were significantly ($p < .05$) negatively correlated (r's ranged from -.42 to -.63), as were residual power values in beta and theta (r's ranged from -.43 to -.49), residual power values in alpha and theta were negatively correlated although not significantly (r's ranged from -.12 to -.35). This pattern fits the findings of Laufs et al. (2006) such that low alpha is accompanied by either high beta or high theta, but that high beta is accompanied by low theta and vice versa. In the present data it appears that low alpha at rest was consistently accompanied by high beta, but not high theta. This indicates that this sample predominantly displayed the high vigilance mode characterized by low alpha and high beta found when alpha correlated negatively with metabolic activity in the fronto-parietal attention network (Laufs et al., 2006).

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[17] Note that the variability referred to in the description of my residualized measure is inter-individual variability in absolute power. This is in contrast to relative power measures, which would concern inter-frequency variability within individuals. Thus my residualized power measures are not equivalent to relative power measures. The issue of inter-individual variability in overall absolute power is common to all individual differences approaches to spectral EEG, as inter-individual variability in overall absolute power can obscure individual differences in power for a given frequency band, e.g. alpha. Klimesch et al. (1999) accounted for this issue using a different method. They created what they termed "normalized percentage power" by calculating the absolute power in each frequency band as the percentage of absolute power in all frequency bands.

Correlations between Resting EEG and AB Performance

Tables 4-1, 4-2, and 4-3 contain the correlations between residual power in the theta, alpha and beta frequency bands at each ROI (frontal, central, parietal, and occipital) and overall across ROIs with various measures of performance on the AB task. Theta power at rest was not correlated significantly with any of the AB task performance measures including AB magnitude (see Table 4-1 and *Figure 4-1a*).

Alpha power at rest was negatively correlated with T2 accuracy, and these relationships were significant for lag 3. Alpha power at rest was also positively correlated with AB magnitude at all ROIs and overall across ROIs (see Table 4-2 and *Figure 4-1b*). This pattern of correlations indicates that greater alpha power at rest is related to greater AB magnitudes, and that this relationship is due to a relationship between alpha power at rest and short lag, not long lag, T2 performance.

Beta power at rest was positively correlated with T2 accuracy, and these relationships were significant for lag 3. Beta power at rest was also negatively correlated with AB magnitude at all ROIs and overall across ROIs (with the exception that the correlation between beta power and AB magnitude at the central ROI that was only marginally significant, $p = .056$). This pattern of correlations indicates that greater beta power at rest is related to smaller AB magnitudes (see Table 4-3 and *Figure 4-1c*), and that this relationship is due to a relationship between beta power at rest and short lag, not long lag, T2 performance as was also the case with resting alpha power.

Table 4-1

Experiment 1 correlations between theta at each ROI, and overall theta, with AB performance measures

	T1 Accuracy	Lag 3 T2 Accuracy	Lag 8 T2 Accuracy	AB Magnitude
Theta (Frontal)	.19	.20	.12	-.19
Theta (Central)	.03	.16	.03	-.18
Theta (Parietal)	-.06	-.07	.07	.12
Theta (Occipital)	-.10	-.09	.14	.19
Theta Overall	.03	.07	.11	-.03

Note: * $p < .05$

Table 4-2

Experiment 1 correlations between alpha at each ROI, and overall alpha, with AB performance measures

	T1 Accuracy	Lag 3 T2 Accuracy	Lag 8 T2 Accuracy	AB Magnitude
Alpha (Frontal)	-.16	-.68***	-.21	.72***
Alpha (Central)	-.12	-.66***	-.24	.69***
Alpha (Parietal)	-.19	-.64***	-.36	.61**
Alpha (Occipital)	-.06	-.55**	-.25	.55**
Alpha Overall	-.14	-.68***	-.28	.69***

Note: * $p < .05$; ** $p < .01$; *** p $< .001$

Table 4-3

Experiment 1 correlations between beta at each ROI, and overall beta, with AB performance measures

	T1 Accuracy	Lag 3 T2 Accuracy	Lag 8 T2 Accuracy	AB Magnitude
Beta (Frontal)	.06	.39*	.19	-.39*
Beta (Central)	.21	.43*	.30	-.37
Beta (Parietal)	.31	.57**	.31	-.55**
Beta (Occipital)	.19	.44*	.11	-.48*
Beta Overall	.21	.50**	.25	-.49*

Note: * $p < .05$; ** $p < .01$

a)

b)

c)
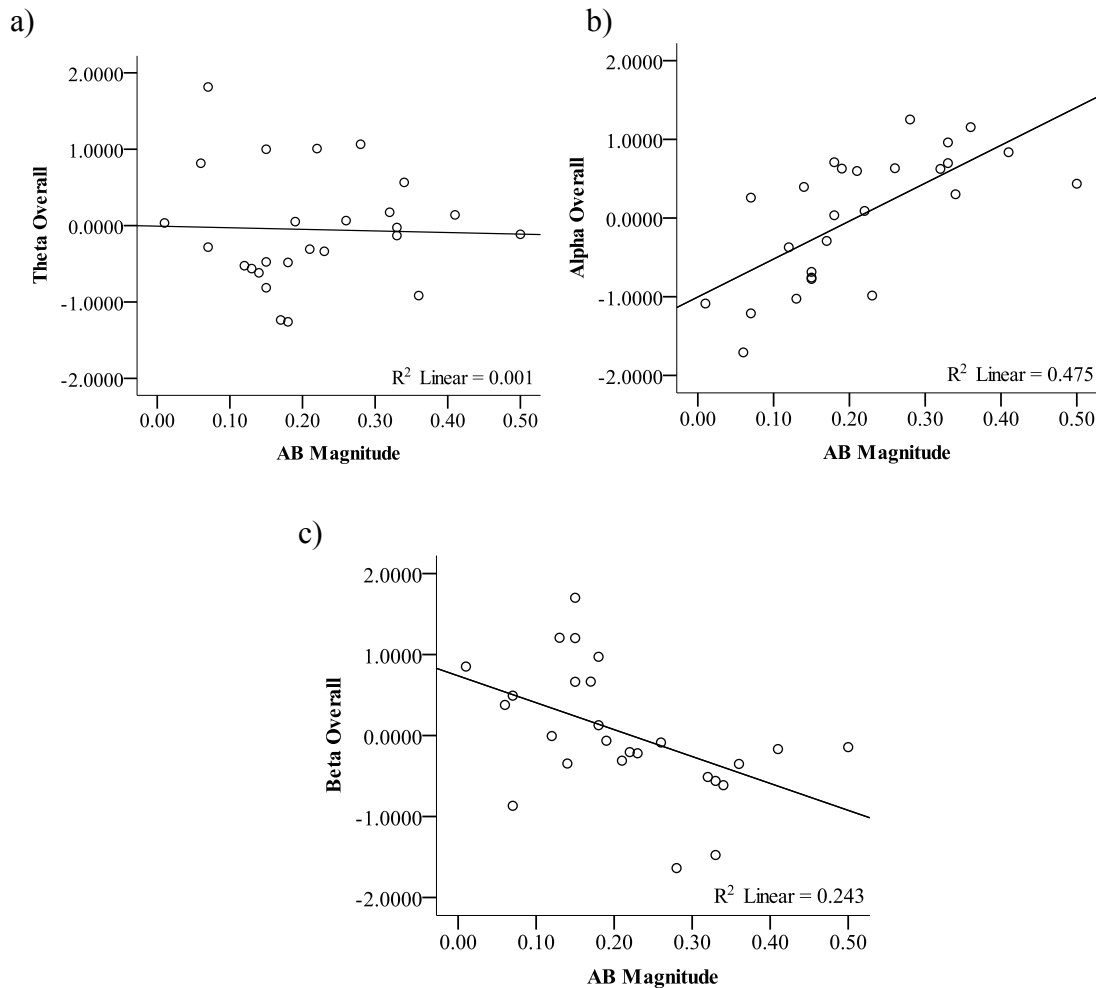
Figure 4-1 a-c. Scatterplots depicting correlation between levels of resting theta (a), alpha (b), and beta (c) with AB magnitude from Experiment 1. The solid line depicts the linear function of the zero-order correlation, and the squared zero-order correlation is indicated in the bottom right corner of the scatterplot.

Since alpha and beta power at rest were significantly negatively correlated with each other and both correlated significantly with AB magnitude I further investigated whether the trade-off between alpha and beta power at rest predicted performance on the AB task (see Table 4-4). The difference score of alpha minus beta power was negatively correlated with T2 accuracy at lag 3, and positively correlated with AB magnitude at all ROIs and overall across ROI. This pattern of correlations indicates that the greater the

preponderance of resting alpha power relative to resting beta power, the larger the AB

magnitude.

Table 4-4
Experiment 1 correlations between alpha-beta at each ROI, and overall alpha-beta with
AB performance measures

	T1 Accuracy	Lag 3 T2 Accuracy	Lag 8 T2 Accuracy	AB Magnitude
Alpha-Beta (Frontal)	-.13	-.63**	-.23	.65***
Alpha-Beta (Central)	-.19	-.64***	-.32	.63**
Alpha-Beta (Parietal)	-.28	-.69***	-.38	.65***
Alpha-Beta (Occipital)	-.14	-.55**	-.20	.57**
Alpha-Beta Overall	-.19	-.67***	-.30	.67***

Note: * $p < .05$; ** $p < .01$; *** $p < .001$

Fourteen participants had positively signed difference scores averaged across

ROIs (+Diff group) indicating more resting alpha than beta power, while twelve

participants had negatively signed difference scores averaged across ROIs (-Diff group)

indicating more resting beta then alpha power[18]. A mixed-model ANOVA of T2 accuracy

with T1-T2 lag (3 or 8) as a within-subject factor and group (+Diff or –Diff) as a between

subjects factor was performed to determine whether the nature of the absolute difference

between resting alpha and beta power interacted with the AB (see *Figure 4-2*). There was

a significant interaction between lag and group ($F (1, 24) = 25.55$, $p < .001$) such that the

effect of lag on T2 accuracy was larger in the +Diff group (*Mdiff* = 29.07; $t (13) = 11.01$,

$p < .001$) than in the –Diff group (*Mdiff* = 12.42; $t (11) = 7.00$, $p < .001$). An additional

post-hoc analysis confirmed that the –Diff group had significantly higher T2 accuracy at

lag 3 than the +Diff group ($t (24) = 4.61$, $p < .001$), but that the two groups did not differ

in T2 accuracy at lag 8 ($t (24) = 1.22$, $p = .236$). This indicates that the AB was greater in

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[18] My independent-groups analysis was intended to function as a median-split approach to a continuous
variable (amount of alpha and beta within an individual). I do not suggest that there is something
qualitatively different between the two groups, as defined by the zero point threshold used here.

the +Diff group than the –Diff group because of differences in short lag performance,

within the AB period, but not because of differences in long lag performance.
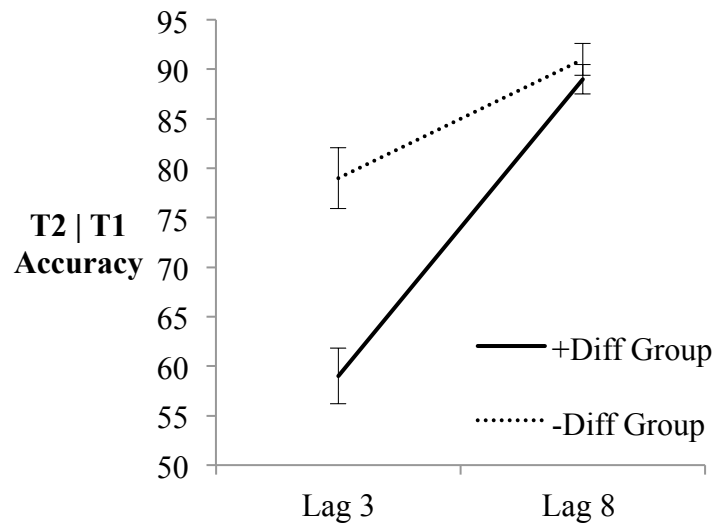
Figure 4-2. Experiment 1 T2 accuracy at each lag (3 & 8) for the +Diff (alpha-beta) and –Diff (alpha-beta) groups, depicting the interaction of alpha-beta group with lag. Error bars denote standard error.

Introduction: Experiment 2

Experiment 1 found that resting alpha and resting beta predicted AB magnitude in opposite directions. Greater resting alpha was correlated with larger AB magnitudes while greater resting beta was correlated with smaller AB magnitudes. Furthermore, a greater preponderance of resting alpha power over resting beta power was related to larger AB magnitudes, and individuals who had greater resting alpha than resting beta power had larger ABs than individuals with greater resting beta than resting alpha power. The purpose of Experiment 2 was to replicate the findings of Experiment 1. The same pattern of results was expected in this independent sample of participants using a different AB task and EEG recording system from a different lab. In addition,

Experiment 2 also had a greater time interval between the AB task and the resting EEG

recordings than Experiment 1.

Methods: Experiment 2

Participants

The participants were 38 individuals recruited from the Brock University

population and surrounding community. The data from six participants were excluded

due to their inability to perform the T2 task at greater than chance levels. The data from

another three participants were excluded due to poor quality EEG data (artifacts or noise).

AB Task

The AB task was modeled on the original AB task of Raymond et al. (1992). The

present task included five blocks of 32 RSVP trials. T1 was present on every trial. Of the

160 total trials, 80 were T2 absent trials (only T1 presented), and 80 were T2 present (T1

and T2) trials. Among the 80 T2 present trials T2 was presented equally often at one of

eight lag positions (1st-8th item, or 100-800 ms following T1 onset). T1 was presented as

either the 7th or 10th RSVP item. T1 was a randomly selected upper-case letter (excluding

X) presented in white font. T2 was the upper-case letter X presented in black font. The

distracter items consisted of black upper-case letters (excluding X). All trial type

combinations were presented randomly and equally within each block.

Each trial began with a fixation cross (1,000 ms), followed by a blank foreperiod

of 1,000 ms before the onset of the RSVP stream. The RSVP stream consisted of 19

alphanumeric stimuli with an SOA of 100 ms per item presented on a gray background.

At the end of each RSVP stream, participants identified the lone white letter, and then

indicated whether an X was present or not. Participants made their T1 and T2 responses

sequentially in an unspeeded manner using specified keys on the keyboard. Each RSVP trial was separated by a 1,000 ms blank ITI. Stimulus presentation and participant responses were controlled using E-Prime software (Schneider et al., 2002).

Resting EEG Procedure

Two blocks of resting EEG were recorded; one block was recorded 2 hours prior to the AB task (at 09:00) and the other 2 hours following the AB task (at 13:00). Each block of recording was performed as in Experiment 1 and resting EEG data was averaged across the blocks.

Resting EEG Acquisition

EEG was recorded continuously using gold-plated electrodes from 12 scalp sites applied according to the 10–20 system, with an electrode placed anterior to Fz as ground. EEG was recorded using Fpz as reference and was re-referenced offline to the average of left and right mastoid sites. EEG data were acquired and analyzed with SPYDER (EBNeuro, Inc., Florence, Italy) software running on a desktop PC, and using Mizar digital amplifiers (Tyco Inc., Ottawa, Canada). Data were sampled at a rate of 128 Hz with 0.0099 to 57.6 Hz hardware filters. Electro-oculogram (EOG) recorded horizontal eye movements using electrodes placed on the outer canthus of each eye, and vertical eye movement and blinks using electrodes placed on the infra- and supra- orbital regions of each eye. Impedance for the EEG was maintained below 5 kΩ and for the EOG was maintained below 10 kΩ.

Resting EEG Analysis

Analysis of resting EEG was identical to that performed in Experiment 1. ROIs were also identical to Experiment 1.

Results: Experiment 2

AB Task Performance

Mean T1 accuracy was 93.83% (SD = 4.18, range 82% to 100%). T2 sensitivity at

each lag was calculated as d′ (Z[hits] – Z[false alarms]). T2 sensitivity was

conditionalized on correct T1 performance. A repeated-measures ANOVA of T2

sensitivity with lag as the factor yielded a significant effect of lag (F (7, 196) = 33.04, $p <$

.001). T2 sensitivity increased from lag 2 to lag 8 and showed lag 1 sparing, indicating

the presence of an AB. Individual AB magnitude was represented by subtracting each

participant's T2 sensitivity at lag 2, the lag where T2 sensitivity was lowest in the AB,

from their highest long lag (6-8) sensitivity (baseline). Mean T2 sensitivity at lag 2 was

.89 (SD = 1.40, range -2.33 to 2.63). Mean T2 sensitivity at the baseline long lag was

2.32 (SD = 1.15, range -.44 to 4.08). Mean AB magnitude was 2.24 (SD = .88, range .19

to 3.61)[19].

Spectral EEG

A repeated measures ANOVA was performed on the resting EEG power values

with frequency range and ROI as factors. A significant main effect of frequency band

was observed (F (2, 56) = 21.31, $p <$ .001), such that power was greatest in alpha band,

and least in the beta frequency band. There was also a significant main effect of ROI (F

(3, 84) = 349.68, $p <$ .001) and a significant interaction of frequency band and ROI (F (6,

168) = 184.24, $p <$ .001). Power decreased from occipital to frontal ROIs but this

decrease was mostly seen in the alpha frequency band and less so in the beta and theta

frequency bands.

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[19] The results of Experiment 2 are identical when the residual difference measure used in Study 1 was
correlated with resting EEG (see Appendix B.6).

As in Experiment 1, power in the different frequency bands for each ROI was significantly positively correlated (r's ranged from .46 to .82), indicating that individuals differed in magnitude of log-transformed power values overall regardless of frequency band. Thus, the same residualized power values for each frequency band were created as in Experiment 1.

Residual power values in alpha and beta within the same ROI were negatively correlated (r's ranged from -.31 to -.43) although only significantly at the parietal ROI. The residual power values in beta and theta also correlated negatively although not significantly (r's ranged from -.13 to -.37), and residual power values in alpha and theta were significantly negatively correlated at all ROIs (r's ranged from -.48 to -.69). This pattern fits the findings of Laufs et al. (2006) such that low alpha is accompanied by either high beta or high theta, but that high beta is accompanied by low theta and vice versa. Contrary to what was observed in Experiment 1, however, in the Experiment 2 it appears that low levels of alpha at rest were accompanied, although not exclusively, by high beta but also high theta. This indicates that some participants displayed the low vigilance mode characterized by low alpha and high theta found when alpha correlated negatively with metabolic activity in parieto-occipital regions while others displayed the high vigilance mode (Laufs et al., 2006).

Correlations between Resting EEG and AB Performance

Tables 4-5, 4-6, and 4-7 contain the correlations between residual power in the theta, alpha and beta frequency bands at each ROI (frontal, central, parietal, and occipital) and overall across ROIs with various measures of performance on the AB task.

Theta power at rest was not correlated significantly with any of the AB task performance measures including AB magnitude (see Table 4-5 and *Figure 4-3a*).

Alpha power at rest was negatively correlated with T2 sensitivity at lag 2 at the frontal ROI, and positively correlated with AB magnitude at each of the ROIs and overall across ROIs (see Table 4-6 and *Figure 4-3b*). This pattern indicates that greater alpha power at rest is related to greater AB magnitudes, and that this relationship is due to a relationship between alpha power at rest and short lag, not long lag, T2 performance.

Beta power at rest was correlated positively with T2 sensitivity at lag 2 at the frontal ROI and negatively with AB magnitude at the frontal ROI. This pattern of correlations indicates that greater frontal beta power at rest is related to smaller AB magnitudes (see Table 4-7 and *Figure 4-3c*), and that this relationship is due to a relationship between beta power at rest and short lag, not long lag, T2 performance.

Table 4-5
Experiment 2 correlations between theta at each ROI, and overall theta with AB
performance measures

	T1 Accuracy	Lag 2 T2 Sensitivity	Baseline Long Lag	AB Magnitude
Theta (Frontal)	-.08	-.04	-.14	-.12
Theta (Central)	-.04	-.09	-.27	-.20
Theta (Parietal)	.02	-.02	-.26	-.32
Theta (Occipital)	-.02	-.11	-.29	-.20
Theta Overall	-.03	-.07	-.27	-.24

Note: * $p < .05$; ** $p < .01$; *** $p < .001$

Table 4-6
Experiment 2 correlations between alpha at each ROI, and overall alpha with AB
performance measures

	T1 Accuracy	Lag 2 T2 Sensitivity	Baseline Long Lag	AB Magnitude
Alpha (Frontal)	.06	-.37*	-.17	.38*
Alpha (Central)	.02	-.32	-.01	.50**
Alpha (Parietal)	-.01	-.28	.04	.49**
Alpha (Occipital)	-.09	-.16	.11	.39*
Alpha Overall	-.01	-.31	-.01	.49**

Note: * $p < .05$; ** $p < .01$; *** $p < .001$

Table 4-7
Experiment 2 correlations between beta at each ROI, and overall beta with AB
performance measures

	T1 Accuracy	Lag 2 T2 Sensitivity	Baseline Long Lag	AB Magnitude
Beta (Frontal)	-.13	.42*	.21	-.39*
Beta (Central)	-.12	.18	.06	-.21
Beta (Parietal)	-.18	-.07	-.12	-.05
Beta (Occipital)	-.09	-.23	-.22	.07
Beta Overall	-.15	.08	-.02	-.16

Note: * $p < .05$; ** $p < .01$; *** $p < .001$
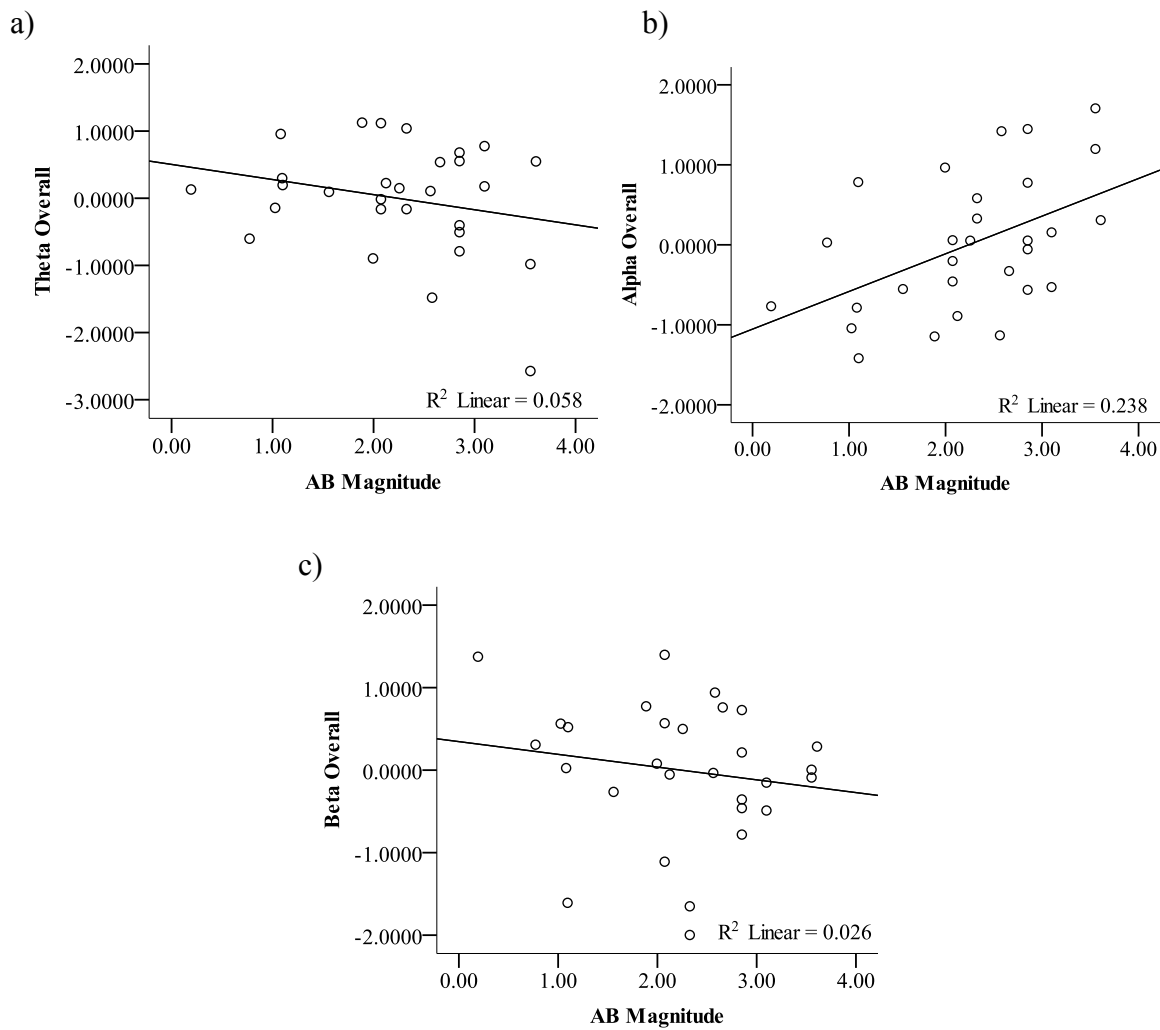
Figure 4-3 a-c. Scatterplots depicting correlation between levels of resting theta (a), alpha (b), and beta (c) with AB magnitude from Experiment 2. The solid line depicts the linear function of the zero-order correlation, and the squared zero-order correlation is indicated in the bottom right corner of the scatterplot.

The difference score of alpha minus beta power was negatively correlated with T2 sensitivity at lag 2 at the frontal ROI and positively correlated with AB magnitude across all ROIs and individually at frontal and central ROIs (see Table 4-8). This pattern of

correlations indicates that the greater the preponderance of resting alpha power relative to

resting beta power, the larger the AB magnitude[20].

Table 4-8
Experiment 2 correlations between alpha-beta at each ROI, and overall alpha-beta with
AB performance measures

	T1 Accuracy	Lag 2 T2 Sensitivity	Baseline Long Lag	AB Magnitude
Alpha-Beta (Frontal)	.12	-.49**	-.23	.48**
Alpha-Beta (Central)	.09	-.31	-.04	.44*
Alpha-Beta (Parietal)	.09	-.13	.09	.32
Alpha-Beta (Occipital)	.00	.04	.20	.20
Alpha-Beta Overall	.08	-.24	.01	.39*

Note: * $p < .05$; ** $p < .01$; *** $p < .001$

Thirteen participants had positively signed difference scores averaged across

ROIs for the alpha-beta difference score (+Diff group) indicating more resting alpha than,

while sixteen participants had negatively signed difference scores averaged across ROIs

for the alpha- difference scores (-Diff group) indicating more resting beta than alpha

power. A mixed-model ANOVA of T2 sensitivity with T1-T2 lag (2 or long lag baseline)

as a within-subject factor and alpha-beta group (+Diff or–Diff) as a between subjects

factor was performed to determine whether the nature of the absolute difference between

resting alpha and beta power interacted with the AB (see *Figure 4-4*). There was a

significant interaction between lag and group (F (1, 27) = 7.03, p = .013) such that the

effect of lag on T2 sensitivity was larger in the +Diff group (*Mdiff* = 2.67; t (12) = 13.35,

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[20] Note that another way to measure the combination of alpha and beta resting power is to investigate the relationship between their interactions and AB magnitude. In order to investigate this possibility a hierarchical multiple regression was performed predicting AB magnitude with resting alpha, beta and theta power on the first step and their interactions (alpha X beta, beta X theta, alpha X theta) in the second step for Experiment 1 and 2 separately. In the both the first and second step alpha is the only significant unique predictor of AB magnitude. Beta was not a significant unique predictor over and above alpha and theta in either Experiment 1 (see Appendix C.3) or Experiment 2 (see Appendix C.4). This suggests that the relationship between resting alpha and beta power accounts for the relationship between resting beta power and AB magnitude. Further, of the resting EEG measures, only resting alpha is a unique predictor of AB magnitude.

p < .001) than in the –Diff group (*Mdiff* = 1.89; *t* (15) = 8.86, *p* < .001). An additional

post-hoc analysis confirmed that the –Diff group had significantly higher T2 accuracy at

lag 3 than the +Diff group (*t* (27) = 2.38, *p* = .025), but that the two groups did not differ

in T2 accuracy at lag 8 (*t* (27) = .85, *p* = .405). This indicates that the AB was greater in

the +Diff group than the –Diff group because of differences in short lag performance,

within the AB period, but not because of differences in long lag performance.
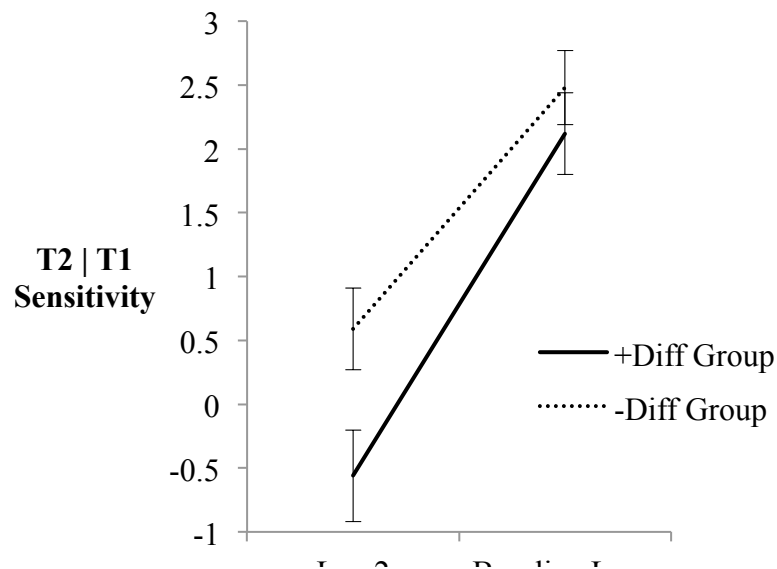
 Figure 4-4. Experiment 2 T2 accuracy at each lag (2 & baseline long lag) for the +Diff
(alpha-beta) and –Diff (alpha-beta) groups, depicting the interaction of alpha-beta group
with lag. Error bars denote standard error.

Except that the correlations between beta and AB task performance were

primarily at the frontal ROI, the correlations of resting theta, alpha, and beta with AB

task performance in Experiment 2 replicate the results of Experiment 1.

General Discussion

In the current study I investigated whether tonic EEG power in alpha (8-12 Hz)

and beta (15-35 Hz) frequency oscillation ranges (i.e., power values when the participant

was at rest and not engaged in a goal-directed task), could predict individual differences

in AB magnitude. I operationalized greater attentional investment at rest as less alpha and greater beta power. I hypothesized that attentional investment at rest, as measured by alpha and beta power, would correlate negatively with AB magnitude (i.e., that larger ABs would be associated with higher alpha and lower beta power). This hypothesis followed from evidence that greater alpha at rest (tonic alpha) was associated with a greater decrease in alpha (alpha ERD) during a task (Doppelmayr, et al., 1998; Klimesch, 1999) and that good performance on difficult perception tasks has been shown to be related to low alpha power (Ergenoglu, et al., 2004; Hanslmayr, et al., 2005).

The results of both Experiment 1 and Experiment 2 supported my hypothesis. Resting alpha power correlated positively with AB magnitude, while resting beta power correlated negatively with AB magnitude. These correlations suggested that greater attentional investment at rest (less alpha and more beta power) was associated with smaller AB magnitudes. In support of this interpretation, it was observed that the relative trade-off between resting alpha and beta power (which were negatively correlated with each other) also predicted the AB and that individuals with greater resting alpha than beta power had larger ABs than individuals with greater resting beta then alpha power. The size of these relationships was surprisingly large in that individual differences in relative alpha-beta power across all ROIs predicted 45% of the variability in AB magnitude in Experiment 1, and 16% in Experiment 2.

It is important to note that the correlations between resting EEG measures and short lag T2 accuracy were considerably stronger than those with long lag T2 accuracy, indicating that the relationships with AB magnitude (a difference measure) are primarily due to variance in short lag T2 accuracy (i.e. variability during the AB interval).

However, it is worth noting that, although they were not significant at the traditional level, the correlations of resting alpha, beta, and alpha-beta with long lag T2 accuracy in Experiment 1 were still of a considerable size (r's ranging from .25 to .30). These correlations were all in the same direction as the significant correlations of alpha and beta with short lag T2 accuracy. This was also the case at the frontal site for Experiment 2. Thus, in addition to predicting AB magnitude, the present results provide evidence that resting alpha is associated with lower T2 accuracy overall, and resting beta with higher T2 accuracy overall.

While ROI did not appear to play a role in the relationships between resting state EEG and AB performance in Experiment 1, in Experiment 2 the relationship involving beta and AB performance was limited to frontal electrode sites. That ROI was relevant to the relationship between resting beta power and AB performance in Experiment 2 but not Experiment 1 could be attributed to different kinds of resting states being present in participants from the two different studies (i.e. participants feeling and/or thinking differently while at rest). The different spectral power results also support this hypothesis. Although speculative, this could result from the different experimental protocols used in the two studies given that Experiment 1 was part of a three-hour test session, whereas Study 2 was part of a full-day in-lab testing session.

We also hypothesized that correlations between power at rest and the AB would be confined to the alpha and beta ranges and would not include the theta range, given that theta power while at rest is indicative of drowsiness not attentional investment. My results also support this hypothesis given that there were no associations between theta power and any AB variables. As noted by Laufs et al. (2006), low relative alpha power

may be accompanied by high relative theta power (indicative of low vigilance at rest) or accompanied by high relative beta power (indicative of high vigilance at rest). The present results suggest that it is not merely that lower alpha is associated with smaller AB magnitudes; but that the combination of higher beta and lower alpha indicative of high investment is associated with smaller AB magnitudes (see Laufs et al. 2006).

Our findings replicate the negative correlations between alpha and beta/theta as well as the negative correlation between beta and theta observed by Laufs et al. (2006), and support the conception of levels of alpha at rest in combination with levels of beta and theta as indicative of different attentional states as suggested by Laufs et al. (2003b, 2006). Specifically, the combination of low levels of alpha and high levels of beta at rest predict the AB, as a state of vigilance or attentional engagement was expected to, while the combination of low levels alpha and high levels of theta did not predict the AB, as was expected from a state of drowsiness.

The Relevance of Attention at Rest to the AB

Our results are in line with previous evidence that the attentional approach adopted by the individual can predict the magnitude of their AB (Arnell & Stubitz, 2010; Dale & Arnell, 2010; Dux & Marois, 2008; MacLean & Arnell, 2010; MacLean, et al., 2010; Martens & Valchev, 2009). It is unclear from my evidence whether the attentional approach represented by attentional investment at rest is indicative of an enduring trait or a labile state. However, in Experiment 2, resting EEG recordings were obtained in two sessions, one two hours before and the other two hours after the AB task was performed. That measures derived from resting EEG at such a distance in time from the AB task can predict performance on that task, suggests that levels of theta, alpha, and beta at rest

could represent a more stable, trait-like measure (for cross-session and inter-session reliabilities of resting alpha, beta, and theta power see Appendices B.5, 6, & 9). Further investigation would be required to determine whether attentional investment at rest, measured by relative levels of theta, alpha and beta remains consistent within the individual at longer intervals. Certainly it is possible that attentional investment at rest may predict AB magnitude in a trait-like fashion, as personality variables have also shown such an association with the AB (MacLean & Arnell, 2010).

Our hypotheses and results are consistent with the resting alpha and alpha ERD literature (see Klimesch et al., 2007 for a review). It has been suggested that levels of alpha are indicative of different orientations of attention (Cooper et al., 2003; Hanslmayr et al., 2011). Specifically, high levels of alpha are thought to indicate a state of internally oriented attention, such that attention is directed toward internally-generated information, for example, memories. In opposition to that, low levels of alpha are thought to indicate a state of externally oriented attention, such that attention is directed toward incoming, externally-generated information, for example, an RSVP. In this case when alpha is high and attention is directed toward internally-generated information perception of externally-generated information would be impaired. In the case of my results this would suggest that the larger AB magnitudes observed in individuals with greater levels of alpha at rest could be the consequence of displaying an internally oriented state of attention while at rest. For example, one possibility is that the state of internally oriented attention is persistent in individuals who display greater levels of alpha at rest. In this case, if the internally oriented state of attention persisted during the AB task, it is possible that impaired perception of externally-generated information accounts for the relationship

between greater alpha at rest and larger AB magnitudes. In other words, the internally oriented state of attention results in reduced perceptual identification which is exacerbated when identification is especially difficult at short lags within the time window of the AB, leading to larger AB magnitudes and poorer performance.

The present results appear to be at odds with findings that suggest that diffused, or decreased attentional investment is associated with smaller AB magnitudes (e.g., Dale & Arnell, 2010; MacLean & Arnell, 2010; MacLean et al., 2010, Olivers & Nieuwenhuis, 2005, 2006) and that greater alpha ERD (a measure of anticipatory attention) in the foreperiod of the RSVP stream is associated with lower T2 accuracy during the AB period (MacLean & Arnell, 2011). Furthermore, the overinvestment hypothesis of Olivers and Nieuwenhuis (2005, 2006) suggests that the AB is caused by too much attentional investment. However, it is important to remember that the overinvestment hypothesis, and those results that support it, are concerned with attentional investment, or attentional approach, relative to a goal-directed task. In the present studies I examined attentional investment specifically in the absence of a goal-directed task, and there is evidence to suggest that indices of attention at rest and relative to a task are not necessarily positively correlated (Doppelmayr, et al., 1998; Klimesch, 1999).

There is an alternative hypothesis for how a state of internally oriented attention at rest could account for the larger AB magnitudes of individual who display greater alpha at rest. One way to characterize the possible relationship between attentional investment at rest and during a task is to conceptualize greater alpha (synchrony) as a state of readiness (e.g., Klimesch et al., 2007). Higher alpha power at rest has also been proposed to reflect an "idling", or alternatively, an actively inhibited cortex (Klimesch, et

al., 2006; Klimesch, et al. 2007; Pfurtscheller, et al., 1996). Regardless of whether one

conceptualizes alpha as idling or inhibited, either way the cortex is relatively unoccupied.

When accompanied by lower levels of beta power, this is indicative of a disengaged

attentional network (Laufs et al., 2006), in a state of readiness (Klimesch et al., 2007).

Specifically, the cortex is unoccupied and the attentional network is disengaged such that

the relevant resources remain available for the processing of incoming, externally-

generated information. Individuals in a state of readiness at rest may then approach a task

with a high degree of readiness, and this may account for the positive correlation

observed between resting alpha and alpha ERD (Doppelmayr et al., 1998). Thus, an

individual in this increased state of readiness may be able to deploy more resources to a

subsequent stimulus. In contrast, individuals who are preoccupied (occupied cortex),

rather than idling at rest may also be preoccupied during the AB task and therefore

deploy fewer resources to the AB task. Thus, according to the overinvestment hypothesis

(Olivers & Nieuwenhuis, 2005, 2006), the high alpha individuals in a state of readiness at

rest would be more likely to overinvest in the AB task and would have larger AB

magnitudes compared to the individuals preoccupied at rest. In this case I might expect

that levels of alpha at rest should correlate with measures of attentional investment during

the AB task, for example alpha ERD (see MacLean & Arnell, 2011). However, I found

no evidence of a correlation ($r = -.005$, $n = 21$) in the sample from Experiment 1 between

resting alpha as measured in this study and alpha ERD during the foreperiod that

preceded each RSVP trials in the AB task.

Interestingly, there was evidence of a correlation between both levels of beta and

theta at rest as measured in Experiment 1 with alpha ERD, although these correlations

were not significant with the sample of 21 individuals. Specifically, resting beta

correlated negatively with alpha ERD ($r = -.25, p = .28$) such that greater resting beta,

which in this study was associated with smaller AB magnitudes, was associated with

smaller reductions in alpha (ERD) preceding RSVP trials, which was found to relate to

better T2 performance at the short lag (i.e. smaller AB; MacLean & Arnell, 2011). The

opposite trend was observed for resting theta, such that greater resting theta was

associated with larger reductions in alpha (ERD) preceding RSVP trials ($r = .27, p = .24$).

That the correlations between alpha ERD and resting theta is in the opposite direction

from that observed with resting beta indicates that the relationship with resting beta is

unique, not a function of total resting power regardless of frequency. These correlations

support the interpretation that less attentional investment at rest was associated with

larger AB magnitudes in the current study because less attentional investment is

indicative of readiness to invest attention, leading to overinvestment in the AB task and

larger AB magnitudes.

Following this supposition, I wondered what attention was being invested in, at

rest, when there is no goal-directed task. Although it is possible that my instructions to sit

quietly and try not to move around demanded attentional investment, it is also possible

that the attentional investment I observed at rest was the result of mind wandering. Mind

wandering (a.k.a. day dreaming, stimulus independent thought, etc.) occurs when

attention is diverted, usually without awareness, away from external tasks, such as

detecting stimuli, toward internal tasks, such as reflection, and is more likely to occur

when attentional resources are available, such as when engaged in a well-practiced task

or at rest when not engaged in a goal-directed task (Smallwood & Schooler, 2006). Thus,

a complimentary interpretation of my findings is that individuals who invest attention at rest, characterized by lower alpha and greater beta power, are mind wandering. Mind wandering, extended beyond rest to coincide with performance of a task, may divert attention away from the task. In the case of the AB task, mind wandering, and the accompanying diversion of attention, would prevent overinvestment, which according to the overinvestment hypothesis (Olivers & Nieuwenhuis, 2006) should reduce the AB. Indeed, in their 2005 study, Olivers and Nieuwenhuis may have induced mind wandering during the AB task by having participants think about holidays or dinner with friends in an unconstrained fashion such that they could allow their thoughts to wander from theme to theme ("free association" condition). They found that AB magnitude was reduced in the free association condition compared to the control condition.

Mind wandering has been associated previously with greater cortical activation at rest as measured by metabolic activity, specifically in areas of the default mode network (Christoff, et al., 2009; Mason et al., 2007). The default mode network (DMN) is a network of cortical areas observed to decrease in activity, in most cases observed using PET or fMRI, when engaging in a goal-directed task from an awake, resting state such as the state from which I derived my measures in the current study (Raichle et al., 2001; Raichle & Snyder, 2007). Mason et al. (2007) observed that metabolic activity was greater in areas of the DMN during a well-practiced task with a greater prevalence of mind wandering than during a novel task with less mind wandering. It was also observed that a greater tendency to mind wander correlated positively with the increase in DMN metabolic activity observed during periods with greater opportunity for mind wandering.

So, it appears that when mind wandering occurs the DMN is engaged and that higher trait levels of mind wandering are also related to the greater DMN engagement.

Relevant to the findings of the current study, Laufs et al. (2003a) found that beta range oscillations (17-23 Hz) correlated with activity in cortical areas such as the posterior cingulate cortex and precuneus, areas thought to be part of the DMN. So, it is possible that the greater beta power I observed at rest in my study, which correlated with smaller AB magnitudes, was accompanied by the activated DMN which appears to characterize cortical activity when mind wandering occurs.

Conclusion

In conclusion, I observed that individual differences in both alpha and beta power during rest predicted individual differences in AB magnitude. Specifically, in two independent studies it was found that greater alpha at rest, less beta at rest, and greater alpha than beta at rest were related to larger AB magnitudes. I interpret these results as evidence that reduced attentional investment during rest is associated with larger AB magnitudes. It is possible that this relationship reflects the consequences of internally versus externally oriented attention (Cooper et al., 2003; Hanslmayr et al., 2011).

We further suggest that reduced attentional investment during rest may represent a state of readiness or anticipation that could contribute to greater investment in the AB task, and thus according to the overinvestment hypothesis (Olivers & Nieuwenhuis, 2006), a larger AB. I also suggest that individuals who display greater attentional investment during rest could be engaging in mind wandering. If mind wandering should occur during the AB task it could reduce investment in the AB task, and thus according to the overinvestment hypothesis (Olivers & Nieuwenhuis, 2006), reduce the AB.

Replication Study

As part of two separate studies, resting EEG data was collected from an additional 105 participants. With this additional sample I will (1) attempt to replicate the effects observed in the original study (Study 3 – Experiment 1, as described above) in the additional sample, (2) examine differences in the data from the sample in the original study and the additional sample, and finally (3) examine the effects observed in the original study with increased power by combining the original and the additional sample into one, composite sample.

Note that although an AB detection task was included in both of the additional studies that extreme methodological differences between these studies and Experiment 2 of Study 3 do not make it possible to "replicate" those findings. However, the relationships between resting EEG and AB detection performance will still be explored in the additional sample.

Methods

Participants

Participants were 105 Brock University undergraduate students (90 females; *M* age = 20 years), recruited through the Brock Psychology Department's online system for participant recruitment.

Procedure

A similar procedure was used in both studies. Participants first completed a resting EEG procedure, then AB tasks (AFC then detection), and finally the time production task.

AB Detection Task

The AB detection task was identical to that used in Study 1.

AB AFC Task

The AB AFC (alternative forced choice) task was identical to that used in Experiment 1 of the present study.

Resting EEG and Acquisition and Analysis

Resting EEG acquistion and analysis procedures were identical to that used in Experiment 1 of the present study, with the following exceptions. EEG was recorded continuously using tin electrodes embedded in an Electro-Cap© from 29 sites distributed according to the 10–20 system, with an electrode placed anterior to Fz as ground. EEG data were acquired with Neuroscan acquisition software using a 32-channel NeuroScan SynAmp.

<div align="center">

Results

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AB AFC Task Performance

AB AFC performance data for 16 participants were excluded due to either poor T1 performance (< 70% where 50% was chance) or poor T2 performance at lag 8 (< 50%). Mean T1 accuracy was 87.16% (SD = 8.37, range 70% to 99%). T2 accuracy was conditionalized on T1 performance. Mean T2 accuracy at lag 3 was 60.52% (SD = 15.96, range 19% to 91%). Mean T2 accuracy at lag 8 was 86.85% (SD = 10.06, range 54% to 100%). A paired-samples t-test indicated significantly lower T2 accuracy at lag 3 than lag 8 indicating the presence of an AB (t (88) = 20.01, $p < .001$).

AB magnitude was calculated as the difference measure, T2 performance on lag 3 trials was subtracted from T2 performance on lag 8 trials creating a difference measure

for each individual. Mean difference score was 26.34% ($SD = 12.42$). Note that the

following results were the same when a residual version of the difference measure was

used as in Studies 1 and 2.

Although accuracy was generally lower in the additional sample and AB

magnitude larger than in the original sample of Study 3 – Experiment 1, only T2 accuracy

on lag 8 trials was significantly lower (see Table 4-9).

Table 4-9 Comparisons with AB performance from Study 3

| | Additional | | Original | |
	M		M	t
T1	87.16	=	90.52	1.85
Lag 3 T2 Accuracy	60.52	=	66.89	1.79
Lag 8 T2 Accuracy	86.85	<	89.96	2.08*
AB Magnitude (Lag 8 – Lag 3)	26.34	=	23.07	1.45

*p < .05

AB Detection Task Performance

AB detection performance data for 35 participants were excluded due to either

poor T1 performance ($< 40\%$) or poor T2 performance at lag 8 ($< 50\%$). Mean T1

accuracy was 64.30% ($SD = 13.86$, range 41% to 90%). T2 performance was

conditionalized on T1 performance. T2 performance was measured as hits (correct on T2

present trials) minus false alarms (incorrect on T2 absent trials). Mean T2 performance at

lag 3 was 26.57% ($SD = 18.05$, range -8% to 71%). Mean T2 performance at lag 8 was

73.94% ($SD = 9.22$, range 51% to 93%). A paired-samples t-test indicated significantly

lower T2 performance at lag 3 than lag 8 indicating the presence of an AB ($t (69) =$

$23.60, p < .001$). Mean difference score was 47.37% ($SD = 16.79$).

Spectral EEG

A 3 X 4 repeated measures ANOVA performed on power values, with frequency range (theta, alpha, and beta) and ROI (frontal, central, parietal, and occipital) as factors, indicated a significant main effect of frequency band (F (2, 208) = 473.93, $p < .001$) such that power was greatest in alpha band and least in the beta band. There was also a significant main effect of ROI (F (3, 312) = 226.16, $p < .001$) and a significant interaction of frequency band and ROI (F (6, 624) = 235.86, $p < .001$) such that that power decreased from occipital to frontal ROIs in the alpha and beta band but was similar across ROI for the theta band. This pattern is identical to that observed in the original samples in Study 3.

A 2 X 3 mixed model ANOVA performed on power values, with frequency range (theta, alpha, and beta) as a within-subjects factors and sample group (original or additional) as indicated a significant interaction of frequency range and group (F(2, 226) = 6.29, $p = .002$). Follow-up independent samples t-tests indicated that the original sample and additional sample did not differ in either alpha or theta power (p's > .27), but that beta power was significantly higher in the additional sample ($M = -.11$, $SD = .11$) than in the original sample ($M = -.19$, $SD = .11$; t (113) = 3.40, $p = .001$).

As in Study 3 absolute power in the different frequency bands for each ROI (e.g. the correlation between alpha at the frontal ROI with beta at the frontal ROI, etc.) was significantly positively correlated (r's ranged from .37 to .76, all p's < .001), indicating that some individuals had higher power values than others across all frequency bands. Thus, the same residualized power values for each frequency band were created as in Study 3.

Residual power values in alpha and beta within the same ROI were negatively correlated at all ROIs (*r*'s ranged from -.26 to -.34, all *p*'s < .01). The residual power values in beta and theta also correlated negatively although only significantly (*p* < .01) at the occipital ROI (*r*'s ranged from -.15 to -.29), and residual power values in alpha and theta were significantly negatively correlated at all ROIs (*r*'s ranged from -.50 to -.59, all *p*'s < .001). These results are similar to those found in Study 3.

Correlations between Resting EEG and AB Performance

Tables 4-10, 4-11, and 4-12 contain the correlations between residual power in the theta, alpha and beta frequency bands at each ROI (frontal, central, parietal, and occipital) and overall across ROIs with various measures of performance on the two different AB tasks. Neither theta, alpha or beta power at rest were correlated significantly with any of the AB task performance measures including AB magnitude for either the AFC or detection tasks. Thus, the pattern of correlations observed in the original samples (Experiment 1 and 2) from Study 3 was not replicated in the additional sample[21]. Except for the higher level of beta in the additional sample there are no significant differences in either the AB performance or resting EEG measures. The only other difference between the two samples is their size, the additional sample is much larger than either of the two original samples and thus power was much greater in the additional sample. Note that when the additional sample was combined with the original sample from Study 3 there were also no significant correlations between resting alpha, beta and theta power and AB performance.

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[21] A hierarchical regression predicting AB magnitude was performed with resting alpha, beta and theta power on the first step and their interactions on the second step as was done in Experiments 1 and 2 of Study 3. None of the predictors or their interactions were significant unique predictors of AB magnitude in the additional sample (see Appendix C.5).

Table 4-10
Correlations between theta at each ROI, and overall theta with AB performance measures (additional sample only)

	T1 Accuracy	T2 Lag 3 Accuracy	T2 Lag 8 Accuracy	AB Magnitude
Theta (Frontal)	.02/-.03	-.05/-.18	-.12/-.21	-.03/.08
Theta (Central)	.01/-.00	.09/-.17	-.03/-.23	-.14/.06
Theta (Parietal)	.06/.17	.15/-.20	-.05/-.20	-.15/.11
Theta (Occipital)	.03/.00	.09/-.26	.07/-.19	-.06/.18
Theta Overall	.03/-.02	.07/-.22	-.01/-.22	-.10/.11

Note: AB AFC task ($n = 89$)/AB detection task ($n = 70$); * $p < .05$

Table 4-11
Correlations between alpha at each ROI, and overall alpha with AB performance measures (additional sample only)

	T1 Accuracy	T2 Lag 3 Accuracy	T2 Lag 8 Accuracy	AB Magnitude
Alpha (Frontal)	-.14/.16	.02/-.01	-.04/.10	-.06/.07
Alpha (Central)	-.11/.15	-.06/.00	-.05/.09	.03/.05
Alpha (Parietal)	-.07/.17	-.11/.04	-.10/.15	.06/.03
Alpha (Occipital)	-.08/.11	-.02/.18	-.07/.24	-.03/-.07
Alpha Overall	-.11/.16	-.05/.06	-.07/.16	.00/.02

Note: AB AFC task ($n = 89$)/AB detection task ($n = 70$); all p's > .05

Table 4-12
Correlations between beta at each ROI, and overall beta with AB performance measures (additional sample only)

	T1 Accuracy	T2 Lag 3 Accuracy	T2 Lag 8 Accuracy	AB Magnitude
Beta (Frontal)	.01/-.09	-.04/-.06	.02/.09	.06/.11
Beta (Central)	-.02/-.09	-.05/-.10	-.04/.13	.03/.18
Beta (Parietal)	-.09/-.07	-.10/-.09	-.11/.09	.04/.15
Beta (Occipital)	-.04/-.09	-.09/-.11	-.10/-.00	.03/.11
Beta Overall	-.04/-.09	-.08/-.09	-.06/.08	.04/.15

Note: AB AFC task ($n = 89$)/AB detection task ($n = 70$); all p's > .05

Discussion

In Study 3 I found that resting alpha and beta power predicted AB magnitude such

that greater resting alpha and less resting beta power were associated with larger AB

magnitudes. I conceptualized greater resting alpha and less resting beta power as indicative of less attentional engagement at rest, thus less attentional engagement at rest was associated with larger AB magnitudes.

As part of two other studies I collected resting EEG and AB performance data from an additional 105 participants. The topographical distribution and inter-correlations among resting power in the alpha, beta, and theta band, and the distribution of AB task performance measures observed in the additional sample replicated those observed in Study 3. Despite this, neither resting alpha nor beta power predicted AB magnitude. The only significant difference between Study 3 and the additional studies was a higher level of resting beta power in the sample from the additional studies. As the samples in the additional study were larger than those in Study 3 it is unlikely that the failure to replicate the findings of Study 3 were due to low power. It is possible that discrepancies between the procedure used to collect data for the replication study and those used in the original experiments of Study 3 could have lead to this failure to replicate the findings of Study 3, for example in both experiments of Study 3 there were repeated measures of resting EEG, as opposed to the single recording of resting EEG in the replication study. These possibilities are discussed further in the general discussion (Chapter 6). It is important to note, however, that the findings of Study 3 were observed in two independent experiments with different samples, and thus were already replicated.

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Chapter 5

Introduction

As mentioned in the General Introduction, the overinvestment that is

hypothesized to lead to an AB (Olivers & Nieuwenhuis, 2006), may be a result of

maladaptive cognitive control (Di Lollo, Kawahara, Ghorashi, & Enns, 2005; Olivers &

Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen, Juvina, Schipper, Borst, &

Martens, 2009). If so, measures of overinvestment would be expected to relate to each

other and to indicators of cognitive control. It is also possible that the inter-relations

among measures of investment and cognitive control could influence each other's

relationship with the AB. The following study will test these possibilities.

Recall that in Study 1 the feedback-related N2 predicted AB magnitudes, such

that individuals who had larger feedback-related N2 amplitudes, especially to feedback

that their performance was correct, had larger AB magnitudes, and that this relationship

was replicated in an additional sample. The data from that same additional sample,

however, did not replicate the correlations between resting alpha/beta power and the AB

that were observed in the original samples from Study 3. Furthermore, although the main

finding from Study 2, that trial to trial fluctuations in alpha ERD predict AB

performance, was replicated at right-frontal sites in the additional sample, the finding that

individual differences in ERD predicted individual differences in AB magnitude was not.

Thus, it is not possible to use the additional sample to test whether the inter-relations

among the feedback N2, alpha ERD, and resting alpha/beta power mediate or moderate

the relationships with AB magnitude[22]. However, it is possible to examine the inter-relations among the predictors as a means to better understand the nature of these measures which have previously been shown to predict the AB. Specifically, if overinvestment is a result of the maladaptive cognitive control, then stronger cognitive control, as indicated by stronger feedback-related N2s, should be associated with greater investment on task, as indicated by greater alpha ERD, and greater readiness at rest, as indicated by greater resting alpha and less resting beta power. Furthermore, recall that previous work showed that baseline alpha power was positively correlated with alpha ERD (Doppelmayr, Klimesch, Pachinger, & Ripper, 1998). It is possible in the additional sample to examine whether baseline alpha and alpha ERD were positively correlated as in this work.

It is also possible to examine whether the measures of investment (alpha ERD and resting alpha/beta power) influence the relationships between the feedback-related N2 and AB magnitude. Specifically, it is possible that the variability in cognitive control (feedback-related N2) which relates to the AB is either unique from the variability cognitive control may share with investment (alpha ERD, resting alpha/beta power), or that this variability may be shared to some degree. If the variability in the feedback-related N2 that relates to the AB is unique it may even be possible to improve the predictive strength of the feedback-related N2 by controlling for investment.

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[22] It is possible that, while the main effect of alpha ERD and resting EEG did not significantly predict AB magnitude, the interaction terms of those predictors may significantly predict AB magnitude. In order to test this hypothesis additional analyses were performed. A hierarchical multiple regression predicting AB magnitude with the main effects on the first step and the interaction terms on the second and third steps was performed. None of the predictors, main effects or interactions, were significant predictors of AB magnitude (see Appendix C.6).

The purpose of Study 4 was not to further address the relationship between cognitive control and the AB as Studies 1-3 did, but rather to further explore previous predictors of the AB. Specifically, the measures used in Studies 1-3 were conceptualized as indicators of attentional investment and cognitive control. According to those conceptualizations it is possible to hypothesize about how such measures might relate to one another. Testing the hypotheses described above, which are based on conceptualizations of the measures from Studies 1-3, will help to refine those conceptualizations.

Method

Participants

Participants were 63 Brock University undergraduate students (57 females; M age = 20 years), recruited through the Brock Psychology Department's online system for participant recruitment. Due to the length of the total testing time (~ 4 hours) some participants left before completing the experimental procedure, due to this attrition the sample size for each task is not equivalent (see Table 5-1).

Table 5-1 Sample size by task

Task (listed in order completed)	n
Resting EEG	63
AB Detection	62
AB AFC	58
Time Production	51

Procedure

Participants completed first the resting EEG procedure, then the AB detection task, followed by the AB AFC (alternative-forced choice) task, and finally the time production task. Stimuli were displayed and responses were logged using E-Prime, running on a desktop PC and presented on a CRT monitor (refresh rate of 60 Hz).

EEG Procedures

ERP acquisition and scoring of the FRN was identical to that in Study 1 (see Chapter 2). The acquisition and scoring of alpha ERD was identical to that in Study 2 (see Chapter 3). The resting EEG procedure and analysis was identical to that used in Study 3 (see Chapter 4).

Tasks

The AB detection task was identical to that used in Study 1 (see Chapter 2), except that in this case the feedback (correct and incorrect) was presented in black font for all participants. The AB AFC task was identical to that used in Study 2 (see Chapter 3). The time production task was identical to that used in Study 1 (see Chapter 2), except that in this case the feedback (correct, incorrect and uninformative) was presented in white font for all participants.

Results

Correlations between Investment and Cognitive Control

None of the N2 residual measures correlated with alpha ERD overall or with resting alpha or theta power (all p's > .10; see Table 5-2). Resting beta power was negatively correlated with N2 residual measures to feedback that performance was incorrect, such that greater resting beta was associated with more negative (i.e. larger) N2 amplitudes to feedback that performance was incorrect (see Table 5-2, *Figure 5-1*). This relationship was significant with the N2 to incorrect feedback overall and in the AB task, but not in the time production task (p = .142)[23].

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[23] In order to examine unique relationships among measures of cognitive control and investment multiple simultaneous regressions were performed with the N2s (correct and incorrect) from each task as predictors of alpha ERD, resting alpha, resting beta, and resting theta power (separately). None of the N2s were significant unique predictors of alpha ERD, resting alpha, resting beta, or resting theta power (see Appendix C.7).

Table 5-2 Correlations between N2 amplitude measures and resting power

	Beta	Alpha	Theta
N2 (overall) correct	-.09	.12	-.13
N2 (overall) incorrect	-.31*	-.05	.05
N2 (AB) correct	-.14	.19	-.14
N2 (AB) incorrect	-.33**	-.01	.05
N2 (TIME) correct	-.12	-.22	.13
N2 (TIME) incorrect	-.21	-.11	.02
N2 (TIME) uninformative	-.05	-.14	-.09

Note: * $p < .05$, ** $p < .01$, $n = 51\text{-}63$
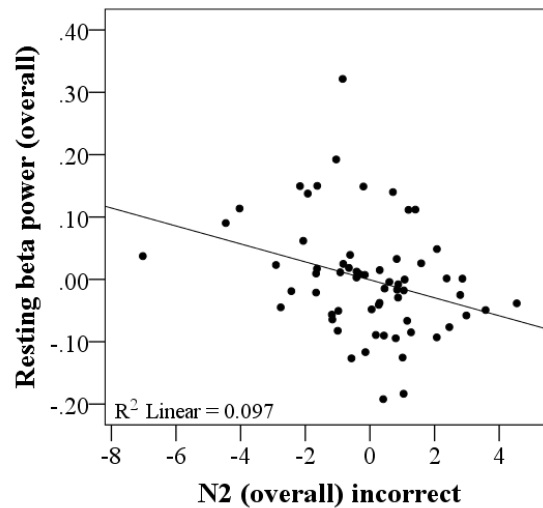
Figure 5-1 Scatterplot of the relationship between resting beta power and the N2 to incorrect feedback overall.

As greater resting beta power was previously found to be associated with smaller AB magnitudes, while larger N2 amplitudes were previously found to be associated with larger AB magnitude, it was expected that greater resting beta power would be associated with smaller N2 amplitudes. However, the opposite pattern was observed here. It is important to note, however, that while the relationship between N2 amplitudes and AB magnitude previously reported (Study 1, Chapter 2) was replicated in the current study, that relationship was only significant with the N2 to correct feedback. Furthermore, the relationship between resting beta power and AB magnitude previously reported (Study 3,

Chapter 4) was not replicated in the current study. Thus, the conditions that led to my hypothesis concerning resting beta power and N2 amplitudes were not replicated.

Despite not being significant, the correlation between AB magnitude and N2 amplitudes to feedback that performance was incorrect approached significance here. Resting beta power was not correlated with AB magnitude in this sample, thus it is unlikely that the variance shared between resting beta power and the N2 to feedback that performance was incorrect is the same variance that predicts AB magnitude. In this case it is possible that by accounting for that variance in N2 that is related to beta, the relationship between the N2 to feedback that performance was incorrect may be strengthened and become significant. In order to test this hypothesis a simultaneous multiple regression was performed with N2 incorrect amplitude (overall) and resting beta power (overall) as predictors of AB magnitude. Neither predictor was a significant unique predictor of AB magnitude over-and-above each other. However the semi-partial correlation ($sr = -.33$) between N2 incorrect amplitude and AB magnitude was slightly larger than the zero-order correlation ($r = -.31$) indicating that this relationship was not harmed once variability in resting beta power was accounted for.

Correlations among Measures of Investment: Alpha ERD and Resting EEG

Alpha ERD (overall) was not significantly correlated with either resting beta or theta power (see Table 5-3). Resting alpha power was significantly correlated with alpha ERD, both overall (i.e. across ROIs) and at every ROI except the occipital one (see Table 5-4). My hypothesis was that resting alpha power would be positively correlated with alpha ERD based on a previous observation of this relationship (Doppelmayr et al., 1998), such that greater resting alpha power would be associated with greater alpha ERD

on task. However, contrary to my hypothesis resting alpha power was negatively

correlated with alpha ERD such that greater resting alpha power was associated with

lesser alpha ERD on task (see *Figure 5-2*).

Table 5-3 Correlations between alpha ERD overall and resting alpha, beta and theta power

	ERD Overall	ERD Frontal	ERD Central	ERD Parietal	ERD Occipital
Resting alpha	-.35*	-.31*	-.36*	-.35*	-.26
Resting beta	.07	.11	.07	.00	.06
Resting theta	.19	.26	.22	.14	.04

Note: * $p < .05$, $n = 49$
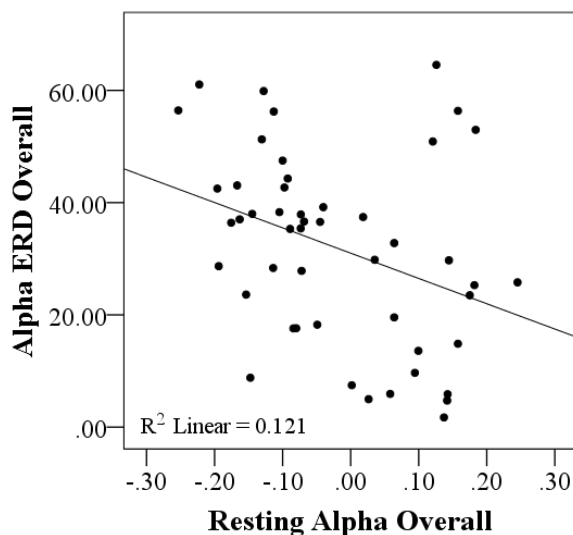
Figure 5-2 Scatterplot of correlations between resting alpha overall and alpha ERD overall

It is possible that resting alpha power as measured in this task is not equivalent to

the baseline alpha that was measured during the reference period that was found to

correlate positively with alpha ERD previously (Doppelmayr et al., 1998). In order to test

this hypothesis I measured baseline alpha during the reference period used to calculate

alpha ERD on task and correlated it with resting alpha power and alpha ERD. Baseline

alpha power from the reference period was significantly positively correlated with resting

alpha power ($r = .47$, $p = .001$) and significantly negatively correlated with alpha ERD ($r = -.37$, $p = .009$). Thus, my failure to replicate a positive correlation between resting/baseline alpha power and alpha ERD is not, apparently, due to the source of the baseline alpha measurement.

Discussion

The purpose of these additional analyses was to determine whether the relationship among measures previously found to predict the AB support the idea that they may represent interrelated aspects of cognitive control. Previously feedback-related N2 amplitude was found to predict AB magnitude (Study1, Chapter 2), such that greater amplitudes were associated with larger AB magnitudes. I conceptualized feedback-related N2 amplitude as a measure of the strength of cognitive control, such that larger amplitudes reflected stronger cognitive control. Alpha ERD (Study2, Chapter3) and resting alpha and beta power (Study3, Chapter 4) were both previously found to predict AB magnitude as well, such that greater alpha ERD and greater resting alpha were associated with larger AB magnitudes. Greater alpha ERD was conceptualized as greater anticipatory investment and greater resting alpha power as greater readiness, both, I hypothesized, were results of strong cognitive control. According to these conceptualizations I hypothesized that stronger cognitive control should result in greater attentional investment (greater anticipatory investment and readiness), thus greater feedback-related N2 amplitude should be associated with greater alpha ERD and greater resting alpha. These hypotheses were not supported by my additional analyses.

Neither resting alpha power nor alpha ERD correlated with feedback-related N2 amplitude. Thus, from these analyses it does not appear that individuals with stronger

cognitive control, as measured by feedback-related N2 amplitude, display greater

investment in the form of readiness, as measured by resting alpha power, or anticipation,

as measured by alpha ERD. That neither of my investment measures, either resting alpha

power or alpha ERD, were related to my measure of cognitive control suggests that

individuals with strong cognitive control are not necessarily overinvestors, and vice

versa. Thus, cognitive control and investment may possibly influence AB magnitude via

independent channels, such that either or both strong cognitive control and attentional

investment result in the AB, but are not mutually inclusive.

Resting beta power was correlated with feedback-related N2 amplitude, such that

greater resting beta power was associated with larger feedback-related N2 amplitudes,

however, this was only the case for N2 amplitude following feedback that performance

was incorrect. Resting beta power, I conceptualized, as indicative of a state of vigilance

(Laufs et al., 2006). Thus, the relationship between feedback-related N2 amplitude

following feedback that performance was incorrect and resting beta power indicates that

greater vigilance is associated with stronger responses to negative feedback. This pattern,

such that greater vigilance is associated with stronger responses to feedback, is sensible if

one were naïve to the relationship of these two measures with AB magnitude as

previously observed. Whereas I did not have specific hypotheses concerning the

relationship between resting beta power and feedback-related N2 amplitude, based on the

previous correlations with AB magnitude discussed in this dissertation I would have

expected the opposite pattern of relationship. Previously, resting beta power was found to

negatively predict AB magnitude, thus greater resting beta power was associated with

smaller AB magnitudes, and feedback-related N2 amplitudes were found to positively

predict AB magnitude, such that greater feedback-related N2 amplitude was associated with larger AB magnitudes.

Thus, based on that pattern of relationship, I would hypothesize that resting beta power and feedback-related N2 would be negatively correlated. However, the relationship between AB magnitude and feedback-related N2 amplitude was only significant for the N2 following positive feedback (i.e. feedback that performance was correct), while the current relationship between resting beta power and feedback-related N2 amplitude was only significant for the N2 following negative feedback (i.e. feedback that performance was incorrect). So, the current findings are not inconsistent with my previous findings, but do suggest that the relationship between feedback-related N2 amplitude and AB magnitude is not due to variability in vigilance (i.e. resting beta power). This is further supported by the finding that the relationship between the feedback-related N2 to negative feedback and AB magnitude was improved, although not significantly, when variability in vigilance as measured by resting beta power was accounted for as compared to the zero-order correlation.

I also hypothesized that measures of investment, readiness and anticipation, should correlate positively with each other, thus greater alpha ERD should be associated with greater resting alpha power. This relationship was also hypothesized as baseline alpha was previously shown to be positively correlated with alpha ERD (Doppelmayr et al., 1998). This hypothesis was also not supported by my additional analyses. Baseline alpha as measured by both resting alpha power and alpha power in the reference period used to calculate alpha ERD, was negatively correlated with alpha ERD, such that greater baseline alpha was associated with less alpha ERD.

One possible reason for the failure to replicate a positive relationship between baseline alpha power and alpha ERD as observed by others (Doppelmayr et al., 1998) is the difference in the conditions of the task used to elicit alpha ERD and the location of alpha ERD. Doppelmayr et al. used a simple oddball task where alpha ERD was measured as the change in alpha power from baseline, the interval in between a warning stimulus, and "test" - the interval following the presentation of the oddball stimuli. Thus, alpha ERD in this case is not "anticipatory" as it was in the current study where alpha ERD was measured as the change in alpha power from baseline (the interval prior to a fixation warning stimulus) and the foreperiod interval that preceded each RSVP trial. So, it is possible that relationship between baseline alpha and alpha ERD depends on whether the change in alpha power (i.e., ERD) is due to anticipatory processes or due to processing the task stimulus.

Another important factor to consider in these additional analyses is that the hypothesized relationships among these predictors were based on the conceptualization of these measures in the context of their relationship with AB magnitude as previously observed (see Studies 1-3, Chapters 2-4). However, only one of those previously observed relationships was replicated in the current study, the relationship between feedback-related N2 amplitude and AB magnitude. Thus, it is difficult to conclude whether my failure to find the hypothesized relationships between these predictors is due to my incorrect conceptualization of these predictors and their relationship with each other, or whether in the current sample the variability I captured in these predictors is not the same that was previously shown to predict AB magnitude.

In conclusion, in the current study the measure of cognitive control was not related to the measures of attentional investment, and the measures of attentional investment (anticipation and readiness) were not related to each other either. Thus, my hypotheses that stronger cognitive control should be associated with greater investment was not supported, indicating the possibility that cognitive control and investment may predict AB magnitude independently from each other. The only significant relationship among these predictors was one where greater resting beta power was associated with larger feedback-related N2 amplitudes to negative feedback. This indicates that greater vigilance is associated with stronger responses to negative feedback. This relationship was confined to negative feedback, while the relationship between the feedback-related N2 amplitude and AB magnitude was confined to positive feedback. Furthermore, the relationship between feedback-related N2 amplitude and AB magnitude was not attenuated when vigilance was accounted for. This suggests that the relationship between AB magnitude and the feedback-related N2 is not due to greater vigilance.

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Chapter 6

General Discussion

This dissertation is composed of four studies directed at investigating whether cognitive control can explain performance on trials where an AB did or did not occur (Study 2), trial to trial (Study 1) and individual to individual differences in AB performance (Studies 1,3, and 4). In line with the recent models of the AB (Di Lollo, Kawahara, Ghorashi, & Enns, 2005; Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen, Juvina, Schipper, Borst, & Martens, 2009), I proposed that while the AB may be the result of the limitations of a particular cognitive process, such as encoding into visual short term memory/working memory (Chun & Potter, 1995; Jolicoeur & Dell'Acqua, 1998; Shapiro, Raymond, & Arnell, 1994), the mechanism that imposes that limitation is cognitive control. More specifically, the premise of this dissertation was that strong cognitive control is maladapted to the dual-task conditions of the AB task because cognitive control optimizes information processing in favor of a temporally proximate goal, T1, at the cost of an equally important (as defined by the task), but more temporally distant goal, T2. Furthermore, it was suggested that the degree to which a given individual enforces cognitive control would determine the degree of cost to T2, that is individual differences in cognitive control would predict individual differences in AB magnitude.

It was proposed that overinvestment is a result of strong cognitive control, in other words, that strong cognitive control is accompanied by greater investment of attentional resources. Whereas previous evidence had shown that overinvestment in the task (Olivers & Nieuwenhuis, 2005; 2006) resulted in larger ABs, and individual

differences in linked to investment (Arnell & Stubitz, 2010; Dux & Marois, 2008; MacLean & Arnell, 2010; MacLean, Arnell, & Busseri, 2010; Martens & Valchev, 2009) predicted AB magnitude, it was proposed that more direct electrophysiological measures of investment would also predict AB magnitude. Additionally, if overinvestment was a result of strong cognitive control, then individual differences in cognitive control should predict individual differences in investment.

The first three studies of this dissertation were designed to investigated particular measures of cognitive control and attentional investment, and were used to explain performance on trials where an AB did or did not occur (Study 2), trial-to-trial variability in performance in the AB (Study 1) as well as individual-to-individual variability in AB magnitude (Studies 1 and 3). While the final study (Study 4) was designed to investigate the hypothesized relationships among the predictors from Studies 1-3. The following is a brief summary of the findings and conclusions of those studies.

Study 1: Cognitive Control as Indicated by Cortical Responsiveness to Performance Feedback

My assertion was that cognitive control is maladapted to the specific dual-task conditions of the AB task because cognitive control serves goals, but that the management of goals results in a bias towards optimizing processing for T1 at the cost of processing T2, which is adaptive in the majority of cases. Thus, measures related to the goal-management aspect of cognitive control (i.e., caring about whether outcomes match goals) should predict individual difference in AB magnitude. Feedback-related negativity amplitude has been associated with the degree to which individuals adopt and enforce a strategy to achieve goals (Bellebaum & Daum, 2008; van der Helden, Boksem, & Blom,

2009). Thus, I hypothesized that feedback-related N2 amplitude would predict AB

magnitude such that greater N2 amplitude will be associated with larger AB magnitudes.

In an AB task and a time production task, feedback-related N2 amplitude was measured

following positive feedback, indicating that performance (T2 performance or time

estimate accuracy) was correct, and negative feedback, indicating that performance was

incorrect. Feedback-related N2 amplitudes were also measured following uninformative

feedback (i.e. did not indicate whether performance was accurate or not) in the time

production task. Larger feedback-related N2 amplitudes to all types of feedback from

both tasks were associated with larger AB magnitudes. The relationship was strongest

with the feedback-related N2 amplitudes to positive feedback. The valence-modulation of

the feedback-related N2 (commonly referred to as the FRN), as measured by the

difference in N2 amplitude following positive and negative feedback (i.e. negative N2

amplitude – positive N2 amplitude), did not predict AB magnitude. This pattern of

findings was also replicated in the additional sample. I speculated that while most people

would be responsive to negative feedback, only individuals with very strong cognitive

control, who were highly motivated, would be highly responsive to positive feedback in

particular. Another possibility is that the variability in responsiveness to reward may

underlie the unique relationship between AB magnitude and the feedback-related N2 to

correct feedback (Holroyd et al., 2008). In that case it is possible that individuals who are

more sensitive to reward have larger ABs. In conclusion, my hypothesis was supported,

stronger responsiveness to signals regarding whether outcomes matched goals or not, as

measured by feedback-related N2 amplitude, was associated with larger AB magnitudes.

Study 2: Attentional Investment as Indicated by Anticipatory Cortical Activation

Based on the overinvestment theory (Olivers & Nieuwenhuis, 2006), and evidence that overinvestment of attentional resources results in the AB and individuals who invest more have larger AB magnitudes (Arnell & Stubitz, 2010; Dux & Marois, 2008; Martens & Valchev, 2009), I predicted that electrophysiological measures of investment would predict AB magnitude. Alpha event-related desynchronization (ERD), an event-related decrease in alpha power, is thought to reflect an anticipatory investment of attentional resources when measured following a warning stimulus, prior to the presentation of goal-relevant stimuli (Bastiaansen & Brunia, 2001; Bastiaansen, Böcker, & Brunia, , 2002; Bastiaansen, Böcker, Brunia, de Munck, & Spekreijse 2001; Capotosto, Babiloni, Romani, & Corbetta, 2009; Onoda et al., 2007; Yamagashi, Goda, Callan, Anderson & Kawato, 2005).

Thus, I hypothesized that alpha ERD would be greater prior to short lag trials where T2 performance was incorrect (a blink), than on short lag trials where an AB did not occur, but that this pattern would not be observed at long lags (the AB being a temporary impairment of T2 performance at short lags that is absent at longer lags). Furthermore, I predicted that individuals with greater alpha ERD across all trials would have larger AB magnitudes, although this hypothesis was not supported. The former hypotheses, however, were supported: Alpha ERD was larger on incorrect short lag trials than correct short lag AB trials, and was larger on T1 correct trials and on long lag T2 correct trials than on T1 incorrect and long lag T2 incorrect trials. Thus, while greater anticipatory investment benefitted T1 and long lag T2 performance, when T2 appeared during the AB interval following T1 (i.e. shorter lags), greater anticipatory investment was detrimental to T2 performance. This pattern of results was replicated in the

additional sample, but only at right frontal sites where the effect was strongest in the original sample.

These data fit with my assertion that cognitive control optimizes information processing to the benefit of goals, for example by increasing anticipatory investment benefitting task performance, but is maladapted to the dual-task conditions of the AB task, biased towards T1 at the cost of T2 when T2 appears at shorter lags. Individual differences in alpha ERD were also positively associated with individual differences in AB magnitudes. However, this relationship was not significant, and was not replicated in the additional sample. In conclusion, my hypothesis was supported as the AB was associated with greater anticipatory investment of attention in that alpha ERD differed according to T2 performance, and this pattern differed according to lag, such that greater anticipatory attentional investment (alpha ERD) was found on T2 incorrect trials at shorter lags and T2 correct trials at longer lags. However, individual differences in overall alpha ERD did not predict individual differences in AB magnitude. This suggests that anticipatory investment is associated with whether T2 performance is impaired at shorter and longer lags, but is not associated with the overall tendency for T2 performance to be correct or not (i.e. is not associated with the overall probability with which T2 performance is correct or incorrect).

Study 3: Readiness as Indicated by Resting Cortical Activation

Another manner of measuring investment is to examine readiness during the resting state, when not engaged in a goal-directed task. The level of alpha frequency (8-12 Hz) power is related to attentional investment such that when alpha is high, attention is not engaged and the cortex is "idling" (Brunia & van Boxtel, 2001; Pfurtscheller,

Stancák, & Neuper, 1996) or inhibited (Klimesch, Sauseng, & Hanslmayr, 2007). Thus, while at rest, when not engaged in a goal-directed task, readiness would be indicated by higher levels of alpha power. I hypothesized that greater readiness, indicated by greater resting alpha power would be associated with larger AB magnitudes. Furthermore, occupation at rest, the opposite of readiness, would be indicated by an increase in beta frequency (15-35 Hz) power accompanying a decrease in alpha (i.e. an active cortex). Thus, I hypothesized that greater resting beta power would be associated with smaller AB magnitudes. In two separate samples, as part of two different studies, I found that greater alpha, and less beta, and greater alpha than beta power at rest were associated with larger AB magnitudes. In conclusion, my hypothesis that greater readiness (to invest) would be associated with larger AB magnitudes was supported. However, these findings were not replicated with the additional sample.

Study 4: Relationships among Measures of Cognitive Control and Investment that Predict AB magnitude

As mentioned above, I proposed that the maximal investment of attentional resources (i.e. overinvestment in the case of the AB task) was a mechanism of cognitive control that optimizes information processing. Thus, measures of investment should relate to each other, such that greater anticipation (alpha ERD) should be associated with greater readiness (alpha power at rest), and both should be predicted by strength of cognitive control, such that stronger cognitive control (feedback-related N2 amplitude) should be associated with greater investment. As part of a separate study, all four of the measures found to predict AB magnitude in the first three studies (feedback-related N2, alpha ERD, and resting alpha and beta power), were collected together in an additional

sample in order to examine (1) whether they related to each other in the hypothesized manner and (2) whether they influenced each other's relationships with AB magnitude.

My hypotheses were not supported. Most of these measures were not correlated with each other (feedback-related N2 amplitude with alpha ERD and resting alpha), and those relationships that were observed related to each other in the direction opposite to what was predicted. Specifically, greater anticipation (i.e. greater alpha ERD) was associated with less readiness (less alpha power at rest). It was observed that greater beta power at rest was associated with larger N2s to feedback. As higher beta power at rest is thought to be indicative of a state of vigilance (Laufs et al., 2006), it is consistent that individuals who are more vigilant would react more strongly to performance feedback, which could also be thought of as an indication of vigilance (i.e. paying consistent attention), as a failure to respond to performance feedback could be indicative of a failure to be consistently vigilant to all relevant information. In conclusion, my hypothesis that overinvestment is a result of strong cognitive control was not supported by the relationship among measures of cognitive control and investment previously found to predict AB magnitude in separate studies.

Implications for Theoretical Accounts of the AB

The Role of Cognitive Control in the AB

The studies that comprise this dissertation were driven by hypotheses derived from theoretical models of the AB. Here I sought to explore those hypotheses from an individual differences perspective using electrophysiological measures of investment and cognitive control. While not explicitly proposed in any one theory of the AB, when examined together I found that an account of maladaptive cognitive control fit with many

of the ideas contained within several recent cognitive control models of the AB. These models no longer conceptualized the AB as only the inevitable result of a fundamental, capacity limited processing bottleneck (e.g. Chun & Potter, 1995; Jolicoeur & Dell'Acqua, 1998), but posited that the limitations that underlie the AB are a demonstration of cognitive control. In other words, while information processing limitations (i.e. bottlenecks) may ultimately prevent T2 from being encoded, the degree to which that limitation is encountered depends on how task goals are conceptualized and how information processing is optimized in order to meet goals.

For example, the bottleneck is thought to occur while T1 is being encoded into VSTM/WM (Chun & Potter, 1995; Jolicoeur & Dell'Acqua, 1998). The P3 component, thought to reflect updating of WM, is larger for T1 when the AB deficit is larger, and is earlier and faster for T1 in non-blinkers (Martens et al., 2006). Thus it is possible to observe the timing of encoding T1 into WM with the P3, and thus to observe the bottleneck. Measures of cognitive control and investment in the task goals should predict the timing of T1 encoding, where strong cognitive control and investment in the task goals (e.g. larger alpha ERD, larger feedback-related N2s) would be associated with a larger and longer P3 to T1. This example demonstrates that cognitive control models of the AB and bottleneck models of the AB are not incompatible, but rather approach the AB from different perspectives (e.g., what initial conditions create a bottleneck vs. at what stage of information processing the bottleneck is located). It is important to note that the hypotheses that this dissertation was designed to test were not meant to distinguish among models of the AB. Rather my hypotheses were based on common themes present in multiple models of the AB.

There is plenty of behavioral and electrophysiological evidence supporting

models of the AB that propose that limitations (bottlenecks) on information processing at

post-perceptual stages ultimately cause the AB to occur. For example the P3, an

electrophysiological measure thought to be related to VSTM/WM encoding processes, is

absent following T2 at shorter lags (Vogel, Luck, & Shapiro, 1998), although only when

T1 requires identification (Sessa, Luria, Verleger, & Dell'Acqua, 2007), and the AB is

larger when the P3 to T1 is larger (Martens, Elmallah, London, & Johnson, 2006).

Furthermore, when T2 is not masked, the P3 to T2 returns although is delayed by around

100 milliseconds when T2 is presented at a shorter lag as compared to a longer lag

(Vogel & Luck, 2002). Similarly, when T2 is unmasked and the T2 task is speeded,

reaction times (RTs) to T2 are longer when T2 is presented at shorter rather than longer

lags (Jolicoeur & Dell'Acqua, 1998). Thus, as suggested by bottleneck models (Chun &

Potter, 1995; Jolicoeur & Dell'Acqua, 1998), the encoding of T2 into working memory

does appear to be impaired/delayed when T2 is presented during the AB period as

indicated by the absent/late P3 and longer RTs to T2. Furthermore, the AB deficit is

modulated by the encoding of T1 as indicated by larger deficits accompanying larger P3s

to T1 and the reappearance of the P3 to T2 when T1 no longer requires identification.

Similarly, the AB is larger when RTs to a speeded T1 task are longer (Jolicoeur, 1998,

1999a), and when the WM demands of the T1 task are greater (Jolicoeur, 1999b).

However, information processing models (Bowman & Wyble, 2007; Chun &

Potter, 1995; Jolicoeur & Dell'Acqua, 1998; Shapiro, et al., 1994) cannot provide a full

account of the AB findings. Specifically, whereas plenty of evidence supports these

models assertions that bottlenecks of limited processing resources interfere with the

necessary and timely processing of T2, they do not account for some instances when an AB does not occur or is significantly reduced (e.g. Di Lollo et al., 2005; Jeffries, Smilek, Eich, & Enns, 2008; Nieuwenstein & Potter, 2006, Olivers & Nieuwenhuis, 2006), or why individuals have reliably smaller or larger AB deficits (Dale, Dux, & Arnell 2013; Dale & Arnell, 2013; McLaughlin, Shore & Klein, 2001), or sometimes no deficit at all, as in the case of non-blinkers (Martens et al., 2006). For example, the AB is markedly attenuated when participants perform an additional task (Olivers & Nieuwenhuis, 2005), when participants are asked to report the entire RSVP sentence (Nieuwenstein & Potter, 2006), or when three targets appear in succession, relative to when two targets are separated by a distractor (Di Lollo et al, 2005). In each of these cases information processing models of the AB would expect the AB to increase, as opposed to decrease, given the increased information processing demands. Furthermore, although individual differences on various measures of information processing speed have been shown to predict overall T2 accuracy in the AB task, they do not predict the lag-dependent T2 accuracy change that is the AB (Arnell, Howe, Joanisse & Klein, 2006).

Cognitive control, however, is a concept that does accommodate such evidence. Specifically, the proposal of this dissertation is that cognitive control is the optimization of information processing in order to achieve goals, thus setting-up the process limitations and bottlenecks that cause the AB. Cognitive control is maladapted to the particular conditions of the AB as information processing is optimized in favor of the temporally proximate goal of T1 at the cost of the temporally distant T2. As an extension of this, the motivation to accomplish goals and the manner in which goals are framed will

determine whether cognitive control optimizes information in a maladaptive fashion as described above, leading ultimately to bottlenecks and the AB, or not.

Thus, I propose that how the participant frames the goals of the task influences the magnitude of the AB deficit. For example, when participants were instructed to try hard and concentrate on the AB task the resultant AB was significantly larger than when they were instructed to "pay less attention" to the RSVP items (Olivers & Nieuwenhuis, 2006). This provides evidence that the motivation with which an individual approaches the AB task influences the AB, such that when they are instructed to be less motivated to accomplish the task goals (i.e. report on T1 and T2) the AB is reduced as compared to when they are instructed to be very motivated to accomplish the task goals. The affective state that an individual is induced into also influences the size of the AB deficit, such that individuals in high arousal and negative affect conditions (Jefferies, et al., 2008; Olivers & Nieuwenhuis, 2006); associated with greater focus of attention (Ashby, Isen, & Turken, 1999), tend to have larger AB deficits. This also supports the idea that the motivational approach influences the AB, such that greater motivation to accomplish task goals is associated with the AB. Similarly, individual differences in measures related to motivation to accomplish goals, such as naturally-occurring state and trait affect, as well as personality traits, predict individual differences in AB magnitude, such that negative state and trait affect (rigid, focused attention; Ashby et al., 1999) are associated with larger AB magnitudes (MacLean et al. 2010; MacLean & Arnell, 2010) while positive affect, extraversion and openness (flexible, diffused attention; Dreisbach, 2006; Fredrickson, 2001; Fredrickson & Branigan, 2005; Le Pine, Colquitt, & Erez, 2000) are

associated with smaller AB magnitudes (MacLean et al., 2010; MacLean & Arnell, 2010).

Not only does the motivation to accomplish goals influence the AB, but also the manner in which task goals are framed. A typical AB occurs when only the two red target words must be reported from the RSVP sentence, but performance for the same target items was not impaired when the participants was asked to report the entire RSVP sentence (Nieuwenstein & Potter, 2006). This provides strong evidence that how the participant frames the goals of the task (e.g., either as performing two separate tasks on two distinct target words or as one task to report a sentence) influences their target performance. Specifically, it appears that when the relevant information is framed as part of one goal report on that information is better than when that same information is framed as being relevant to two separate goals.

The novel assertion in this dissertation is that the management of multiple goals is maladapted to the dual-task conditions of the AB, prioritizing T1 at the cost of T2 that initiates the conditions that result in an information processing bottleneck and the resultant AB. So, it was hypothesized that measures of functions related to the management of multiple goals would be related to the AB. The anterior cingulate cortex (ACC) is thought to be one location involved in the management of goals (Botvinick, Cohen, & Carter, 2004). The ACC is also one structure in the neuro-cognitive system thought to be involved in the AB (Hommel et al., 2006). The ACC is thought to be one generator of the feedback-related negativity (FRN; Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997; Ruchsow, Grothe, Spitzer, & Kiefer, 2002), a cortical response shown to reflect the degree to which individuals adopt and enforce strategy to

achieve goals (Bellebaum & Daum, 2008; van der Helden et al., 2009). I found that individuals with larger feedback-related N2s had larger AB magnitudes, indicating that stronger responses to information regarding whether goals were achieved or not were associated with larger AB magnitudes. This supports the assertion that cognitive control can modulate the AB, and furthermore, the specific claim of this dissertation that it is the goal-management aspect of cognitive control (i.e., caring about whether outcomes match goals) specifically that promotes conditions that lead to the AB.

This proposed role of cognitive control in the AB, as suggested in this dissertation, and the supporting evidence, both that contained within this dissertation and elsewhere (e.g. Jeffries et al., 2008; Nieuwenstein & Potter, 2006; MacLean & Arnell, 2010; MacLean et al., 2010; Olivers & Nieuwenhuis, 2006), do not contradict existing theoretical accounts of the AB. Rather, by providing an account that accommodates variability in the AB, this model compliments pre-existing models of the AB and leads to interesting ideas for further study. For example, I would hypothesize that individuals who have strong cognitive control, as indicated by feedback-related N2 amplitude, would more reliably enforce the limitations which optimize information processing for T1, e.g. they would more reliably enforce the "boost and bounce" (Olivers & Meeter, 2008), overinvest attention in the RSVP items (Olivers & Nieuwenhuis, 2006), abandon control over input filters in favor of focusing on T1 (Di Lollo et al., 2005), all these resulting in larger AB magnitudes. Individuals with stronger cognitive control, as indicated by feedback-related N2 amplitude, may enforce greater restrictions on capacity for encoding information into VSTM/WM, as is suggested to lead to the AB in bottleneck models of the AB (e.g. Chun & Potter, 1995; Jolicoeur & Dell'Acqua, 1998; Shapiro et al., 1994).

So, the findings of Study 1, that greater feedback-related N2 amplitude was associated with larger AB magnitudes, supports my assertion that the goal-management aspect of cognitive control contributes to the production of the AB.

This hypothesis compliments other theoretical accounts of the AB, suggesting that the specific limited cognitive mechanism that leads to an AB is the result of cognitive control, which is ultimately responsible for the AB and furthermore explains individual differences in AB magnitude. Other theoretical accounts of the AB do not explicitly provide an account for individual differences in the magnitude of the AB. The hypothesis of this dissertation, however, explicitly addresses inter-individual variability in AB magnitude by proposing that individual differences in the strength of cognitive control determine whether the limitations discussed in other theoretical accounts of the AB will be reliably enforced. When those limitations, for example the capacity limitation of encoding into VSTM (Shapiro, Raymond, & Arnell, 1994), are reliably enforced (i.e. rigidly enforced) as in people with strong cognitive control, AB magnitudes will be larger than when less reliably enforced (i.e. flexibly enforced).

Investment

I have also asserted in this dissertation that maximal investment of attention was one such condition determined by cognitive control as a means for optimizing information processing in order to achieve goals. Olivers and Nieuwenhuis (2006) proposed that overinvestment may be one cause of the AB, such that T1 and distractors items presented in the RSVP stream during an AB task receive an unnecessarily large investment of attention, allowing them to interfere with T2 (Olivers & Nieuwenhuis, 2006). This hypothesis is supported by evidence that preventing overinvestment reduces

the magnitude of the AB (Olivers & Nieuwenhuis, 2005; 2006), and that individual differences in investment predicted AB magnitude (Arnell & Stubitz, 2010; Dux & Marois, 2008; Martens & Valchev, 2009). Thus, I hypothesized that electrophysiological indicators of investment should also relate to the AB. The results of both Study 2 and Study 3 of this dissertation support that hypothesis. Greater anticipatory investment, as indicated by greater alpha ERD, and greater readiness (to invest), as indicated by greater resting alpha power, were both found to relate to the AB and to larger AB magnitudes respectively. The results of Study 2 and 3 also indicate that trait-like investment and investment as measured outside of the RSVP are also related to the AB. Thus, the degree of attentional investment in general is associated with the AB, not just investment in RSVP items, although it may be related to investment in RSVP items. Furthermore, that investment predicts the AB both trial-to-trial (alpha ERD, Study 2) and person-to-person (resting alpha power, Study 3).

An additional hypothesis examined in this dissertation was that overinvestment was a result of strong cognitive control, and thus cognitive control should predict investment. In Study 4, however, cognitive control as measured by feedback-related N2 amplitude was not related to either measure of investment. Furthermore, the measures of investment were negatively related to each other such that greater readiness (resting alpha power) was associated with less anticipation (alpha ERD), in contrast to both my hypothesis and previous findings that baseline alpha power and alpha ERD were positively correlated (Doppelmayr, Klimesch, Pachinger, & Ripper,, 1998). That my measure of the goal-management aspect of cognitive control did not predict measures of investment does not, of course, rule out my original hypothesis that overinvestment is a

result of cognitive control. It could be that only variability in overinvestment during the RSVP is predicted by cognitive control, or that while cognitive control does predict overinvestment it is not the goal-management aspect of cognitive control that does so.

However, there is another possibility. It is possible that alpha ERD and alpha power at rest (i.e. baseline alpha) are also demonstrations of cognitive control and that while cognitive control is defined here as information processing in the service of achieving goals, the various aspects of controlled information processing can affect the AB independently. There is evidence in the literature to support the idea that alpha power, and modulations in alpha power (e.g. ERD) are not simply indicators of the degree to which attention is invested, but are rather the mechanism for controlling the investment of attention over time in a goal-consistent fashion (i.e. cognitive control; Klimesch, 2012).

Alpha as Cognitive Control

In a recent review Klimesch (2012) provided a comprehensive account of the evidence of a connection between alpha frequency cortical activity and attention, and suggested that alpha, and modulations in alpha (event-related desynchronization and synchronization; ERD and ERS) are the means of controlling the timing of attention, and thus optimizing information processing in order to achieve goals. The account of cognitive control provided in this dissertation is that cognitive control reflects the optimization of information processing in order to achieve goals. One way that information processing would be optimized to achieve goals is to precisely time the application of attention. Klimesch suggests that the timing of the inhibition of cortical activity, a mechanism of selective attention, is controlled by alpha frequency oscillations,

or more specifically modulations of alpha oscillations. Specifically, ERS, representing an increase in alpha power, inhibits cortical activity while ERD, representing a decrease in alpha power, releases inhibition (or allows attention to be invested). So, patterns of ERS and ERD can precisely and selectively time the application of inhibition and thus attention.

As reviewed by Klimesch (2012), the evidence of task-related modulations of alpha oscillations suggests that the timing of inhibition by alpha is goal-driven. Thus, modulations of alpha oscillations represent the optimization of the timing of information processing in order to achieve goals (i.e. the temporal aspect of cognitive control). Alpha, then, is an especially good candidate for accounting for the AB and individual differences in AB magnitude, as the AB requires efficient timing of attention, that is, the application of sufficient attention to T1, the suppression of attention to intervening distractors, and the application of sufficient attention to T2. This would be evident in a decrease in alpha to T1 (ERD), followed by an increase in alpha to trailing distractors (ERS) and another alpha ERD to T2. This is one possibility among many that have been suggested for the role of alpha in the AB (see Hanslmayr, Gross, Klimesch, Shapiro, 2011).

Originally in this dissertation resting alpha power and alpha ERD were conceptualized as measures of investment such that resting alpha power indicated readiness to invest and alpha ERD represented anticipatory investment. This conceptualization fits with the idea that the level of alpha power is inversely related to the level of cortical activity (Goldman, Stern Engel & Cohen, 2002; Laufs et al. 2003a, 2003b, 2006; Moosman et al., 2003), and in as much as greater cortical activity represents attention-driven information processing, then, when less attention is invested there should

be less cortical activity and thus greater alpha. However, the idea that modulations in alpha oscillations represent not simply the presence or absence of attention-driven cortical activity, but rather reflect cognitive control via the timing of inhibition (i.e. attention) in the service of goals, suggests that alpha, and modulations in alpha, are not simply the outcome of cognitive control, but rather are indicators of controlled information processing.

In this regard the findings of Studies 2 and 3 also support the hypothesis of this dissertation that cognitive control (i.e. goal-driven optimization of information processing) creates the conditions which lead to the AB, and that individual with stronger will have larger AB magnitude. Specifically, Klimesch (2012) proposes that individuals with greater resting alpha power, (associated with larger AB magnitudes in Study 3), will more efficiently filter information via inhibition, and thus have stronger cognitive control. One way to think of individuals with greater resting alpha power is that these individuals will inhibit cortical activation unless engaged in a goal-directed task, in other words they "save" cortical resources for goal-relevant information. Klimesch proposes that anticipatory alpha ERD, (found to be larger on AB trials than on no-AB trials in Study 2), reflects the activation of goal-relevant information prior to its presentation. This would suggest that on AB trials (trials where T2 was incorrect at short lags and trials where T2 was correct at long lags) there was stronger activation of goal-relevant information than on no-AB trials (trials where T2 was correct at long lags and trials where T2 was incorrect at long lags). So, greater alpha ERD prior to an RSVP trial may not only indicate greater recruitment of attention in anticipation of performing an attention demanding task, but also stronger investment in task goals more generally.

That readiness, as indicated by resting alpha power, and anticipation, as indicated by alpha ERD, were negatively related to each other may indicate individual differences in the style of cognitive control that an individual employs. It is possible that some individuals are high inhibitors; these individuals would strictly inhibit information processing except in the presence of goal-relevant information. As a result, these individuals would maintain high levels of alpha power except in the presence of goal-relevant information and thus would not show alpha ERD (i.e. would not invest attention) in anticipation of goal-relevant stimuli. Other individuals are low inhibitors; these individuals are more open to processing information even when goal-relevant information is not present. As a result, these individuals would display greater fluctuations of alpha power in the absence of goal-relevant information, and in anticipation of goal-relevant information.

While both the feedback-related N2 and alpha measures can be thought of as indicators of controlled cognition, they may capture different and independent demonstrations of controlled cognition. For example, variability in the responsivity to information regarding whether goals were achieved or not may indicate the degree to which individuals are concerned with achieving goals, but may not predict how effectively an individual employs cognitive control in order to achieve those goals. However, it does appear from the findings of Studies 1-3 of this dissertation that the stronger need for, and/or application of, cognitive control is associated with larger AB magnitudes.

Cognitive Control: A Unified Function or a Diversity of Controlled Cognitive Functions?

Originally in this dissertation cognitive control was conceptualized as functions related to optimizing information processing in the service of goals. This perhaps suggests that cognitive control is a unified function that directs and coordinates information processing functions (e.g. working memory, selective attention); in other words, that there is a cognitive functions which "controls" and optimizes these other cognitive functions. This conceptualization led to an idea that measures of cognitive control should relate to each other positively, such that indicators of more/stronger cognitive control would be associated with other indicators of more/stronger cognitive control. In the case of this dissertation, I predicted that stronger investment in feedback regarding whether goals were achieved (larger feedback-related N2) should be associated with greater investment of attention in goal-directed tasks (greater alpha ERD), and greater readiness to perform goal-directed tasks (resting alpha). These measures were not found to relate to each other in this way, suggesting that cognitive control is not a unified function. However, the purpose of this dissertation was not to find evidence that cognitive control is unified.

It is perhaps not useful to think of cognitive control as a unified function that controls and directs other cognitive functions (i.e. a homunculus). However, the original definition of cognitive control that I laid out in this dissertation, that cognitive control is information processing optimized for achieving goals, still holds without the need for a homunculus. Specifically, cognitive control is controlled cognition, or more specifically, controlled attention. In other words, cognitive control *is* the allocation of attention in such a way as to process relevant information such that it can influence behavior to be

consistent with goals, and a lack of cognitive control *is* the allocation of attention in such a way that irrelevant information is processed. So, for example, as task goals are served by greater anticipatory attentional investment (alpha ERD) then alpha ERD is a demonstration of cognitive control. Furthermore, as greater alpha ERD is associated with better goal-related behavior *in most* cases (i.e. correct T1 performance and correct T2 performance at long lags, see Study 2) then greater alpha ERD is an indicator of stronger cognitive control. It also happens that stronger cognitive control (greater alpha ERD) is detrimental to T2 performance at shorter lags. Similarly, task goals are also served by paying attention to feedback regarding whether performance is goal-consistent or not (feedback-related N2s), and keeping attention ready while at rest until goal-relevant information appears (resting alpha power). Thus, rather than being indicators of information processing which has been controlled by "cognitive control", these measures are indicators of control over attention. If this is the case, then it appears that individuals may differ in the strength of control of attention, but not consistently across all instances of controlled attention (i.e. alpha ERD, resting alpha, and feedback-related N2).

Replicability of Effects

Data were collected for Study 4 in order to examine hypothesized relationships among measures related to the AB in Studies 1-3. However, these data also provided an opportunity to attempt to replicate the relationships and effects observed in Studies 1-3. The relationship between feedback-related N2 amplitude and AB magnitude (such that greater feedback-related N2 amplitude was associated with larger AB magnitudes), appears to be a reliable finding, as it was replicated in the additional sample collected for Study 4.

The key findings of Study 2 were also replicated in the additional sample, although not exactly as was observed in Study 2. Specifically, the lag X T2 correct/incorrect interaction was significant only at right frontal sites. Right frontal sites were where alpha ERD was observed to be largest in both the original sample of Study 2. Similarly, in Study 2 it was observed that while greater alpha ERD was associated with worse T2 performance at the short lag, it was associated with better T2 performance at the long lag. In the additional sample the short lag effects were again significant, where greater alpha ERD was observed for incorrect trials than correct trials. However, while alpha ERD was larger on T2 correct than T2 incorrect trials when T2 was presented at a longer lag, this difference was no longer significant. That alpha ERD was greater prior to T1 correct than T1 incorrect trials at frontal sites as found in Study 2 was replicated in the additional sample. So, in general the effects of Study 2 appear reliable, however only at right frontal sites where alpha ERD is observed to be largest.

However, the relationship between alpha ERD and individual differences in AB magnitude, which was positive as hypothesized but non-significant in the original sample of Study 2, was not replicated in the additional or combined samples. Thus, the evidence presented in this dissertation does not support the hypothesis that individual differences in anticipatory attentional investment predict individual differences in AB magnitude. So, it appears that while anticipatory attentional investment does differ according to T2 performance differently according to lag (i.e. differs according to whether an AB occurs or not), it does not differ between individuals according to the magnitude of their AB.

The resting EEG and AB findings of Study 3 were not replicated in the additional sample. Despite being replicated already - the main findings of Study 3 were observed in

two separate samples as part of two separate studies - resting alpha and beta power were not correlated with AB magnitude, or any other AB performance measure in the additional sample. This was not due to power, as the sample size of the additional sample was larger than either of the original samples, and the effects were not observed in a combined sample as well. Nor does it appear that the descriptive characteristics of the measures differed for the different samples.

Why might the results of Study 3 have been observed twice in the main report, yet not be replicated with the Study 4 sample? The conditions for replicating the findings of Studies 1 and 2 in the additional sample were ideal as the procedures were closely replicated. The conditions for replicating the findings of Study 3, however, were less ideal, owing to more substantial differences in experimental procedures. Specifically, in both experiments of Study 3 resting EEG measures were taken repeatedly over an extended period of time. In the first experiment of Study 3 resting EEG measures were taken following each block of the AB task, for a total of five blocks. Thus resting EEG was measured five times in a period of approximately 1-1.5 hours, and resting theta, alpha, and beta power were averaged across these five times. In the second experiment of Study 3 resting EEG measures were taken twice, once before and once after the AB task, thus resting EEG was measured twice in a period of four hours. In contrast, however, resting EEG was measured only once in the additional sample from Study 4. Repeated measures over an extended period of time may yield a much more valid measure of trait-like individual differences in resting EEG power. Measuring only a single instance of resting EEG may not capture accurately the trait-like variability due to insufficient

sampling, and therefore may not be able to predict other trait-like measures such as AB magnitude.

It is also important to note that in the additional sample T1 accuracy on both AB tasks was poorer than typically observed, and lower than observed in either Study 2 or 3. This means that a substantial proportion of the data from the additional Study 4 sample was excluded due to; 1) the removal of several participants who failed to reach the minimum T1 accuracy criterion for inclusion, and 2) the removal of all T1 incorrect trials for those participants who remained in the sample. It is important to exclude T1 incorrect trials and data from individuals with very low T1 performance as in both cases there is no evidence that T1 had been attended to, and one would not expect an AB on trials where T1 was not attended. However, it is possible that the inter-individual variability in AB magnitude in the additional sample was altered systematically by the removal of data from individuals with poor T1 performance. This was not the case in Studies 2 and 3, where T1 performance was at typically high levels. It is possible, for example, that the unusually poor T1 performance in the additional sample is due to some systematic difference in the experimental procedure, e.g. greater fatigue, which alters variability in AB performance in such a way to eliminate the relationships previously observed in Studies 2 and 3. It is also possible that AB scores were less reliable for those who remained given that a substantial proportion of the trials were removed for many of those participants who remained in the sample. Note also, that these two alternatives are not mutually exclusive.

In summary[24], the findings of Study 1 and 2 were replicated in the additional

sample and thus appear to be reliable. The findings of Study 3, which were found in two

separate samples from two separate experiments, and thus were replicated before, were

not replicated in the additional sample. I suggest that this may be due to insufficient

sampling of resting EEG in order to capture trait variability, and/or differences in AB

performance variability. However it is also possible that the relationships observed in the

smaller samples of Experiments 1 and 2 of Study 3 were spurious, possibly Type I errors.

Concluding Summary

Selective attention serves goal-consistent behavior. Cognitive control is required

in order to direct selective attention, in order to optimize information processing to

achieve goals. Thus, in general strong cognitive control should lead to goal-consistent

behavior (i.e. to performing accurately). The attentional blink (AB) is a deficit in

performance to a second target observed when that target closely follows the first target

in time but not at longer lags (Raymond, Shapiro, & Arnell, 1992). Based on existing

cognitive control models of the AB (Di Lollo et al., 2005; Olivers & Meeter, 2008;

Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009), I proposed in this dissertation that

the AB is caused by the conditions of information processing determined by cognitive

control, such that cognitive control optimizes information processing for the benefit of

the first target, but at the cost of the second target. Thus, stronger cognitive control

should be associated with larger deficits (i.e. larger AB magnitudes). Following from the

overinvestment hypothesis of the AB (Olivers & Nieuwenhuis, 2006), in this dissertation

I examined whether greater investment of attention, proposed to be a result of the

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[24] In all cases it is possible that reliability, i.e. internal consistency, of the measures may have contributed to
whether effects were observed or whether observed effects were replicated. Internal consistency reliabilities
for all measures for Studies 1-4 are located in Appendices B.1-9.

cognitive control that contributes to the AB, predicted individual-to-individual and trial-to-trial differences in AB performance.

Study 1 found that the feedback-related N2, a measure related to the goal-management aspect of cognitive control, predicted AB magnitude, such that individuals who responded more strongly to feedback regarding whether goals had been met had larger AB magnitudes. Study 2 found that trials where an AB occurred were preceded by greater anticipatory investment of attention (i.e. greater alpha ERD) as compared to trials where no AB occurred, but alpha ERD did not predict individual differences in AB magnitude. Study 3 found that greater readiness (i.e. greater alpha power at rest) was associated with larger AB magnitudes. Thus, all three studies supported my general hypotheses that stronger cognitive control and greater investment of attention would be associated with larger AB magnitudes.

However, Study 4 found that the measure of cognitive control (feedback-related N2) was unrelated to either measure of investment of attention. Thus, my hypothesis that investment of attention was a result of cognitive control was not supported. Furthermore the measures of investment were negatively correlated with each other, such that greater readiness (resting alpha power) was associated with less anticipatory investment (alpha ERD). Based on more recent characterizations of alpha oscillations, however, I reconceptualized both alpha and alpha ERD as measures of cognitive control and suggested that the reason why feedback-related N2 was not related to the alpha measures (alpha ERD and resting alpha power) was because they represented independent aspects of cognitive control. Furthermore, that alpha ERD and alpha reflect different cognitive control strategies, that is that people tend to be either strict suppressors (high alpha

power) except in the presence of target information or more flexible suppressors (lower alpha power) who would not necessarily suppress all but target information, and may even respond to information more tangentially related to goals, e.g. a warning stimulus. This interpretation accommodates the finding of a negative relationship between resting alpha power and anticipatory alpha ERD, as well as evidence that greater baseline alpha is associated with larger alpha ERD to *target* information (Doppelmayr et al., 1998).

In general the findings of this dissertation support the hypothesis that cognitive control creates the conditions that lead to the AB and that stronger cognitive control, which would typically be associated with better performance, is associated with greater impairments to performance in the form of larger AB magnitudes. This conclusion provides a basis to explain individual differences in the AB, and is consistent with numerous individual difference studies showing that cognitive and dispositional measures associated with a flexible, diffuse and less invested processing style predict smaller ABs, while measures associated with a more invested and focused processing style show larger ABs (Arnell & Stubitz, 2010; Dale & Arnell, 2010; MacLean et al., 2010; MacLean & Arnell, 2010). This suggests that while cognitive control and selective attention may serve goal-consistent behaviors, under some conditions, for example when the optimal processing for one goal conflicts with the optimal processing of another, cognitive control and selective attention may be costly.

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A.1 Research Ethics Board Approval Letters

REB APPROVAL Study 2 & 3

DATE: September 8, 2008

FROM: Michelle McGinn, Chair

                   Research Ethics Board (REB)

TO: Dr. Karen M. Arnell, Psychology

                   Mary H. MacLean

FILE: 08-041 ARNELL/MACLEAN

TITLE: Brain Waves and Personality

The Brock University Research Ethics Board has reviewed the above research proposal.

DECISION: Accepted as clarified.

This project has received ethics clearance for the period of **September 8, 2008 to August 31, 2009** subject to full REB ratification at the Research Ethics Board's next scheduled meeting. The clearance period may be extended upon request. ***The study may now proceed.***

Please note that the Research Ethics Board (REB) requires that you adhere to the protocol as last reviewed and cleared by the REB.   During the course of research no deviations from, or changes to, the protocol, recruitment, or consent form may be initiated without prior written clearance from the REB. The Board must provide clearance for any modifications before they can be implemented. If you wish to modify your research project, please refer to http://www.brocku.ca/researchservices/forms to complete the appropriate form Revision or Modification to an Ongoing Application.

REB APPROVAL Study 1 & 4

Brock University
Research Ethics Office
Tel: 905-688-5550 ext. 3035
Email: reb@brocku.ca

Bioscience Research Ethics Board

Certificate of Ethics Clearance for Human Participant Research

DATE: 1/25/2012

PRINCIPAL INVESTIGATOR: ARNELL, Karen - Psychology

FILE: 11-146 - ARNELL

TYPE: Ph. D.   STUDENT: Mary MacLean
                  SUPERVISOR: Karen Arnell

TITLE: Investigations into autonomic measures of cognitive control and their relationship with the attentional blink

ETHICS CLEARANCE GRANTED

Type of Clearance: NEW   Expiry Date: 1/31/2013

The Brock University Bioscience Research Ethics Board has reviewed the above named research proposal and considers the procedures, as described by the applicant, to conform to the University's ethical standards and the Tri-Council Policy Statement. Clearance granted from 1/25/2012 to 1/31/2013.

The Tri-Council Policy Statement requires that ongoing research be monitored by, at a minimum, an annual report. Should your project extend beyond the expiry date, you are required to submit a Renewal form before 1/31/2013. Continued clearance is contingent on timely submission of reports.

To comply with the Tri-Council Policy Statement, you must also submit a final report upon completion of your project. All report forms can be found on the Research Ethics web page at http://www.brocku.ca/research/policies-and-forms/research-forms.

In addition, throughout your research, you must report promptly to the REB:
   a) Changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
   b) All adverse and/or unanticipated experiences or events that may have real or potential unfavourable implications for participants;
   c) New information that may adversely affect the safety of the participants or the conduct of the study;
   d) Any changes in your source of funding or new funding to a previously unfunded project.

We wish you success with your research.

Brock University
Research Ethics Office
Tel: 905-688-5550 ext. 3035
Email: reb@brocku.ca

Bioscience Research Ethics Board

Certificate of Ethics Clearance for Human Participant Research

DATE: 3/5/2013

PRINCIPAL INVESTIGATOR: ARNELL, Karen - Psychology

FILE: 11-146 - ARNELL

TYPE: Ph. D.   STUDENT: Mary MacLean
                                          SUPERVISOR: Karen Arnell

TITLE: Investigations into autonomic measures of cognitive control and their relationship with the attentional blink

ETHICS CLEARANCE GRANTED

Type of Clearance: RENEWAL   Expiry Date: 3/31/2014

The Brock University Bioscience Research Ethics Board has reviewed the above named research proposal and considers the procedures, as described by the applicant, to conform to the University's ethical standards and the Tri-Council Policy Statement. Clearance granted from **3/5/2013 to 3/31/2014**.

The Tri-Council Policy Statement requires that ongoing research be monitored by, at a minimum, an annual report. Should your project extend beyond the expiry date, you are required to submit a Renewal form before **3/31/2014**. Continued clearance is contingent on timely submission of reports.

To comply with the Tri-Council Policy Statement, you must also submit a final report upon completion of your project. All report forms can be found on the Research Ethics web page at
http://www.brocku.ca/research/policies-and-forms/research-forms.

In addition, throughout your research, you must report promptly to the REB:
   a)   Changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
   b)   All adverse and/or unanticipated experiences or events that may have real or potential unfavourable implications for participants;
   c)   New information that may adversely affect the safety of the participants or the conduct of the study;
   d)   Any changes in your source of funding or new funding to a previously unfunded project.

We wish you success with your research.

A.2 Consent Forms

CONSENT FORM Study 2 & 3

Consent to Participate

Brain Waves and Personality

Fall/Winter 2008

Principal Investigator:	Faculty Supervisor:
Mary MacLean, MA candidate	Dr. Karen Arnell, Advisor
Department of Psychology	Department of Psychology
Brock University	Brock University
(905) 688-5550 Ext. 5872	(905) 688-5550 Ext. 3225
mmaclean@brocku.ca	karnell@brocku.ca

INVITATION

You are invited to participate in a psychological investigation. The purpose of this investigation is to examine the relationship between various individual differences and performance on a cognitive task using brain waves as a measure of that interaction.

BASIS FOR PARTICIPANT SELECTION

Our experiment requires you to look at visual words and letter strings that will be rapidly presented on the computer screen. Persons who have poor visual acuity so that they are unable to read fairly large words on the computer screen, or persons who learned English after the age of 9 will be unable to participate in the experiment. Please tell the experimenter now if these apply to you. Also, only those without known neurological conditions or history of neurological damage should perform the experiment. Neurological conditions we would be concerned about include epilepsy, previous coma or extended period of unconsciousness, previous stroke, known malformations of the brain, difficulty perceiving shapes/forms, or difficulty understanding spoken speech or sounds. You do not have to provide any specifics about your neurological condition, or which condition you have, but please tell the experimenter now if you have a neurological condition. Also, if you are left-handed you can still run the experiment, but please tell the experimenter now so we can note this in our records. We plan to include about 20 people in each of our experiments like this one, all of whom have these characteristics.

EXPLANATION OF PROCEDURES

As a participant, you will be asked to first complete a questionnaire asking some questions about your daily habits and any medications or health-related issues you may have. You will also be asked to fill out several other questionnaires that provide statements and ask you to indicate how well they represent you, or will provide you with several adjectives and ask you to indicate whether they represent how you feel. Following this an electrode cap will be placed on your head. The cap is snug fitting, and we will need to squirt some gel onto each electrode to help us get good measurements of your brain waves. This means your hair will get some gel in it, and you will feel some slight scratching on your scalp as we put the gel into the electrodes. We will not break the skin or scratch hard enough for you to feel any discomfort. If you are ever uncomfortable while we are setting-up the electrodes, then please tell us right away. It will take us about half an hour to get the cap fitted properly, and to make sure we are getting good brain-wave signals from each electrode. The brain waves we will record are just like EEG recordings done in hospitals. The electrodes only measure electrical activity in your brain; they will not emit signals into your brain, and will not shock or harm you. The electrodes will just sit onto of your head, they will not be inserted into your head. We will be able to monitor your brain waves, but we will not be able to "read your mind", tell what you are thinking, or how you are feeling.

While we record your brain waves you will perform a computer task that requires you to observe items on a screen and respond to specific items by pressing buttons on a keyboard. The task will last approximately 2 hours, and you will be given 5 minute breaks at regular intervals. After you have completed the computer task we will ask you to fill out another questionnaire about your mood and personality. You will then be given your compensation for participation. Participation will take approximately 3.5 to 4 hours of your time.

POTENTIAL BENEFITS AND RISKS

This research will help us develop our understanding of how our brains focus attention, while attempting to make sense of information presented very quickly. If you are curious, after the experiment we can show you the types of brain activation we are examining, and what your brain waves look like when recorded by the computer. If you are not familiar with cognitive neuropsychology, then the experiment will expose you to a new area of psychology.

In order to ensure that we get a good signal from each of the electrodes, the cap needs to be snug fitting, and electrolytic gel solution needs to be applied to the electrodes. After wearing the cap for a couple of hours, you may feel that the cap feels somewhat tight. While we are putting the gel into each electrode you may feel some slight temporary scratching on your scalp. The gel will come in contact with your skin, but is safe and harmless unless you have skin allergies or sensitivities. If you are concerned

about the gel aggravating your skin allergies or sensitivities, you should not participate in this study. The study is a fairly long, and we encourage you to walk around and stretch when needed. You may experience mild fatigue while performing the trials. Feel free to take a short break whenever you require one. If you are uncomfortable with performing one or more of the tasks, or answering one or more of the questions, then please just make this clear to the experimenter, and you will be allowed to omit that portion of the experiment.

The electrode cap and individual electrodes are cleaned thoroughly with detergent after each use, and disinfected periodically. A new syringe used to squirt gel into the electrodes is used with each participant and the blunt end of the syringe will not touch any surface other than the cap, the electrodes and the participants scalp.

CONFIDENTIALITY

The information we collect from you in this study (your responses and brain activity records) will be coded by a number, not your name. Your identity will not be revealed or connected with the experimental results. We are interested in combining data from all of the participants, not in separately examining the pattern for each person. Your data will be combined with the data from other participants, and reported in summary form. Data and records created by this project are the property of the University and the investigator. You may have access to the overall results of the experiment by making a written request to Dr. Karen Arnell (Department of Psychology, Brock University, St. Catharines, ON, L2S 3A1). A copy of the summary results will then be sent to you when the experiment has been completed. This right of access extends only to the data combined from all participants, and not to your individual data nor the individual data of other participants.

VOLUNTARY PARTICIPATION

Your participation is voluntary, and you may withdraw from the study at any time without penalty, loss of credits or compensation (you will still receive 2 participation hours and $20.00, or $40.00). Your decision of whether or not to participate will not affect your course grades or your eligibility for other studies. If you decide to participate now, you are free to withdraw your consent and to discontinue participation at any time.

PUBLICATION OF RESULTS

Results of this study may be published in professional journals and presented at conferences. Feedback about this study will be available from the faculty supervisor approximately 9 months after completion, and can be obtained by contacting them through e-mail at karnell@brocku.ca or by phone at (905) 688-5550 ext. 3225.

CONTACT INFORMATION AND ETHICS CLEARANCE

If you have any questions about this study or require further information, please contact the Principal Investigator or the Faculty Supervisor using the contact information provided above. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University (file #08-041). If you have any comments or concerns about your rights as a research participant, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, reb@brocku.ca.

Thank you for your assistance in this project. Please keep a copy of this form for your records.

CONSENT FORM

I agree to participate in this study described above. I have made this decision based on the information I have read in the Information-Consent Letter. I have had the opportunity to receive any additional details I wanted about the study and understand that I may ask questions in the future. I understand that I may withdraw this consent at any time.

Name (Please print): _____

Signature: _____

Date: _____

COMPENSATION

Please complete *one* of the following options:

(1)	I agree to participate in this study for $40.00, and understand that I will not receive participation hours.

Name (Please print): _____

Signature: _____

Date: _____

OR

(2)	I agree to participate in this study for 2 hours of participation and $20.00.

Name (Please print): _____

Signature: _____

Date: _____

Informed Consent Form

Principal Student Investigator: **Faculty Supervisor:**
Mary MacLean, PhD Candidate   Dr. Karen Arnell, Professor
Department of Psychology   Department of Psychology
(905) 688-5550 x5872, mm07fi@brocku.ca   (905) 688-5550 x3225,
karnell@brocku.ca

INVITATION

You are invited to participate in this study. The purpose of this study is to investigate
individual differences in attention by examining brain and heart activity.

BASIS FOR PARTICIPANT SELECTION

Our experiment requires you to look at visual words and letter strings that will be rapidly
presented on the computer screen. Persons who have poor visual acuity so that they are
unable to read fairly large words on the computer screen, or persons who learned English
after the age of 9 will be unable to participate in the experiment. Please tell the
experimenter now if these apply to you. Also, only those without known neurological
conditions or history of neurological damage should perform the experiment.
Neurological conditions we would be concerned about include epilepsy, previous coma
or extended period of unconsciousness, previous stroke, known malformations of the
brain, difficulty perceiving shapes/forms, or difficulty understanding spoken/written
speech or sounds. Participants also must not have any physiological conditions that
influence their cardiac and/or circulatory functions (e.g. heart arrhythmia). You do not
have to provide any specifics about your neurological or physiological condition, or
which condition you have, but please tell the experimenter now if you have any such
neurological or physiological condition described above. Participants in this study must
not be taking any psychoactive medication (e.g. anti-anxiety, anti-depressants, Adderall,
etc.), or medication to regulate blood pressure or heart function (e.g. beta-blockers).
Again, you do not need to provide any details about medication you may be taking but
please tell the experimenter now if you are taking any such medications described above.
Also, if you are left-handed you can still run the experiment, but please tell the
experimenter now so we can note this in our records.

WHAT'S INVOLVED

As a participant, you will be asked to indicate your emotional state and personality. You
will also be asked to perform two computer tasks. One computer task will require you to
view and respond to words presented on the screen at a fast pace. The other computer
task will require you to estimate 1 second time intervals. Brain activity will be recorded
using an electrode cap placed on your head and heart activity will be recorded using

electrodes attached to your torso. The electrode cap is tight fitting, and we will need to insert some gel onto each electrode to help us get good measurements of your brain waves. This means your hair will get some gel in it, and you will feel some slight scratching on your scalp as we put the gel into the electrodes. The brain waves we will record are just like EEG recordings done in hospitals. The electrodes only measure electrical activity in your brain; they will not emit signals into your brain, and will not shock or harm you. The electrodes will just sit on top of your head they will not be inserted into your head. We will be able to monitor your brain waves, but we will not be able to "read your mind", tell what you are thinking, or how you are feeling. Participation will take approximately 3 hours of your time.

POTENTIAL BENEFITS AND RISKS
This research will help us to understand individual differences in attention and limits of attention by examining brain and heart activity. After you complete your participation you will be provided with a more detailed description of exactly what individual differences we are interested in and why we use these tasks and questionnaires to study this. If you are not familiar with cognitive, affective psychology, then this study will expose you to a new area of psychology. It is also a chance to experience a psychological experiment first-hand.

There also may be risks associated with participation. The electrode cap is tight fitting and can become uncomfortable. In order to record brain activity we will need to squirt electrode gel into the electrode cap. In order to do this we use a blunt ended, large gauge syringe. This syringe cannot pierce your skin although it is possible for the syringe to lightly scratch your scalp. If the use of the syringe should irritate you at all please inform the experimenter immediately. In order to record heart activity it is necessary for us to apply electrodes to your torso. One electrode will be placed just below your right collarbone on your chest, between the last two ribs on the side of your torso, at the base of your neck (back and front), on your stomach in the middle just below where your ribcage ends, and one on your lower back over your spine (see the picture below to that indicates where electrodes will be placed).
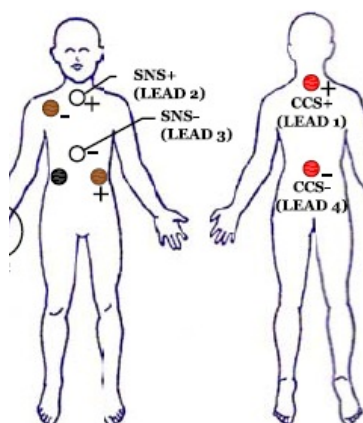
In order to apply these electrodes it is necessary for us to lift your shirt high enough to reveal the bottom of your ribcage. We may also need to move your top aside enough to place the electrodes just below your collarbone and between your shoulder blades. The application of these electrodes will take place in a private room with only the experimenter present. If you are uncomfortable with this procedure at any point please inform the experimenter immediately. It is also possible that the rapid presentation of visual stimuli could induce seizures in some people. If you have ever had seizures, or are on medication that puts you at risk of seizures, you should not participate in this study. If you experience discomfort during the rapid presentation of the visual stimuli you should stop your participation and inform the experimenter immediately.

The study is a fairly long, and we encourage you to walk around and stretch when needed. You may experience mild fatigue while performing the trials. Feel free to take a short break whenever you require one. If you are uncomfortable with or have questions about any of the risks described above please inform the experimenter now.

CONFIDENTIALITY
All information you provide is considered confidential; your name will not be included or, in any other way, associated with the data collected in the study. Furthermore, because our interest is in the average responses of the entire group of participants, you will not be identified individually in any way in written reports of this research. Data collected during this study will be stored either in a locked cabinet or password-protected computer inside an alarmed lab on campus. Data will be kept for 10 years after which time all data will be destroyed. Access to this data will be restricted to the primary investigators and a research assistant.

VOLUNTARY PARTICIPATION
Participation in this study is voluntary. If you wish, you may decline to answer any questions or participate in any component of the study. Further, you may decide to

withdraw from this study at any time and may do so without any penalty or loss of benefits to which you are entitled. However as the data we collect is anonymous once the data is submitted it cannot be withdrawn.

PUBLICATION OF RESULTS

Results of this study may be published in professional journals and presented at conferences. Feedback about this study will be available in eight months from Dr. Karen Arnell.

CONTACT INFORMATION AND ETHICS CLEARANCE

If you have any questions about this study or require further information, please contact the Principal Investigator or the Faculty Supervisor (where applicable) using the contact information provided above. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University (11-146). If you have any comments or concerns about your rights as a research participant, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, reb@brocku.ca.

CONSENT

I, (print) _____, agree to participate in this study described above. I have made this decision based on the information I have read in the Informed Consent Letter. I have had the opportunity to receive any additional details I wanted about the study and understand that I may ask questions in the future. I understand that I may withdraw this consent at any time.

Participant Signature: _____

Date: _____

PARTICIPATION

I, (print) _____, agree to participate in this study described above for four (4) hours of participation credit.

Participant Signature: _____

Date: _____

Researcher's Signature: _____

A.3 Demographic and Participant Information Questionnaire

QUESTIONNAIRE Study 2 & 3

Sex: **M / F**

Date of Birth (MM/DD/YY):

Handedness: **L / R**

Is English your **first** language? **Y / N**

Do you have any **uncorrected** vision or hearing impairments? **Y / N**

Have you ever suffered mild to severe head trauma (e.g. concussion, loss of consciousness)?

Y / N

If 'yes', how long ago? _____

Have you ever been diagnosed with a perceptual or cognitive impairment (e.g. ADD/ADHD, epilepsy, color-blindness, etc.)?

Y / N

Have you ever been diagnosed with an affective disorder (e.g. depression, bipolar, etc.)?

Y / N

Are you currently taking any medication(s)? **Y / N**

If yes, what medication(s)?

___

Do you consume nicotine? **Y / N**

In what form do you usually consume nicotine (e.g. cigarettes, chewing tobacco, etc.)?

___

How often to you consume nicotine?

Never   1-2/Month   1-2/Week   3+/Week   Daily

When did you last consume nicotine?

_____

Do you consume caffeine? **Y / N**

In what form do you usually consume caffeine (e.g. coffee, tea, cola, etc.)?

_____

How often to you consume caffeine?

Never   1-2/Month   1-2/Week   3+/Week   Daily

When did you last consume caffeine?

_____

How many hours of uninterrupted sleep do you **typically** get on weekdays?

_____

How many hours of uninterrupted sleep did you get **last** night?

_____

QUESTIONNAIRE Study 1 & 4

Demographic Information

Sex (circle one):

Male   Female

Age: _____ years old

Handedness (circle one):

Left-handed   Right-handed

Are you wearing contact lenses right now?

Y   N

Are you currently taking any psychoactive medications (e.g. Adderal, anti-anxiety, etc.)? (circle one)

Y   N

Are you currently taking and medications to regulate your heart function or blood pressure (e.g. beta-blockers)? (circle one)

Y   N

Do you have any perceptual or cognitive impairments (e.g. colour-blindness, difficulty reading or comprehending written words, etc.)? (circle one)

Y   N

Do you have any conditions that influence your heart or circulatory functions? (circle one)

Y   N

Appendix B.1

Correlation Table for Primary Analysis Variables – Study 1 (Chapter 2)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
Attentional blink													
1. AB magnitude (residual)	**.55**												
ERN-related measures (Flanker task)													
2. MFNresid-correct	-.19	**.78**											
3. MFNresid-incorrect	.09	.01	**-.03**										
4. MFNresid-diff(ERN)	.22	-.77*	.63*	**.38**									
FRN-related measures (TIME task)													
5. N2.time-resid-corr	-.31*	.02	.13	.06	**.78**								
6. N2.time-resid-incor	-.25	.18	.31	.06	.71*	**.67**							
7. N2.time-resid-diff(FRN.time)	.08	.21	.24	-.01	-.38*	.38*	**.39**						
FRN-related measures (AB task)													
8. N2.AB-resid-corr	-.39*	.21	.27	.02	.73*	.63*	-.13	**.93**					
9. N2.AB-resid-incor	-.26	.32	.26	-.07	.65*	.67*	.02	.86*	**.75**				
10. N2.AB-resid-diff(FRN.AB)	.25	.22	-.03	-.18	-.15	.06	.27	-.27*	.27*	**-.15**			
FRN-related measures (combined)													
11. N2.combo-resid-corr	-.38*	.12	.22	.05	.93*	.72*	-.27*	.93*	.82*	-.22	**.91**		
12. N2combo-resid-incor	-.29*	.26	.31	-.00	.75*	.92*	.23	.82*	.92*	.19	.84*	**.79**	
13. N2.combo-resid-diff(FRN.combo)	-.21	.27	.17	-.09	-.35*	.32*	.87*	-.23	.16	.72*	-.29*	.27*	**.21**

Note: Sample sizes ranged across correlations from 36 to 54 participants. Bolded coefficients are split-half (even vs. odd trial) reliabilities. Significance of reliabilities is not indicated.

Appendix B.2

Correlation Table for Primary Analysis Variables – Chapter 2 replication sample

Variable	1	2	3	4	5	6	7	8	9	10
Attentional blink										
1. AB magnitude (residual)	**.31**									
FRN-related measures (TIME task)										
2. N2.time-resid-corr	-.52*	**.31**								
3. N2.time-resid-incor	-.02	.51*	**.49**							
4. N2.time-resid-diff(FRN.time)	.51*	-.49*	.49*	**.09**						
FRN-related measures (AB task)										
5. N2.AB-resid-corr	-.49*	.57*	.31	-.26	**.52**					
6. N2.AB-resid-incor	-.38*	.49*	.34	-.16	.88*	**.62**				
7. N2.AB-resid-diff(FRN.AB)	.22	-.17	.06	.23	-.24	.24	**.20**			
FRN-related measures (combined)										
8. N2.combo-resid-corr	-.56*	.88*	.46*	-.43	.89*	.78*	-.23	**.46**		
9. N2combo-resid-incor	-.26	.62*	.81*	.19	.73*	.83*	.21	.76*	**.63**	
10. N2.combo-resid-diff(FRN.combo)	.47*	-.48*	.43*	.92*	-.31	-.01	.62*	-.43*	.26	**.24**

Note: Sample sizes ranged across correlations from 22 to 24 participants. Bolded coefficients are split-half (even vs. odd trial) reliabilities. Significance of reliabilities is not indicated.

Appendix B.3

Correlation Table for Primary Analysis Variables –Chapter 3, Study 2

Variable	1	2	3	4	5
Attentional blink					
1. T2 performance – short lag	**.94**				
2. T2 performance – long lag	.67*	**.82**			
3. AB magnitude (residual)	-.73*	.00	**.86**		
Alpha ERD-related measures					
4. alpha ERD (average, all ROIs)	-.29	-.21	.26	**.93**	
5. alpha ERD (right/frontal sites only)	-.44	-.26	.41	.91*	**.93**

Note: *n* = 21; Bolded coefficients are split-half (even vs. odd trial) reliabilities. Significance of reliabilities is not indicated.

Appendix B.4

Correlation Table for Primary Analysis Variables – Chapter 3, replication sample

Variable	1	2	3	4	5
Attentional blink					
1. T2 performance – short lag	**.92**				
2. T2 performance – long lag	.62*	**.92**			
3. AB magnitude (residual)	-.79*	.00	**.80**		
Alpha ERD-related measures					
4. alpha ERD (averaged, all ROIs)	.04	-.01	-.07	**.91**	
5. alpha ERD (right/frontal sites only)	-.06	.05	.12	.79*	**.90**

Note: *n* = 40; Bolded coefficients are split-half (even vs. odd trial) reliabilities. Significance of reliabilities is not indicated.

Correlation Table for Primary Analysis Variables – Chapter 4, Study 3, Experiment 1

Variable	1	2	3	4	5	6	7	8	9	10
Attentional blink										
1. T2 performance short lag	**.94**									
2. T2 performance long lag	.53*	**.88**								
3. AB-difference score	-.95*	-.23	**.87**							
4. AB-difference.residual	-.93*	-.17	.99*	**.85**						
Resting EEG measures										
5. resting alpha (all ROIs)	-.68*	-.28	.69*	.68*	**.69**					
6. resting beta (all ROIs)	.50*	.25	-.49*	-.48*	-.58*	**.72**				
7. resting theta (all ROIs)	.07	.11	-.03	-.03	-.19	-.43*	**.55**			
8. resting alpha (frontal sites)	-.68*	-.21	.72*	.72*	.92*	-.52*	-.11	**.71**		
9. resting beta (frontal sites)	.39*	.19	-.39*	-.38	-.51*	.89*	-.39*	-.48*	**.57**	
10. resting theta (frontal sites)	.20	.12	-.19	-.16	-.19	-.34	.82*	-.22	-.49*	**.43**

Note: *n* = 27; Bolded coefficients are split-half (even vs. odd trials for AB performance; first 1/3rd of recording session vs. second 1/3rd of recording session for EEG) reliabilities. Significance of reliabilities is not indicated.

Correlation Table for Primary Analysis Variables – Chapter 4, Study 3, Experiment 2

Variable	1	2	3	4	5	6	7	8	9	10
Attentional blink										
1. T2 performance short lag	x									
2. T2 performance long lag	.78*	x								
3. AB-difference score	-.58*	.06	x							
4. AB-difference.residual	-.63*	.00	.99*	x						
Resting EEG measures										
5. resting alpha (all ROIs)	-.31	-.00	.49*	.49*	**.92**					
6. resting beta (all ROIs)	.08	-.02	-.16	-.16	-.33	**.94**				
7. resting theta (all ROIs)	-.07	-.27	-.25	-.23	-.57*	-.22	**.87**			
8. resting alpha (frontal sites)	-.37*	-.17	.37*	.38*	.88*	-.25	-.43*	**.90**		
9. resting beta (frontal sites)	.43*	.22	-.39*	-.41*	-.31	.82*	-.24	-.28	**.90**	
10. resting theta (frontal sites)	-.03	-.14	-.13	-.13	-.65*	-.01	.78*	-.72*	-.19	**.86**

Note: *n* = 29; Bolded coefficients are between session (9:00 am session vs. 1:00 pm session) reliabilities. Significance of reliabilities is not indicated.

Appendix B.7

Correlation Table for Primary Analysis Variables – Chapter 4, Replication study

Variable	1	2	3	4	5	6	7	8	9	10
Attentional blink										
1. T2 performance short lag	**.92/.58**									
2. T2 performance long lag	.63*/.39*	**.86/.09**								
3. AB-difference score	-.78*/-.86*	.00/.13	**.76/.31**							
4. AB-difference.residual	-.89*/-.92*	-.19/.00	.98*/.99*	**.78/.52**						
Resting EEG measures										
5. resting alpha (all ROIs)	-.05/.06	-.07/.16	.00/.02	.02/.00	**.90**					
6. resting beta (all ROIs)	-.08/-.09	-.06/.08	.05/.15	.06/.14	-.29*/-.25*	**.77**				
7. resting theta (all ROIs)	.08/-.22	-.01/-.22	-.10/.11	-.09/.14	-.53*/-.51*	-.18/-.24*	**.86**			
8. resting alpha (frontal)	.02/-.01	-.04/.10	-.06/.07	-.05/.05	.92*/.93*	-.16/-.16	-.41*/-.38*	**.90**		
9. resting beta (frontal)	-.04/-.06	.02/.09	.06/.11	.06/.10	-.31*/-.32*	.90*/.89*	-.15/-.17	-.20/-.34*	**.73**	
10. resting theta (frontal)	-.05/-.18	-.12/-.21	-.03/.08	-.01/.11	-.50*/-.48*	-.17/-.22	.90*/.93*	-.48*/-.44*	-.20/-.23	**.82**

Note: AFC/Detection; *n* = 89/70; Bolded coefficients are split-half (even vs. odd trials for AB performance; first 1/3rd of recording session vs. second 1/3rd of recording session for EEG) reliabilities. Significance of reliabilities is not indicated.

Correlation Table 1 for Chapter 5 – Correlations Among AB measures

Variable	1	2	3	4	5	6	7	8	9
1. T2 performance – short lag, detection task	**.58**								
2. T2 performance – long lag, detection task	.43*	**.09**							
3. AB magnitude (residual), detection task	-.90*	.00	**.52**						
4. T2 performance – short lag, AFC task	.35	.23	-.27	**.92**					
5. T2 performance – long lag, AFC task	.07	.31	.10	.68*	**.86**				
6. AB magnitude (residual), AFC task	-.39*	-.08	.39*	-.73*	.00	**.78**			
7. T2 performance – short lag, combined tasks	.88*	.38*	-.79*	.91*	.61*	-.68*	**.75**		
8. T2 performance – long lag, combined tasks	.35*	.89*	.04	.63*	.91*	-.01	.59*	**.48**	
9. AB magnitude (residual), combined tasks	-.78*	.07	.90*	-.66*	-.03	.89*	-.80*	.00	**.65**

Note: Sample sizes ranged across correlations from 29 to 37 participants. Bolded coefficients are split-half (even vs. odd trial) reliabilities. Significance of reliabilities is not indicated.

Correlation Table 2 for Chapter 5 – Correlations Among Cognitive Control / Attentional Investment Variables

Variable	10	11	12	13	14	15	16	17	18	19	20	21	22	23
10. N2.time-resid.correct (TIME task)	**.31**													
11. N2.time-resid-incor (TIME task)	.56*	**.49**												
12. N2.time-resid.correct (detection task)	.30*	.18	**.52**											
13. N2.time-resid-incor (detection task)	.47*	.39*	.59*	**.62**										
14. N2.time-resid.correct (combined tasks)	.68*	.37*	.94*	.57*	**.46**									
15. N2.time-resid-incor (combined tasks)	.55*	.65*	.57*	.95*	.59*	**.63**								
16. Alpha-ERD (averaged, all ROIs)	.11	.17	.05	.21	.02	.21	**.91**							
17. Alpha-ERD (right/frontal sites only)	.28	.00	.07	.19	.08	.16	.79*	**.90**						
18. Resting alpha (all ROIs)	-.22	-.11	.19	-.01	.12	-.05	-.35*	-.39*	**.90**					
19. Resting beta (all ROIs)	-.12	-.21	-.14	-.33*	-.09	-.31*	.07	.06	-.21	**.77**				
20. Resting theta (all ROIs)	.13	.02	-.14	.05	-.13	.05	.19	.18	-.54*	-.25*	**.86**			
21. Resting alpha (frontal sites)	-.23	-.18	.12	-.11	.06	-.12	-.31*	-.34	.89*	-.12	-.32*	**.90**		
22. Resting beta (frontal sites)	-.18	-.24	-.17	-.34*	-.14	-.32*	.11	.10	-.24	.93*	-.23	-.17	**.73**	
23. Resting theta (frontal sites)	.21	.21	-.00	.22	.00	.22	.26	.14	-.51*	-.23	.86*	-.44*	-.27*	**.82**

Note: Sample sizes ranged across correlations from 49 to 63 participants. Bolded coefficients are split-half (even vs. odd trial for N2s and alpha ERD; first 1/3rd of recording session vs. second 1/3rd of recording session for resting EEG) reliabilities. Significance of reliabilities is not indicated.

Appendix B.10

Correlation Table 3 for Chapter 5 – Correlations Attentional Blink Variables and Cognitive Control / Attentional Investment Variables

Cognitive control / Investment variables	Attention Blink variables (see Table 1 for variable labels)								
	1	2	3	4	5	6	7	8	9
10. N2.time-resid.correct (TIME task)	.22	-.32	-.39*	-.25	-.15	.23	-.09	-.22	-.05
11. N2.time-resid-incor (TIME task)	-.06	-.14	-.00	-.25	-.09	.29	-.17	-.16	.09
12. N2.time-resid.correct (detection task)	.33*	-.14	-.43*	.13	.16	-.03	.28*	.14	-.24
13. N2.time-resid-incor (detection task)	.19	-.28	-.34*	-.06	-.09	-.01	.07	-.15	-.20
14. N2.time-resid.correct (combined tasks)	.33*	-.21	-.47*	.14	.19	-.01	.27*	.17	-.21
15. N2.time-resid-incor (combined tasks)	.15	-.29	-.31	-.01	-.07	-.04	.11	-.14	-.24
16. Alpha-ERD (averaged, all ROIs)	.27	.39*	-.12	.04	-.01	-.06	.04	.12	.04
17. Alpha-ERD (right/frontal sites only)	.20	-.00	-.23	-.05	-.08	.00	-.05	-.10	-.02
18. Resting alpha (all ROIs)	-.06	.19	.16	.04	.03	-.02	.08	.11	-.02
19. Resting beta (all ROIs)	.01	.07	.02	.11	.07	-.09	.04	.08	.01
20. Resting theta (all ROIs)	-.06	-.15	-.01	-.08	-.13	-.02	-.14	-.16	.06
21. Resting alpha (frontal sites)	.14	.19	.25	.10	.08	-.07	.08	.14	.01
22. Resting beta (frontal sites)	.13	.08	-.11	.14	.10	-.09	.11	.11	-.06
23. Resting theta (frontal sites)	-.06	-.18	-.02	-.28*	-.29*	.11	-.27*	-.31*	.10

Note: Sample sizes ranged across correlations from 25 to 50 participants.

Appendix C.1

Results from additional multiple regression analysis – Chapter 2, Study 1

Predictors	Criterion = AB magnitude (resid difference)		
	Step 1	Step 2	Step 3
MFNresid.correct	-.10	.01	-.01
MFNresid.incorrect	.19	.36	.41
N2.TPtask.resid.correct		-.03	.07
N2.TPtask.resid.incorrect		-.39	-.44
N2.ABtask.resid.correct			-.40
N2.ABtask.resid.incorrect			.41
Model R^2	.04	.19	.25
Change in model R^2	.04	.16	.06

Note: unstandardized beta weights; $n = 34$

Appendix C.2

Results from additional multiple regression analysis – Chapter 2, replication sample

Predictors	Criterion = AB magnitude (resid difference)	
	Step 1	Step 2
N2.TPtask.resid.correct	.69*	-.51
N2.TPtask.resid.incorrect	.33	.33
N2.ABtask.resid.correct		-.41
N2.ABtask.resid.incorrect		.13
Model R^2	.36*	.43
Change in model R^2	.36*	.07

Note: unstandardized beta weights; $n = 22$

Appendix C.3

**Results from additional multiple regression analysis – Chapter 4, Study 1
Experiment 1**

Predictors	Criterion = AB magnitude (resid difference)	
	Step 1	Step 2
Resting alpha	.58*	.55*
Resting beta	-.09	-.11
Resting theta	.03	.06
Alpha by beta interaction		.04
Alpha by theta interaction		.07
Theta by beta interaction		.08
Model R^2	.49*	.50
Change in model R^2	.49*	.01

Note: unstandardized beta weights; $n = 28$

Appendix C.4

**Results from additional multiple regression analysis – Chapter 4, Study 1
Experiment 2**

Predictors	Criterion = AB magnitude (resid difference)	
	Step 1	Step 2
Resting alpha	.63*	.72*
Resting beta	.05	.14
Resting theta	.12	.31
Alpha by beta interaction		.44
Alpha by theta interaction		-.07
Theta by beta interaction		.44
Model R^2	.24	.34
Change in model R^2	.24	.09

Note: unstandardized beta weights; $n = 29$

Appendix C.5

Results from additional multiple regression analysis – Chapter 4, Replication study

Predictors	Criterion = AB magnitude (resid difference)	
	Step 1	Step 2
Resting alpha	-.08	-.12
Resting beta	.03	.08
Resting theta	-.06	-.10
Alpha by beta interaction		.07
Alpha by theta interaction		-.14
Theta by beta interaction		.07
Model R^2	.01	.03
Change in model R^2	.01	.02

Note: unstandardized beta weights; $n = 89$

Appendix C.6

Results from additional multiple regression analysis – Chapter 5

Predictors	Criterion = residual AB magnitude		
	Step 1	Step 2	Step 3
N2.TPtask.resid.correct	-.43/.11	-.15/.15	-.12/.26
N2.TPtask.resid.incorrect	.31-.29	.04/.17	.10/-.03
N2.ABtask.resid.correct	-.51/.25	-.62/.30	-.57/.29
N2.ABtask.resid.incorrect	.31/-.26	.39/-.16	.33/-.11
Alpha ERD	-.16/.05	-.05/.01	-.01/.13
Resting alpha	.09/-.22	.39/-.02	.42/.05
Resting beta	-.01/-.16	-.48/.06	-.64/.10
Resting theta	.03/-.15	.06/-.21	.05/-.27
Alpha by beta interaction		.05/.29	-.27/.42
Alpha by theta interaction		.41/-.07	.56/-.03
Theta by beta interaction		-.48/-.30	-.49/-.39
Alpha ERD x resting alpha			-.16/-.32
Alpha ERD x resting beta			-.43/.32
Alpha ERD x resting theta			-.15/-.19
Model R^2	.30/.19	.41/.33	.47/.39
Change in model R^2	.30/.19	.11/.13	.06/.07

Note: unstandardized beta weights; AB detection/AB AFC; $n = 25/31$; * $p < .05$

Appendix C.7

More results from additional multiple regression analysis – Chapter 5

Predictors	Criterion			
	Alpha ERD	Resting alpha	Resting beta	Resting theta
N2.TPtask.resid.correct	.02	-.14	.06	.07
N2.TPtask.resid.incorrect	.07	-.08	-.14	.09
N2.ABtask.resid.correct	-.03	.24	-.07	-.06
N2.ABtask.resid.incorrect	.21	-.19	-.29	.22
Model R^2	.06	.08	.15	.08
Change in model R^2	.06	.08	.15	.08

Note: unstandardized beta weights; $n = 45$-50