ABSTRACT

Title of Dissertation: DYNAMIC CHANGES IN FEEDBACK PROCESSING AND COGNITIVE CONTROL NETWORKS DURING A GAMBLING TASK: A SOURCE ANALYSIS OF EEG AMPLITUDE AND PHASE MEASURES

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A wealth of cognitive and clinical psychology research has been devoted to better understanding the mechanism underlying goal-directed behavior. Decades of research in this area have highlighted the importance of salience and cognitive control networks in processing task feedback for improving goal-directed performance on subsequent trials. However, discrepancies in findings and methodological limitations have led to competing conceptual models of task performance with untested theoretical hypotheses. The current study aimed to evaluate a leading integrative theory of task performance (the Expected Value of Control theory), by assessing activity and functional connectivity of ACC (indexing salience) and dlPFC (indexing control) regions on the timescale of milliseconds. Time-frequency event-related potential (TF-ERP) measures were assessed in the theta (3-7 Hz) frequency range based on cortical source localization analysis. Hypotheses centered on the theory that ACC activity precedes engagement of the dlPFC and cognitive control network. Analyses are based on an archival dataset of 154

undergraduates who completed a gambling task with the goal to win the most money possible. Supporting the hypotheses, TF and source localization analyses revealed that activity in the ACC did precede functional connectivity with the dlPFC. In fact, both the ACC and dlPFC became active before functional connectivity was observed between these regions, suggesting that initial feedback processing occurred separately in each area before broader inter-region communication. This novel finding supports the Expected Value of Control theory and adds to the field's current understanding of feedback and cognitive control processing during task performance.

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by

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Chapter 1: Introduction

1.1 Goal-directed Behavior

A large portion of research in cognitive psychology and cognitive neuroscience has focused on better understanding how humans set and achieve goals. The ability to achieve one's goals is vital for successful learning, achieving rewards, and adaptive behavior in all domains of life (for reviews see Ridderinkhof et al., 2004; van Veen & Carter, 2006). Cognitive psychologists have investigated the mechanism underlying the selection and execution of adaptive and appropriate behavior. This mechanism, termed *Cognitive Control,* is a system capable of selecting thoughts and actions that match an internally represented goal (Miller, 2000; Miller & Cohen, 2001; Ridderinkhof et al., 2004; Kouneiher et al., 2009). In the laboratory, psychologists were able to study goal-directed behavior and cognitive control through analysis of behavioral and brain-based measures during task performance. Cognitive tasks vary in design and ultimate goal, but are distinct from resting and free form tasks that do not contain task rules, instructions, or goals and are thought to index another process and brain network entirely (Greicius et al., 2003; 2009).

Within the lens of task performance, cognitive control allows the individual to adhere to the internal representation of the task demands by supporting appropriate goal-orientated information processing and action planning (van Garavan et al., 2002; Kerns et al., 2004; Veen & Carter, 2006). Thus, the cognitive control system must include (1) a mechanism for monitoring on-going task demands and performance and (2) a system for adapting behavior when current task performance signals a need for behavior modification. This latter system is more effortful and cautious in nature and requires top-down processing to bias appropriate actions. Therefore, the top-down modulation of

behavior cannot be active consistently throughout the task due to the inherent energy costs of employing such a system (Ridderinkhof et al., 2004). Knowledge of the parameters and limitations of a cognitive control system led psychologists to formulate several pertinent lines of research. What signals the recruitment of top-down processing, how does the cognitive system modulate behavior to optimize performance, and how is this recruitment discontinued when no longer necessary (Botvinick et al., 2001)? Finally, what are the neuroscientific mechanisms underlying these processes? Many theories developed over the past decades have sought to answer these questions with evidence from behavioral, EEG, and fMRI studies. Though there is disagreement regarding the exact mechanism of cognitive control recruitment, behavior modification, and withdrawal, cognitive psychologists agree on the importance of the anterior cingulate cortex (ACC) and lateral prefrontal cortices in achieving goal-directed behavior.

1.2 Role of ACC in Goal Achievement and Task Performance

For decades the function of the ACC has been hotly debated. Studies utilizing ERP and fMRI methodology in a wide-range of tasks found ACC activation during response conflict (Berns et al., 1997; Carter et al., 1998; Botvinick et al., 1999; Botvinick et al., 2001) error processing (Gehring et al., 1993; Dahaene et al., 1994; Kiehl, et al., 2000; Garavan et al., 2002) and aversive feedback, including monetary loss, pain, and social rejection (Miltner et al., 1997; Gehring & Willoughby, 2002; Bush et al., 2002; Holroyd & Coles, 2002; Eisenberher et al., 2003; Neieuwenhuis et al., 2004; Shackman et al., 2011). Further investigation of the structural and anatomical nature of the ACC did not provide definitive evidence for one functional role of the ACC over another. Cytoarchitecture of the ACC is varied, suggesting the functions of the ACC could be as

heterogeneous as the structure (Paus, 2001). Further, the ACC has bidirectional projections to prefrontal, motor, and limbic regions, which implicate it in cognitive, motor/action-planning, and motivational/evaluative processes (Barbas & Pandya, 1989; Van Hoesen et al., 1993; Morecraft & Van Hoesen, 1998; Paus, 2001). With the abundance of results regarding ACC activity, cognitive psychologists began to develop conceptual theories about the functional role of the ACC in task performance.

Among the first theories developed was Botvinick and colleague's *Conflict Monitoring Hypothesis.* Botvinick's investigation into the function of the ACC focused on ACC activity in the flanker task. An fMRI study of this task showed ACC activity was enhanced for incongruent vs. congruent trials and further enhanced on incongruent trials that followed congruent trials vs. incongruent trials that followed another incongruent trial (Botvinivk et al., 1999). Botvinick and colleagues (1999, 2001, 2004) interpreted these findings and similar findings in other fMRI studies (Berns et al., 1997; Carter et al., 1998; Kerns et al., 2004) as evidence for the role of the ACC in detecting conflict in information processing. In the Flanker task, individuals are instructed to press a button depending on the direction of an arrow in the middle of five presented arrows. On incongruent trials the arrow is pointing in the opposite direction of the flanking arrows (<<><<), presenting conflict between the competing correct and incorrect responses. This competition between correct and incorrect responses is relatively weaker on congruent trials where the response-relevant arrow and flanking arrows are pointing in the same direction. Botvinick and colleagues (1999) conclude that conflict between competing responses is further heightened when an individual has become accustomed to congruent trials and then encounters an incongruent trial. Further research into the conflict-

monitoring hypothesis broadened the definition of conflict beyond just incongruent flanker trials to tasks involving selection among equally permissible outcomes and overriding automatic, prepotent responses (Botvinick et al., 2004; Cohen & Carter, 2004; Ridderinkhof et al., 2004). Thus, the conflict-monitoring hypothesis proposes an information processing system, located in the ACC, which detects heightened conflict in competing sensory stimuli to monitor the changing cognitive demand of the task and support the most appropriate response pathways.

In parallel to the development of the conflict-monitoring hypothesis, EEG methodology uncovered an ERP component associated with errors in speeded tasks, such as the Go/No-go task. This error-related component (ERN) is characterized by a negative deflection approximately 100 ms after the error (Falkenstein et al., 1990; Gehring, 1992; Gehring et al., 1993), and localizes to the ACC region (Dehaene et al., 1994; Gehring et al., 2000). Further, through simultaneous EEG-fMRI methodology, the ERN was found to be correlated with fMRI error-related activity in the ACC and behavioral post-error reaction time slowing (Debener et al., 2005). As evidence of this error-related (ACC localized) component piled up, researchers began to conceptualize the ACC as an error-processing unit (Gehring et al., 1995; Holroyd et al., 1998; Falkenstein et al., 2000). The proposed function of this error-processing unit is to detect the occurrence of errors in a task and use this information to make adjustments to avoid future errors and optimize task performance (Gehring et al., 1995; Holroyd et al., 1998; Falkenstein et al., 2000). In fact, ERN amplitude, and in turn ACC activity, has been shown to increase with the relative task-importance of the error (Gehring, 1992; Gehring et al., 1993) and the magnitude of the error (i.e. the discrepancy between the error response and theoretical

correct response) (Bernstein et al., 1995), suggesting the importance of the ERN and error detection for overall task performance. Despite strong evidence for the ACC being sensitive to the occurrence of errors, this theory of ACC function quickly transformed to include a more general definition of performance error.

With growing investigation of the ERN in multiple task and performance contexts, researchers began to focus on the positive and negative valence of feedback more broadly. Models of reinforcement learning and reward processing began to inform the conceptualization of ACC function due to studies focused on the importance of rewarding outcomes as well as errors (Holroyd & Coles, 2002; Holroyd et al., 2003; Niewenhaus et al., 2004). These studies focused on a newer ERP component designated the feedback ERN or FN, which was characterized by a negative deflection 180-350 ms after the presentation of task feedback (Miltner et al., 1997; Gehring & Willoughby, 2002). Thus, the FN component reflected ACC activity resulting from the processing of feedback representing incorrect behavior/actions more broadly (i.e. monetary loss, aversive feedback) and not just the occurrence of an error (Miltner et al., 1997; Holroyd & Coles, 2002). Reinforcement learning proposes that learning optimal behavior in an uncertain environment and maximizing task performance are dictated by the principle that individuals will repeat an action in the future if the outcome tied to that action is desirable (Sutton et al., 1992; Sutton & Barto, 1998). Holroyd and Coles (2002) integrated previous ERN and error-related activity in the ACC with reinforcement learning theory and the FN to extend the functional model of the ACC in task performance. Holroyd and Coles (2002) argued that since ACC activity had been found to be associated with task difficulty and early learning in uncertain tasks (Gabriel, 1993;

Bussey et al., 1996; Bush et al., 1998), integration with the reinforcement learning theory was appropriate. This modified model stated that the ACC evaluates the desirability and representational reward of all ongoing outcomes to improve task importance (Holroyd & Coles, 2002).

Previous neuroscience research in mesocortical dopamine neurons bolstered this reinforcement-learning model of the ACC. In primate studies, an increase in mesocortical dopamine produced in the ventral tegmental area (VTA) has been associated with rewarding stimuli (Schultz et al., 1995; Schultz, 1997; Schultz, 1998), but only in the early learning stages of the task. Once the monkey has optimized task performance, rewarding stimuli do not lead to an increase in dopamine release (Ljunhberg et al., 1992; Schultz et al., 1993). Correspondingly, dopamine release in the VTA decreases in response to the presentation of punishment and aversive stimuli for primates (Mirenowicz & Schultz, 1996), a finding that was later replicated in rats (Ungless, 2004). Finally, dopaminergic neurons in the VTA have been found to have substantial projections to the ACC (Gasper et al., 1989; Lewis, 1992). These neuroscience results informed the mechanism of Holroyd and Coles's (2002) reinforcement learning model: 1) when a monitoring system detects an outcome that was better than expected (i.e. rewarding) dopamine release increases, 2) when a detected outcome is worse than expected (i.e. errors, aversive stimuli) dopamine release decreases, and 3) these dopaminergic signals target the ACC and train the ACC to learn the most appropriate motor response. Holroyd and Coles (2002) suggest that within the ACC exist motor command units or "motor controllers", which encode for different actions. Motor controllers are biased by signals from mesocortical dopamine to choose which motoric action is most appropriate and

optimize task behavior over time. Therefore, in line with past dopaminergic studies, the ACC would no longer be activated once an individual has thoroughly learned the rules of the task (Holroyd & Coles, 2002). The reinforcement-learning model of the ACC, and in turn the conceptualization of ERN and FN data, would affect future studies investigating task outcome evaluation.

The reinforcement-learning model broadened the field's definition of task "error" to any performance feedback that was worse than expected. This theory also predicted that more unexpected outcomes would elicit larger ERNs than outcomes that were less unexpected (Holroyd & Coles, 2002; Holroyd et al., 2003). Supporting this hypothesis, Holroyd and colleagues (2003) found greater FN amplitude associated with processing of unexpected aversive feedback than the FN associated with expected aversive feedback, regardless of the fact that both feedbacks were equally aversive. Further studies found enhancement of the FN component for incorrect trials and/or monetary losses dependent on the feedback's relevance to task performance (Gehring & Willoughby, 2002; Niewenhaus et al., 2004), supporting the notion that the ACC evaluates all task feedback on a good-bad continuum (Niewenhaus et al., 2004). Thus, the performance-monitoring system in the ACC must evaluate feedback not simply for the occurrence of errors but for the magnitude, valence, and expectancy of each presented feedback. Task performance is not optimized solely through aversive learning and avoidance of future errors, but through the comparison of actual and expected feedback and the ultimate desire to maximize reward. By this logic, predictions about the likelihood of positive and rewarding feedback are also processed in the ACC, and are reflected by a relative decrease in ACC activation (Holroyd, 2004; Holroyd et al., 2008). The role of the ACC

had quickly extended to encompass processing of performance feedback on a number of dimensions, leading to a more nuanced picture of ACC function.

Though all these theories had different views of the exact function of the ACC in optimizing behavior, no model claimed that the function of the ACC comprised a complete picture of performance optimization. These models conceptualized the ACC as a monitoring system (though there was debate concerning what exactly the ACC monitored) that would signal activation of top-down regions to elicit changes that would increase task performance and allow the individual to achieve their intended goal. Therefore, these models needed to theorize about the processes underlying the top-down control.

1.3 Top-down Cognitive Control in Task Performance

The concept of cognitive control was implicated in goal-directed behavior due to the belief that in order to achieve long-term goals, humans have adapted a system to override automatic and impulsive behaviors (Miller, 2000; Miller & Cohen, 2001). Long-term goals can be very complicated and involve plans of behavior that may overlap or change over time (Miller, 2000). For example, to achieve the goal of buying a house, one needs to develop strategies to save money, augment and replace strategies that do not match with the goal, and override automatic behavior, such as spending money on non-essential items. Arguably, the definition of cognitive control can be broad and far-reaching, intersecting with concepts of executive functioning, inhibition, working memory, and cognitive flexibility. However, Miller and Cohen (2001) were able to articulate cognitive control within task performance and goal-directed behavior as "the active maintenance of patterns of activity that represent goals and the means to achieve

them." Miller and Cohen (2001) argue a neural mechanism for cognitive control would include differential patterns of neuronal firing that resolve conflict, bias the occurrence of appropriate behavior, and create associations between action and resulting feedback. These neurons must integrate information from areas involved with motivations, actions, and sensory processing in order to learn and implement the appropriate goal-directed behavior (Miller & Cohen, 2001).

The criteria laid out by Miller and Cohen quickly implicated the lPFC in cognitive control processing due to its extensive interconnection to areas associated with motivation/reward, motor, and high-order sensory processing (Barbas & Pandya 1987; 1991; Pandya & Yeterian, 1991; Barnes & Pandya, 1992; Seltzer & Pandya, 1994; Miller, 2000; Miller & Cohen 2001) and activation during experience-dependent learning tasks, in which different strategies resulted in reward or goal-attainment (Bichot et al., 1996; Bichot & Schall, 1999; Fuster et al., 2000; Miller, 2000). In fact, primate studies demonstrated that differential firing in lPFC neurons was associated with the primate's utilization of different task rules (i.e. learned association with stimulus shape vs. spatial location) (Hoshi et al., 1998; White & Wise, 1999), congruent with the conceptual framework of cognitive control as an internal representation of the long-term goal and the relevant rules to achieve it (Miller 2000; Miller & Cohen, 2001). Further, the lPFC is not active during the performance of more automatic, bottom-up behaviors (Miller & Cohen, 2001). These early primate findings led Miller and Cohen (2001) to assert that the neuronal activity associated with this top-down cognitive system resides in the lPFC. Miller and Cohen's theory of cognitive control in the lPFC was quickly integrated into theories of task monitoring and the ACC, which was also found to have strong structural

connections with the lPFC (Morecraft & Van Hoesen, 1991; Van Hoesen et al., 1993; Bates & Goldman-Rakic, 1993; Paus, 2001). Miller and Cohen (2001) provided the cognitive community with a well-articulated model of prefrontal cognitive control, but relied on previous theories of the ACC to describe when and how lPFC and cognitive control would become activated; their theory does not provide definitive evidence for one model of ACC function over another.

Botvinick and colleagues seamlessly included the lPFC model of cognitive control in the conflict-monitoring hypothesis of the ACC. This model had already proposed that the ACC detected conflict in task stimuli in order to bias information processing toward relevant information and away from competing task-irrelevant information (Botvinick et al., 1999; 2001). Therefore, Miller and Cohen were able to provide a neural mechanism for the information biasing integral to Botvinick's conflict-monitoring model. On trials with high levels of conflict, such as an incongruent flanker trial, the ACC would detect the conflict and signal to lateral prefrontal regions the need for biasing of information processing in order to successfully choose the correct response. Neurons in the lPFC associated with processing of task-relevant stimuli and correct behavior would then fire more rapidly and become more active than neurons associated with competing responses, allowing the individual to execute the correct behavior despite the conflict. Botvinick (2007) later built upon this theory to include a cost-benefit model of recruiting additional top-down control in the lPFC. Botvinick argued that the occurrence of conflict in task stimuli represented a cost because of the necessary involvement of effortful and taxing cognitive control. Due to the aversive nature of conflict, cognitive control processing would then bias away from strategies of

information processing that would allow for continued occurrence of information conflict (Botvinick, 2007). Through this aversive learning, response decision-making would be biased toward more efficient and appropriate strategies over time, suggesting conflict could signal top-down processing to improve performance on a single trial as well over the course of the task. A similar process would occur during the aversive learning of error monitoring. Errors, like conflict, represent aversive and costly events in task performance that should be avoided in the future (Gehring et al., 1995; Gehring & Fencsik, 2001). If the ACC detected the occurrence of an error, a signal to the lPFC would call for corrective or compensatory strategies, such as slowing reaction time on the next trial (Gehring & Fencsik, 2001).

The Miller and Cohen (2001) model of cognitive control also fits nicely with Holroyd and Cole's (2002) proposed reinforcement learning model of ACC function. Unlike conflict and error monitoring, the reinforcement-learning model argues that the ACC monitors all response outcomes, regardless of valence or magnitude, to create more accurate associations between possible actions and outcomes (Holroyd & Coles, 2002; Holroyd et al., 2004; Holroyd et al., 2008). Decisions concerning future behavior are not solely based on avoidance of aversive outcomes but also on the optimization of reward. Because the ACC contains nuanced information regarding the potential reward associated with possible outcomes, Holroyd and colleagues propose that decision-making and the resulting motor controller signals occur in the ACC (Holroyd & Coles, 2002; Holroyd et al., 2004; Holroyd & Yeung, 2011). So how is control processing in the lPFC involved? Holroyd and Yeung (2011) stated that the ACC also contains a cost-benefit analysis before signaling the appropriate motor controller; the ACC choses the best option for the

next trial based on internally held action outcome associations and calculates the amount of effort needed to execute this choice. When the estimated effort reaches some substantial threshold the ACC will recruit the lPFC to add effortful, top-down processing to bias toward that appropriate pathway (Holroyd & Yeung, 2011). Thus, Holroyd and Yeung (2011) posit that it is not just the detection of costly errors or conflict that signals for increased cognitive recruitment of lPFC, but a cost-benefit analysis that includes the potential reward of the action and the difficulty of the action. Because Miller and Cohen's (2001) model of the role of cognitive control in goal-directed behavior supported models of the ACC, the debate over what exactly is monitored by the ACC and when cognitive control is recruited remained.

The above theories conceptualize cognitive control as a response to feedback processing – a mechanism to bias performance depending on on-going outcome information. Recent cognitive control theories have designated this type of cognitive control processing as *reactive* due to its reliance on responding to feedback. Braver (2012) succinctly articulates the difference between this reactive cognitive control and *proactive* cognitive control. Reactive cognitive control is stimulus-driven and involves feedback monitoring in the ACC, while proactive cognitive control is independent of on-going task feedback. Instead, proactive control represents the sustained maintenance of the task's goal and the pertinent behavior to achieve this goal. Thus, researchers theorize that proactive control in the dlPFC does not involve the ACC (and feedback monitoring) as does reactive control. Importantly, theories of proactive and reactive control hypothesize differential patterns of proactive and reactive cognitive control engagement through time (Amodio, 2010; Braver, 2012; Schmid et al., 2015). As detailed in the

description of reactive cognitive control above, engagement of the dlPFC is hypothesized to occur post-stimulus as needed depending on feedback processing in the ACC. On the other hand, proactive control is sustained throughout the task and thought to be engaged in anticipation and preparation for the stimulus (Braver, 2012). Therefore, studies aimed at investigating individual differences in proactive control measure dlPFC engagement during the inter-trial interval (ITI) prior to stimulus exposure (Amodio, 2010; Schmid et al., 2015). Though the scope of this current study is limited to the timing of reactive cognitive control, these theoretical timing differences between proactive and reactive control lend itself to interesting future investigation (see section 4.6 for further discussion).

1.4 Inconsistent Findings for ACC and lPFC Function in Task Performance

As the models of task performance and goal-directed behavior advanced, so too did the quality and quantity of neuroscientific studies investigating aspects of task performance. This led to over a decade of research that attempted to provide evidence supporting one conceptual model of ACC-lPFC function over others. Additionally, as neuroscientific methodology improved, particularly fMRI, researchers were able to better localize task-related activity to the dorsal ACC (dACC) and dorsolateral prefrontal cortex (dlPFC). Thus, depending on the spatial resolution inherent in the design of the following studies, findings of activity in the ACC/dACC and lPFC /dlPFC were interpreted as functionally similar. However, despite the advancement and proliferation of neuroscience studies focused on the topic of cognitive control in task performance, consensus for one model remained elusive.

Based on the conflict monitoring and error related models of the ACC in task performance, cognitive neuroscientists set out to collect consistent evidence of ACC activity during conflict and error trials. Several studies investigated ACC activity on both error and conflict-related trials and found enhanced ACC activation for both (Carter et al., 1998; van Veen & Carter, 2002; Baker & Holroyd, 2011; Ebitz et al., 2015). ERP studies conducted by van Veen and Carter (2002) and Baker and Holroyd (2011) were able to dissociate ACC activity related to conflict and that related to errors because of the temporal resolution of EEG. Both found that an ERP component associated with stimulus conflict (N200) and later components associated with error and feedback (ERN and FN) localized to the ACC. Van Veen and Carter (2002) interpreted these results as evidence that the detection of conflict and errors consisted of the same underlying process but at different time points during task performance. Thus, the potential difference in timing between conflict and error processing was missed in fMRI experimental designs that lacked the necessary temporal sensitivity.

Despite the lack of temporal resolution inherent in fMRI methodology, these studies are integral to understanding the neural processing of task performance due to the spatial resolution of fMRI. Are conflict and error processing actually occurring in the same region of the brain or does it just appear that way in low-resolution methodology? To investigate this question, fMRI studies had to address the confound plaguing this debate: trials that have high conflict tend to be the trials that result in participant error (Ulsperger et al., 2001; Garavan et al., 2002; Garavan et al., 2003; Botvinick, 2004). Garavan and colleagues (2003) attempted to disentangle effects of stimulus conflict and errors by modifying a go/no-go task to consist of some blocks that had faster stimulus

presentation and enhanced prepotency of go trials. Therefore, this experimental design could compare ACC activity on errors to that of high-conflict correct trials. Garavan and colleagues (2003) found spatial dissociation between error and high conflict activity, with error processing activating the ACC and high conflict activating the pre-supplementary motor area (pre-SMA). This same spatial pattern of error processing localizing to the ACC and conflict processing localizing more posteriorly was also found within other modified tasks (Kiehl et al., 2000; Braver et al., 2001; Ulsperger et al., 2001), suggesting the processes that underlie error and conflict detection may not be identical. Further evidence for the functional separation between conflict and error processing was found in an ERP study based on more recent models of conflict and outcome evaluation. Maier and colleagues (2008) tested whether error detectability or importance predicted ACC activation by instructing participants to monitor and report their errors on some blocks and by including errors of varying task-relevance. According to a later account of the conflict-monitoring hypothesis, conflict can also refer to the discrepancy between the actual and desired response when an individual realizes the correct behavior post-error (Yeung et al., 2004). Following this definition of conflict, the conflict-monitoring model predicts greater ACC activity and enhanced ERNs for errors that were detected by the participant (Yeung et al., 2004). Conversely, error-related and reinforcement learning theories predicted greater ERN amplitude for those errors that are more relevant and vital to task performance (Gehring 1992; Gehring et al, 1993; Holroyd & Coles, 2002). Results of the study revealed greater ERNs for task-relevant errors than detected errors, lending support to the reinforcement-learning model of ACC function over conflict (Maier et al., 2008). These contradictory findings beg the question whether the processes that underlie

error and conflict processing are truly synonymous. Fortunately, researchers recognized that in order to understand the importance of conflict and error processing within task performance they needed to associate error/conflict processing with cognitive control and behavioral adjustment.

Both conflict monitoring and error monitoring models predict increased cognitive control recruitment (dlPFC activity) and behavioral modification after the ACC detects the occurrence of conflict (Botvinick et al., 1999; 2001; 2004; 2007) or errors respectively (Gehring, 1992; Gehring et al., 1993; Gehring et al., 1995; Holroyd et al., 1998; Falkenstein et al., 2000). A slowed fMRI task further bolstered the functional dissociation between ACC and dlPFC, revealing dlPFC activation during the instructions of the task and ACC activation during the presentation of task stimuli (MacDonald et al., 2000). As predicted by all performance monitoring models, these results indicate ACC functioned as a stimulus evaluator while dlPFC was involved in a more top-down representation of task rules and cognitive demands. MacDonald and colleagues (2000) also showed that individuals with the greatest dlPFC activity during instruction presentation displayed the fastest reaction times on correct trials, demonstrating the importance of dlPFC for efficient task performance. In a modified stroop task, Gehring and Knight (2000) found that individuals with lesion damage to lPFC regions displayed error-related ACC activity (ERN components) on both error and correct trials. Further, the individuals with lateral prefrontal damage performed less corrective behavior on trials following an error compared to age-matched controls (Gehring & Knight, 2000). These results suggest that the lPFC is a necessary component of successful error detection and optimized performance. On the other hand, a conflict-monitoring study found that ACC

activity during incongruent trials on the flanker task predicted lPFC activity on the subsequent trial as well as behavioral adjustment (i.e. slowed reaction time) (Kerns et al., 2004). These studies provide evidence for the importance of error and conflict processing for later dlPFC activation and behavior modification, yet none contain explicit findings that would contradict one performance model.

Another fMRI study conducted by Garavan and colleagues (2002) further complicated the conflict and error monitoring hypotheses of lPFC activation during task performance. In this study, researchers modulated task difficulty by decreasing and increasing stimulus presentation in a go/no-go task and found greater ACC activation on the more difficult speeded no-go trials but greater dlPFC activation on slower no-go trials (Garavan et al., 2002). Garavan and colleagues (2002) interpreted these surprising findings as evidence for greater ACC involvement on more "urgent" inhibition and longer, more controlled processing in the dlPFC on inhibition trials that allow for more time to respond. These findings oppose predictions that both enhanced ACC and enhanced dlPFC activity would be associated with more difficult conflict or errors, further complicating the theory of ACC-dlPFC interactions within task performance. Despite the proliferation of well-designed studies focused on investigating the neuroscientific mechanism of task performance, results continued to provide some support for the available conceptual models without strong evidence arguing against other models. It became clear that the system involved in efficient task performance could not just be driven by conflict, error detection, or surprising outcomes alone. Cognitive psychologists recognized the merit of each of the proposed models and sought to integrate the aspects of the model supported by data, which has led to the development

of more nuanced and comprehensive models of goal-directed behavior in the current literature.

1.5 A Push Toward an Integrative Model

With the continuation of conflicting findings and further evidence ostensibly in support for conflict monitoring, error processing, and reinforcement learning theories of task optimization, it became clear that a more comprehensive model of cognitive control and task performance needed to be developed to encompass the varied experimental results. The first comprehensive model to account for evidence of the importance of conflict, errors, reward, and outcome probability was the *predicted response-outcome model (PRO)* developed by Alexander and Brown (2011). This model is based on learning principles of reward and possible actions available to the learning agent, and most closely resembles Holroyd and Coles' reinforcement learning perspective of task performance. Similar to the reinforcement-learning model, the PRO model proposes that the ACC must detect and evaluate all performance outcomes, regardless of valence (i.e. error/correct or loss/reward), in order to ensure efficient task performance (Alexander & Brown, 2011). The hypothesized function of the ACC within a task is to detect discrepancies between actual and predicted outcomes and update predictions if a discrepancy exists. Alexander and Brown delve further into the neural mechanism behind outcome predictions, stating that activity in individual neurons represent learned associations between particular actions and outcomes that contains information about the probability of an action resulting in a specific outcome and the probable time in which the outcome will occur (Alexander & Brown, 2011). When neurons in the ACC detect that the actual action-outcome association matched the internal prediction, activity in

those neurons is inhibited. Conversely, if a predicted outcome does not occur, the appropriate neurons fire and ACC activity in enhanced. Though at first glance this mechanism can appear synonymous with reinforcement learning, Alexander and Brown (2011) articulate key differences. Unlike reinforcement learning models, the PRO model is concerned with mapping associations between the individual's actions and outcomes rather than that task stimulus and outcomes. Further, the PRO model consists of separate predictions for all possible action-outcome associations, not just the overall prediction of reward or loss inherent in the reinforcement-learning theory. Most importantly, instead of tracking the occurrence of surprising outcomes, the PRO model states that ACC neurons become active when a predicted outcome did not occur, termed *negative surprise* (Alexander & Brown, 2011). The occurrence of negative surprise signals to the appropriate neurons that the probability value within that action-outcome association needs to be updated.

How does the PRO model better account for the diverse results in the field? Alexander and Brown (2011) propose ACC activity related to errors reflects negative surprise in the comparison of the predicted and observed outcome. ACC activity became enhanced because the individual believed their action would result in correct feedback – but that was not the case. Alexander and Brown (2011) also suggest findings that associated conflict with ACC activity can be better explained via the PRO model. They pose that it is not the occurrence of conflicting stimuli itself that causes ACC activation, but that conflicting stimuli often coincide with the simultaneous activation of several appropriate action-outcome predictions. The PRO model is able to articulate an internally consistent mechanism that explains the plethora of evidence connecting error, conflict,

and outcome likelihood to ACC activity. Though more focused on elucidating task-based processing in the ACC, the PRO model does suggest possible functions of cognitive control and the dlPFC. Alexander and Brown (2011) hypothesize that top-down cognitive control might be necessary when the task is particularly novel and action-outcome predictions are uncertain. In this situation, cognitive control would aid in the cost-benefit analysis of deciding which action seems the most appropriate given the limited available information. Additionally, the occurrence of negative surprise could indicate to prefrontal regions the possibility to enact a different strategy (Alexander & Brown, 2011). This latter proposal of ACC-dlPFC collaboration in the on-going analysis of task strategy and decision to implement alternate strategies has become the defining feature of the most recent performance theories.

Focus on the overall strategies adopted during completion of a task is the result of the aggregation of recent neuroscientific studies focused on updating task models (Amiez et al., 2012; O'Reilly et al., 2013; Sellet et al., 2013) and theories of exploratory and exploitative strategies in adaptive behavior (Mangel & Clark, 1986; Jones & Cohen, 2005; Kolling et al., 2016a; Kolling et al., 2016b). In both foraging value theory (Mangel & Clark, 1986) and adaptive gain theory (Jones & Cohen, 2005), individuals switch between exploratory and exploitative strategies depending on which strategy garners the greatest success in the current environment. Exploratory strategies are utilized in novel environments when the association between possible actions and outcomes is unknown or when a past strategy is no longer a good fit with the environment (i.e. when aversive outcomes outnumber rewarding ones). Conversely, exploitative strategies are applied when action-outcome associations are well understood and the individual is able to

optimize behavior or task performance (Mangel & Clark, 1986; Jones & Cohen, 2005). Amiez and colleagues (2012) applied the principles of exploration and exploitation to ACC and dlPFC activity during task performance. In this study, participants were able to choose between four stimuli and were provided feedback communicating whether they chose the correct stimulus. Participants understood that one of the four stimuli was associated with correct feedback and continued to search behind the remaining stimuli until the correct feedback was revealed. Once revealed, the participants were free to choose this stimulus in subsequent trials to elicit further correct feedback. Thus, Amiez and colleagues were able to investigate activity associated with exploratory strategies (searching for the correct feedback) and exploitative feedback (continuing to choose the stimulus that elicits the correct response). Results from this fMRI study showed enhanced ACC and dlPFC activity during the exploratory phase of the task with increased ACC and dlPFC activity on trials eliciting incorrect feedback *and* the first trial that garnered correct feedback (Amiez et al., 2012). Therefore, it is not the error feedback itself that leads to ACC activity and cognitive control recruitment, but the presence of feedback that indicates further application of exploratory strategies. Additional studies replicated these findings in the ACC, demonstrating enhanced ACC activity on trials calling for a change in task strategy (O'Reilly et al., 2013; Sellet et al., 2013). Of particular interest is the fMRI experiment by O'Reilly and colleagues (2013) that dissociated task difficulty from trials requiring a change in task strategy. In this task, participants were asked to look as quickly as possible at a dot that appeared on the screen. Dots were most likely to appear near the spot of the previous dot unless the dot changed color, signaling that future dots were likely to appear around this new location. The appearance of white dots appeared in

a different location on the screen but did not signal any information about future trials. Though both white dot trials and color-change trials were equally difficult and displayed relatively longer reaction times, the ACC was activated only for the trials that required the model change and not the white dot trials (O'Reilly et al., 2013).

These innovative findings informed the development of the foraging value theory (FVT) of task performance (Kolling et al., 2016a; Kolling et al., 2016b). Within this conceptualization of task performance, Kolling and colleagues posit that ACC and dlPFC are involved in a cost-benefit analysis to decide whether to continue with the current task strategy or experiment with a new one (Kolling et al., 2016a; Kolling et al., 2016b). More specifically, populations of neurons in the ACC encode values associated with every possible action/strategy, such as the potential reward that action may elicit. Different populations of neurons become excited or inhibited throughout the task based on learned outcomes, which informs the resulting cost/benefit analysis and action decision. When values of opposing actions are close together (e.g. during conflict) choosing a particular action is more difficult and results in increased reaction time on the task (Kolling et al., 2016a). If a decision were easier, Kolling and colleagues (2016) hypothesize that the cost/benefit and decision-making processes in the ACC might occur so quickly that current methodology would miss the ACC activity associated with those trials. Finally, the foraging value theory conceptualizes the role of dlPFC and cognitive control as a system utilized to help execute the action decision made within the ACC; congruent with other performance models and recent strategy-focused findings, this model would predict heightened dlPFC activity when ACC is also activated (Kolling et al., 2016a; Kolling et al., 2016b). The FVT provides a broader perspective to outcome evaluation in task

performance while still introducing a mechanistic model that accounts for the varied research findings. To Kolling and colleagues, conflict, errors, rewards, etc. are all indicators of the success and efficiency of the current strategy and contribute to the cost/benefit analysis in the ACC.

A final integrative theory of dACC and dlPFC function extends the cost/benefit analysis and decision-making of the foraging value theory to include the inherent cost of recruiting top-down cognitive control. Shenhav, Botvinick, and Cohen (2013, 2016) have kept abreast of recent research in performance literature and developed the Expected Value of Control theory (EVC). Within EVC, the role of the ACC is not only to calculate the potential costs and benefits of maintaining the current strategy or seeking another strategy for optimal reward attainment, but also the potential costs and benefits of utilizing effortful control systems to execute change (Shenhav et al., 2013; 2016). EVC theory accounts for the costly and effortful nature of cognitive control that has been discussed in the task performance literature for years (Botvinick et al., 2001; Botvinick, 2004; Ridderinkhof et al., 2004; Kool et al., 2010; Kool & Botvinick, 2014; Zink et al., 2018). EVC states that all behavior lies on a continuum of automatic to controlled behavior, which is monotonically related to the amount of effort necessary to execute that behavior (Shenhav et al., 2013, 2016). If the amount of effort needed to execute a behavior outweighs any potential benefit that might arise from the behavior, it would not be adaptive to drain the resources necessary to implement the behavior. Therefore, the ACC must calculate the amount of control and effort a behavior requires before deciding to recruit the dlPFC. Shenhav and colleagues (2016) suggest that the ACC must calculate and signal to the dlPFC the necessary identity (what control processes do we need?) and

intensity (how much control do we need?) of control required for a behavior in the current environment. The primary function of the ACC in the EVC model is to evaluate and signal for the appropriate level of control, which is heavily dependent on the specific context of the environment (Shenhav et al., 2013, 2016). Further, because the context of a task varies wildly from study to study, so too does the cost/benefit decision for control, which can help explain the conflicting ACC and lPFC findings in task performance literature (Shenhav et al., 2016). Because of the importance of task context, Shenhav, Botvinick, and Cohen (2016) highlight the necessity of thorough neural methods in future research.

1.6 Time-Frequency Amplitude and Phase Indices of ACC and dlPFC

Significant advances have been made in ERP methodology and analyses since the discovery of ERN and FN components that can aide in the continued investigation of task performance and goal directed behavior. Previous work concerning the FN involved debate over whether FN amplitude primarily reflected processing of rewarding or aversive feedback (Holroyd et al., 2008; Potts et al., 2006; Holroyd et al. 2011; Proudfit, 2015). This debate highlighted an inherent confound in interpreting FN findings: processing related to rewards and losses overlapped in time. In order to evaluate the conceptual models of task performance, processing related to differentially valenced feedback needed to be isolated. This methodological limitation led to the development of time-frequency analysis, which decomposes the time-domain ERP waveform into its composite frequencies. These frequencies can then be extracted and analyzed separately for modulation related to different aspects of task stimulus. In fact, several time-frequency analyses of the FN component have shown that the FN can be decomposed

into delta and theta frequencies, which are differentially sensitive to gain and loss feedback, respectively (Nelson et al., 2011; Bernat et al., 2008, 2011, 2015; Foti et al., 2015; Watts et al., 2017). Foti and colleagues (2015) source localized feedback-related theta and delta activity, suggesting that gain processing in delta localized to the striatum and replicating that loss processing occurred in theta localized to the ACC. Therefore, theta activity within the FN time window can be seen as an index for ACC activity during feedback processing.

Time-frequency ERP analyses also offer the opportunity to isolate frequency activity with a fine temporal resolution based on the data reduction method used in the creation of the time-frequency transform. The most widely used time-frequency transforms in ERP work are wavelets (Daubechies, 1990; Graps, 1995; Torrence & Compo, 1998), which are effective but have the widely understood property of smearing energy in time at low frequencies and smearing in frequency at high frequencies. The current project instead employed Cohen's class reduced interference distributions (RIDs; Cohen, 1992; 1995), offering several advantages relative to wavelets, including better time-frequency support, and computing instantaneous frequency in such a way that does not smear energy in time or frequency (for a more detailed listing of differences between RID and wavelet approaches please see Bernat et al., 2005). The time-resolution is particularly relevant to the current project, which aims to disentangle activity occurring in the theta band across only 100-200 ms. For these reasons, the Cohen's class RID transform was used in this study to create the time-frequency surfaces for analysis (see section 2.10).

Further advancement of ERP analyses and integration with fMRI and diffusion tensor imaging (DTI) techniques advanced a new neuroscientific conceptualization of cognition. Instead of perceiving different cognitive functions in a modular fashion localized to specific brain regions, cognitive neuroscientists began to explain cognition in terms of dynamic changes in distinct brain networks (Fuster, 2006; Bressler & Menon, 2010). Neuroscience research, utilizing EEG, fMRI, and DTI methods, identified several large-scale functional networks that were consistently associated with separable cognitive functions (Greicius et al., 2003; Fox et al., 2006; Seeley et al., 2007; Golland et al., 2007; Bressler & Menon, 2010). The default mode network (DMN) is characterized by functional connectivity among regions such as the ventromedial prefrontal cortex and posterior cingulate cortex during self-referential autobiographical processing and socially cognitive tasks (Gusnard et al., 2001; Shannon & Buckner, 2004; Szpunar et al., 2007; Harrison et al., 2008; Buckner et al., 2008; Spreng et al., 2009; Bressler & Menon, 2010). The DMN has also been shown to be consistently deactivated during cognitively demanding tasks (Raichle et al., 2001; Singh & Fawcett, 2008; Sridharan et al., 2008). In contrast with the DMN, research uncovered a central-executive network (CEN) active during cognitively demanding tasks, which included activation within dlPFC, the posterior parietal cortex, and other cortical regions (Seeley et al., 2007; Sridharan et al., 2008; Bressler & Menon, 2010). Thus, the CEN was conceptualized as the neuroscientific network associated with top-down cognitive control recruitment (Sridharan et al., 2008; Bressler & Menon, 2010). A third network, termed the salience network, is hypothesized as the network responsible for switching between the DMN and CEN (Sridharan et al., 2008, Bressler & Menon, 2010; Menon & Uddin, 2010; Hellyer et

al., 2014; Goulden et al., 2014). The salience network is comprised largely of the ACC and anterior insula and has been associated with orientating processing toward external stimuli and allocating attention (Seeley et al., 2001; Sridharan et al., 2008; Eckert et al., 2009; Menon & Uddin, 2010). Therefore, the salience network could facilitate appropriate switching between the CEN and DMN based on the integration of external and internal information (Bressler & Menon, 2010; Menon & Uddin, 2010). Activation of the CEN and salience networks can easily be applied to results showing dlPFC activity and ACC activity respectively. In accordance with conceptual models of task performance, network models describe the salience network (ACC activation) as an allocator of attention based on external cues that can activate the CEN (dlPFC) when appropriate. It is not just modular activation that is important to understanding behavior, but the functional integration and communication between disparate brain regions.

Development of time-frequency phase-synchrony approaches has led to a theta-filtered measure that indexes lPFC cognitive control and activation of the CEN. Inter-channel phase synchrony (ICPS) calculates the degree of phase-synchrony between channels (regions) at different points in the ERP waveforms, and is a demonstrated measure of functional integration among different brain regions. Brain regions that have a high degree of ICPS are operating in coordination (i.e. in phase with each other). ICPS is a useful measure of functional connectivity that has been shown to index the recruitment of cognitive control during task performance. Theta-filtered ICPS between medial frontal areas associated with ACC activity and lateral prefrontal areas has been associated with trials that are predicted to require greater cognitive control (Cavanaugh et al., 2009; Van de Vijver et al., 2011; Bolanos et al., 2013; Moran et al., 2015; Aviyente et al., 2017).

Enhanced theta-ICPS has been observed on trials presenting errors and incorrect feedback (Cavanaugh et al., 2009; Van de Vijver et al., 2011; Bolanos et al., 2013; Moran et al., 2015) as well as aversive loss feedback (Aviyente et al., 2017). Further, heightened theta-ICPS predicted post-error slowing on the subsequent trial (Cavanaugh et al., 2009; Moran et al., 2015) a well-established behavioral index of increased cognitive control. These new time-frequency RID ERP approaches allow researchers to index ACC activity, as well as ACC/lPFC functional integration subserving cognitive control, at the fine time-scale inherent to EEG.

Finally, burgeoning work focused on phase-synchrony may help elucidate the mechanism underlying communication between ACC and dlPFC neurons and switching between salience and control networks. Fries (2005) presented neuronal coherence as the ultimate method of communication between disparate brain regions. When disparate neuronal populations are coherently oscillating (i.e. in-phase), the time windows for information input and output are the same in both brain regions, fostering communication between them (Fries, 2005). Further evidence of the communication potential of phase coherence within a region involves research of cross-frequency coherence, also termed cross-frequency coupling (Sauseng & Klimesch, 2008; Cohen, 2009). Cross-frequency phase-amplitude coupling refers to the occurrence of phase oscillation in one frequency (usually the lower frequency) inducing activity (amplitude) changes in another frequency (Sauseng & Klimesch, 2008; Cohen, 2009; Cohen et al., 2009). Theta frequency phase peaks, for example, have been found to correspond to increased gamma and alpha power (Canolty et al., 2006; Demiralp et al., 2007; Cohen et al., 2008, Tort et al., 2009). Cohen and colleagues (2008) also found that theta-gamma phase-amplitude coupling was

associated with processing of valenced feedback and was enhanced on loss trials compared to gains. This approach can also be extended to include phase-phase coupling and coupling between phase in one region and amplitude in another region. This represents an exciting new area of methodological development.

Another more widely used approach is to index coherence between trials, assessing the consistency in responding relative to an event. This can be indexed using inter-trial phase synchrony (ITPS) where greater ITPS indexes the coherence of phase among trials of a specific stimulus type. Thus, researchers have begun to conceptualize enhanced ITPS as an indicator of increased coordination and communication between distant brain areas (Sauseng et al., 2007; Cavanaugh et al., 2009; Burwell et al., 2014; Aviyente et al., 2017; Tootell et al., 2018). Time-frequency decomposition of ITPS has shown theta-ITPS in medial-frontal electrodes is associated with theta amplitude and increased ACC activity (Cavanaugh et al., 2009; Cohen & Cavanaugh, 2011; Burwell et al., 2014). Therefore, trials that are predicted to have increased ACC activity are predicted to have heightened ITPS as well. Several studies have confirmed this prediction by demonstrating increased theta-ITPS for incorrect feedback (Cohen et al., 2008; Cavanaugh et al., 2010; van Noordt et al., 2017) and more cognitively demanding inhibition trials (Papenberg et al., 2013). ERP theta-ITPS may become crucial in our understanding of communication between different brain regions, which can aide in understanding the monitoring and control processes underlying task performance.

Though these ERP time-frequency and phase measures have been associated with ACC and prefrontal activity in the past, the spatial resolution of EEG measures is inherently lower than that of fMRI methodology, making inferences about underlying

sources more complicated (Tenke & Kayser, 2012; Kayser & Tenke, 2015; Bradely et al., 2016). Past studies have utilized cortical source localization techniques, such as that of Foti and colleagues (2015), to compute the corresponding activity in the underlying cortex most likely responsible for the observed electrical activity at the scalp. Thus, source localization analyses can transform 2-D montages of electrical activity measured at EEG electrodes into 3-D models of the electrical dipoles that generated that measured activity, which allows for localization of ERP activity to cortical regions like the ACC and dlPFC (see Methods section for a description of this process). With source localization, researchers are able to make far stronger inferences about the cortical structures involved in producing the recorded ERP than amplitude and phase measures can alone (Reynolds & Richards, 2005; Reynolds & Richards, 2008). Once activity has been computed into 3-D source space, measures of ERP amplitude, frequency coherence, and functional connectivity can be calculated from dipole activity in 3-D voxels as opposed to electrical activity in EEG electrodes. Incorporating source localization analyses and time-frequency analyses sheds light on ACC and dlPFC activity on the scale of milliseconds, offering the opportunity to uncover novel neural findings that can aide in the ongoing discussion of comprehensive models of task performance.

1.7 Deficits of ACC and dlPFC Functioning in Psychopathology

One final research area of interest within task performance literature is the effect of ACC and lPFC dysfunction on goal-directed behavior. Studies on the structural damage of ACC and lPFC regions have shown resulting deficits in task performance (Gehring & Knight, 2000; Swick & Turken, 2002; Ullsperger et al., 2002), further supporting the importance of both these regions for efficient task performance. From

these lesion and damages studies, psychologists began to question whether individuals who demonstrated poor ability to execute goal-directed behavior also had measurable neural deficits. Clinically, many psychopathological disorders are characterized by maladaptive decision-making, inhibition, and behavior (Schachar & Logan, 1990; Sergeant et al., 2002; Bachara & Damasio, 2002; Pickup, 2008; Snyder et al., 2015).

Because of the observed clinical deficits associated with several mental disorders, studies investigated dysfunction in ACC and lPFC regions early on in the development of task performance models. Experiments utilizing inhibition tasks found that individuals with attention-deficit hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD), and schizophrenia displayed poorer accuracy on inhibition trials and decreased ACC activity compared to age matched controls (Enright et al., 1993; Pliszka et al., 2000; Carter et al., 2001; Curtis et al., 2001). Individuals with ADHD were also found to have smaller volume in lPFC regions compared to controls, and this anatomical deficit predicted poorer accuracy and longer mean reaction times on an inhibition task (Casey et al., 1997). Interestingly, though anxious individuals demonstrate similar task performance to that of controls (Roche et al., 2005; Righi et al., 2009), they exhibit increased error-related ACC activity thought to represent their increased vigilance and attention to the task in order to avoid errors (Gehring et al., 2000; Hacjak et al., 2003; Ladouceur et al., 2006; Olvet & Hajcak, 2008; Righi et al., 2009; Weinburg et al., 2010; Cavanaugh & Shackman, 2014). Increased error-related ACC activity has also been shown in other internalizing disorders such as depression (Steele et al., 2004; Pizzagalli et al., 2006; Holmes & Pizzagalli, 2007; Holmes & Pizzagalli, 2008; Olvet & Hajcak, 2008). Despite robust evidence for decreased error-related ACC activity in internalizing

disorders, findings related to feedback processing, as indexed by the FN component, are less clear. While there is some evidence for increased FN for negative feedback in anxious individuals (Cavanaugh & Shackman, 2014), individuals with depressive symptoms have exhibited increased *and* decreased FN and ACC activity to negative feedback (Tucker et al., 2003; Santesso et al., 2008; Foti & Hajcak, 2009; Mueller et al., 2015). Due to a well-known negativity attention bias in those with depression, most researchers predicted increased attention toward negative feedback and thus enhanced FN (Santesso et al., 2008; Olvet & Hajcak, 2008; Foti et al., 2009). Mueller and colleagues (2015) attempted to explain these discrepant results suggesting that depression with anhedonia would moderate this effect. In support of this hypothesis, Mueller and colleagues (2015) found that individuals with major depressive disorder (MDD) displayed enhanced FN activity toward errors, while individuals with MDD with high levels of anhedonia showed decreased FN and processing of negative feedback. In comparison to the discrepant and nuanced findings for ACC activity in internalizing disorders, results for externalizing disorders are well established. Individuals with externalizing disorders, such as ADHD, conduct disorder, antisocial personality disorder, and substance use, exhibit reductions in ACC-related error and feedback processing as indexed by ERP ERN, FN, P3 and time-frequency delta components (Justus et al., 2001; Iacono et al., 2003; Bauer & Hesselbrock, 2003; Ruchsow et al., 2005; Patrick et al., 2006; Potts et al., 2006; Franken et al., 2007; Fein & Chang, 2008; Olvet & Hajcak, 2008; Gilmore et al., 2010; Bernat et al., 2011). Studies employing fMRI techniques have provided further evidence for decreased ACC activity during errors in subjects dependent

on a variety of substances (Kaufman et al., 2003; Forman et al., 2004; London et al., 2005; Gruber & Yurgelun-Todd, 2005; Goldstein et al., 2007).

In addition to findings regarding ACC activity on task performance by individuals with internalizing and externalizing disorders, studies have begun to investigate deficits in communication among regions (i.e. ACC and dlPFC) in psychopathology. On a flanker task, anxious individuals displayed increased error-related activity in the ACC but lower functional connectivity (indexed by theta-ICPS) to lPFC (Moran et al., 2015), suggesting that dysfunction in psychopathology may be better conceptualized as a lack of communication between regions rather than solely anatomical deficits. The importance of neural communication in understanding psychopathology was further evinced in studies focused on deficits in large-scale networks. Research has shown abnormalities within and between DMN and salience networks in Alzheimer's Disorder, schizophrenia, depression, anxiety, PTSD, and ADHD (Wang et al., 2006; Stam et al., 2006; Feinstein et al., 2006; Greicius et al., 2007; Dickerson & Sperling, 2009; Bressler & Menon, 2010; White et al., 2010; Liddle et al., 2011; Sripada et al., 2012; Moran et al., 2013). The behavioral and cognitive complexity of psychopathology is likely due to some sort of dysfunction at the network level as opposed to deficits contained within specific brain areas.

Due to evidence of hypoactivity in ACC and lPFC regions in several mental health disorders, researchers investigated whether these regions could be predictors of treatment response. Even the most evidence-based treatments and psychotropic medications are not effective for treating all individuals with mental health symptoms, so psychologists sought to uncover why treatments work for some and not others. Could we

identify some neural indicator that would allow clinicians to choose the most effective treatment for that individual, saving both time and money? Emerging work that attempts to answer this question has found conflicting evidence regarding the possibility of ACC and lPFC activity as a reliable treatment outcome predictor. Studies focused on depression, which has been characterized by hypoactivity in the dACC (Heller & Nitschke, 1997; Drevets, 1999; Davidson et al., 1999), found greater symptom reduction after medication in individuals with higher baseline activity in the rostral ACC (Pizzagalli et al., 2003; Brockmann et al., 2009; Keedwell et al., 2010; Pizzagalli et al. 2010). This same pattern was found in studies focused on treatment of anxiety as well (Shin et al., 2013; Ball et al., 2014). However, other studies concluded that *decreased* ACC activity at baseline predicted better treatment outcomes for psychotropic medication as well as cognitive behavioral therapy (Dichter et al., 2010; Siegle et al., 2006, 2012). These studies also associated better therapy and medication outcomes with reduced baseline lPFC activity (Dichter et al., 2010; Siegle et al., 2006, 2012), while others found the opposite (Ritchey et al., 2011; Samson et al., 2011). Clearly there is no consensus on the utility of ACC or lPFC activity as a treatment predictor, but the many results implicating ACC and lPFC dysfunction in psychopathology point to the importance of these regions in the conceptualization of psychopathology and in better understanding the processes underlying behavioral change in treatment.

1.8 Current Study

The current study offers a unique opportunity to investigate ACC and dlPFC activity in response to rewarding and aversive feedback in a gambling task with time-frequency amplitude, ITPS, ICPS, and source localization methodology. The innovative

ERP analyses included in this study will allow us to compute ACC and dlPFC activity on sets of trials to make an inference of processing *within* a trial, instead of relying on post-error slowing and dlPFC activity on subsequent trials to infer increased reactive cognitive control in response to ACC activity. The fine timescale of these techniques will also allow us to investigate ITPS on the scale of milliseconds. Because ITPS is hypothesized to index the mechanism behind communication between distant regions, it will be imperative to uncover the exact time ITPS becomes enhanced post-stimulus. If ITPS were indeed the mechanism for communication between the ACC and dlPFC we would predict enhanced ITPS activation would correlate with ACC and dlPFC activation. Additionally, with time-frequency and source localization analyses, modulation of ERP amplitude, ITPS, and ICPS can be calculated for rewarding and aversive stimuli separately and localized to underlying cortical regions.

Because the proposed time-frequency approaches can provide additional information about the timing of ACC and dlPFC interaction within a trial, this study can also evaluate predictions of conceptual models of task performance and psychopathology. Recent integrative theories of task performance focus on context and strategy change as signals for increased ACC and dlPFC activation (Shenhav et al., 2013, 2016; Kolling et al., 2016). These theories predict greater ACC activation and reactive cognitive control processing when the environment signals a change in strategy. Within a gambling task, this signal could be conceptualized as aversive feedback or change in feedback (i.e. loss feedback or gain feedback after consecutive losses) and the resulting change in strategy could be operationalized as a change in the riskiness of subsequent gambles. Once a change in strategy is indicated due to aversive feedback, the individual might become

more risky in their gambling choices in an attempt to explore other potential gambling strategies to obtain reward (Masakia et al., 2006). Finally, though the sample included in this study is not clinical, a range of individual difference measures was collected for each participant. With a range of data on externalizing (aggression, disinhibition) and internalizing (distress, anxiety, depression) dimensions of psychopathology in this sample, this study can investigate differences in the timing of ACC/dlPFC activation and communication (ITPS), depending on self-reported behavioral deficits.

1.9 Hypotheses

Timing of ACC/dlPFC connectivity

Aim: Assess timing of interactions between ACC and dlPFC.

Current time-frequency approaches provide increased resolution for assessing ACC/dlPFC functional connectivity, relative to what has been done before. This can provide information about the nature of ACC and dlPFC activity and what the time lag will be between peak ACC and dlPFC activation

Hypotheses:

- In line with all proposed conceptual models of task performance, it is predicted that ACC activation will precede dlPFC activation.
- Peak ACC activity, as defined by the greatest difference between loss and gain trials on source-localized theta measures, will occur earlier in time than peak dlPFC activity, which will be similarly defined.

Inter-trial Phase Synchrony

Aim: Assess increased ITPS as a necessary component of event related ICPS.

It has been hypothesized that inter-trial synchrony in theta oscillations measures neuronal coherence, which is thought to be integral to functional connectivity; however, little work has assessed inter-trial and inter-channel phase synchrony with high time resolution to assess their relationship during ACC/dlPFC interactions.

Hypothesis:

- Theta-band inter-trial phase synchrony (ITPS) will be enhanced in the same time window as peak ACC and dlPFC activity, as indexed by theta-amplitude and theta inter-channel phase synchrony (ICPS).

Sequence Effects and Change Trials

Aim: Assess ACC/dlPFC interactions relative to sequences of gains and losses.

Task performance models now highlight the importance of task context and behavioral strategies underlying ACC and dlPFC activity. Relative to widely reported increases in ACC/dlPFC activity on trials with aversive feedback, I predict that loss trials will show increased ACC/dlPFC activation compared to gain trials (see hypotheses 1-4 below). Within this, loss trials can be understood to signal a need for alteration in task strategy because the predicted rewarding outcome did not occur. Further, the preceding context can exaggerate or attenuate the impact of a current loss. Relative to this, I predict that valence on preceding trials affects ACC/dlPFC activity, in addition to the valence of the current trial. With additional preceding loss outcomes, the participant will develop a greater urgency to change their current strategy. Therefore, I predict greater ACC/dlPFC activation and implementation of a riskier task strategy on sequences ending in a loss, but with preceding losses (LLL) versus gains (GGL) (see hypotheses 1 and 2 below). Similarly, sequences ending in a gain and preceded by losses (LLG) are hypothesized to

be increased relative to gain outcomes preceded by gains (GGG) (hypotheses 3 and 4), where consecutive rewarding feedback will not signal any need to update task strategy or employ a riskier strategy (hypothesis 4).

Sequences of three trials will be extracted for strategy change analyses. On the third trial in these sequences the following is predicted from greatest to least ACC/dlPFC activation, % risk, and conceptual need for strategy switching.

Hypothesis:

1. Loss – Loss – **Loss** ➔ **Greatest ACC/dlPFC activity, highest % risk, greatest need to switch strategy and obtain reward**
2. Gain – Gain – **Loss** ➔ **Relatively greater ACC/dlPFC activity than LLG sequence, % risk relatively greater than LLG**
3. Loss – Loss – **Gain** ➔**Relatively lower ACC/dlPFC activity than GGL sequence, % risk relatively lower than GGL**
4. Gain – Gain – **Gain** ➔ **Lowest ACC/dlPFC activity, lowest % risk, lowest need to switch strategy**

Psychopathology and Individual Differences

Aim: Assess impact of internalizing and externalizing on ACC/dlPFC.

It is widely predicted that goal-directed behavior and thus task performance processing would be deficient in individuals with high levels of internalizing and/or externalizing symptoms.

Hypotheses:

- In line with previous research detailed above, it is predicted that ACC activity will be increased during task performance for participants high in internalizing and decreased for participants high in externalizing.
- Impaired recruitment of cognitive control resources for both high internalizing and externalizing individuals is predicted, as indexed by decreased dlPFC activity and decreased theta-ICPS.

Chapter 2: Methods

2.1 Participants

Participants (n = 159) were undergraduate students recruited from Florida State University. Two participants were excluded due to a problem with the EEG recording (e.g., experimenter error and software malfunction) and three participants were excluded due to an excessive number of EEG artifacts (>33% of trials rejected using methods described below). The final sample contained 154 participants 18 years of age or older (84 females; M age = 19.52, SD = 1.61). This final sample was not significantly different from the original sample on key demographic variables, including gender and age. Exclusion criteria for participation in this study included uncorrectable visual impairments, diagnosed neurological conditions, and traumatic brain injuries. Participants were provided informed consent before starting the study and were offered monetary compensation ($10/hr) or course credit for participation. Data from the first 50 subjects of this sample were analyzed in a prior study (Aviyente et al., 2017), and basic gain/loss results from this sample have been recently published (Tootell et al., 2018). Timing and individual difference effects as well as cortical source localization analyses have not been previously investigated in this sample (n = 154). These novel analyses are detailed below.

2.2 Questionnaires/Surveys

Participants completed well validated internalizing and externalizing measures. To index internalizing psychopathology, participants were administered the Inventory of Depression and Anxiety Symptoms (IDAS; Watson et al., 2007). The IDAS is a 99-item self-report questionnaire indexing symptoms of panic, social anxiety, generalized anxiety

and depression, and suicidality. Additionally, externalizing symptoms were measured through administration of the 159-item Externalizing Inventory (EXT-159; Krueger et al., 2007). This survey evaluates externalizing symptoms on three subscales: aggression, disinhibition/impulsivity, and substance use behavior. Four additional participants did not complete these questionnaires, leaving data from 150 participants available for psychopathology statistical analyses (see section 2.13).

2.3 Behavioral Data - Percent Risk

For each gambling trial, participant evaluation time and participant choice (left or right box, 5¢ or 25¢) were recorded. Higher risk choices were defined as choosing 25 over the alternative option of 5, regardless of position (i.e. 25 value was in the left or right box). Low risk choices were defined as choosing 5 over the alternative option of 25. Trials that consisted of the same monetary value in both left and right boxes (e.g. 5-5 and 25-25) were removed from the computation of risk. Percent risk for the trial type in question was calculated by dividing the total number of trials by the number of high-risk trials.

2.4 EEG Procedure

EEG data were collected in a sound-attenuated, dimly lit room. Experimental stimuli were presented on a 21-inch Dell high-definition CRT color monitor, centrally placed at a viewing distance of 100 cm and a visual angle of 3.5°. E-Prime version 1.1 was used to present the stimuli, and a PST Serial Response Box (Psychology Software Tools, Inc.) was used to collect responses to the task.

Participants performed a modified version the gambling task (Gehring & Willoughby, 2002), as shown in Figure 1. Each trial consisted of two adjacent squares

presented side-by-side with a number (5 or 25) representing a monetary value inside each square. Participants were instructed to select one of the squares by pressing the left or right button on a button box. The squares remained on the screen until the participant made a selection through their chosen button press. After a selection was made and 100 ms passed, the chosen square turned either green or red to signify a win or a loss (with green or red as the winning color counterbalanced across participants). At the same time, the unselected square turned either green or red to indicate what the outcome of the trial would have been had that square been chosen instead. The colored feedback stimuli remained on the screen for 1000 ms, followed by a blank screen for 1000 ms preceding the onset of the next trial. All combinations of 5 and 25 (i.e., 5-5, 5-25, 25-5, and 25-25) were presented as targets, with each combination occurring an equal number of times in a randomized sequence. The task was composed of 4 blocks of 32 trials. All participants were given $5.00 at the end of the task. Before the task began, participants completed a brief practice to ensure understanding of the task.

2.5 Psychophysiological Data Acquisition

Data were recorded using a Neuroscan 128-channel Quik-Cap (sintered Ag-Ag/Cl; non-standard layout) and a 128-channel Synamps RT amplifier (Neuroscan, Inc.). Ten electrodes around the ears were removed from analysis due to inconsistent connection to the scalp across participants, leaving a total of 113 EEG channels available for analysis. Horizontal electrooculogram activity was recorded from electrodes placed on the outer canthus of both eyes, while vertical electrooculogram activity was recorded from electrodes placed above and below the left eye. Impedances were kept below 10 kΩ. EEG signals were vertex referenced during recording (directly between Cz and CPz), and re-

referenced to averaged mastoid signals offline, collected using an analog 0.05 to 200 Hz bandpass filter and digitized at 1000 Hz using Neuroscan Acquire (Neuroscan, Inc.).

2.6 Data Preprocessing for Source Analysis

Epochs of three seconds were then taken from 1000 ms pre- to 2000 ms post-feedback stimulus onset with a 150 ms pre-stimulus baseline and were re-referenced to averaged mastoid sites. Ocular artifacts were corrected with a regression-based algorithm developed by Semlitsch, Anderer, Schuster, & Presslich (1986) in the Neuroscan Edit 4.5 software (Neuroscan, Inc.) and downsampled to 128 Hz using the Matlab resample function (Mathworks, Inc.), which utilizes an anti-aliasing filter before resampling. Data were further cleaned according to the following criteria. To remove larger face or eye artifacts not appropriately handled by the Semlitsch algorithm, single trials were rejected if activity at F3 or F4 exceeded ±100 μV in either the pre-stimulus period of -1000 to -1 ms, or the post stimulus period of 1 to 2000 ms. Further trials were rejected if activity in any electrode exceeded ±150 μV during the same pre- and post-stimulus time periods. With these data cleaning criteria applied, 11% of all trials were removed from analysis. Visual analysis of the averaged waveforms indicated that 0.005% of electrodes were disconnected during recording and were replaced with the mean of the nearest neighbors.

2.7 Cortical Source Localization

The spatial distributions of the ERP data at each of these time points were then calculated through cortical source localization in the open-source FieldTrip program (Oostenveld et al., 2011). Cortical source localization provides the likely source dipole or dipoles that could potentially generate electrical current that would fit with the measured

ERP data at the scalp electrodes (Reynolds & Richards, 2008). This process is done through equivalent current dipole analysis (ECD), which analyzes the fit between the observed scalp current and hypothetical source dipoles (Reynolds & Richards, 2007; DeLorme, et al., 2002; Jung et al., 2001; Michael et al, 2004; Huizenga & Molenaar, 1994; Scherg, 1990, 1992; Scherg & Picton, 1991). For each of these hypothetical dipole sources, a forward solution is calculated to create the current distribution at scalp electrodes that matches the hypothetical dipole location. This hypothetical scalp distribution is then compared to the observed scalp potentials. The difference between the hypothetical and observed scalp potentials is then minimized through an iterative process of modifying the source dipole. Thus, the final computed source dipole solution is one that generates a hypothesized scalp distribution that most closely resembles that of the observed data (Reynolds & Richards, 2007). This iterative process can further be improved by placing a minimum norm constraint on the dipole solution. Applying a minimum norm constraint significantly cuts down the infinite available solutions to those that fit the observed data with the least amount of energy. Several algorithms have been developed to serve this purpose, such as low-resolution brain electromahnetic tomography (LORETA), standardized LORETA, and exact LORETA (Pascual-Marqui et al., 1992; Pascual-Marqui et al., 2002; Pascual-Marqui, 2002; Pascual-Marqui, 2007; Pascual-Marqui et al., 2011). Due to evidence that sLORETA and eLORETA provide more accurate constraints on analyses than the original LORETA algorithm (Ding et al., 2004; Wagner et al., 2004; Bradley et al., 2016), and that eLORETA provides less-blurred spatial solutions than those of sLORETA (Wagner et al., 2008; Jatoi et al., 2014), eLORETA was used in the source localization analyses in this study.

In order to generate dipole solutions with the most accurate hypothesized current scalp distribution, ECD analyses require information about head and brain region locations and the specific electrode placement on the scalp (Michel et al., 2004). For our source localization analysis, we utilized the standard Montreal Neurological Institute (MNI) structural MRI, averaged from 152 healthy adults, as our model of head and brain region locations (Collins et al., 1994). Accurate electrode placement for the Neuroscan 128-channel Quik-Cap was generated through recording the exact electrode placement on the head through Polhemus FASTRAK® digitization software (Polhemus, Colchester, VT, USA). Electrode placement was digitized for 12 individuals and then averaged to create a 3D electrode map that represented the likely placement of each electrode relative to the head. For each individual, 7 fiducials (nasion, inion, preauriculars, mastoids, and vertex) were recorded before digitizing each electrode to better orient the electrodes in 3D space relative to the individual's head. Integration of the likely placement of electrodes on the scalp and the cortical structure of brain regions in aged-matched individuals allows for more accurate dipole solutions in our ECD analyses (Reynolds & Richards, 2005; Reynolds & Richards, 2008). A visual representation of the source analysis process can be seen in Figure 2.

2.8 Selection of Regions of Interest (ROIs)

Source localization techniques can produce dipole solutions for each voxel created in the source model. However, the time and computational effort necessary to compute such results increases as the number of voxels increases. Thus, it is recommended that individual voxel size not become too small (5x5x5mm voxels were chosen for this study) and voxels utilized in the analysis be limited to brain regions of theoretical interest. Due

to a priori hypotheses in this study, voxels associated with the ACC and dlPFC were extracted for source localization and subsequent time-frequency analysis. That said, research investigating the function of these regions, the ACC in particular, have included a variety of spatial definitions. As described in section 1.4 of this paper, some researchers believed the conflicting findings of the function of the ACC in feedback processing were due to the low spatial resolution of previous studies. Ensuing high-resolution fMRI studies localized feedback-related processing to dorsal, anterior, and posterior regions of the ACC and even as posterior as the pre-SMA (Kiehl et al., 2000; Braver et al., 2001; Ulsperger et al., 200; Garavan et al., 2003). Due to these varied results, this study did not have an a priori hypothesis regarding the specific sub-regions of the ACC and their respective feedback processing functions. Therefore, multiple ROIs were selected for ACC and dlPFC areas to provide a comprehensive spatial analysis of these functional regions.

ROIs were selected using the Brainnetome Atlas (https://atlas.brainnetome.org/bnatlas.html), which was constructed from an average of 20 healthy individuals (10 male, 10 female) from ages 19-25. The Brainnetome Atlas was selected for this study due to the congruence in age between the individuals used to create this atlas and the participants in our study. Additionally, the Brainnetome Atlas includes relatively large ROIs based on functionally defined Brodmann areas, which well-suited the aims of this study. A full picture of the parcellation of regions within the Brainnetome Atlas can be seen in Figure 3. Brainnetome ROIs chosen to encompass functional ACC and dlPFC regions in this study are listed in Table 1. These ROIs were selected based on visual inspection of the atlas as well as research regarding the Brodmann areas most associated with the ACC and

dlPFC to create ROIs as comprehensive as possible. Figures 4 and 5 display sagittal and coronal views, respectively, of these chosen Brainnetome regions. Research showed that the ACC was most associated with Brodmann areas 24 and 32 (Tölle et al., 1999; MacDonald et al., 2000; Peyron et al., 2000; Pizzagalli et al., 2001, 2003, 2006; Kitayama et al., 2006; Palomero-Gallagher et al., 2009) and the dlPFC was associated with areas 9 and 46 (MacDonald et al., 2000; Fassbender et al., 2006; Zhou et al., 2007; Grimm et al., 2008). Note that the Brainnetome regions depicted in Table 1 include numbers that are representative of these Brodmann areas. Source analysis was computed for each ROI included in Table 1 and Figures 4 and 5.

2.9 Subsampling of Source Waveforms

ERP waveforms computed through the source analysis were then prepared for time-frequency analyses. Subsampling is a method that is particularly useful for equating the number of trials in each subaverage during processing, thus handling the uneven numbers of trials across outcome types and participants due to data cleaning and artifact rejection. An equal number of trials for each outcome type is crucial for inter-channel phase synchrony analyses (see section 2.12) because these analyses are biased by trial numbers with greater trials resulting in smaller inter-channel phase synchrony values. Because samples within each waveform contain dependencies, full waveforms were the unit on which subsampling was performed (e.g. all samples within each waveform were treated together in the process). Subsampling involved making multiple averages for each condition (50), each from a random subset of trials (20) for each outcome (i.e., 50 subsampled averages, each created from 20 randomly selected trials, with replacement, were produced).

2.10 Time-Frequency Amplitude.

To evaluate the time-frequency (TF) evoked power, TF decompositions were performed on condition averages (as detailed above in section 2.9). The goal in starting with condition averages is to use the same ERP activity conventionally studied using time-domain components. This approach has been used in previous work (Bernat et al., 2011; Harper et al., 2016, 2014; Nelson et al., 2011). First, 3rd order Butterworth filters were used to isolate activity the theta frequency range, based on the visual inspection of the unfiltered representation of time-frequency energy following the outcome stimulus for one second. A 2 Hz highpass filter in conjunction with an 8 Hz lowpass filter were used to isolate the theta frequency. TF transforms were produced using a binomial reduced interference distribution (RID) variant of Cohen's class of time-frequency transformations, using the full epoch of the filtered signals (-1 s to 2 s, relative to feedback stimulus onset). To statistically investigate timing effects, the time-frequency distribution was computed at a high time resolution of 128 time bins per second and 2 frequency bins per Hz. Figure 6.a displays the grand average theta-filtered amplitude (AMPL) decomposition for gain and loss trials. Within the theta-filtered TF decomposition, TF windows (shown in Figure 6.c) were chosen and extracted for statistical analyses. A larger FN-P3 window encompassing theta TF power during time-domain FN and P3 components was extracted to analyze gain/loss differences over time. Smaller TF windows, designated the FN_{Theta} and $P3_{Theta}$ windows, were chosen for statistical analyses detailed in section 2.13 to investigate differences in feedback processing across TF measures (AMPL, ITPS, and ICPS) between the FN and P3 time components. The FN_{Theta} and $P3_{Theta}$ windows were defined by peak gain/loss differences in TF-ITPS (described in section 2.11).

2.11 Time-frequency ITPS

Average inter-trial phase synchrony (ITPS) was computed separately for gain and loss trials in the theta frequency range. Creating these averages involved taking a set of trials, and computing the phase difference between each trial and the average phase across trials, and then averaging the phase differences to create a phase locking value (PLV) across the trials (Aviyente et al., 2011). This process was conducted iteratively using the same subsampling approach defined in section 2.9, producing condition average ITPS surfaces of the same dimensions as the amplitude measures, for each ROI within participants. Visual inspection of the ITPS TF surface revealed significant gain/loss differences under the FN time component and the rise of the P3 time component (see Figure 6.b) Due to a priori hypotheses regarding the necessity of ITPS in communication among disparate brain regions (described in section 1.6), significant gain/loss activity within TF-ITPS determined the TF windows (Figure 6.c) chosen for statistical analyses in AMPL, ITPS, and ICPS measures.

2.12 Time-frequency ICPS

Cognitive control functional network activity was assessed through theta-filtered inter-channel(in this case region) phase synchrony (ICPS) within and between ACC and dlPFC regions consistent with our work (Aviyente et al., 2017; Moran, Bernat, Aviyente, Schroder, & Moser, 2015) and others (Cavanagh et al., 2009). Theta-band ICPS was calculated within ACC ROIs, within dlPFC ROIs, and between ACC and dlPFC regions. Seed ROIs for ACC and dlPFC regions were selected through visual inspection of maximal loss-gain AMPL differences throughout the FN/P3 window (ACC seed - A24cd_r; dlPFC seed - A8dl_l; see **Appendix A** for loss-gain difference plots for individual ROIs used for

seed selection). Thus, theta-ICPS was computed between 1. ACC seed A24cd_r and the average of the remaining ACC ROIs 2. dlPFC seed A8dl_l and the average of the remaining dlPFC ROIs and 3. ACC seed A24cd_r and the average of all dlPFC ROIs. For all ROIs, theta-ICPS was calculated through phase synchrony computations based on Cohen's class of time-frequency distributions (Aviyente et al., 2011). As with ITPS computation, the time-frequency windows chosen for the theta amplitude measure were applied to the ICPS measure, targeting phase synchrony within the TF windows depicted in Figure 6.c.

2.13 Data Analytic Plan

The source localization analyses detailed in sections 2.7 and 2.8 computed ERP waveforms in each of our selected ROIs at each pre-selected time point post-stimulus. Time-frequency and phase analyses (see sections 2.10 – 2.12) were then applied to these waveforms, generating theta-filtered AMPL, ITPS, and ICPS measures within each ROI included in Table 1 and Figures 4 and 5. Gain and loss activity within these measures was visually inspected to determine the ROIs appropriate for subsequent statistical analyses of ACC and dlPFC activity. Inspection of feedback processing in these ROIs revealed similar patterns of gain and loss activity within all ROIs associated with ACC and dlPFC regions, respectively (refer to loss-gain difference plots of individual ROIs in appendix). Therefore, AMPL, ITPS, and ICPS activity was averaged across all ROIs within the ACC and dlPFC regions. These averaged ACC and dlPFC ROIs were used for subsequent statistical analyses detailed below.

To investigate hypothesized differences in timing of peak ACC and dlPFC activity within AMPL, ITPS, and ICPS measures, t-tests between average activity on loss and gain trials for each measure were computed at every time point within FN-P3 time-

frequency window (see Figure 6.c). The t-values at each time point for each measure were then plotted to visually analyze the differential activation in ACC and dlPFC regions throughout the FN/P3 time window. To statistically investigate differences in timing among AMPL, ITPS, and ICPS measures for each region, time-frequency windows corresponding to peak gain/loss differences in theta-ITPS were extracted, designated FN_{Theta} and $P3_{Theta}$ windows (see Figure 6.c). A 2x3 repeated measure ANOVA was computed to assess differences in feedback processing among each measure between the FN_{Theta} and $P3_{Theta}$ windows in the averaged ACC and dlPFC ROIs. Post-hoc analyses were calculated to further investigate the effects of feedback type (gains and losses), ERP measure (Theta-AMPL, ITPS, and ICPS), and time window (FN_{Theta} and $P3_{Theta}$).

To evaluate the effect of context and task strategy on feedback processing in the ACC and dlPFC ROIs 2x3 repeated measures ANOVAs were computed for each theta measure with feedback (gain, loss) and feedback sequence (the first, second, and third gain after a loss/first, second, and third loss after a gain) as within-subject factors. Percent risk values were included in the 2x3 ANOVAs as a numerical covariate to analyze the association of feedback sequence and measure with a behavioral measure of risky gambling decisions. Bayesian 2x3 repeated measures ANOVAs (feedback x sequence) were computed to further investigate the likelihood of interaction, main effect, and null models.

To evaluate the effect of individual differences in internalizing and externalizing symptoms on AMPL, ITPS, and ICPS measures over time, scores on internalizing and externalizing measures (IDAS and EXT-159, respectively) were correlated with average activity on gain and loss trials within FN_{Theta} and $P3_{Theta}$ time windows. Correlations

among psychopathology and ERP measures were computed for averaged ACC and dlPFC ROIs (AMPL & ITPS) and functional connectivity between the seed ACC ROI and the averaged dlPFC ROI (ICPS). Bayesian Pearson correlations between individual difference scores and ERP amplitude and phase measures were computed to estimate the likelihood of alternative versus null models.

The Bayesian analyses included in this study calculated posterior probabilities that estimated the likelihood of the null (H_0) and alternative models (H_1) given the observed data (Masson, 2011). Posterior probabilities that provided support for the alternative model are displayed as $p(H_1|D)$, while posterior probabilities that support the null model are displayed as $p(H_0|D)$. Posterior probabilities can range from 0 (no evidence for the model) to 1 (very strong evidence for the model). The current standard for interpreting these values dictates that $p(H_1|D)$ values between .50-.75 represent weak evidence for the model, values between .75-.95 represent support for the model, values between .95-.99 are strong support for the model, and values >.99 are strong support for the model (Rafferty, 1995). Inclusion of Bayesian statistical analyses allows for a more complete investigation of effects due to the interpretation of posterior probabilities on a continuum of likelihood as opposed to the either/or nature of p-value significance. Further, Bayesian analyses can incorporate previous data in the field through modulation of prior likelihoods.

Chapter 3: Results

3.1 Timing differences in ACC and dlPFC activity

Figure 7 presents the results for feedback processing in theta-AMPL, ITPS, and ICPS during FN-P3, FN_{Theta}, and $P3_{Theta}$ time windows. Line plots were used to display loss-gain t-values at each time bin (i.e. approximately every 8ms from 175-420ms post feedback presentation) within the FN/P3 window for AMPL, ITPS, and ICPS measures. Bar charts exhibit loss-gain t-values for each ERP measure within the FN_{Theta} and $P3_{Theta}$ windows, which were used in the statistical analyses discussed below. As described in sections 2.12 and 2.13, AMPL and ITPS activity within these windows was averaged separately across all ACC and dlPFC ROIs for statistical analysis, while ICPS functional connectivity was assessed within the ACC region, within the dlPFC region, and between ACC and dlPFC regions. The line and bar plots computed for each of these averaged ROIs can be seen in figure 7 a. b. and c.

Overall 2x3 ANOVAs were performed to assess the effects of time post-feedback and time-frequency measure on gain/loss differences in averaged ACC and dlPFC ROIs. Due to the large magnitude difference between theta-AMPL values and theta-ITPS and ICPS values, all gain/loss differences for the three measures were converted to z-scores prior to ANOVA analyses. In the 2x3 ANOVA for the ACC region, the interaction between time (FN_{Theta} and $P3_{Theta}$ windows) and theta measure (AMPL, ITPS, and ICPS) was not significant ($F(2,154)=0.63$, $p=.53$, $p(H_0|D)=0.99$). Interpretation of main effects models revealed no significant effect of theta measure but a significant effect of time on gain/loss differences (see Table 2). Post-hoc Bonferroni corrected t-tests were computed to parse the nature of gain/loss differences among the three theta measures. For all three

theta measures, feedback-related ACC activity was greater in the later $P3_{Theta}$ window compared to the earlier FN_{Theta} window (Theta-AMPL: $t(154)=3.72$, $p<.001$; Theta-ITPS: $t(154)=5.03$, $p<.001$; Theta-ICPS: $t(154)=4.57$, $p<.001$, see Figure 7 a.).

The overall 2x3 ANOVA within the dlPFC ROI also demonstrated no significant interaction between time and theta-measure ($F(2,154)=0.12$, $p=.88$, $p(H_0|D)=0.99$), yet a significant main effect of time (see Table 3). Similar to patterns seen in the ACC region, paired t-tests in the dlPFC region showed greater dlPFC activation during the $P3_{Theta}$ window in comparison to the FN_{Theta} window (Theta-AMPL: $t(154)=3.42$, $p=.009$; Theta-ITPS: $t(154)=2.87$, $p=0.048$; Theta-ICPS: $t(154)=2.87$, $p=0.049$, see Figure 7 b.).

In addition to assessing the functional connectivity within ACC and dlPFC ROIs, ICPS between ACC and dlPFC regions was computed. In line with the results for within region ICPS, ICPS between ACC and dlPFC ROIs was greater during the $P3_{Theta}$ window than the FN_{Theta} window ($t(154)=5.25$ $p<.001$; see Figure 7 c.).

These results indicate several patterns of feedback processing throughout the FN/P3 time window. In both ACC and dlPFC regions, gain/loss differences in theta-AMPL were seen early during the FN_{Theta} time window and were sustained through the $P3_{Theta}$ window. Feedback-related activity in ITPS and ICPS measures in both ACC and dlPFC regions peaked later during the $P3_{Theta}$ window. Interestingly, theta-ITPS in ACC and dlPFC regions exhibited a "double-peak" pattern of feedback-related activity during the FN/P3 time window (displayed in the left column of figure 7). Gain/loss differences in ITPS peaked once during the FN time-domain window and again during the rise of the P3 time-domain window to a greater degree. Further discussion of this "double-peak" pattern and its potential role in inter-region communication during feedback processing is

discussed in section 4.1.

3.2 Effect of task context on feedback processing in ACC and dlPFC

To evaluate the effects of task context on feedback processing in time-frequency measures, 2x3 ANOVAs were conducted within the FN_{Theta} and $P3_{Theta}$ windows. These ANOVAs included feedback (i.e. gains and losses) and sequence (i.e. the first, second, or third consecutive feedback of that type) as within-subjects factors. Participant percent risk was included in these analyses as a covariate. Because percent risk did not have a significant effect in any of the subsequent analyses, percent risk is not included as a predictor in the following results. ANOVAs were computed within the FN_{Theta} and $P3_{Theta}$ windows for ACC and dlPFC ROIs. Full results of these ANOVAs for theta-AMPL, theta-ITPS, and theta-ICPS measures can be seen in Tables 4, 5, and 6.

The interaction between feedback and sequence was not significant across all measures and ROIs (see Tables 4, 5, and 6). Additional Bayes analyses further supported the null hypothesis in these analyses, indicating a significant interaction between feedback and sequence was not likely (evidence for the null hypotheses ranged from $p(H_0|D)=0.98$ to $p(H_0|D)=0.99$ for all ANOVAS computed). Main effects of feedback were significant for all measures, with loss feedback eliciting greater theta-AMPL, ITPS, and ICPS relative to gain feedback (see Figures 8, 9, and 10 and Tables 4, 5, and 6). In contrast, main effects of sequence were significant in theta-AMPL analyses (see Table 4), but not in theta-ITPS or ICPS. Post-hoc Bonferroni corrected t-tests were computed to better understand the sequence main effect in theta-AMPL. Paired t-tests in all four AMPL ANOVAs revealed no significant differences between the first and second feedback trials in sequence for gains and losses (ACC/FN_{Theta}: gain $t(154)=1.00$, p=.92, loss $t(154)=0.60$, p=.99; ACC/$P3_{Theta}$: gain $t(154)=0.50$, p=.99, loss $t(154)=1.50$, p=.68;

dlPFC/FN_{Theta}: gain $t(154)=2.10$, p=.28, loss $t(154)=0.60$, p=.99; dlPFC/$P3_{Theta}$: gain $t(154)=1.70$, p=.56, loss $t(154)=0.90$, p=.94), but significant differences between the first/second trials and the third consecutive feedback in the sequence (third vs. first trials: ACC/FN_{Theta}: gain $t(154)=6.00$, p<.001, loss $t(154)=6.10$, p<.001; ACC/$P3_{Theta}$: gain $t(154)=3.00$, p=.03, loss $t(154)=6.10$, p<.001; dlPFC/FN_{Theta}: gain $t(154)=7.70$, p<.001, loss $t(154)=6.70$, p<.001; dlPFC/$P3_{Theta}$: gain $t(154)=6.70$, p<.001, loss $t(154)=6.40$, p<.001). Post-hoc analyses showed that the third gain and loss in the sequence elicited greater theta-AMPL activity than the first two (see Figure 8).

Overall, results of the sequence x feedback ANOVAs for theta measures indicate no significant interaction between feedback valence and feedback sequence. Feedback-related activity in all theta measures demonstrated greater ACC and dlPFC activity for loss trials relative to gains in congruence with timing analyses described in section 3.1. Sequence effects were found only in ACC and dlPFC theta-AMPL activity, in which the third consecutive gain or loss in the sequence exhibited significantly greater activation than the first two.

3.3 Association of ACC/dlPFC activity with psychopathology

Finally, the relationship between feedback processing in the ACC and dlPFC and internalizing and externalizing psychopathology was assessed. Pearson correlations were computed between ERP activity on gain and loss trials and IDAS and EXT-159 scores within FN_{Theta} and $P3_{Theta}$ time windows. Spearman rank-order correlations were also computed to investigate the potential influence of outlier scores. Since correlation values computed through Pearson and Spearman methods were consistent, only the results of the Pearson correlations are included below.

Within the averaged ACC ROI, theta-AMPL activity on gain trials was not significantly correlated with participant scores on the IDAS or EXT-159 measures. Similarly, there was not a significant correlation between AMPL activity on loss trials and IDAS scores. However, Pearson correlations on loss trials revealed a significant negative association between EXT-159 scores and AMPL activity during the FN_{Theta} window and a trend-level negative association during the $P3_{Theta}$ window (FN_{Theta} $r(148) = -.18$, p=.023; $P3_{Theta}$ $r(148) = -.14$, p=.09). Bayesian Pearson correlations revealed weak evidence for alternative models of a significant association between externalizing and AMPL loss activity (FN_{Theta}: $p(H_1|D)=0.69$; $P3_{Theta}$: $p(H_1|D)=0.43$). Within the averaged dlPFC ROI, correlations between theta-AMPL activity on gain and loss trials and IDAS and EXT-159 scores were not significant.

Correlations between theta-ITPS activity on gain and loss trials and psychopathology were also computed within ACC and dlPFC regions. Within the ACC ROI, there was a significant negative correlation between ITPS activity and EXT-159 scores on loss trials during the $P3_{Theta}$ window ($r(148) = -.19$, p=.02). Correlations of ITPS activity and psychopathology within the dlPFC ROI revealed significant negative associations in the $P3_{Theta}$ window between activity on gain trials and IDAS scores ($r(148) = -.16$, p=.045) as well as activity on loss trials and EXT-159 scores ($r(148) = -.18$, p=.034). Bayes analyses for these significant correlations suggested weak support for the alternative models (ACC/EXT: $p(H_1|D)=0.57$; dlPFC/IDAS: $p(H_1|D)=0.57$; dlPFC/EXT: $p(H_1|D)=0.62$).

To assess the association between theta-ICPS and psychopathology measures, correlations were computed for ICPS within ACC ROIs, within dlPFC ROIs, and

between ACC and dlPFC ROIs. Pearson correlations did not reveal significant associations between feedback processing and externalizing/internalizing for any of these ICPS measures. Overall, correlation results suggest limited association between feedback processing in theta ERP measures and internalizing and externalizing scores. Associations were strongest within theta-AMPL measures, in which higher externalizing scores were significantly correlated with reduced AMPL activity on loss trials.

Chapter 4: Discussion

4.1 Results Summary

The current study used RID time-frequency and cortical source localization methodology to investigate feedback processing and recruitment of cognitive control resources within a brief time window (100-200 ms). Past fMRI work regarding feedback processing and cognitive control has provided better understanding of the spatial distribution of these processes. Additionally, time-domain ERP studies have evaluated differences in temporal dynamics of feedback processing while utilizing indirect measures of prefrontal recruitment (i.e. post-error slowing). This study aimed to closely assess the timing of dynamics in valenced feedback and cognitive control processing through analysis of TF-ERP activity localized to ACC and dlPFC ROIs and computed on the scale of milliseconds. The strengths of this approach allowed the evaluation of prevailing conceptual models of feedback processing, recruitment of the cognitive control network, and behavior modification during task performance, such as PRO and EVC theories (detailed in section 1.5), without the use of post-error slowing measures. Further, our methods allowed us to investigate the nascent proposition that inter-trial phase synchrony (ITPS) indexes coherence, which may be a key mechanism facilitating communication between disparate brain regions.

According to these current models of task performance and behavior modification, we outlined several predictions for ERP measures of feedback processing and cognitive control. Firstly, these task performance models theorize that the ACC processes task feedback and analyses the need for subsequent behavior modification. Based on this cost/benefit decision, the ACC then communicates with the dlPFC and

recruits cognitive control resources if appropriate. Thus, it was hypothesized that peak gain/loss activity in the ACC would precede peak gain/loss activity in the dlPFC, as indexed by theta-AMPL and theta-ICPS measures respectively. Our second hypothesis evaluated neuronal coherence as a potential component of inter-region communication. This theory posits that in order for disparate brain regions to communicate, the oscillating nature of neuronal activity must first be firing in synchrony to "open the window" for possible information input and output (Fries, 2005). Therefore, the current study predicted that theta-ITPS, a measure of neuronal synchrony, would peak as communication between disparate regions peaked (theta-ICPS).

In addition to these hypotheses assessing the validity of current models of task performance, this study also investigated the impact of feedback context and participant psychopathology on feedback processing and cognitive control recruitment. With previous research in mind (see sections 1.2-1.7), it was predicted that certain feedback characteristics would affect feedback-related activity in ACC and dlPFC regions. The EVC model in particular proposes that feedback which signals a need for a switch in task strategy would result in enhanced ACC and dlPFC activity. Feedback signaling a change in task strategy would include undesired outcomes, such as monetary loss. This effect would likely be compounded as the number of consecutive undesirable outcomes increased and the need to switch task strategy grew. Therefore, it was hypothesized that loss relative to gain feedback would result in enhanced ACC and dlPFC activity (as indexed by theta ERP measures) and the third consecutive loss in a sequence of losses would result in greater ACC and dlPFC activity than the first loss. Conversely, sequences of gains would result in the opposite pattern of ACC and dlPFC activity with the third

gain in sequence would elicit the least amount of activity. Due to the efficacy of the current task strategy in obtaining desirable after desirable outcome, there would be no need for extensive feedback processing and recruitment of costly cognitive control resources.

Finally, this study assessed the influence of externalizing and internalizing psychopathology on ACC and dlPFC feedback-related activity. Based on previous psychopathology ERP research outlined in section 1.7, it was hypothesized that feedback processing in the ACC would be enhanced in those high in internalizing and blunted in those high in externalizing. Further, due to deficits in goal-directed behavior in those high in externalizing and internalizing, we predicted that dlPFC activity and recruitment of cognitive control resources would be negatively associated with high scores on externalizing and internalizing measures.

Results of this study largely supported the hypotheses founded on the conceptual theories outlined in the EVC and other prevalent task performance models. Peak gain/loss theta-AMPL activity in the ACC did precede peak gain/loss theta-ICPS during the rise of the P3 time component, suggesting that feedback processing in the ACC occurred prior to communication between the ACC and dlPFC, as predicted. However, significant gain/loss theta-AMPL activity in the dlPFC also occurred earlier in the FN time window and prior to peak theta-ICPS, which was not explicitly predicted by the EVC model. Our results suggest that significant feedback-related processing occurred in *both* the ACC and dlPFC during the FN window prior to recruitment of communication between these regions. Implications of this result for the EVC and other current models are discussed in section 4.2.

Evaluation of theta-ITPS results revealed a "double-peak" pattern of gain/loss activity within the FN/P3 window, peaking during the FN and the rise of the P3 (see Figure 7). Further, feedback-related activity in theta-ITPS was significantly greater during the later time window, which coincided with peak gain/loss activity in theta-ICPS. Therefore, theta-ITPS demonstrated an oscillatory pattern of activation with peak activation concurrent with our measure of communication between ACC and dlPFC regions. These findings are congruent with theories of inter-region communication, in which neuronal coherence, or synchrony, is a prerequisite for communication between neuronal populations (Fries, 2005; Uhlhaas & Singer, 2006; Arenas et al., 2008). Overall, these results inspire confidence in the validity and utility of using ITPS as a measure of neuronal synchrony and the opportunity for inter-region communication.

In contrast, results regarding feedback sequence and the impact of psychopathology on feedback processing and cognitive control were weak, failing to provide additional evidence for components of task performance and behavior modification theories. Based on the theorized importance of task context on the processing of specific task feedback, it was hypothesized that feedback on previous trials would influence the context of the current trial. Contrary to these predictions, feedback sequence did not show any effect on feedback processing in theta-ITPS or theta-ICPS measures in ACC or dlPFC regions. Only in theta-AMPL did feedback sequence impact activity, yet not exactly as hypothesized. In both ACC and dlPFC regions, the third feedback in the sequence elicited significantly greater theta-AMPL than the first two. However, this effect was not modulated by feedback valence as predicted; both gain trials and loss trial evinced this pattern. Since the EVC model proposes that feedback which

signals the greatest need for a switch in task strategy results in the most feedback and cognitive control processing, it was predicted that only the third consecutive loss, and not gain, would elicit enhanced ERP activation. Conceptually, three consecutive gains would signal successful task performance with no need to recruit costly resources to change strategies in order to improve performance. These surprising results will be further discussed in the next section.

Finally, results assessing the relationship between psychopathology and ERP measures of feedback processing were not robust, but significant correlations were in the predicted direction. Higher externalizing scores were associated with blunted activity on theta-AMPL and theta-ITPS measures in line with previous research. There were no significant associations between internalizing or externalizing scores and theta-ICPS, which indexed processing in the cognitive control network.

4.2 Implications for Feedback Processing and Cognitive Control Theories

The results described above offer support for many aspects of the current conceptual theories of feedback processing and cognitive control during task performance. Present models of task performance go beyond describing the ACC as a processor of feedback valence or conflict and instead conceptualize the ACC as a cost-benefit analyzer, using on-going feedback to inform behavior on subsequent trials (PRO model: Alexander & Brown, 2011; FVT model: Kolling et al., 2016a; Kolling et al., 2016b; EVC model: Shenhav et al., 2013; 2016). According to FVT and EVC models (introduced in section 1.5), the ACC integrates on-going feedback information to decide whether to continue utilizing the current task strategy or alter the task strategy in the chance of improving task performance. Switching task strategies involves recruiting

costly, top-down cognitive control resources in the prefrontal cortex in order to effectively modify behavior on the next trial in accordance with the new strategy. Thus, feedback that is likely to evoke a desire to switch strategies (i.e. undesirable outcomes such as losing money or committing an error) results in enhanced processing in the ACC and dlPFC regions. These current models explicitly differentiate the role of the ACC from cognitive control processes in the prefrontal cortex, proposing a "call and response" mechanism of cognitive control recruitment; the ACC processes on-going feedback and calls the PFC for cognitive control resources when needed. Therefore, ACC activity is hypothesized to always precede activity in dlPFC regions.

With the fine temporal resolution of the theta-ERP measures in this study, we were able to assess these proposed dynamics in ACC and dlPFC activity. As predicted by the EVC model, feedback-related activity in the ACC preceded communication between ACC and dlPFC regions, as indexed by peak feedback-related activity in theta-ITPS and theta-ICPS. Additionally, significant feedback-related activity was also seen in dlPFC regions during the FN window prior to peak theta-ICPS activation. These results suggest that initial feedback processing occurred in both ACC and dlPFC regions before the "window" of communication was open later between these regions. Therefore, communication during the P3 time window might encapsulate a more two-way communication between ACC and dlPFC regions as opposed to the one-way "call and response" communication proposed by the EVC model. In the EVC model the ACC is conceptualized as the main processer of feedback information and cost/benefit analyzer of whether further dlPFC cognitive control processing is necessary. However, if significant feedback processing occurs in the dlPFC prior to the proposed recruitment of

cognitive resources, the dlPFC may have a larger role in this cost/benefit decision than previously described in the EVC model.

Not only do modern models contain hypotheses regarding the timing of ACC and dlPFC activation post-feedback, but they also discuss aspects of feedback that would differentially elicit ACC and dlPFC activity. This is particularly clear in the EVC model (Shenhav et al., 2013; 2016) which highlights the importance of the broader context of the task on how the ACC processes individual feedback. In this theory the ACC takes into account context to evaluate whether a switch in strategy and behavior modification is needed on the next trial. To make the subsequent modification more effective, the ACC recruits cognitive control resources to ensure proper behavior execution. The important question then becomes what feedback would necessitate a change in task strategy? Broadly, outcomes that interfere with the task's ultimate goal, such as monetary losses or errors, would warrant processing in the ACC devoted to whether a switch in task strategy were appropriate. This prediction has been supported by many ERP studies concerning feedback processing (Miltner et al., 1997; Gehring & Willoughby, 2002; Bush et al., 2002; Holroyd & Coles, 2002; Eisenberher et al., 2003; Neieuwenhuis et al., 2004; Cavanaugh et al., 2009; Shackman et al., 2011; Bernat et al., 2011; Van de Vijver et al., 2011; Bolanos et al., 2013; Moran et al., 2015; Bernat et al., 2015; Aviyente et al., 2017) and was again confirmed in this study through enhanced ACC and dlPFC activity on loss trials relative to gains. In addition to assessing the effect of feedback valence, this study investigated the effect of the valence of previous feedback trials on processing of current feedback. It was hypothesized that previous feedback would influence the complex cost/benefit analysis of whether to change task strategy on subsequent trials: the more

consecutive loss feedback, the more attractive a switch in task strategy would become. Results in this study did not fully support this hypothesis, however. Enhanced feedback processing on the third consecutive loss was only seen in theta-AMPL measures and not in theta-ITPS or ICPS. Furthermore, enhanced theta-AMPL was seen on the third consecutive gain trial compared to the first two gains in sequence, which was not predicted by the EVC model. Within a strategy-switching framework, the third consecutive gain would not be least likely to prompt a change of task strategy because there is no need to improve task performance.

These results do not necessarily negate aspects of the EVC theory. More likely the design or methodology of this study failed to manipulate task context as hoped. First, sequence effects in this sample have not been evaluated outside of source space, so further analysis in this sample would be beneficial to confirm these effects. Next, the specific experimental design of this gambling task might also have influenced task context and sequence results. Other studies in our lab have investigated the effect of feedback sequence on feedback processing through as many as eight consecutive trials (Bachman et al., in review). Perhaps, sequences of three trials were not long enough to create a significant context effect on current feedback processing. In the future it will be important to assess task context effects through varied design manipulations (see section 4.6).

4.3 Clinical Implications

Evaluation of the relationship between psychopathology and the ERP measures of feedback processing included in this study did not culminate in overwhelming results. However, significant associations between externalizing psychopathology and feedback-

related processing in the ACC add to the list of research describing such a relationship (see section 1.7; Justus et al., 2001; Iacono et al., 2003; Bauer & Hesselbrock, 2003; Kaufman et al., 2003; Forman et al., 2004; Ruchsow et al., 2005; London et al., 2005; Gruber & Yurgelun-Todd, 2005; Patrick et al., 2006; Potts et al., 2006; Franken et al., 2007; Goldstein et al., 2007; Fein & Chang, 2008; Olvet & Hajcak, 2008; Gilmore et al., 2010). Our study might have failed to find more significant psychopathology effects because of the sample or the specific context of the task. Our sample included undergraduate students enrolled in a Southeastern State University, which did not recruit for more severe cases of internalizing and externalizing psychopathology. Perhaps, administration of the same experimental design within clinical populations with more severe behavioral dysfunction would evince more significant results. Additionally, if task context is a key component of feedback and cognitive control processing as the EVC model suggests, perhaps the specific context of the gambling task used in our study was not sensitive to potential individual differences. Future studies could assess the relationship between psychopathology and task performance within a wide variety of tasks (see Future Directions section 4.6). Continued research on this topic could reveal dysfunction in feedback processing and cognitive control that can help explain behavioral deficits seen in many suffering from internalizing and/or externalizing disorders. Advances in the understanding of the cognitive processing in psychopathology could also greatly improve clinical treatments available to those with a variety of mental health disorders. Even leading evidence-based treatments that represent the gold-standard of clinical treatment, such as cognitive behavioral therapy and other third-wave cognitive treatments, exhibit varied (and sometimes limited) efficacy across all disorders (Rector &

Beck, 2001; Hides et al., 2010; Hofmann et al., 2012; Arnberg & Ost, 2014; van der Gaag, 2014). If the nature of the cognitive and behavioral processing in externalizing and internalizing disorders were better understood, perhaps treatment could be tailored to increase efficacy and maintain long-term effects for all participants. With these potential benefits in mind, further investigation into the potential deficits in ACC and cognitive control processing in individuals with psychopathology will be likely be both fruitful and invaluable.

4.4 Strengths of Current Study

The current study benefited from several design choices that led to results and implications germane to recent work in task performance and behavior modification research. Firstly, this study utilized time-frequency and source localization ERP analyses to assess feedback processing with increased spatial resolution to add to the high temporal resolution offered by ERP approaches. As discussed previously, most prior ERP work regarding task performance and recruitment of cognitive control resources have relied on indirect measures of cognitive control such as post-error slowing and analyses of behavioral measures on subsequent trials. In contrast our design allowed us to assess feedback-related dynamics within ACC and dlPFC regions with increased time-resolution than previous work relying on wavelets, providing improved time resolution to investigate communication between these regions on the timescale of milliseconds post-feedback. The ability to assess the time-dynamics of ACC and dlPFC activity together also afforded us the opportunity to link our results to current theoretical models of task performance that detail the potential interplay between these regions (and their respective networks) in effective behavior modification. Our inclusion of innovative ITPS and ICPS

methodology served as measures targeting this theorized inter-region communication. Though manipulation of task context (i.e. sequences of gain/loss feedback) and psychopathology (i.e. scores on externalizing and internalizing questionnaires) did not produce robust results, their inclusion speaks to the potential importance of these concepts in efficient task performance and behavior modification and hopefully stimulates further research on these topics (see section 4.6 Future Directions). Finally, our study benefited from a large sample size, which permitted us to investigate the variety of topics included in this design.

4.5 Limitations of Current Study

Many of the limitations in this study reflect potential future topics of investigation pertinent to models of feedback processing, communication between brain networks, and psychopathology. However, one methodological limitation of the current work is our method for electrode digitization within the cortical source localization process. For accurate localization of ERP activity at the scalp to potential cortical sources, it is vital to record EEG electrode locations as precisely as possible in 3D space. In this study, electrode locations were calculated through the averaging of recorded channel locations for 12 same-aged individuals in the same EEG cap used in our original data collection. Though this technique is methodologically sound (Reynolds & Richards, 2005; Reynolds & Richards, 2008), the gold-standard for recording EEG electrode locations is to digitize electrode placement for each participant included in EEG data collection. Source localization analyses can be further improved if structural MRIs are recorded for each participant and included in the creation of the source model (our study used the MNI average brain for this purpose). With individual channel locations and structural MRIs,

cortical source analysis of ERP waveforms could be computed at the individual level. Despite the considerable resources needed for this more individualized data collection, ERP waveforms could be more confidently sourced at a finer spatial resolution. At the same time, source analyses from more broadly defined regions, such as those included in this study, are likely appropriately represented.

An additional limitation to the current design is the relatively narrow age range of participants. The archival sample utilized in this study comprised solely of college-aged individuals; though the results of this study are not directly generalizable to a broader age range and stages in the lifespan, this sample did provide a more accurate picture of feedback and cognitive control processes because age was not a confounding factor. Our priority in this experimental design was to characterize the dynamics of ACC and dlPFC feedback-related activity at an age when cognitive processing is believed to be at peak capacity. Past research regarding cognitive control functioning throughout the lifespan suggests that cognitive control processing develops and improves through childhood and adolescence (Davidson et al., 2006; Luna et al., 2010; Munakata et al., 2012) and declines in the elderly (Braver et al., 2001; Paxton et al., 2008). These deficits and differences in cognitive control capacity throughout the lifespan likely influence feedback processing in the ACC and dlPFC and communication between these regions to modify task behavior, which would be interesting to evaluate in the future.

4.6 Future Directions

As alluded to above, future research assessing EVC and current task performance models would benefit most from evaluation of theta-ERP measures in a variety of experimental designs and participant samples. The ECV model underscores the

importance of task context on feedback processing and recruitment of the cognitive control network for adaptive behavior modification. If task context is a key component of the decision to continue with the current task strategy or switch to a potentially more advantageous one, then assessment of the specific context of the experimental task used in the study is paramount. Would longer sequences of desirable or undesirable feedback change task context? Would different task instructions impact task context? Do different people conceptualize task context differently in the same task? If task context is as important to feedback processing as the EVC model suggests, then differences in task context could account for some of the conflicting findings in the task performance field (discussed in section 1.4). Focus on sample characteristics in task performance research could also reveal individual differences in how learners formulate task goals and apply their chosen strategy to achieve them. As mentioned in section 4.3, it would be valuable to reproduce the methodology detailed in this study in a variety of clinical populations.

Finally, future investigation of task context in proactive as well as reactive cognitive control would likely be especially fruitful. The current study focused solely on reactive cognitive control, or cognitive control processing dependent on on-going feedback processing. On the other hand, proactive control is theoretically independent of task feedback, and is instead engaged in anticipation of stimulus trials (Braver, 2012), and indexed by ERP measures during the inter-trial interval (Amodio, 2010; Schmid et al., 2015). Because proactive control is also tied to improving task performance based on the conceptualized goal of the task, measures of proactive control would also likely be impacted by manipulations of task context. Research by Schmid and colleagues (2015) demonstrated differential activation in reactive and proactive cognitive control in

individuals with social anxiety, with those high in social anxiety exhibiting greater reactive cognitive control yet blunted proactive cognitive control. Therefore, future task performance research would benefit from including measures of both proactive and reactive cognitive control.

4.7 Conclusions

This study sought to evaluate current conceptual models of task performance, in particular the EVC theory, through the fine temporal resolution offered by RID TF-ERP measures coupled with cortical source localization analyses. Overall, results support the proposed feedback and cognitive control processing underlying behavior modification and improvement of task performance. However, the mechanism of communication between ACC and prefrontal cognitive control regions might be more complex than the unilateral "call and response" suggested by the EVC theory. Our results suggest that initial feedback processing occurs in both ACC and dlPFC regions prior to coordination and communication between these regions. Thus, the cost/benefit decision to maintain or switch task strategies hypothesized to occur mainly in the ACC might involve more dlPFC processing than previously suggested. Instead of the ACC calling for cognitive control resources when needed, feedback-related communication between these regions might be more collaborative in nature.

Tables

Table 1
Regions of interest extracted from Brainnetome Atlas for source analysis. Numbers within the Brainnetome labels are representative of Brodmann areas.

ROI	Brainnetome Label	Brainnetome Coordinates (X, Y, Z)
ACC	A24cd_l	(86,134,71)
	A24cd_r	(95,133,70)
	A32p_l	(85,162,87)
	A32p_r	(96,155,80)
	A24rv_l	(88,136,84)
	A24rv_r	(96,149,96)
dlPFC	A9_46d_l	(64,170,76)
	A9_46d_r	(122,164,72)
	A46_l	(63,184,95)
	A46_r	(119,182,90)
	A9_46v_l	(50,168,91)
	A9_46v_r	(133,172,94)
	A8dl_l	(73,150,55)
	A8dl_r	(113,152,56)
	A8vl_l	(59,150,62)
	A8vl_r	(133,154,69)
	A9l_r	(104,175,67)
	A9l_l	(80,176,67)

Table 2
Time X Measure ANOVA results for ACC ROI

Predictor	df_{Num}	df_{Den}	*Epsilon*	SS_{Num}	SS_{Den}	*F*	*p*	η^2_g
(Intercept)	1	154		0.00	444.13	0.00	1.0	.00
Time	1	154		24.69	101.51	37.45	.000	.03
Measure	2	154	0.90	0.00	265.37	0.00	1.0	.00
Time x Measure	2	154	0.98	0.37	90.93	0.63	.533	.00

Note. df_{Num} indicates degrees of freedom numerator. df_{Den} indicates degrees of freedom denominator. Epsilon indicates Greenhouse-Geisser multiplier for degrees of freedom, *p*-values and degrees of freedom in the table incorporate this correction. SS_{Num} indicates sum of squares numerator. SS_{Den} indicates sum of squares denominator. η^2_g indicates generalized eta-squared.

Table 3
Time X Measure ANOVA results for dlPFC ROI

Predictor	df_{Num}	df_{Den}	*Epsilon*	SS_{Num}	SS_{Den}	*F*	*p*	η^2_g
(Intercept)	1	154		0.00	479.91	0.00	1.0	.00
Time	1	154		12.74	94.81	20.69	.000	.01
Measure	2	154	1.00	0.00	224.03	0.00	1.0	.00
Time x Measure	2	154	0.97	0.09	115.42	0.12	.880	.00

Note. df_{Num} indicates degrees of freedom numerator. df_{Den} indicates degrees of freedom denominator. Epsilon indicates Greenhouse-Geisser multiplier for degrees of freedom, *p*-values and degrees of freedom in the table incorporate this correction. SS_{Num} indicates sum of squares numerator. SS_{Den} indicates sum of squares denominator. η^2_g indicates generalized eta-squared.

Table 4
Feedback X Sequence ANOVA results for theta-AMPL

Predictor	df_{Num}	df_{Den}	*Epsilon*	SS_{Num}	SS_{Den}	*F*	*p*	η^2_g
ACC ROI within FN_{Theta} Window								
(Intercept)	1	154		83027847.98	64269027.69	198.95	.000	.38
Feedback	1	154		2728194.00	15137613.73	27.75	.000	.02
Sequence	2	154	0.68	7655189.86	29579417.57	39.86	.000	.05
Feedback x Sequence	2	154	0.70	9660.83	25064904.82	0.06	.883	.00
ACC ROI within $P3_{Theta}$ Window								
(Intercept)	1	154		120415751.38	181864230.03	101.97	.000	.25
Feedback	1	154		8822150.01	42618584.91	31.88	.000	.02
Sequence	2	154	0.65	9797191.98	57415078.88	26.28	.000	.03
Feedback x Sequence	2	154	0.60	1059197.31	74907164.46	2.18	.138	.00

dlPFC ROI within FN_{Theta} Window								
(Intercept)	1	154		81816431.79	88739711.96	141.99	.000	.34
Feedback	1	154		1504492.75	22133450.44	10.47	.001	.01
Sequence	2	154	0.67	9511461.38	28179912.03	51.98	.000	.06
Feedback x Sequence	2	154	0.68	93157.21	22570144.04	0.64	.473	.00
dlPFC ROI within $P3_{Theta}$ Window								
(Intercept)	1	154		139514644.77	18772119907	114.45	.000	.31
Feedback	1	154		3200461.59	35823729.80	13.76	.000	.01
Sequence	2	154	0.72	12677387.94	39534179.61	49.38	.000	.04
Feedback x Sequence	2	154	0.65	35674.14	41838146.00	0.13	.782	.00

Note. df_{Num} indicates degrees of freedom numerator. df_{Den} indicates degrees of freedom denominator. Epsilon indicates Greenhouse-Geisser multiplier for degrees of freedom, *p*-values and degrees of freedom in the table incorporate this correction. SS_{Num} indicates sum of squares numerator. SS_{Den} indicates sum of squares denominator. η^2_g indicates generalized eta-squared.

Table 5

Feedback X Sequence ANOVA results for Theta-ITPS

Predictor	df_{Num}	df_{Den}	*Epsilon*	SS_{Num}	SS_{Den}	*F*	*p*	η^2_g
ACC ROI within FN_{Theta} Window								
(Intercept)	1	154		397.51	0.08	805160.96	.000	1.00
Feedback	1	154		0.01	0.04	22.13	.000	.02
Sequence	2	154	0.90	0.00	0.06	0.53	.571	.00
Feedback x Sequence	2	154	0.85	0.00	0.06	2.07	.136	.00
ACC ROI within $P3_{Theta}$ Window								
(Intercept)	1	154		406.66	0.13	497894.53	.000	1.00
Feedback	1	154		0.01	0.05	46.65	.000	.04

Sequence	2	154	0.94	0.00	0.06	1.42	.244	.00
Feedback x Sequence	2	154	0.93	0.00	0.06	1.11	.327	.00
dlPFC ROI within FN_{Theta} Window								
(Intercept)	1	154		391.49	0.03	1969597.43	.000	1.00
Feedback	1	154		0.00	0.01	11.62	.001	.01
Sequence	2	154	0.94	0.00	0.02	1.49	.227	.00
Feedback x Sequence	2	154	0.78	0.00	0.02	0.46	.585	.00
dlPFC ROI within $P3_{Theta}$ Window								
(Intercept)	1	154		396.87	0.05	1343647.77	.000	1.00
Feedback	1	154		0.00	0.01	32.03	.000	.02
Sequence	2	154	0.92	0.00	0.01	0.67	.498	.00
Feedback x Sequence	2	154	0.92	0.00	0.02	1.19	.305	.00

Note. df_{Num} indicates degrees of freedom numerator. df_{Den} indicates degrees of freedom denominator. Epsilon indicates Greenhouse-Geisser multiplier for degrees of freedom, *p*-values and degrees of freedom in the table incorporate this correction. SS_{Num} indicates sum of squares numerator. SS_{Den} indicates sum of squares denominator. η^2_g indicates generalized eta-squared.

Table 6

Feedback X Sequence ANOVA results for Theta-ICPS

Predictor	df_{Num}	df_{Den}	*Epsilon*	SS_{Num}	SS_{Den}	*F*	*p*	η^2_g
ACC-ACC within FN_{Theta} Window								
(Intercept)	1	154		529.41	1.57	51789.49	.000	1.00
Feedback	1	154		0.00	0.14	3.37	.068	.00
Sequence	2	154	0.90	0.00	0.26	0.32	.702	.00
Feedback x Sequence	2	154	0.88	0.00	0.28	0.58	.539	.00

ACC-ACC within $P3_{Theta}$ Window								
(Intercept)	1	154		538.64	1.64	50517.76	.000	1.00
Feedback	1	154		0.01	0.12	11.13	.001	.00
Sequence	2	154	0.87	0.00	0.24	0.81	.430	.00
Feedback x Sequence	2	154	0.89	0.00	0.25	0.48	.595	.00
dlPFC-dlPFC within FN_{Theta} Window								
(Intercept)	1	154		394.90	0.12	515251.65	.000	1.00
Feedback	1	154		0.00	0.01	8.86	.003	.00
Sequence	2	154	0.88	0.00	0.02	2.90	.064	.00
Feedback x Sequence	2	154	0.88	0.00	0.02	0.68	.489	.00
dlPFC-dlPFC within $P3_{Theta}$ Window								
(Intercept)	1	154		398.01	0.15	422049.78	.000	1.00
Feedback	1	154		0.00	0.01	9.39	.003	.00
Sequence	2	154	0.91	0.00	0.02	0.92	.394	.00
Feedback x Sequence	2	154	0.93	0.00	0.02	1.18	.306	.00
ACC-dlPFC within FN_{Theta} Window								
(Intercept)	1	154		398.71	0.10	587903.40	.000	1.00
Feedback	1	154		0.00	0.01	8.98	.003	.00
Sequence	2	154	0.90	0.00	0.02	2.34	.104	.00
Feedback x Sequence	2	154	0.91	0.00	0.02	0.54	.567	.00
ACC-dlPFC within $P3_{Theta}$ Window								
(Intercept)	1	154		402.65	0.15	426600.46	.000	1.00
Feedback	1	154		0.00	0.01	25.89	.000	.01
Sequence	2	154	0.94	0.00	0.03	0.24	.776	.00
Feedback x Sequence	2	154	0.88	0.00	0.03	0.45	.612	.00

Note. df_{Num} indicates degrees of freedom numerator. df_{Den} indicates degrees of freedom denominator. Epsilon indicates Greenhouse-Geisser multiplier for degrees of freedom, *p*-values and degrees of freedom in the table incorporate this correction. SS_{Num} indicates sum of squares numerator. SS_{Den} indicates sum of squares denominator. η^2_g indicates generalized eta-squared.

Figures

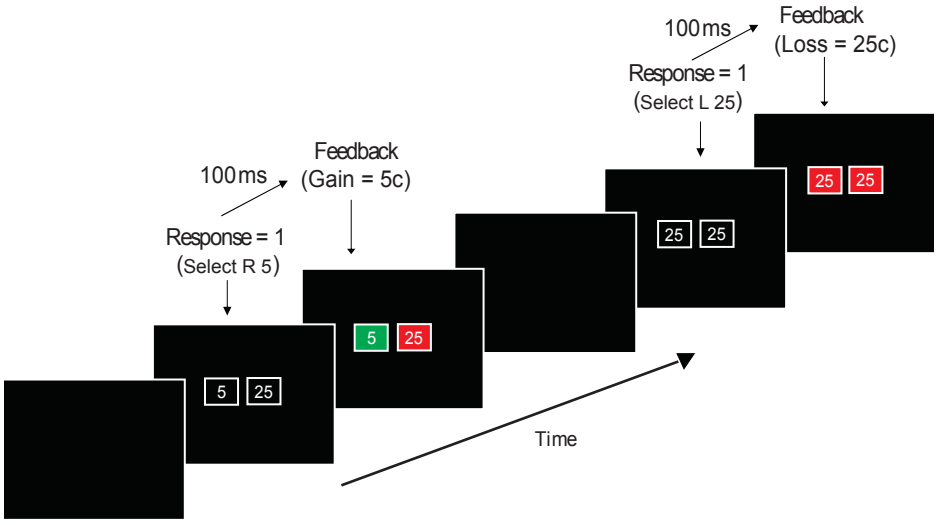

Figure 1 Sequence of stimulus and outcome events in the gambling task.

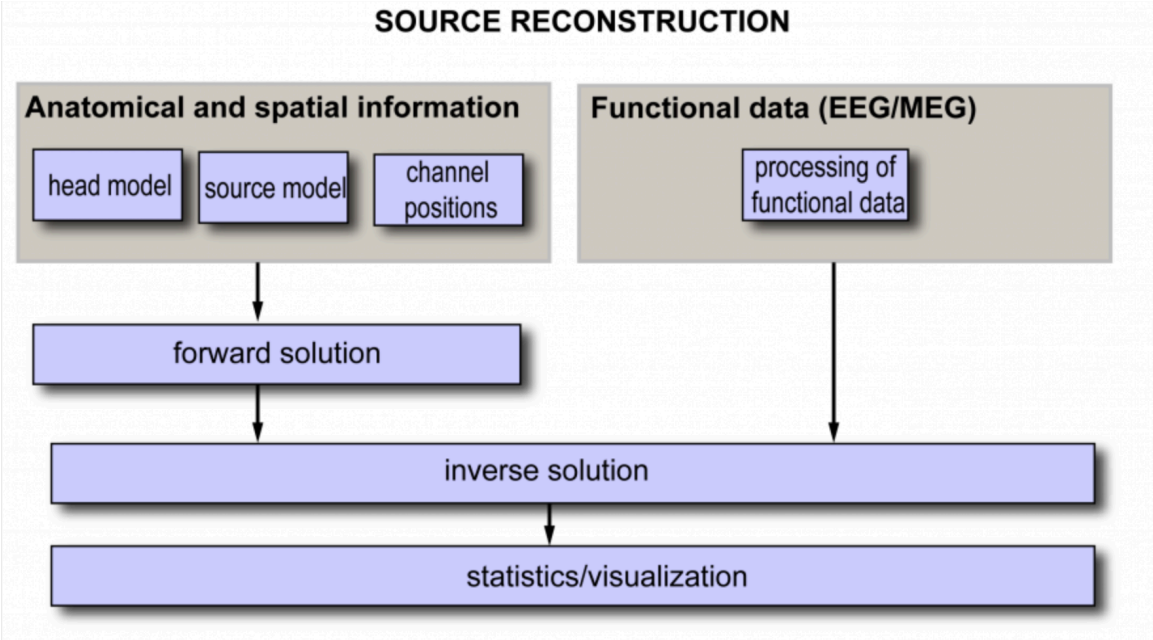

Figure 2 Flow chart of the conceptual steps in the cortical source localization process. Figure was pulled from Oostenveld, R., Fries, P., Maris, E., & Schoffelen, J. M. (2011). FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. Computational intelligence and neuroscience.

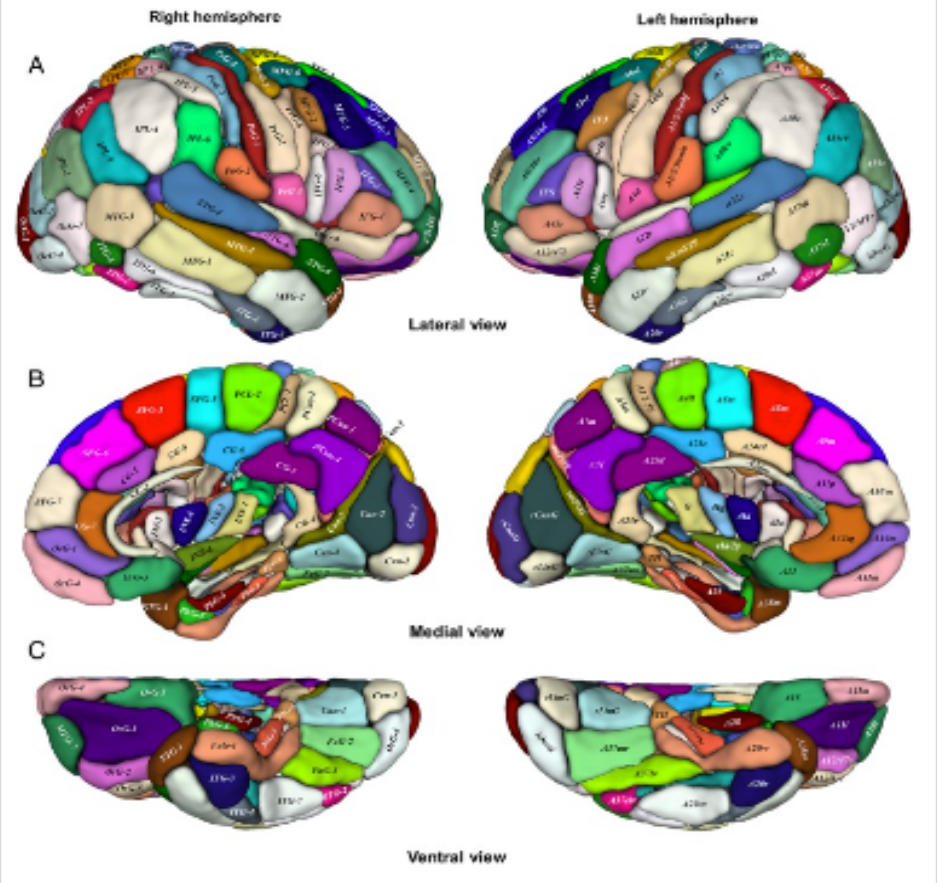

Figure 3 Location of the 246 sub-regions identified in the Brainnetome Atlas. Figure was pulled from Fan, L., Li, H., Zhuo, J., Zhang, Y., Wang, J., Chen, L., ... & Fox, P. T. (2016). The human brainnetome atlas: a new brain atlas based on connectional architecture. *Cerebral cortex*, *26*(8), 3508-3526.

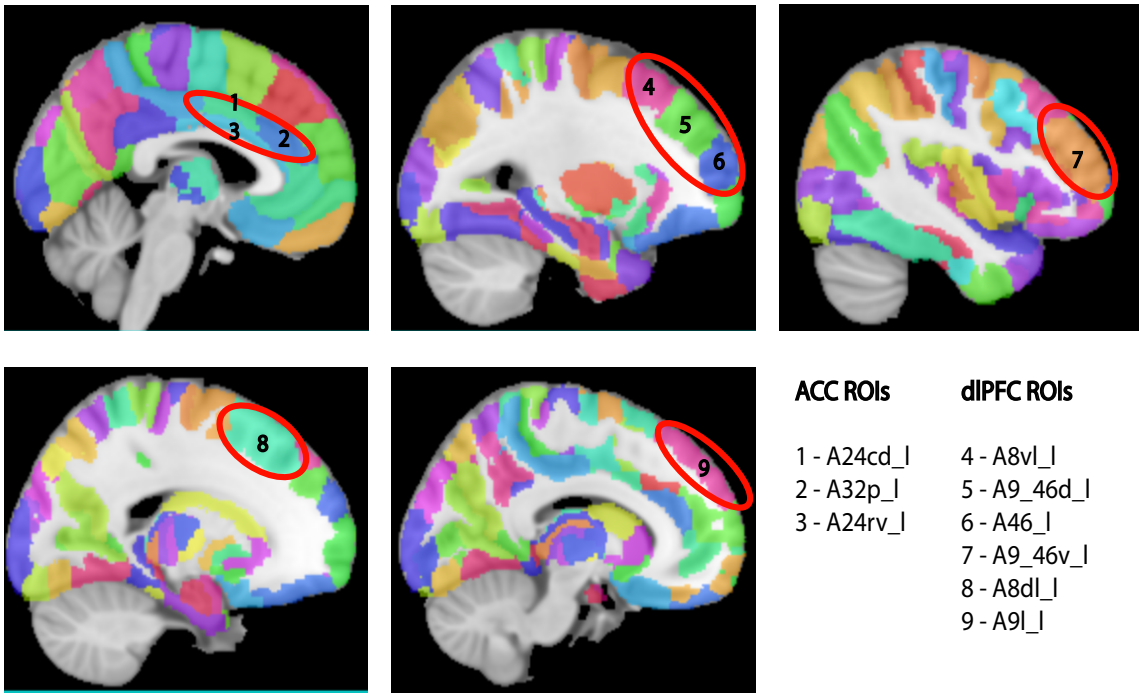

Figure 4 Sagittal views of the chosen ACC and dlPFC ROIs. Corresponding Brainnetome labels are displayed in lower right.

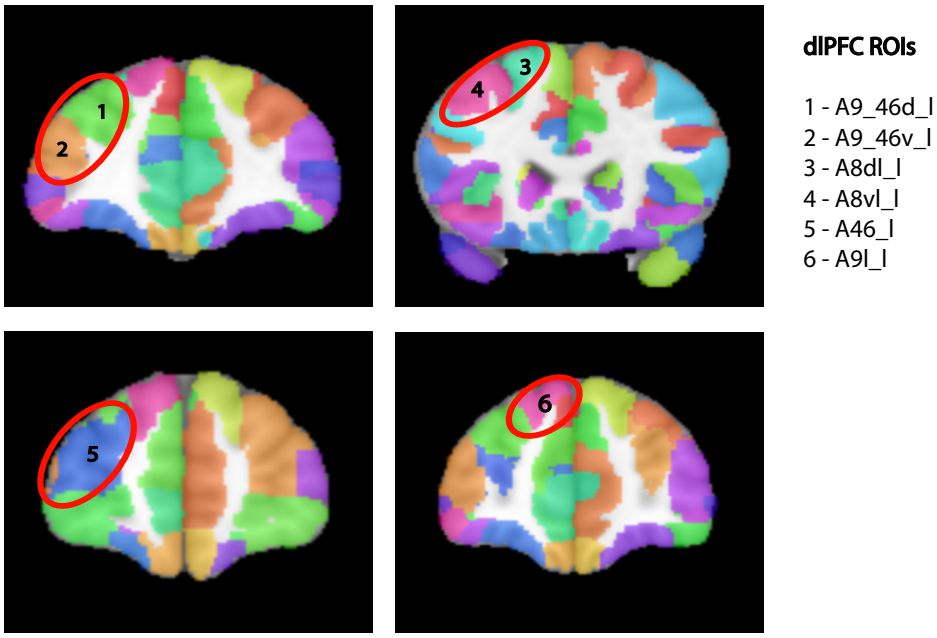

Figure 5 Coronal views of dlPFC ROIs, exhibiting relative laterality of each. Corresponding Brainnetome labels are displayed on right.

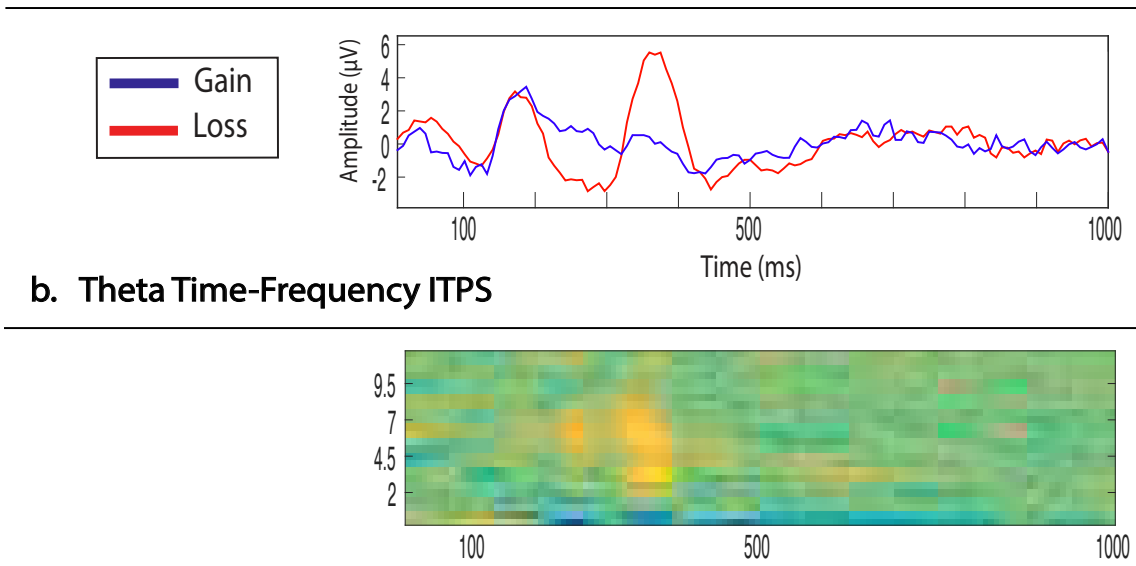

Figure 6 (a) Average theta amplitude waveforms on gain and loss trials. (c) TF theta amplitude plots displaying TF windows chosen for statistical analyses. (b) TF theta ITPS plot with peak loss-gain activity, which determined the boundaries of FN_{Theta} and $P3_{Theta}$ windows.

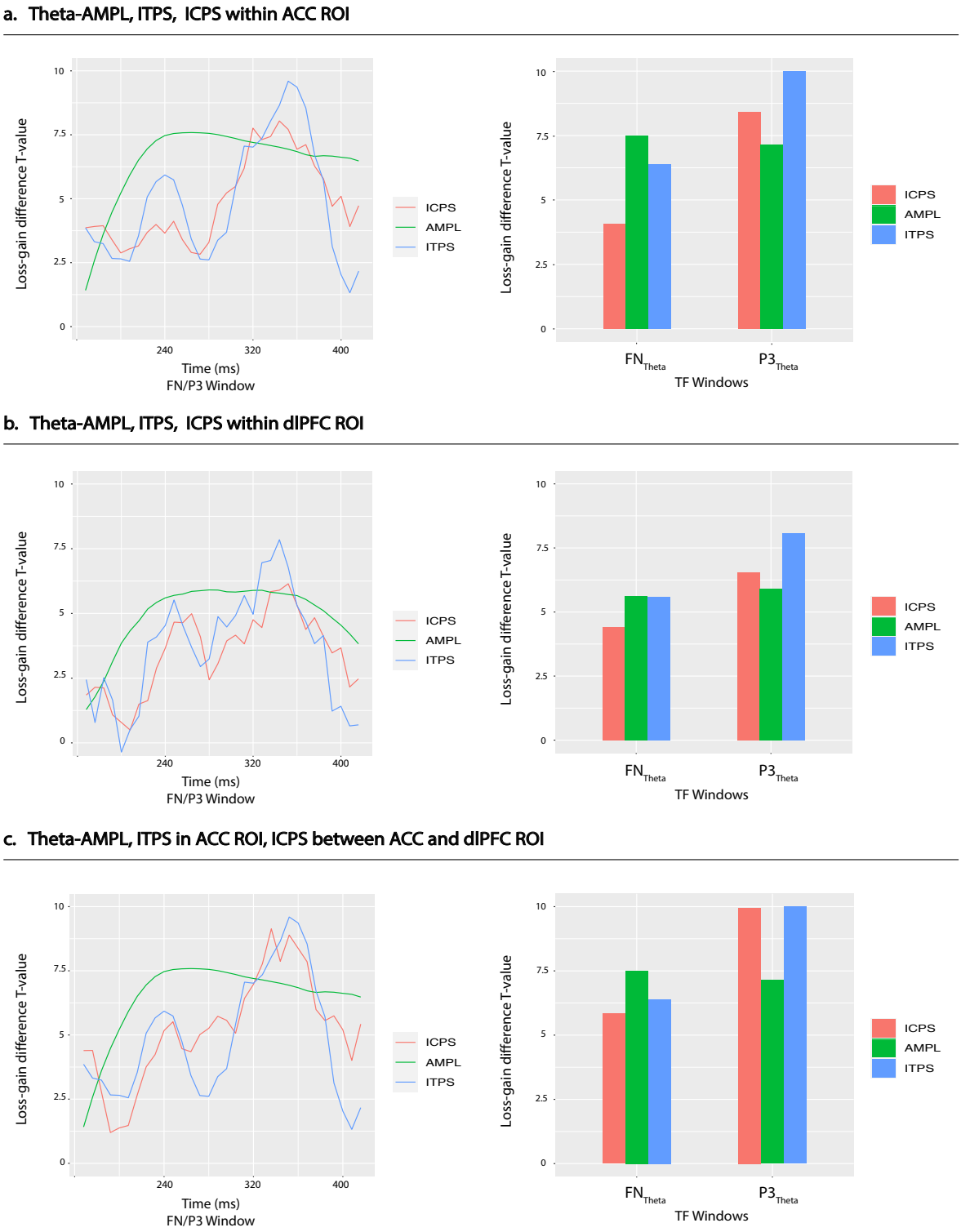

Figure 7 Gain/loss difference t-values at each time-bin (approximately 8ms) within the FN/P3 window (left column) and within FN_{Theta} and $P3_{Theta}$ windows (right column). (a) AMPL and ITPS measures were computed within averaged ACC ROI, while ICPS was computed between the ACC seed ROI (A24cd_r) and an average of the remaining

ACC ROIs. (b) AMPL and ITPS computed within averaged dlPFC ROI and ICPS computed between dlPFC seed ROI (A8dl_l) and remaining dlPFC ROIs. (c) AMPL and ITPS computed within averaged ACC ROI and ICPS computed between ACC seed ROI (A24cd_r) and averaged dlPFC ROIs.

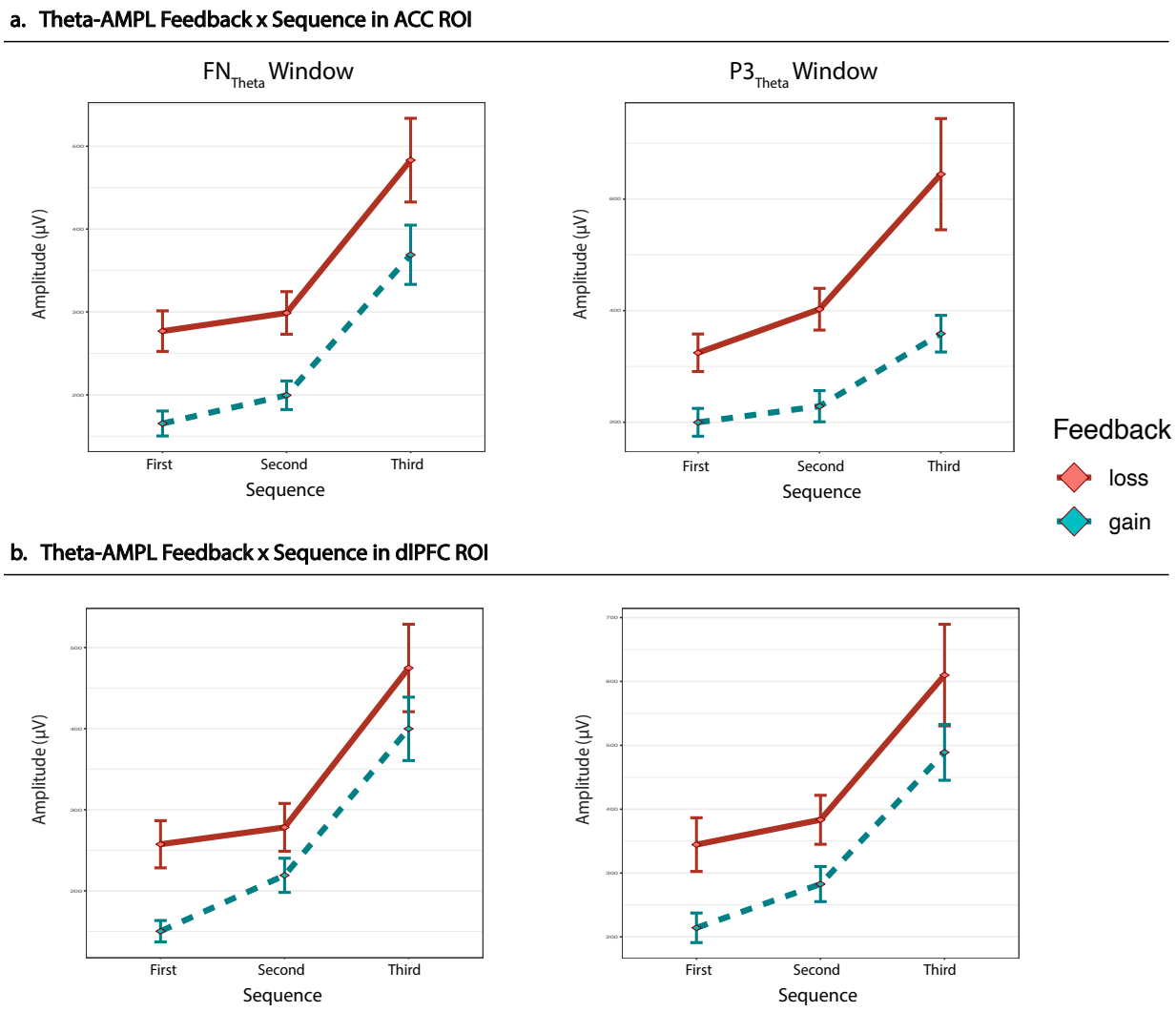

Figure 8 Peak theta-amplitude values on gain and loss trials by feedback sequence (i.e. first, second, or third consecutive gain/loss) within FN_{Theta} and $P3_{Theta}$ windows. Peak theta-AMPL computed within averaged ACC (a) and dlPFC ROIs (b).

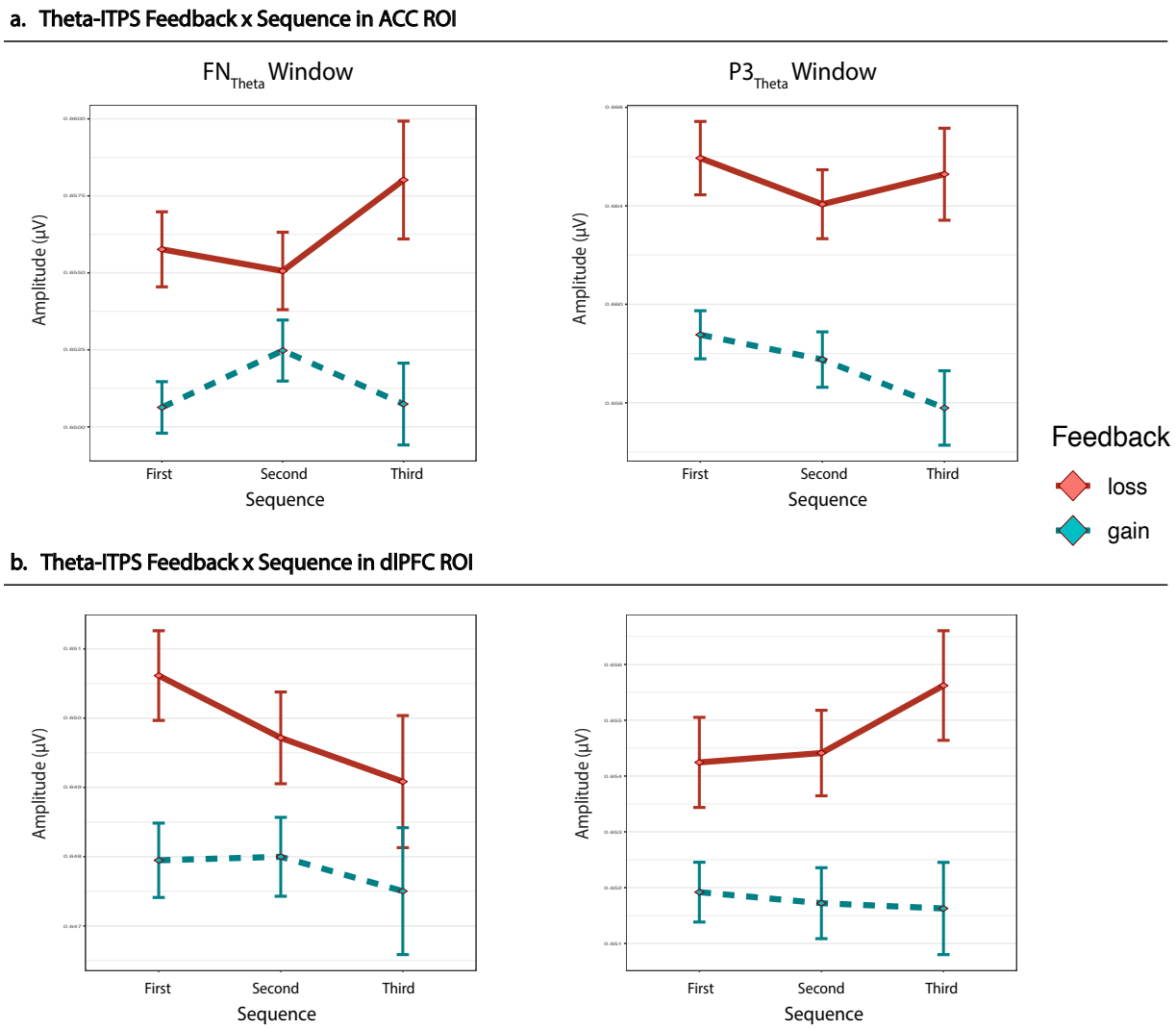

Figure 9 Peak theta-ITPS values on gain and loss trials by feedback sequence (i.e. first, second, or third consecutive gain/loss) within FN_{Theta} and $P3_{Theta}$ windows. Peak theta-ITPS computed within averaged ACC (a) and dlPFC ROIs (b).

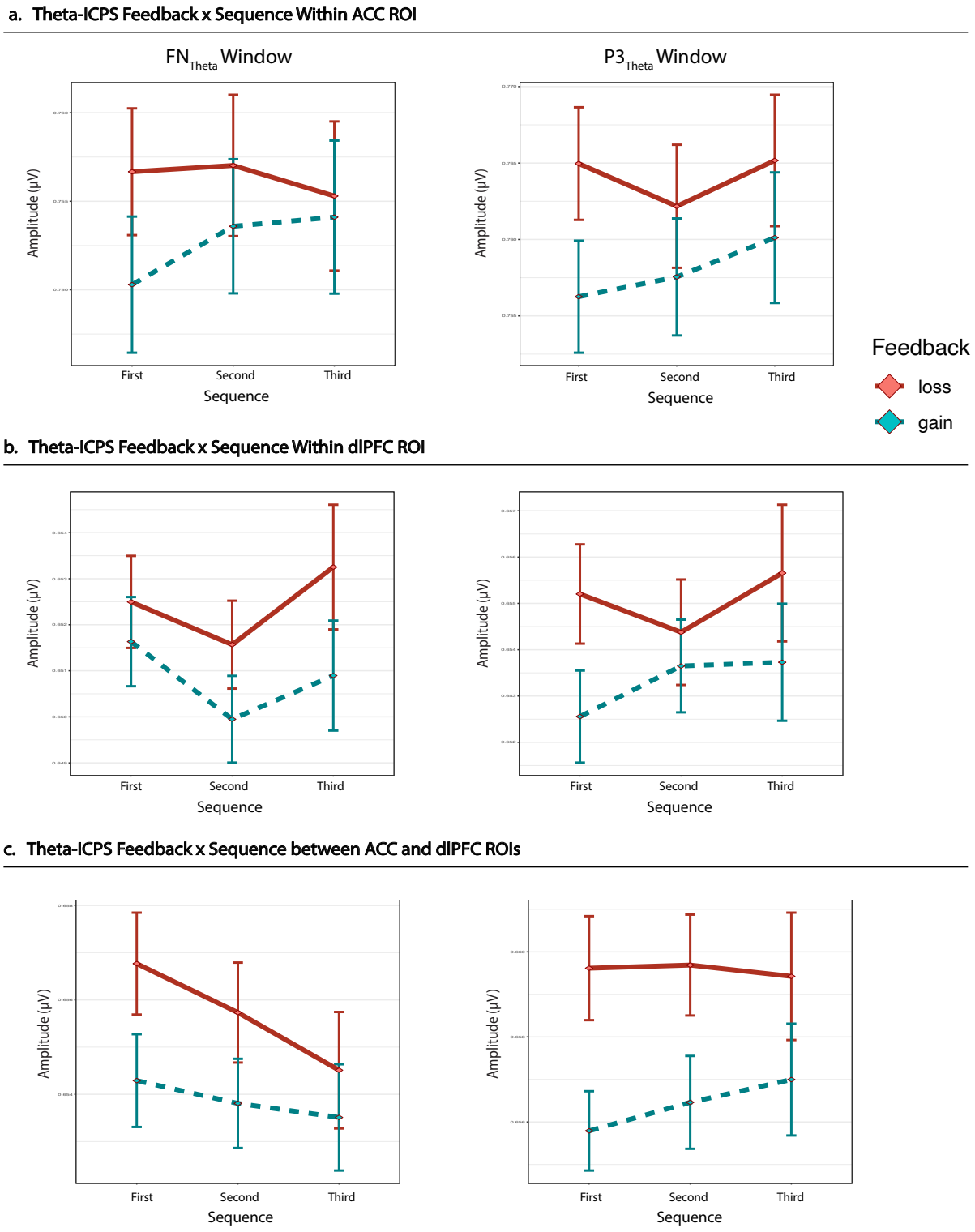

Figure 10 Peak theta-ICPS values on gain and loss trials by feedback sequence (i.e. first, second, or third consecutive gain/loss) within FN_{Theta} and $P3_{Theta}$ windows. Peak theta-ICPS computed within the ACC ROI (a), within the dlPFC ROI (b) and between the ACC seed region and averaged dlPFC ROI (c).

Appendix A

Figures of gain/loss difference t-values at each time-bin within the FN/P3 window computed at each Brainnetome region individually.

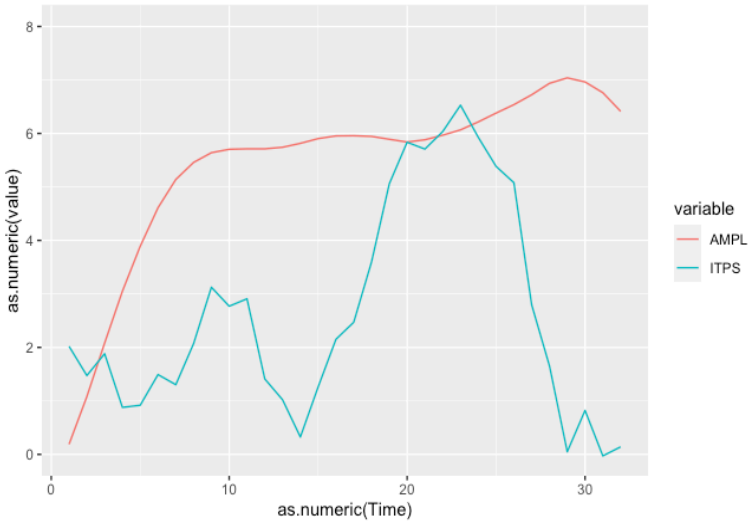

Figure 11 Theta-AMPL and theta-ITPS for A24cd_r

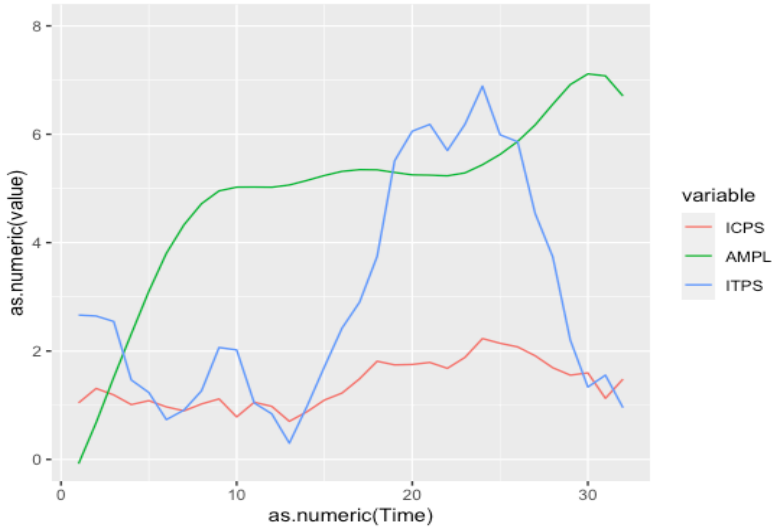

Figure 12 Theta-AMPL, theta-ITPS for A24cd_l, theta-ICPS between seed region (A24cd_r) and A24cd_l

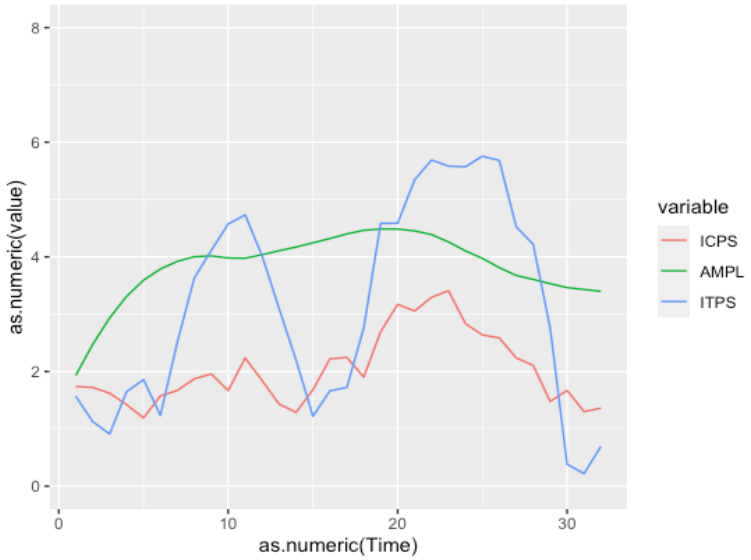

Figure 13 Theta-AMPL, theta-ITPS for A32p_r, theta-ICPS between seed region (A24cd_r) and A32p_r

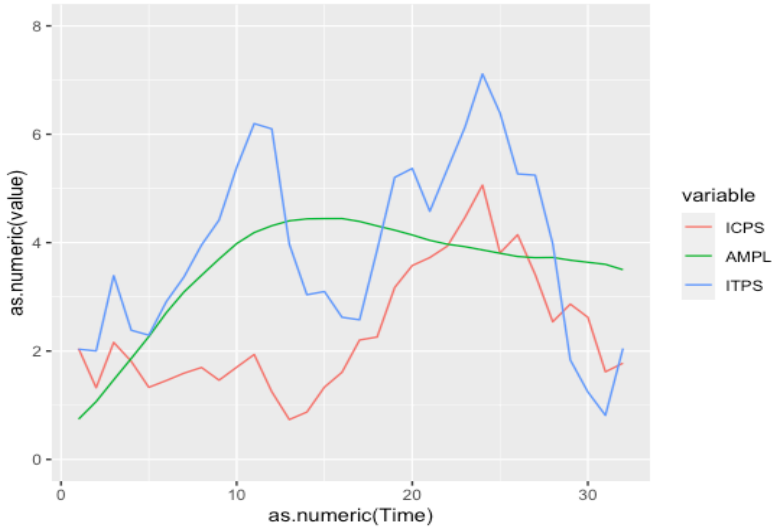

Figure 14 Theta-AMPL, theta-ITPS for A32p_l, theta-ICPS between seed region (A24cd_r) and A32p_l

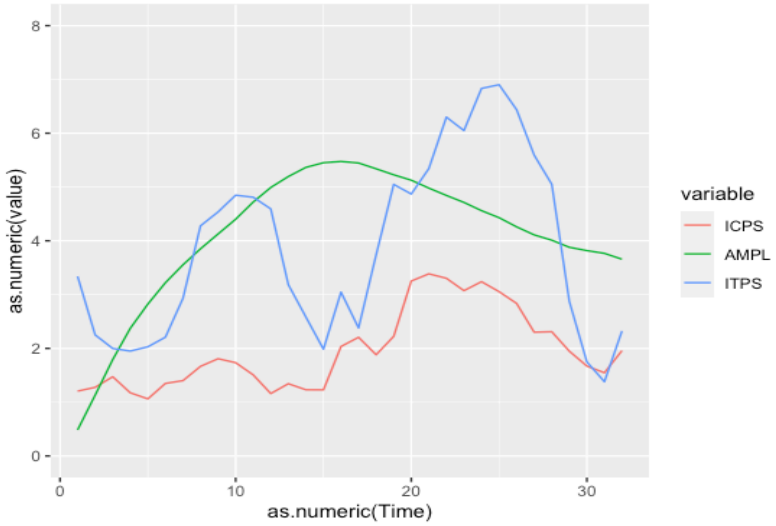

Figure 15 Theta-AMPL, theta-ITPS for A24rv_r, theta-ICPS between seed region (A24cd_r) and A24rv_r

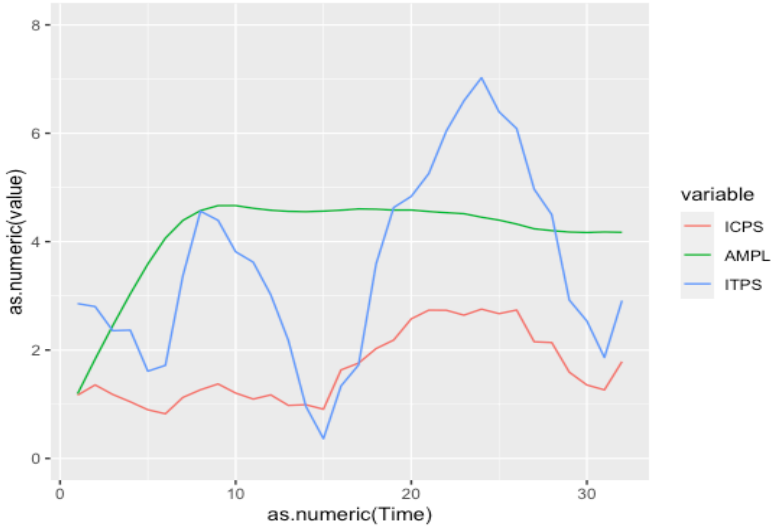

Figure 16 Theta-AMPL, theta-ITPS for A24rv_l, theta-ICPS between seed region (A24cd_r) and A24rv_l

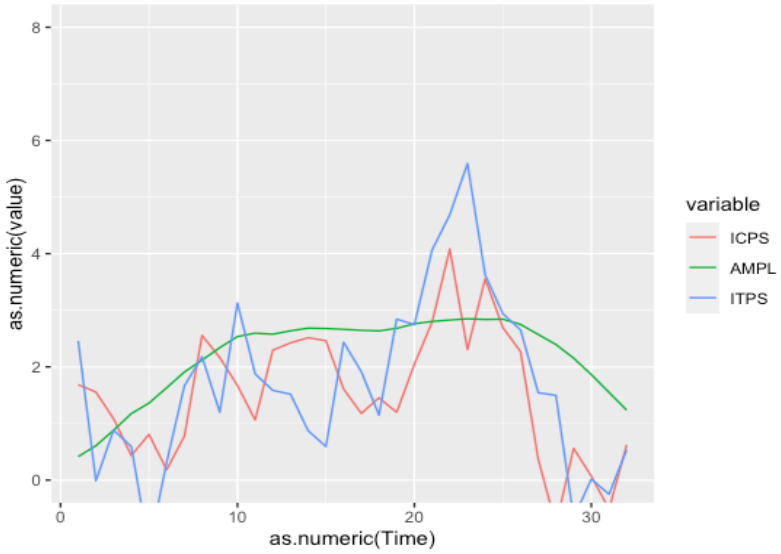

Figure 17 Theta-AMPL, theta-ITPS for A8vl_l, theta-ICPS between seed region (A24cd_r) and A8vl_l

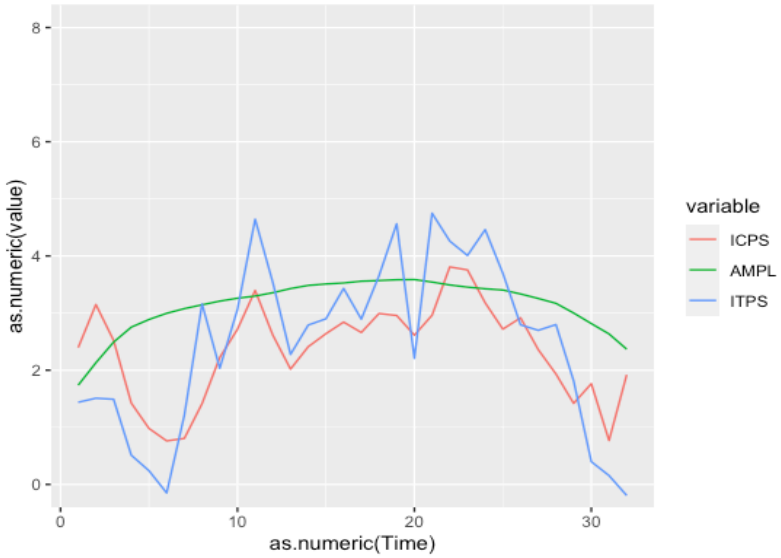

Figure 18 Theta-AMPL, theta-ITPS for A9l_l, theta-ICPS between seed region (A24cd_r) and A9l_l

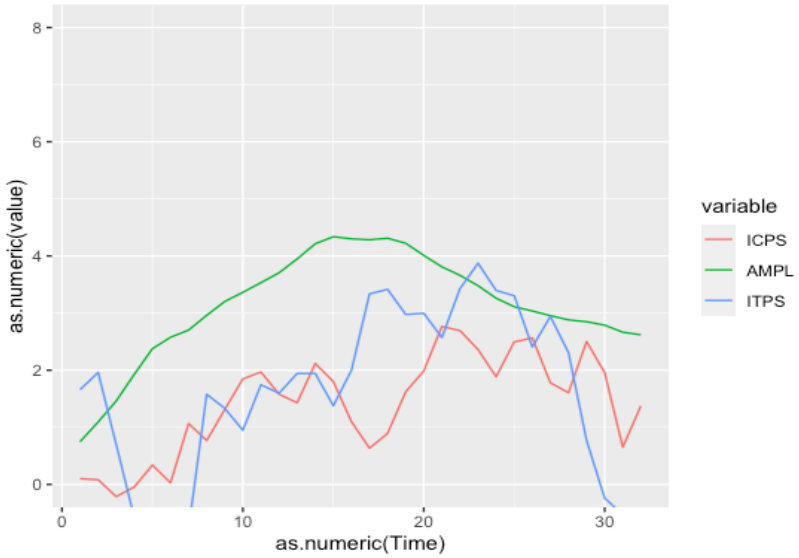

Figure 19 Theta-AMPL, theta-ITPS for A8dl_l, theta-ICPS between seed region (A24cd_r) and A8dl_l

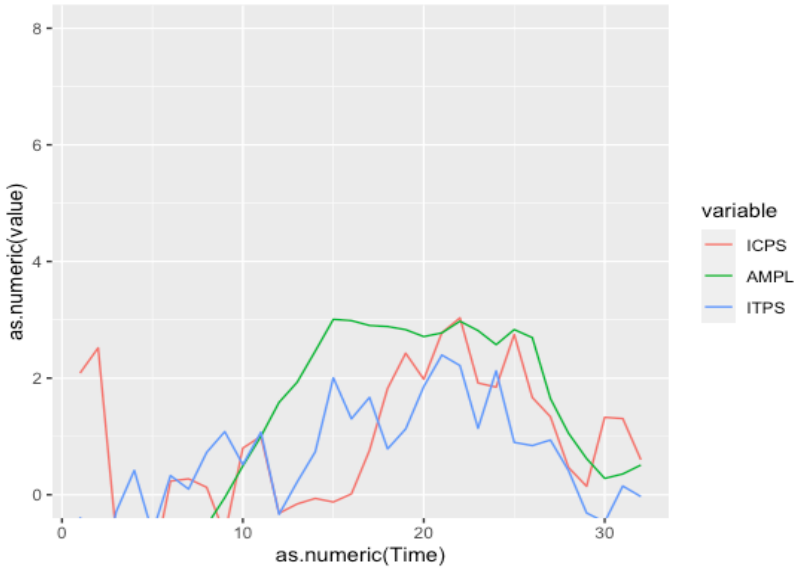

Figure 20 Theta-AMPL, theta-ITPS for A9_46v_l, theta-ICPS between seed region (A24cd_r) and A9_46v_l

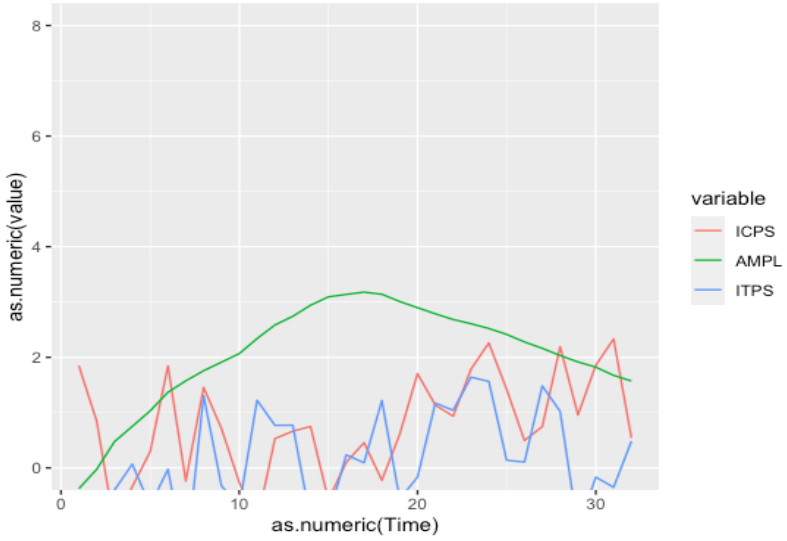

Figure 21 Theta-AMPL, theta-ITPS for A46_l, theta-ICPS between seed region (A24cd_r) and A46_l

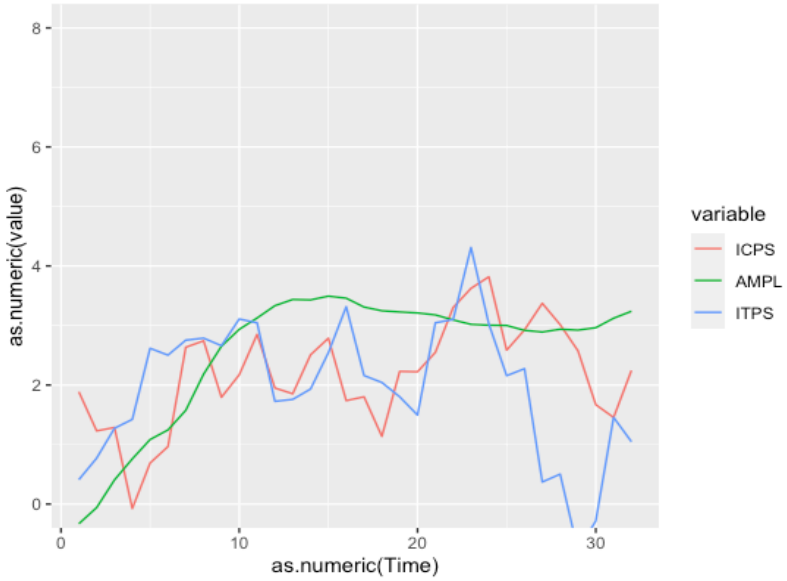

Figure 22 Theta-AMPL, theta-ITPS for A9_46d_l, theta-ICPS between seed region (A24cd_r) and A9_46d_l

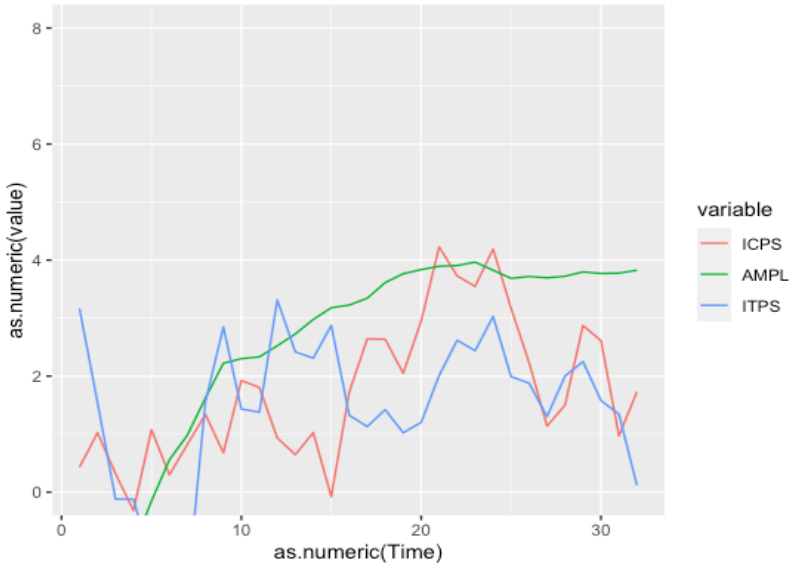

Figure 23 Theta-AMPL, theta-ITPS for A8vl_r, theta-ICPS between seed region (A24cd_r) and A8vl_r

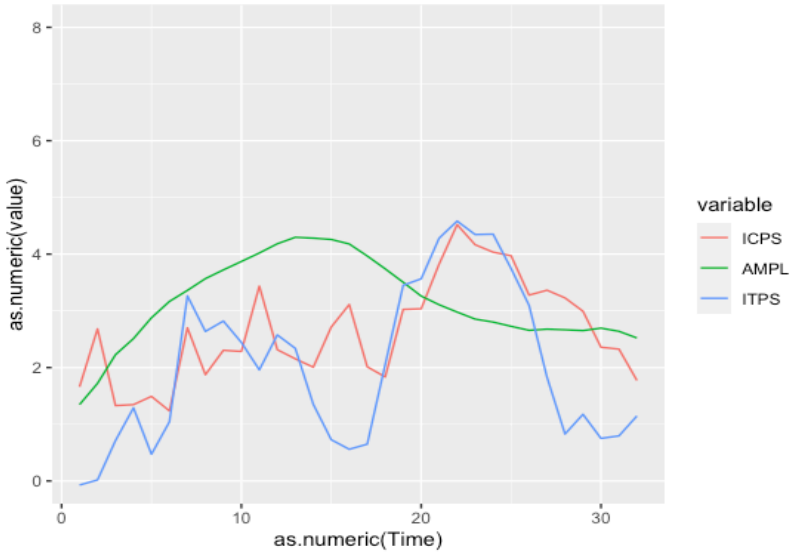

Figure 24 Theta-AMPL, theta-ITPS for A9l_r, theta-ICPS between seed region (A24cd_r) and A9l_r

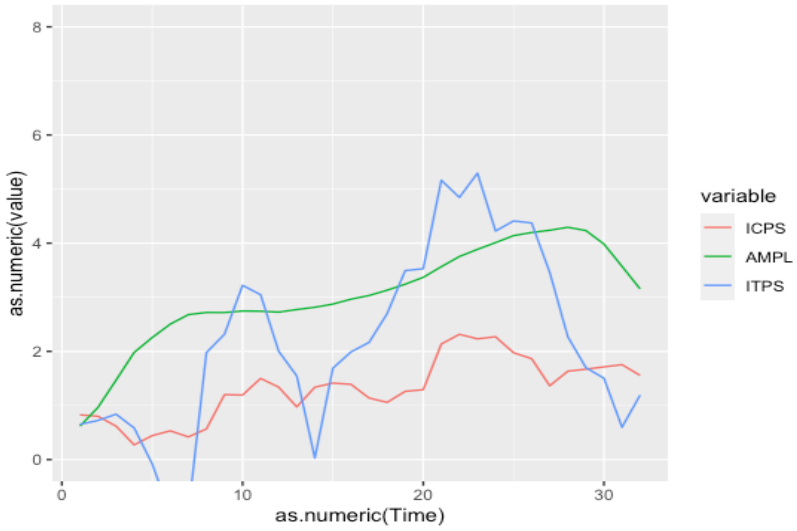

Figure 25 Theta-AMPL, theta-ITPS for A8dl_r, theta-ICPS between seed region (A24cd_r) and A8dl_r

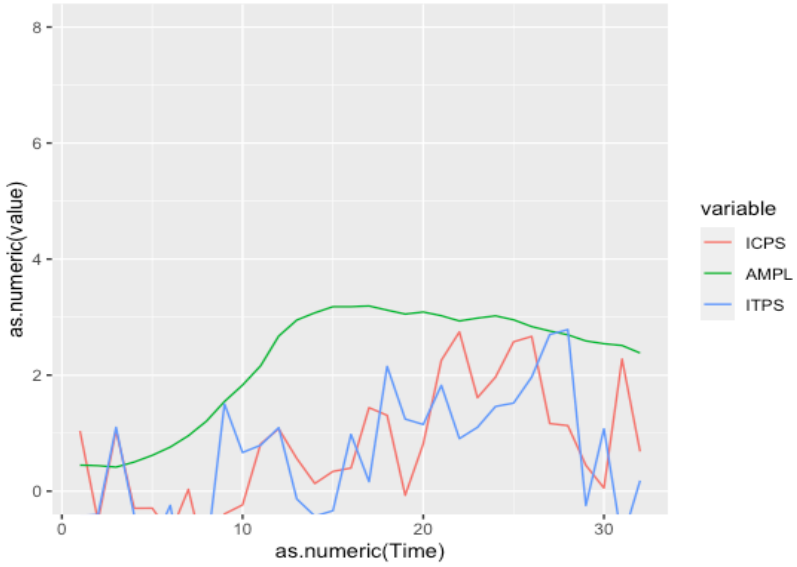

Figure 26 Theta-AMPL, theta-ITPS for A9_46v_r, theta-ICPS between seed region (A24cd_r) and A9_46v_r

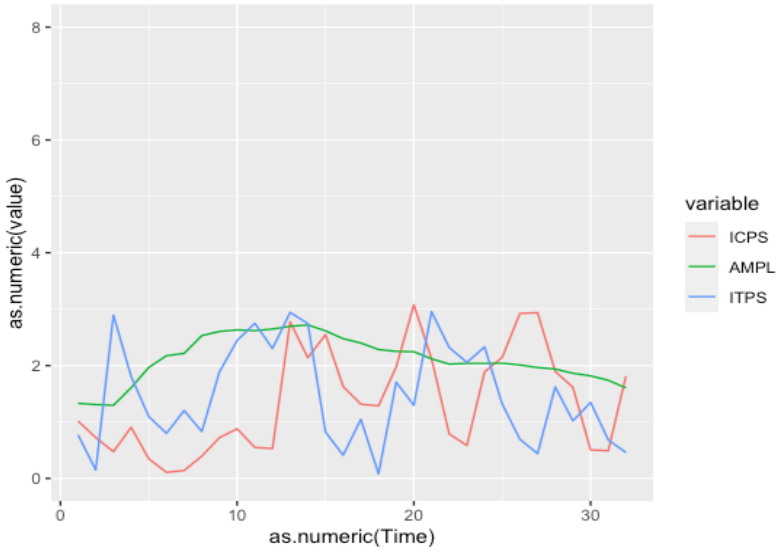

Figure 27 Theta-AMPL, theta-ITPS for A46_r, theta-ICPS between seed region (A24cd_r) and A46_r

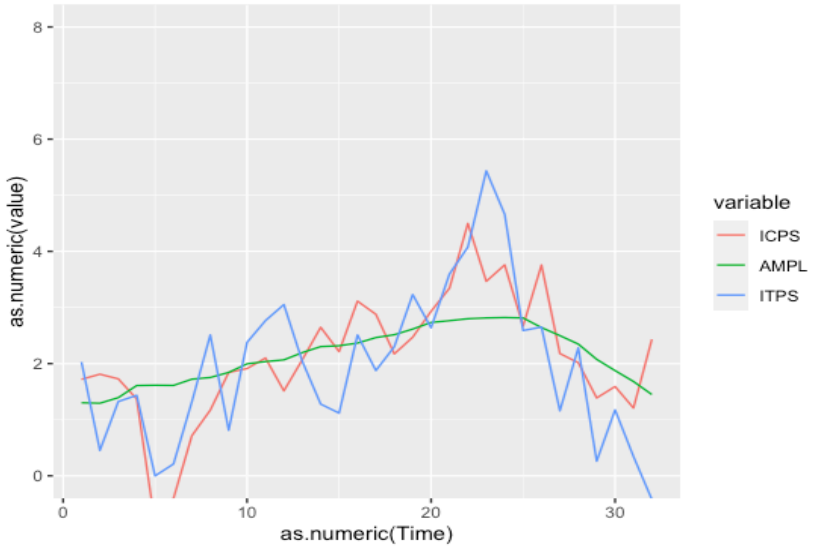

Figure 28 Theta-AMPL, theta-ITPS for A9_46d_r, theta-ICPS between seed region (A24cd_r) and A9_46d_r

Figures of gain and loss values separately at each time-bin within the FN/P3 window.

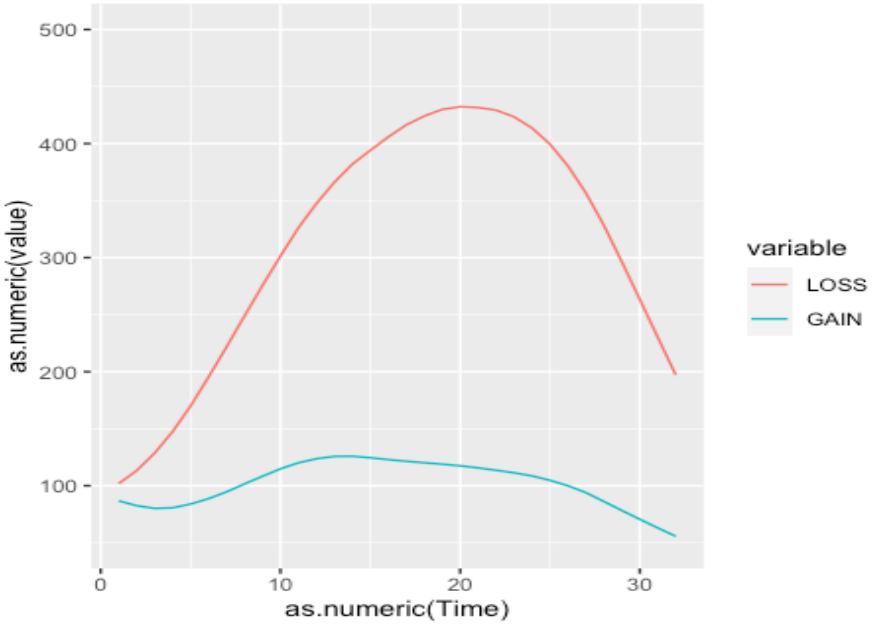

Figure 29 Gain and loss values for theta-AMPL in averaged ACC ROI

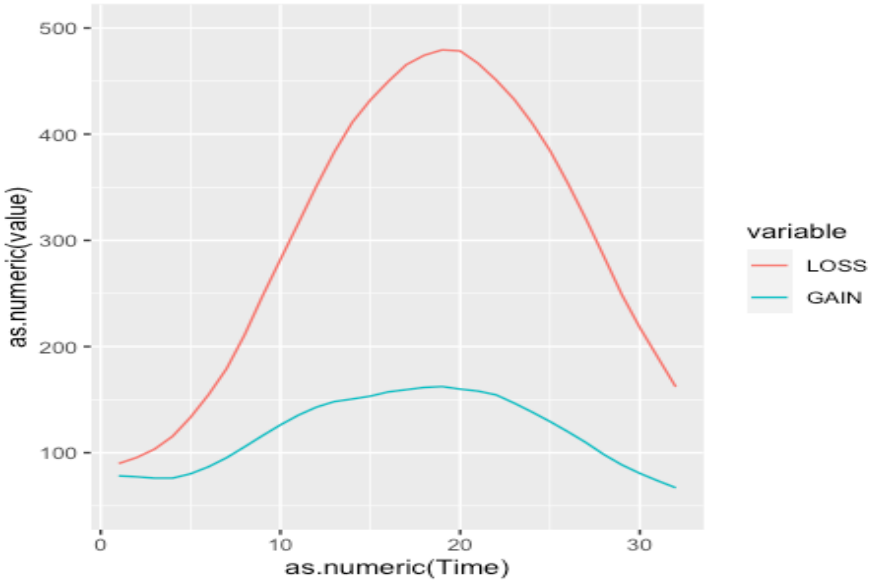

Figure 30 Gain and loss values for theta-AMPL in averaged dlPFC ROI

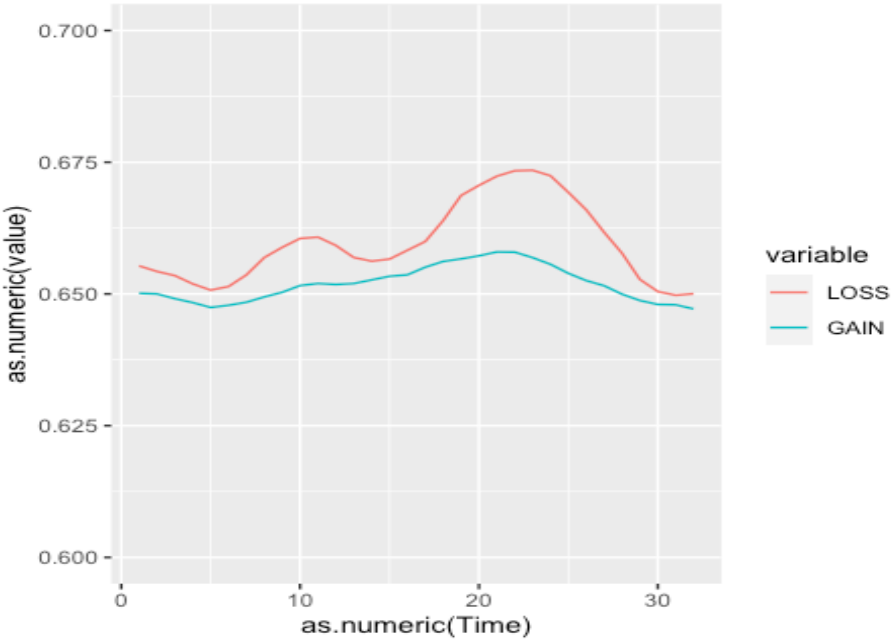

Figure 31 Gain and loss values for theta-ITPS in averaged ACC ROI

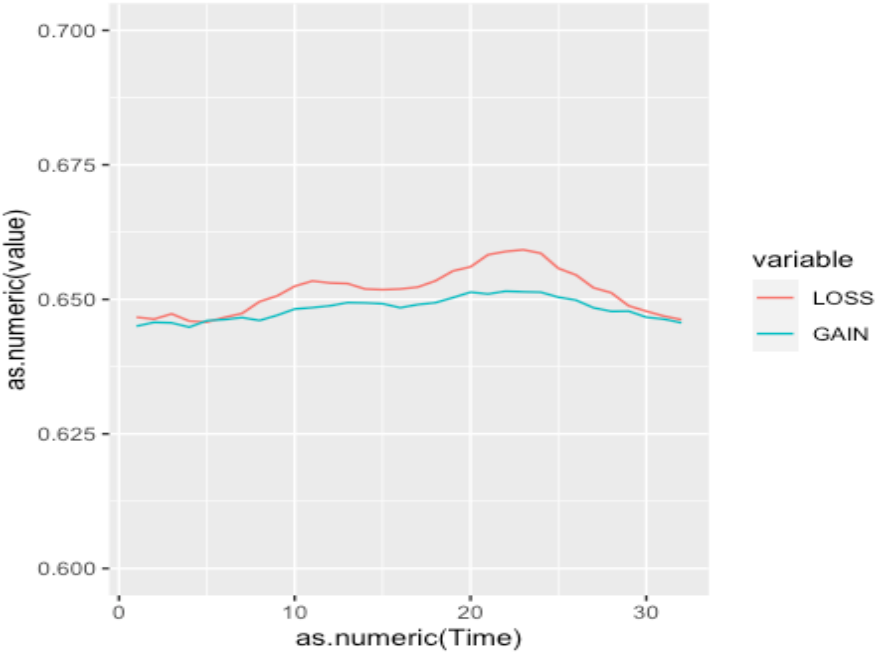

Figure 32 Gain and loss values for theta-ITPS in averaged dlPFC ROI

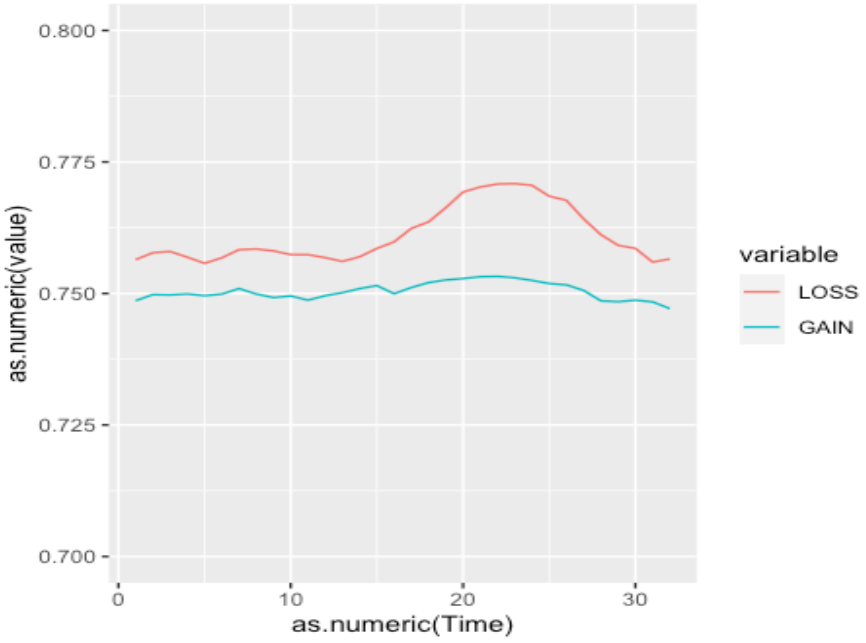

Figure 33 Gain and loss values for theta-ICPS between seed region (A24cd_r) and averaged ACC ROI

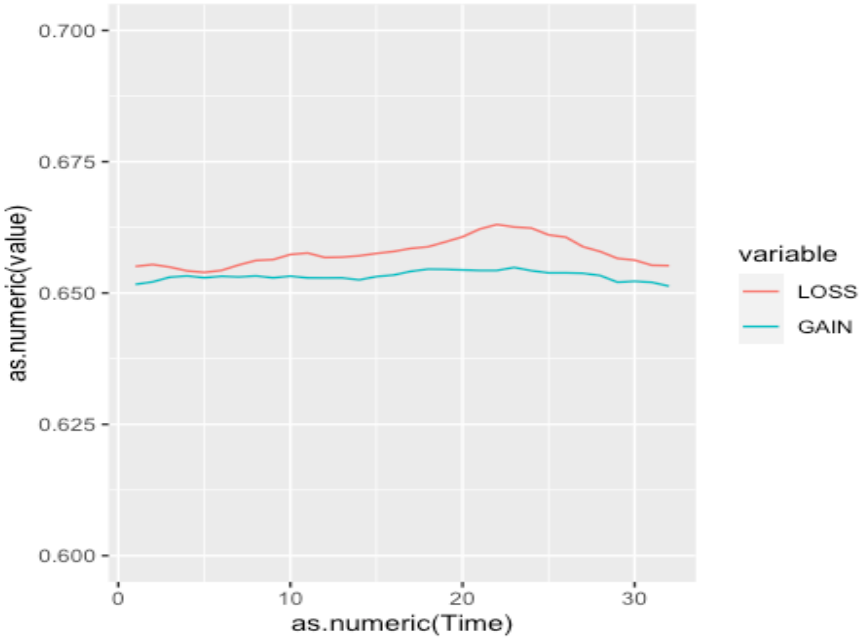

Figure 34 Gain and loss values for theta-ICPS between seed region (A24cd_r) and averaged dlPFC ROI

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