The role of anticipation and an adaptive monitoring system in stuttering: a theoretical and experimental investigation

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THE ROLE OF ANTICIPATION AND AN ADAPTIVE MONITORING SYSTEM IN STUTTERING: A THEORETICAL AND EXPERIMENTAL INVESTIGATION

by

Richard Matthew Arenas

An Abstract

Of a thesis submitted in partial fulfillment of the requirements for the Doctor of Philosophy degree in Speech and Hearing Science in the Graduate College of The University of Iowa

May 2012

Thesis Supervisors: Professor Patricia M. Zebrowski
Associate Professor Prahlad Gupta
ABSTRACT

The focus of this thesis was on the relationship between anticipation of stuttering and its effect on speech production in people who stutter (PWS). In order to aid in the development of a hypothesis based study that is rooted in biological mechanisms, this thesis introduced a new theoretical framework from which to view the factors that contribute to stuttering variability. The speech and monitoring interaction (SAMI) framework proposes that there are two systems that account for stuttering variability: the speech production system and the monitoring system.

Utilizing the SAMI framework, it was hypothesized that there would be a positive correlation between stuttering expectancy ratings and verbal response time. This hypothesis was based on the assumption that the anticipation of stuttering will activate the monitoring system which subsequently initiates a global inhibitory process over action selection. Two studies were conducted to test this hypothesis. Study one was a behavioral task that had two primary goals: 1) Characterize the degree to which PWS anticipate stuttering and how accurately they can predict actual stuttering. 2) Investigate the correlation between stuttering expectancy and the verbal response time in word naming tasks. Study two was a series of simulations using a biological plausible neural network model of the monitoring system. The goal of the simulations was to make a qualitative comparison of the behavioral results and the results from the neural network model.

There were four key findings: 1) Across the group of PWS, there was a positive correlation between stuttering expectancy and verbal response time in the immediate naming task. 2) The degree to which stuttering expectancy was correlated with response
time was found to be positively correlated with stuttering severity. 3) There was a correlation between stuttering severity and the belief that expecting to stutter increases the likelihood stuttering will occur. 4) The results from the simulations were qualitatively comparable to the results of the behavioral task.

This is the first study to show that the expectation of stuttering has an effect on fluent speech production, providing evidence that the anticipation of stuttering is not only correlated with moments of stuttering, but it may also be a causal factor. Further, this relationship is stronger in more severe PWS, indicating that the presence of the anticipation of stuttering may be a factor in determining the severity of stuttering. The fit between the model and the behavioral results provides evidence for the underlying substrates of these effects.

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To Angela, without your endless patience and love this would have never been possible.
Thanks and I love you.
There is still more to this incredible trick you play on yourself when you try not to stutter. It is built upon one of the most powerful and deceptive illusions you are likely ever to know. First, as we have seen, you expect to stutter – and because of what you do to keep from stuttering your expectation is proved correct. That is, you do the things that you are stuttering. This strengthens your tendency to expect stuttering. That is, if you expect to “have trouble” saying a word, and then you do “have trouble”, you will be still more inclined to expect to “have trouble” the next time you have to say that word. And not only that word but also other words that seem to be like it – that begin with the same sound, for example, or that are about the same length, or that are to be spoken to the same sort of listener or in a similar situation, or with a like tone of voice, and so on. You will not expect difficulty in saying all such words every time you have to say them, of course. The number or proportion of them arousing your expectation of trouble will vary, for example, from situation to situation and from listener to listener. The main point is that you do learn to expect trouble, and the more often and the more painfully your expectation is borne out, the more thoroughly you learn to expect trouble.

Wendell Johnson, Stuttering and what you can do about it
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There are many people who played an integral role in the development of these ideas and this study. I would like to thank the members of my committee for providing critical feedback during my prospectus that ultimately led me to the development of the behavioral study. Prahlad Gupta helped a great deal with regard to creating the equation that formalizes the interactions within the SAMI framework. Bruce Tomblin was helpful with the statistical methods and he was very generous with his time when I would continually come in to his office unannounced filled with excitement about the results or just to bounce ideas off of him. Joshua Brown, from the University of Indiana, provided important guidance in the use of the neural network model. I would specifically like to thank my long time mentor Tricia Zebrowski. She has always struck a perfect balance of providing strong mentoring while at the same time taking a hands off approach that has allowed me to pursue every tangential and sometimes crazy ideas that I have had regarding stuttering.

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ABSTRACT

The focus of this thesis was on the relationship between anticipation of stuttering and its effect on speech production in people who stutter (PWS). In order to aid in the development of a hypothesis based study that is rooted in biological mechanisms, this thesis introduced a new theoretical framework from which to view the factors that contribute to stuttering variability. The speech and monitoring interaction (SAMI) framework proposes that there are two systems that account for stuttering variability: the speech production system and the monitoring system.

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INTRODUCTION

Stuttering is a speech disorder characterized by the involuntary repetition, prolongation or cessation of a sound or syllable (World Health Organization, 1977). In short, people who stutter (PWS) exhibit difficulty in the smooth, forward movement of speech. However, within and across PWS the frequency of stuttering varies greatly depending on contextual factors. There are linguistic factors that have been shown to increase stuttering. Stuttering has been shown to occur more frequently in more complex linguistic contexts, and stuttering frequency is affected by word position and word class (Brown, 1935; Buhr & Zebrowski, 2009). There are also several non-linguistic factors that have been found to modulate stuttering frequency. For example, there is a significant amount of empirical and clinical data showing that stuttering is more frequent in contexts of increased emotional arousal or feelings of social pressure to perform (Steer and Johnson, 1936; Porter, 1939; Van Riper and Hull, 1955; Sheehan, Hadley and Gould, 1967; Commodore, 1980; Weber & Smith, 1990; Menzies, Onslow & Packman, 1999). The overall goal of this thesis was to investigate the anticipation of stuttering, which is another non-linguistic factor that has been speculated to play a role in development and moment of stuttering. The anticipation, or expectancy, phenomenon describes the ability of most PWS to predict with a high degree of accuracy the words that they will stutter on (Johnson and Knott, 1937). The nature of the relationship between anticipation and stuttering is not clear. Several theories of stuttering propose a causal relationship, in which the anticipation of stuttering initiates involuntary processes or behaviors that interfere with fluent speech production (Bloodstein, 1960; Johnson, 1959). However, there have been a limited number of studies that have attempted to tease apart the nature of this relationship.

The relatively small amount of research which has examined the relationship between anticipation and stuttering is unfortunate given that conventional stuttering
therapy approaches frequently incorporate techniques aimed at reducing the effects of stuttering anticipation. These therapeutic practices are used despite inadequate data describing the nature of the relationship of anticipation and stuttering. There is also no current theoretical explanation for how anticipation is capable of interfering with speech production to increase the likelihood of stuttering. Presently, there is a need for specific hypotheses and subsequent research investigating the relationship between anticipation and stuttering in order to expand our understanding of both the nature and treatment of stuttering.

An investigation of the relationship between anticipation and stuttering should be rooted in a theoretical model of stuttering that adequately explain the contextual variability of stuttering through the interaction of a wide range factors (linguistic and non-linguistic factors). Ideally the model would describe the functional relationship between factors and provide details regarding the neural substrates of the functional relationships. Two current models of stuttering address the interaction of factors that contribute to the onset and development of stuttering. The Multi-factorial Model of stuttering (Smith & Kelly, 1996) characterizes stuttering as an emergent behavior that results from the dynamic interaction of multiple factors. This model holds that capacities and deficits that exist in the speech production system of PWS can be directly and indirectly affected in a non-linear fashion by non-speech factors. Specific non-speech factors include, but are not limited to, temperament, emotional arousal and characteristics of the environment. The non-linear nature of the interactions results in large changes in speech fluency with relatively small changes in factors such as the speaker’s emotional state. This framework makes the assumption that factors vary across individuals and across time, such that at any given time there is a unique “recipe” that contributes to the stuttering behavior for each PWS (Zebrowski & Kelly, 2002). A second, more recent model is the Dual Diathesis-Stressor (DDS) model (Walden, Frankel, Buhr, Johnson, Conture & Karrass, 2008) which proposes that there are two primary predisposing traits.
that contribute to stuttering: an emotional diathesis and a speech-language diathesis. The DDS model emphasizes that environmental stressors can influence, or exacerbate pre-existing emotional and speech-language diatheses to increase or decrease the stuttering behavior. The interaction of these two diatheses (traits) combined with the influence of internal or environmental stressors (states) affects the frequency of stuttering and potentially the developmental path of the problem.

The Multifactorial and DDS models are widely accepted, with part of their appeal related to their generality with regard to the specific nature of the interaction of factors. This is particularly useful clinically when faced with explaining the disorder to a range of clients who more than likely have disparate constellations of factors that contribute to stuttering. While the DDS model in particular has moved us closer to clarifying the complex state and trait factor interactions underlying stuttering, both it and the Multifactorial model are too general to allow for specific predictions for an empirical investigation of the relationship between anticipation and stuttering. In order to guide the studies in this thesis, a new framework of stuttering is presented that attempts to provide formalized explanations of the functional interactions between linguistic and non-linguistic factors. Further, the framework proposes specific biological mechanisms for these interactions.

The framework presented in this thesis is called the Speech and Monitoring Interaction (SAMI) framework. The purpose of the framework is to explain the functional interaction of factors and how they contribute to stuttering variability. A detailed description of SAMI is provided at the beginning of the literature review. For the purposes of the introduction, the following description of the SAMI framework is provided in order to sufficiently explain the role the framework played in the development of the studies in this thesis. In the SAMI framework there are two independent systems, the speech production system and the monitoring system, that interact to determine the timeliness of the initiation of the next speech motor plan. SAMI
holds that fluent speech is achieved through the timely activation and initiation of the next speech motor plan, while stuttering is defined as the inability to sufficiently activate the next speech motor plan within the time constraints of the current speech rate (see Civier, 2010 for similar criteria). In the SAMI framework, the speech production and monitoring systems are independent, each possessing a unique set of trait and state factors. Within SAMI, the proposed neural substrate for the monitoring system is the anterior cingulate cortex (ACC) which the integrates information across neural systems and one of its primary roles is to optimize performance by initiating a global inhibitory control process over action selection (Alexander & Brown, 2010; Botvinick, 2001; Botvinick, 2007; Bush, Luu & Posner, 2000; Danielmeier, Eichele, Forstmann, Tittgemeyer & Ullsperger, 2011). In general, global inhibition is adaptive because it slows response time and increases the chances that an optimal action will be selected. However, SAMI argues that inhibitory processes from the monitoring system apply increased pressures on the timely selection of the next speech motor plan and this increases the likelihood of stuttering.

In SAMI it is assumed that there are trait and state factors that influence both the speech production system and the monitoring system. Figure 1 depicts a schematic of the SAMI framework with the trait and state factors that are capable of influencing the two systems. What is novel about the SAMI framework is that it focuses on the salient trait and state factors that influence the monitoring system and how this system interacts with the speech production process to create context dependent stuttering. For example, the DDS and Multi-Factorial Model hypothesize that factors like emotional arousal and social pressure are factors that contribute to stuttering likelihood but these models provide no explanation of how these factors interfere with fluent speech. The SAMI framework provides an explicit explanation that these factors modulate the monitoring system (ACC) and the greater the activation of the monitoring system, the greater amount of global inhibition will be exerted on the selection of the next speech motor plan.
The SAMI framework explicitly proposes that the anticipation of stuttering is a factor that modulates the monitoring system. This proposition comes directly from the error-likelihood hypothesis of monitoring that speculates that the ACC is capable of acquiring associations with cues that predict situations in which errors in performance are likely to occur (Brown & Braver, 2005). In the error-likelihood hypothesis it is speculated that the anticipatory activity of the monitoring system is an adaptive process that allows for anticipatory control processes to be initiated sooner in order to decrease the chance of future error commission. However, as speculated earlier within SAMI, the global inhibitory processes that are generally adaptive may become maladaptive when they interfere with the timely initiation of the next speech motor plan. The error-likelihood hypothesis correlates well with the experimental and clinical data showing that contextual cues can acquire the ability to elicit more frequent stuttering if in the past they have been associated with moments of stuttering (Brutten and Shoemaker, 1967; Johnson, Larson and Knott, 1937).

There are two studies reported in this thesis that were motivated by the SAMI framework. Study one utilized a within subject design to investigate the behavioral effects of stuttering expectancy on the verbal response time in word naming tasks. Based on SAMI, it was hypothesized that there would be a positive correlation between stuttering expectancy and verbal response time during fluent speech production. If a behavioral effect of stuttering expectation was present during fluent speech production it would provide evidence that there are motor consequences of anticipation and these consequences may play a causal role in the moment of stuttering. Study two was a series of neural network simulations to test whether results from a model of the error-likelihood hypothesis would qualitatively match the behavioral results from study one. The error rate in the model served as a proxy for the expectation of stuttering from study one. Given that the neural network model is a biologically plausible and well established model, a qualitative match between the results of the two studies would provide support
that the ACC, and the control processes that are initiated by the ACC, are part of the neural substrates related to the causal influences of stuttering anticipation on speech production. Hence, the purpose of the neural network model was to bridge the gap between behavior and the neural substrates such that predictions of neural activity can be empirically tested in a follow-up study using brain imaging techniques.
Figure 1. A schematic of the SAMI framework.
CHAPTER 1: LITERATURE REVIEW

There are three specific aims for this literature review. The first aim is to provide the details of the SAMI framework (section 1.1). The second aim is to provide supporting evidence for the SAMI framework (sections 1.2, 1.3 and 1.4). The third aim is to introduce the error likelihood hypothesis, and to explain how the error likelihood hypothesis may explain the biological foundation of the variability of stuttering that is related to the expectation of stuttering (section 1.5).

1.1 The SAMI framework

The framework presented in this thesis is called the Speech and Monitoring Interaction (SAMI) framework, and it leverages the framework of the DDS with regard to the existence of two independent, but interacting, systems. What makes SAMI unique is that it proposes that specific non-speech traits and states (emotional diathesis and stressors) interact with the speech production system through a domain general monitoring system. In the SAMI framework, the speech production and monitoring systems are independent, each possessing a unique set of trait and state characteristics. While independent, the two systems interact in that the monitoring system continually integrates information from both the environment and the processing of ongoing speech production in order to optimize performance. Further, the monitoring system optimizes performance by initiating a global inhibitory control process over action selection (Alexander & Brown, 2010; Botvinick, 2001; Botvinick, 2007; Bush, Luu & Posner, 2000; Danielmeier, Eichele, Forstmann, Tittgemeyer & Ullsperger, 2011). In general, global inhibition is adaptive because it slows response time and increases the chances that an optimal action will be selected. However, SAMI argues that increased inhibitory processes from the monitoring system in combination with deficits in the speech production system of PWS, interact to increase or decrease stuttering behavior by interfering with the temporal selection and initiation of the appropriate speech motor
plan. Specifically, SAMI holds that fluent speech is achieved through the timely activation and initiation of the next speech motor plan, while stuttering is defined as the inability to sufficiently activate the next speech motor plan within the time constraints of the current speech rate (see Civier, 2010 for similar criteria).

SAMI assumes that there are trait and state factors influencing the stability of speech production process and this ultimately manifest in the temporal variability of the selection and initiation of speech motor plans. Based on robust evidence that there are deficits in the speech production system of PWS (Anderson & Wagovich, 2010; Anderson, Pellowski & Conture, 2005; Brown, Ingham, Ingham, Laird, & Fox, 2005; Chang, Erickson, Ambrose, Hasegawa-Johnson & Ludlow, 2008; De Nil, Kroll, Kapur, & Houle, 2000; Foundas, Corey, Angeles, Bollich, Crabtree-Hartmen, Heilman, 2003; Kleinow & Smith, 2000; Smith, Sadagopan, Walsh, & Weber-Fox, 2010; Wu, Maguire, Riley, Lee, Keator, Tang, 1997), there is an explicit assumption that PWS have a higher level of competitive interference compared to fluent speakers. Within SAMI there is the acknowledgement that trait and state factors within the speech production system contribute to variability in stuttering. For example, the linguistic complexity of an utterance is positively correlated with the amount of stuttering (Buhr & Zebrowski, 2009). However, the variability of stuttering that comes from factors influencing the speech production system is likely due to increased linguistic demands on the system that result in more competitive activation that ultimately slows the initiation of the correct motor plan. This is why models of stuttering that rely solely on deficits with the speech production system cannot adequately explain non-linguistic context dependent stuttering. What is novel about the SAMI framework is that is focuses on the salient trait and state factors that influence the monitoring system and how this system interacts with the speech production process to create context dependent stuttering. Figure 1 depicts the trait and state factors that are capable of influencing the two systems as well as the way that the two systems interact with regard to their influence on motor plan activation.
The equation in figure 2 formalizes the interaction of the two systems in determining whether the next motor plan will be successfully initiated. The variable A is the level of activation of the correct motor plan at the end of the time that is allotted for fluent speech to occur based on the time constraints of the current speech rate. If $A \leq .5$ then the correct motor plan will not be fully activated within the time constraints necessary for fluent speech and stuttering occurs. If $A > .5$ then the motor plan is successfully initiated in sufficient time for fluent speech to occur. The variable m is the level of activation in the monitoring system which in this formalization is the amount of global inhibition on motor plan selection. The variable c is the total activation of the incorrect motor plans that are competing with the correct motor plan. There is an explicit assumption that the more activation of competing units the more inhibition there will be on the correct motor plan. The p variable is the excitatory activation of the correct motor plan. The p variable provides the function with a positive bias such that in the absence of inhibition from the monitoring system ($m = 0$) and absence of competition from incorrect motor plans ($c = 0$), the correct motor plan with any level of excitatory input would have a final activation ($A$) greater than .5 and fluent speech would occur. It is the combined contribution of the competition in the speech production system and the inhibition from the monitoring system that lead to stuttering. Because stuttering results from the combination of these two systems, it explains why stuttering can be absent at times when monitoring is low (e.g. speaking alone) even when the linguistic load is high. Or, why stuttering can occur on linguistically simple utterances (e.g. one’s own name) when the monitoring system is very active. Figure 2 provides detailed examples of both stuttering and fluency.

When comparing PWS to fluent speakers it is possible to use the formalization from SAMI to better define and predict group differences. For example, it is assumed that due to deficits in the speech production system, PWS have greater competitive activation of the incorrect motor plans (c) due to a less efficiently organized and
integrated neural network for speech production. Also, it is speculated that the global inhibition from the monitor (m) is on average greater than fluent speakers during speech production. This speculation maps well to the emotional diathesis that are described in the DDS model. It is, however, assumed that the excitatory input to the correct motor plan (p) is not different between PWS and fluent speakers. This assumption is based on the fact that when PWS have disfluencies, whether it be blocks or repetitions, they know what they are trying to say and they are attempting to say the correct word or sound. It is hypothesized that it is the average cumulative inhibition from competitive activation of the incorrect motor plans (c) and the global inhibition from the monitor (m) that differentiate PWS from fluent speakers.

With the conceptual framework and formalization of SAMI outlined, it is now important to present the proposed neurological substrates and mechanisms that underlie the SAMI framework. Psycholinguists have speculated for several decades that a monitoring system plays an important role in speech production, and the nature of the monitoring system has been a topic of great debate (for a review see Postma, 2000). Until recently most of the hypotheses regarding monitoring in speech production came from psycholinguistic models that were not necessarily concerned with the biological substrates of the monitoring system (e.g. Levelt, 1989). However, in the last decade there has been growing interest in understanding the neural mechanisms of speech production monitoring. One line of research that has proven to be fruitful has been the investigation of the role of a domain general monitoring system in speech production. A domain general monitoring system has been implicated across several modalities, for example, eye movement tracking (Belopolsky & Kramer, 2006), reaction time decision tasks (Falkenstein, Hoorman, Christ & Hohnsbein, 2000), playing musical instruments (Katahira, 2008) and, most importantly for this thesis, in speech production (for a review see Dell, 2011).
Based on imaging studies, brain lesions, and dipole source modeling, the neural substrates for the domain general monitoring system is likely the anterior cingulated cortex (ACC) (Luu, Collins, & Tucker, 2000; Dehaene, Posner & Tucker, 1994). There is broad consensus that the functional role of the monitoring system is to integrate salient information from various sources (motivation, evaluation of errors, and representations from cognitive and emotional networks) in order to optimize performance by modulating cognitive, motor, endocrine, and visceral responses (Bush, Luu & Posner, 2000; Botvinick, 2007; Alexander & Brown, 2010). The specific nature of the modulation from the ACC depend on the particular task being performed, but a general theme is present across tasks in which increased activation is present in perceptual modalities to increase attentional resources toward the ongoing performance, while at the same time inhibitory processes are initiated in the motor domain to slow motor initiation (Egner & Hirsch, 2005; Danielmeier, Eichele, Forstmann, Tittgemeyer & Ullsperger, 2011). An example of inhibitory control over motor selection is in post-error slowing where reaction times increase following error commission (Botvinick et. al., 2001). Functional imaging studies have identified that the neural correlates of the motor inhibition control processes initiated by the ACC are similar to the voluntary stop-signal network that involves the pre-supplementary motor area (preSMA) and the subthalamic nucleus (STN) (Aron & Poldrack, 2006; Aron, Behrens, Smith, Frank & Poldrack, 2007; Danielmeier, Eichele, Forstmann, Tittgemeyer & Ullsperger, 2011). The STN is a structure within the BGTC that has been implicated in a global inhibitory (No GO) response that inhibits all current activation within the BGTC (Frank, 2006). It is explicitly proposed in this thesis that neural substrate of the control mechanism in which the ACC interacts with the speech production process is via the ACC-preSMA-STN inhibitory network. A full review of this network along with imaging studies that provide evidence for its role in stuttering are provided in the literature review of this paper.
Behavioral, electrophysiological, and imaging data have shown that many factors can influence the activation of the domain general monitoring system. For example, emotional arousal and social pressure are positively correlated with ACC activity and response reaction time (Wiswede, Muntere, Goschke & Russeler, 2007; Hajcak, McDonald and Simons, 2003a, 2003b; Hajcak, McDonald and Simons, 2004; Cavahagh and Allen, 2008). Pharmacologic agents such as dopamine antagonists and alcohol have also been found to reduce ACC activation (Ridderinkhof, 2002; Holroyd and Yeung, 2003; Zirnheld, Carroll, Kieffaber, O'Donnell, Shekhar & Hetrick, 2004). Besides the trait and state factors that can modulate the monitoring system, the error-likelihood hypothesis of monitoring speculates that the ACC is capable of acquiring associations with cues that predict situations in which errors in performance are likely to occur (Brown & Braver, 2005). In the error-likelihood hypothesis it is speculated that the anticipatory activity of the monitoring system is an adaptive process that allows for anticipatory control processes to be initiated sooner in order to decrease the chance of future error commission. However, as speculated earlier within SAMI, the global inhibitory processes that are generally adaptive may become maladaptive when they interact with deficits in the speech production. The error-likelihood hypothesis correlates well with the experimental and clinical data showing that contextual cues can acquire the ability to elicit more frequent stuttering if in the past they have been associated with moments of stuttering (Brutten and Shoemaker, 1967; Johnson, Larson and Knott, 1937).

1.1.1 Summary

This section provided a detailed description on the SAMI framework. The formalization within SAMI is intended to provide a quantifiable account of the interaction between the speech production and monitoring systems. The following sections of this literature review provide the rationale for implicating the monitoring system as the means by which non-speech factors affect the speech production system.
1.2 Monitoring and speech production and its role in stuttering

It has been speculated for years that a monitoring system plays an important role in speech production. The Levelt model, which is one of the most prominent psycholinguistic models of speech production, proposed the perceptual loop theory which hypothesizes that there is an independent monitoring system that checks the appropriateness of the speech production plan prior to articulation (the inner loop) as well as the auditory output after articulation (the outer loop) (Levelt, 1989). Within the perceptual loop theory, the monitor is a control mechanism in that it initiates repairs, changes to the speech plan, whenever errors are detected and these repairs result in disfluencies. Disfluent speech can be experimentally induced in normally fluent adults when they are given speaking tasks in which they are primed to have increased phonological, syntactic or semantic errors (Postma and Kolk, 1993). It has also been demonstrated that in normally fluent speakers the disfluent speech resulting from repairs by the monitoring system can be increased or decreased by manipulating factors that influence the vigilance of the monitoring system such as attentional resources being allocated to speech (Arends, Povel, and Kolk, 1988) and social pressure (Baars, Motley & MacKay, 1975; Motley, Camden, Baars, 1982). The psycholinguistic evidence showing that disruptions in speech can be induced in fluent speakers by means of manipulating linguistic factors (priming) and non-speech factors (attention and social pressure) independently, makes a formalized interaction between the monitoring system and the speech production system a good candidate for a mechanistic explanation of stuttering.

The role of a monitoring system in stuttering is not a new concept. The Covert Repair hypothesis of stuttering (Postma and Kolk, 1993) postulates that PWS have language formulation (specifically phonological encoding) deficits that result in a large amount of errors in the speech plan that are detected by the monitoring system prior to
articulation and the subsequent repairs initiated by the monitor result in stuttering
behavior. A similar, but different hypothesis of stuttering was generated based on the
Levelt model called the Vicious Circle hypothesis (VCH) (Vasic & Wijnen, 2005). The
VCH claims that there is no need to assume that PWS have language deficits because
stuttering can be more parsimoniously explained by a hyper-vigilant monitoring system
that detects and repairs erroneous errors. As evidence for the VCH, the authors cite
psycholinguistic studies that demonstrated that the same factors (e.g. attention, social
pressure) that increase or decrease the vigilance of the monitoring system are the same
factors increase or decrease stuttering.

The nature of the monitoring that occurs in speech production has been under
debate for several decades in the field of psycholinguistics (for a review see Postma
2000). The perceptual loop theory (Levelt, 1989) proposes that there is a single
comprehension based system that monitors both the speech plan prior to articulation as
well as one’s own overt speech and other people’s speech. Evidence against a single
comprehension based monitor comes from dissociations in some aphasiacs who have
impaired comprehension but intact monitoring abilities (Nickels and Howard, 1995). An
alternative hypothesis, that does not have the trouble with this dissociation, is a single
production based that utilizes a domain general monitoring system that that evaluates
activation and performance across modalities (Nozari, Dell & Schwartz, 2011). A
domain general monitoring system has been implicated across several modalities, for
example, eye movement tracking (Belopolsky & Kramer, 2006), reaction time decision
tasks (Falkenstein, Hoorman, Christ & Hohnsbein, 2000), playing musical instruments
(Katahira, 2008) and , most importantly for this thesis, in speech production (Masaki,
Tanaka, Takasawa & Yamazaki, 2001; Ganushchak and Schiller, 2006; Ganushchak and
Schiller, 2008; Arnstein, Lakey, Compton and Kleinow, 2011). Based on imaging
studies, brain lesions, and dipole source modeling, the neural substrates for the domain
general monitoring system is likely the anterior cingulated cortex (ACC) (Luu, Collins, & Tucker, 2000; Dehaene, Posner & Tucker, 1994).

One of the primary tools used to investigate the domain general monitoring system is the event related potential called the error-related negativity (ERN) which is a negative going signal which begins shortly after an incorrect motor response begins (Falkenstein, Hohnsbein, Joormann, & Blanke, 1991). The ERN component was initially believed to represent error detection when an incorrect action was performed. However, the ERN component has been found to be present in situations in which no error was committed but instead there are high degrees of response conflict (e.g. Stroop tasks). In light of these findings, an elaborated explanation of the ERN came from Botvinick et. al. (2001), who proposed that the ERN is not elicited by error detection, but rather from the detection of conflicting representations, in which errors are an extreme case of conflict. An alternative to the conflict detection hypothesis is the error-likelihood hypothesis, which speculates that ACC activity comes from anticipatory activation from contextual cues that signal impeding errors (Brown and Braver, 2005). There is evidence showing that both error-likelihood and conflicting representations play a part in activating the ACC. Ridderinkhof et al. (2004) make the argument that regardless of the source of the activation to the ACC, the ERN is an electrophysiological correlate of a signal from the ACC that indicates the need for adjustments in ongoing processes.

The first study to find an ERN in a speaking task was a stroop color-word task in which the participants had to verbally say the color of the written words on the screen (Masaki, Tanaka, Takasawa & Yamazaki, 2001). On some of the trials the written word was a color that was incongruent with the color of the text. The incongruent trials created more errors than the congruent trials. They found that in the error trials there was an ERN similar to that of the ERNs that are present in stroop tasks in which the response is a button press. However, the stroop task is inherently a conflict response task and it is not language specific, so it is difficult to determine if the ERN was due only to response
conflict and was not representative of error monitoring processes in ongoing speech production.

Ganushchak and Schiller (2006) conducted a study looking at ERN in a verbal error monitoring task in which they manipulated time pressure. In choice reaction time tasks, increased time pressure, by means of stressing speed over accuracy, has been found to decrease the amplitude of the ERN. Psycholinguistic studies have found that increased time pressure increases verbal errors (Ooman & Postma, 2001), presumably by reducing error monitoring. Based on these findings, Ganushchak and Schiller hypothesized that an ERN would be present during verbal monitoring errors in both low and high time pressure trials, but the ERN amplitude would be smaller during the increased time pressure trials. Their hypothesis was confirmed. In a follow up study, Ganushchak and Schiller (2008) manipulated motivation to see whether verbal monitoring was similar to other action monitoring in terms of increased ERN amplitudes in high motivation situations compared to low motivational situations. They used a monetary reward for correct responses in the high motivation trials and no reward for correct responses in the low motivation trials. They found that similar to ERNs in non-verbal action monitoring, the ERNs in verbal monitoring showed greater amplitude in high motivation trials compared to low motivation trials.

Another study using ERPs to look at speech monitoring used a task that has been used extensively in psycholinguistics to elicit spoonerisms. Spoonerisms are speech errors in which a phoneme (or phonemes) is placed in the wrong position. An example of this might be, if a person meant to say “I don’t want to burn my toast” but they actually said “I don’t want to turn my boast”. Experimentally, spoonerisms are elicited using a task called spoonerisms of laboratory induced predisposition (SLIP), in which a series of word pairs are presented on the screen and the participant is instructed to silently read the words. Occasionally a “target” pair of words is marked to elicit overt speech. The production of the “target” pair can be influenced by the preceding word pairs because the
initial phonemes in the preceding word pairs have a constant initial phoneme configuration that is incongruent with the “target” pair. For example, initial word pairs may be “ball doze”, “bash door” and “bean deck”, these would be followed by the “target” pair “darn bore” which may result in the spoonerism “barn door”. The trials with spoonerism errors showed ERP negativity that closely resembled the classic ERN found in non-speech errors. Based on psycholinguistic explanations for errors in the SLIP paradigm (Dell, 1986, Levelt, Roelofs & Meyer, 1999), the authors concluded that the ERNs were the result of a conflict between competing phonological representations.

Arnstein, Lakey, Compton and Kleinow (2010) specifically investigated error monitoring in PWS during a speech task and a non-speech task in order to test the hypothesis put forth in the VCH that PWS have a hyper-sensitive error monitoring system. They found that similar ERN components were present in both the non-speech task and the speech task, which further confirmed that the same monitoring mechanism in the ACC is involved in speech and non-speech tasks. They also found that PWS had greater ERN amplitudes compared to the fluent speakers in both the speech and non-speech tasks. Arnstein et al. concluded that this supports the view of a hyper-sensitive monitoring system in PWS that is proposed in the VCH, and they stated that the monitoring system may be an important factor for understanding stuttering.

1.2.1 Summary

This section provided a review of the psycholinguistic evidence that a monitoring system plays in integral role in speech production by means of influencing ongoing speech in order to maximize performance. The role of the monitoring system in stuttering has been proposed in two separate hypotheses. It must be stated clearly that the VCH captures much of the essence of the SAMI framework and the VCH was highly influential on the development of SAMI. However, the VCH is based purely on psycholinguistic models, specifically the perceptual loop model, and makes no attempt to
explain the biological nature of the interaction between the monitoring system and the speech production process. Also, the VCH disavows the notion that there are deficits in the speech production system of PWS and attributes the entirety of the disorder on a hyper-vigilant monitoring system. Within SAMI there is an acknowledgment that deficits within the speech production system are likely to exist and play an important role in the development of the disorder. The SAMI framework also expands on the VCH by extending past psycholinguistic models and proposes specific biological substrates and mechanisms that are outlines below. Based on recent research there is robust evidence that the ACC is neurological substrate to the monitoring system described in the SAMI.

1.3 Trait and state factors that influence stuttering and the monitoring system

Many early theories of stuttering were based on the psychological construct of anticipation, and the notion that stuttering was an anticipatory reaction to cues that signal an impending disfluency. In the words of Wendell Johnson, “Stuttering is everything we do trying not to stutter”, this profound statement carries with it the assumption that at some level the PWS is scanning ahead in time and anticipating imminent stuttering. In the 1930’s Johnson and colleagues conducted a series of studies to understand the nature of stuttering variability. Johnson and Knott (1937) found that for most people who stutter, when reading a passage, the distribution of stuttered words was markedly consistent from one reading to the next. They described this as the consistency effect. Johnson and Millsap (1937) tried to eliminate stuttering through successive readings of the same passage by covering the words that were stuttered after each reading of the text and telling the person to read only the visible words. They found that new words adjacent to the previously stuttered words were stuttered with subsequent readings and these were adjacent to the covered words. They called this the adjacency effect and attributed the new instances of stuttering to a response to contextual cues that were associated with past
stuttering. Many variations of experiments have corroborated the consistency and adjacency effects in both children and adults who stutter (Johnson and Inness, 1939; Hawkins and Brutten, 1964; Neelly and Timmons, 1967; Rappaport and Bloodstein, 1971; Stefankiewicz and Bloodstein, 1973). In subsequent studies, investigations were conducted to understand whether PWS were able to consciously predict the moment of stuttering. Knott, Johnson and Webster (1937) investigated whether PWS are able to predict, with a visual signal, the words that they will stutter on prior to saying the word while reading a text passage. The results showed that across all ten PWS who participated in the study, 94 percent of the disfluencies were anticipated prior to saying the word. These findings were replicated several times with larger groups of PWS, with the results indicating that no less than 85 percent of the disfluencies were predicted prior to saying the word (Bloodstein, 1995).

The association between anticipation and consistency was also investigated by Martin and Haroldson (1967) in a study where they had 30 PWS assign each word in a reading passage an expectancy score according to the following scale: definitely will stutter, 5; probably will stutter, 4; might stutter, 3; probably won’t stutter, 2; definitely won’t stutter, 1. The subjects then read the passage five times in a row and the results showed that words that had higher expectancy ratings were stuttered more frequently and more consistently across readings. Table 1 shows the number and percentage of words at each expectancy rating across all 30 subjects. The table also shows the number and percentage of words that were stuttered across each expectancy rating during the first reading. What is striking is that words with expectancy ratings of 3, 4 or 5 make up 22.8% percent of the words in the passage, but they account for 51.1% of the total number of disfluencies. Also, words with expectancy ratings of 3, 4 or 5 were stuttered 31.4% of the time while words with expectancy ratings of 1 or 2 were only stuttered 8.9% of the time. Supporting the idea that anticipation is related to the consistency effect, there were 136 words where the same subject stuttered on in all five readings and the median
expectancy rating of those words was 3. However, there were 190 words in which a subject never stuttered across all five readings and the median expectancy rating of those words was 1. There is not a one to one relationship between anticipation (expectancy) and actual stuttering, as observed by the fact that 57% of the words with an expectancy rating of 5 were not stuttered, however, there is a strong predictive relationship between anticipation and stuttering.

There is evidence that, even early in the development of stuttering, PWS develop a sense that particular words or sounds are difficult to say. Bloodstein (1960) found that 82 percent of children who stutter where able to list specific words or sounds that they anticipated they would stutter on. In essence, this is a kind of long term representation in which certain contexts (sounds or words) have deep associations indicating that stuttering is imminent. Johnson and Solomon (1937) provided empirical evidence that, at least for words, some form of long term anticipation does exist. In the study they had PWS mark words in a reading passage on which they expect to stutter. They then had the individuals read the passage aloud after an interval of 10 to 15 minutes and then again at least a day later. In both readings about 50 percent of the words that were predicted to be stuttered were stuttered. And only 10 percent of the words that were stuttered were not predicted.

Non-linguistic cues can also become associated with stuttering and subsequently contribute to the likelihood that stuttering will occur in the future. For example, Johnson, Larson and Knott (1937) had PWS read a passage with colored border in front of a large audience. The large audience was used to ensure that the PWS would experience a large amount of stuttering. Subsequently, they found that reading a different passage to a single listener in the presence of the colored border resulted in more stuttering compared to reading to the single listener without the colored border. The authors hypothesized that the colored border had acquired the ability to elicit more stuttering due to its association with previous stuttering. Using similar paradigms in which previously neutral cues become associated with moments of stuttering and subsequently increases
disfluencies when the cue is presented in the future has been further replicated (Brutten and Shoemaker, 1967).

The evidence provided above suggests that PWS acquire associations between cues (e.g. particular phonemes, words, people, contexts) and moments of stuttering and these cues are capable of influencing the likelihood that stuttering will occur in the future when presented with the cue. Based on the ability of people to consciously anticipate upcoming stuttering, it is reasonable to hypothesize that the manner in which the cues elicit their influence over speech production is through some kind of anticipatory influence. From the perspective of the SAMI framework in which a monitoring system is capable of acquiring error likelihood representation, stuttering can be explained as the proactive intervening on current activity through global inhibition.

Similar phenomena of anticipation have been measured in the monitoring system by measuring the ERN component. For example, Krigolson and Holroyd (2007) presented visual stimuli prior to trials in a manual tracking task were ERN components are elicited. They manipulated the pairings of these cues so that some cues were more likely to presented prior to trials in which the experimenters had rigged to tracking task to ensure performance errors. The results showed that after many trials in which certain stimuli (negative cues) were frequently paired with error trials, the trials that were preceded by negative cues began to have ERN components that appeared more quickly after an error compared to errors in trials that were preceded by neutral cues. The authors hypothesized that this shift in the time in which the ERN was initiated provides evidence that the ACC is able to utilize statistical knowledge about past errors that are associated with cues present in the environment. They further speculated that this anticipatory activation makes the ACC able to initiate control processes more rapidly in contexts in which errors are more likely.

A study conducted by Dunning and Hajcak (2007) provided even more compelling evidence that the ACC utilizes statistical knowledge of error likelihood to
generate anticipatory responses. This study was similar to the study by Krigolson and Holroyd (2007) in that certain neutral cues were paired with trials that frequently resulted in errors and other cues were paired with trials in which few errors were elicited. What was different about the Dunning and Hajcak (2007) study was that after the cues had been strongly associated with error trials, they measured ERP that were time locked only to the presentation of the visual cues. The results showed that the ERP component measured after the presentation of the negative cues was observed as a negative deflection that was similar in latency and morphology to the ERN that is traditionally observed after behavioral error commission. This same ERN like component was not observed after the presentation of cues that were not associated with previous errors. This provides evidence that the ACC anticipatory response to likely errors is robust enough that it is capable of eliciting ERN-like responses in the absence of errors. A more detailed discussion of learned cues is provided later in the review of the error likelihood hypothesis of monitoring.

Social pressure is a state factor that is correlated with the monitoring system and stuttering. It has been a clinical observation that most, if not all, PWS speak fluently when talking to one’s self, talking to inanimate objects (e.g. stuffed animals), pets or infants (Bloodstein, 1995). In all of these situations social pressure is very low because nobody is listening to cast judgment upon their speech. The influence of perceived listener judgment on stuttering has been investigated using several different methods. Hansen (1956) experimentally manipulated an audience’s reaction to PWS while they read a passage. Results of the study showed that when the audience reactions were negative the frequency of stuttering increased and conversely when audience reactions shifted positively during the course of the reading the amount of stuttering decreased. Another method of investigating social pressure is by manipulating audience size with the assumption that increased numbers of people provide greater communicative pressure due to increased communicative responsibility, increased threat of unfavorable listener
reactions to stuttering and greater concern about social approval. There are robust findings that there is a positive relationship between audience size and the frequency of stuttering (Steer and Johnson, 1936; Van Riper and Hull, 1955; Commodore, 1980). A classic example of this relationship was provided by a study in which the percent of words stuttered was measured throughout the experiment while the audience size was progressively increased from one, two, four and eight listeners (Porter, 1939). The results showed that stuttering increased in a fairly linear fashion: more people in the audience (more communicative pressure) resulted in more stuttering. Not only does the number of people one is speaking to make a difference in fluency, but the status of the person/s make a difference as well. Sheehan, Hadley and Gould (1967) experimentally manipulated the social status of two listeners that PWS had to speak with. One of the listeners was addressed as “Doctor” and this listener dressed in a suit and tie, while the second listener was not addressed as “Doctor” and dressed in casual clothing. They found that PWS stuttered more frequently when speaking with the “Doctor”, a person of presumably higher social status, compared to the less formal listener.

It is not clear what aspect of social pressure is most salient with regard to increasing stuttering. The effects could be due to emotional arousal, increased sense of risk due to perceived negative consequences or an interaction of these factors with temperamental characteristics. All of these factors have been found to influence the monitoring system in investigations looking at the ERN component (Boksem, Tops, Wester, Meijma, & Lorist; 2006; Hajcak, McDonald and Simons, 2003a, 2003b; Hajcak, McDonald and Simons, 2004). For example, in a study to specifically investigating the effects of social evaluation and the interaction of temperament on ERN, Cavahagh and Allen (2008) conducted a study to record ERN while subjects performed similar tasks while alone (not being judged) and while performing a task while being monitored via a video camera by an examiner who specifically told the subjects that they were going to be evaluated on their performance. To further increase the likelihood that
they would feel pressure of being judged, the subjects were asked for their SAT scores and then told that the task that were about to take was a very good judge of their overall intelligence. The results showed that the ERN magnitude was greater in the scenario where the subjects were being “judged” compared to when they were not. There was also a significant interaction between temperament (degree of behavioral inhibition) and ERN magnitude in the evaluative scenario. The results were interpreted as consequences of different motivational and affective reactivities under social evaluative threat.

Another example of a factor that has been identified to modulate both stuttering and the monitoring system is alcohol. It has been clinically observed that some PWS speak more fluently while under the influence of alcohol (Bloodstein, 1995). It is well known that alcohol decreases the effectiveness of the monitoring system. In error monitoring tasks, blood alcohol level correlates with decreased ERN magnitude and more behavioral errors (Ridderinkhof, 2002). Holroyd and Yeung (2003) reviewed potential mechanism by which alcohol effects the monitoring system and they concluded that alcohol consumption could either directly affect the monitoring system (the ACC), or it could indirectly affect monitoring by impairing the cognitive processes that the monitoring system depends on for generating the error/conflict signals. There is certainly evidence that alcohol impairs efficient cognitive processing, however it is speculated here that, given that PWS may possess deficits in the speech production system, it is unlikely that fluency would be facilitated by further impairments to cognitive processes. Within SAMI it is likely that alcohol would increase the deficits in the speech production system. So, the most likely means by which alcohol would be fluency facilitating would be from reducing activity in the monitoring system which would subsequently reduce the amount of global inhibition on motor plan activation.
1.3.1 Summary

The robust evidence regarding the influence of contextual cues and social pressure on stuttering frequency highlights how pervasive these factors can be in modulating the stuttering behavior. There is also empirical evidence that these same factors correlate with activation of the monitoring system. Although there are several factors that correlated well with stuttering frequency and monitoring system activity (e.g. emotional arousal, pharmacological agents), learned cues and social pressure were discussed in greater detail because they seem to be the two factors that are amenable to change within the therapy context. For example, social pressure is driven primarily from internal forces like concern over people’s reactions. These internal forces can, to a certain degree, be modified through changes in attitude toward stuttering. This is in fact what many stuttering therapies focus on. Therapy techniques like stuttering on purpose or acceptance of stuttering are successful for many PWS. The anticipatory global inhibition on motor activation due to cues that are associated with past stuttering are amenable to change because they are learned representations. Because they are learned they are capable of unlearning through pairing those same cues with successful speech (fluent). This gets to the heart of why understating how these techniques are successful at the biological level can help to create better and more targeted therapies.

Despite the robust evidence of a relationship between anticipation and stuttering, there has been no research that has investigated the influence of anticipation on speech production. Particularly, there have been no studies that have looked at the influence of anticipation on fluent speech in PWS. It is critical to investigate fluent speech because if fluency is viewed on a continuum from highly stable fluent speech on one extreme to highly variable and unstable disfluent speech on the other extreme, data showing that the expectation of stuttering is capable of creating increased variability in speech production
would provide evidence that anticipation is capable of pushing speech toward the more unstable and disfluent edge of the continuum.

1.4 Hypothesized role of the ACC-preSMA-STN network on motor plan inhibition

Within the SAMI framework, stuttering is defined as the inability to sufficiently activate the next speech motor plan within the time constraints of the current speech rate. The inability to successfully initiate the correct motor plan is due to the combined effects of inhibition from competing activation of other motor plans and global inhibition initiated by the monitoring system. This section reviews the literature that provides support that inhibition from the monitor utilizes a “brake” network that involves the ACC-preSMA-STN.

A paradigm that has been used to understand the neural correlates motor inhibition is the stop-signal paradigm. The stop-signal paradigm is an experimental procedure where subjects are prompted to engage in an action at the beginning of each trial (e.g. push and hold one of two buttons). The majority of trials are “Go” trials, which the subject just has to press and hold the correct button for the duration of the trial. In some of the trials, there is a stop signal in which the subject has to inhibit the “Go” action (prevent pressing the button or depress the button). Aron, Behrens, Smith, Frank and Poldrack (2007) used a stop-signal paradigm to investigate the hypothesis that the mechanism of controlling response selection is via the initiation of “Global NoGo” signals in the STN. Using diffusion-weighted magnetic resonance imaging (DWI) and functional MRI (fMRI), Aron et al. investigated the brain regions that are associated with motor inhibition. The DWI results discovered that there is a three-way anatomical network in the right hemisphere that includes the right inferior frontal cortex (IFC), the preSMA and the STN. The fMRI results corroborated this anatomical network by
showing evidence that the activation of these three structures in this network were significantly correlated with behavioral measures of motor inhibition. The authors specifically stated that this network between these three structures seems to be a functional “braking” or inhibitory network.

The stop-signal paradigm is a voluntary global inhibition of all current motor activity, therefore the “braking” or inhibitory network that was discovered by Aron et al. (2007) is not easily mapped directly to the phenomenon of adaptive motor inhibition that is initiated by the monitoring system. However there is significant evidence that the preSMA is consistently found to be activated in the same tasks and paradigms that elicit ACC activation and subsequent control processes (Garavan, Hester, Murphy, Fassbender & Kelly, 2006; Miller & D’Esposito, 2005; Rushworth, Walton, Kennerley & Bannerman, 2002; Wittfoth, Buck, Fahle, & Herrmann, 2006). But even more directly, it has been shown that interfering with the function of the pre-SMA specifically alters task switching performance which relies on inhibition of the ongoing task (Nakamura et al., 1999; Rushworth et al., 2002; Shima and Tanji, 1998). An investigation of post error slowing was conducted using fMRI in an attempt to directly test whether the monitoring system utilizes the “braking” network to optimize performance by inhibiting motor activity (Danielmeier, Eichele, Forstmann, Tittgemeyer, & Ullsperger, 2011). The results from fMRI showed that post-error slowing was correlated with activity in the ACC, the preSMA and STN. These findings support the hypothesis that the monitoring system uses the motor inhibition network involving the preSMA and STN and provides further evidence to support the hypothesis that the ACC is capable of initiating control processes that directly inhibit motor responses (Marco-Pallares, Camara, Münte & Rodriguez-Fornells, 2008; Tanimura, King, Williams & Lewis, 2011).

If the ACC-preSMA-STN inhibitory network does play a role in stuttering variability it is important to show how this network could be integrated with current models of stuttering that have investigated deficits within the speech production system.
Civier (2010) used a computational model of speech production, which incorporated the dynamic interactions within BGTC circuits, to investigate whether two separate hypothesized deficits both lead to disfluent behaviors. In one simulation he tested the hypothesis that stuttering is caused by too much dopamine neurotransmitter in the striatum which leads to delays in the initiation of speech motor plans (Wu et al., 1997; Alm, 2004). The second simulation tested the hypothesis that there is a deficit in the connectivity between the primary motor cortex and the basal ganglia (Chang, Erickson, Ambrose, Hasegawa-Johnson & Ludlow, 2008; Sommer, Koch, Paulus, Weiller & Büchel, 2002; Watkins, Smith, Davis & Howell, 2008). Within both simulations he found that the lesioned models showed delays in initiating motor plans, which was equated to disfluent behavior. By utilizing a computational model, Civier provided a clearer understanding of how two separate subtypes of deficits within the BGTC could result in stuttering behavior. However, within these simulations the stuttering behavior would be consistent across all words unless some stochastic noise is implemented that make the disfluencies occur in a random fashion devoid of contextual influence. The model used in these simulations was a simplified model of the basal ganglia that did not include the STN. By including the STN, which has been shown to be an important structure for normal functioning within the BGTC, the model used by Civier (2010) could incorporate the ACC-preSMA-STN inhibitory network that is proposed in the SAMI framework. By including this network, the Civier (2010) model could possibly achieve context dependent stuttering.

1.4.1 Summary

Within the SAMI framework it is proposed that the ACC-preSMA-STN network is the means by which the monitoring system casts its influence over the speech production process by globally inhibiting the activation of speech motor plans. There is DWI and fMRI data supporting the existence of this network and its utilization in motor
inhibition to optimize performance. This proposed mechanism would fit well within models of stuttering that hypothesize that delays in the selection of the motor plan are responsible for stuttering behavior.

1.5 Error likelihood hypothesis of monitoring and stuttering expectancy

The error likelihood hypothesis of monitoring (Brown and Braver, 2005) states that the ACC learns to predict error likelihood in a given context based on reinforcement learning from previous errors. This is in contradiction to the conflict monitoring theory (Botvinick, Nystrom, Fissel, Carter, & Cohen, 1999) which hypothesizes that ACC activity is positively correlated with activation of conflicting (or competing) representations. The conflict hypothesis views the ACC as a reactive monitor that is only capable of initiating control processes after conflicting activations are already present. The error likelihood hypothesis views the ACC as a proactive monitor that is adaptively able to anticipate contexts in which it would be advantageous for the ACC to assert control processes to reduce the likelihood of an error (Alexander & Brown, 2010).

Brown and Braver (2005) used two computational models, one that instantiated a conflict monitor and one that instantiated an adaptive monitor that learns error likelihood representations, to compare the two hypotheses of monitoring. The two models were compared against each by determining which model best matched behavioral and fMRI data in a go/change task. The two models were similar in that they have identical input layers and response (output) layers, and they both had an ACC layer that activates a control process that interacts with the response (output) layer by means of global inhibition. However, the error-likelihood model had no connections from the response layer to the ACC. In the error-likelihood model the ACC layer becomes activated based on connections from the input layer only. These connections are modified based on co-activation of ACC and Input units when there is an incorrect (error) response. The
experiment entailed the subjects responding with a button press of the left or right index finger corresponding to the direction of an arrow that appears in the center of the screen. One-third of the trials were “change” trials where a second arrow appeared that is larger than the first arrow and above it, and points in the opposite direction. The appearance of the second arrow instructs the subject to, if possible, withhold the response to the first arrow and instead substitute a response to the second arrow. The change signal delay (CSD) is the time difference between the onset of the first and second arrows. The likelihood of an error can be increased by increasing the CSD, which increases the likelihood that subjects will be committed to responding to the first arrow before they can cognitively process and respond to the second arrow. In the task, one cue color (e.g. white) is associated with a short CSD and therefore a low error likelihood (about 4% errors), if a change signal (second arrow) should appear. Another cue color (e.g. blue) is associated with a long CSD and therefore a higher error likelihood (about 50%). The task dissociates among error, conflict, and error likelihood effects as follows. When the change signal appears, the contrast of error commission vs. correct response yields the error effect. The contrast of correct change (two arrows) minus correct go (single arrow) trials yields the response conflict effect. In correct trials with only a single arrow (go trials), the contrast between high vs. low error likelihood color cues yields the error likelihood effect.

Using the two computational models, simulations were run with the same inputs across both models. The nature of the input to the models was used to simulate the real experiment that the humans performed. The activation of the ACC layer in both of the models were compared to the fMRI results from real subjects. Both models demonstrated similar ACC activation in the high conflict trials (trials with long a delay for the change signal), but only the error-likelihood model fit the fMRI data where there was increased ACC activation in the low conflict trials where there was a high error-likelihood cue. The increased ACC activity in the low conflict trials with the high error-likelihood cues
also correlated with increased reaction times in those trials. The increased reaction time in these trials was speculated to be the results of inhibitory processes from the ACC to the pre-motor cortex. The authors interpreted the results as evidence that the activity in the ACC in high conflict contexts could be explained through anticipatory activation of the ACC to error likelihood based on contextual cues, instead of the ACC directly monitoring current conflict. However, in the initial trials of the experiment the ACC of the real subjects did respond to the high conflict trials regardless of the color cue that preceded the trial. The error likelihood computational model did not match this response in the early trials because it had not yet acquired the association between the cues and error likelihood. This does provide evidence that the monitor also responds to conflict as well. Several studies since the Brown and Braver (2005) publications have provided evidence for both the conflict and error likelihood hypotheses (Krigolson & Holroyd, 2007; Dunning & Hajcak, 2007). This supports the notion that the ACC may be monitoring several things including the evaluation of errors, conflict monitoring, and error likelihood (Bush, Luu & Posner, 2000; Botvinick, 2007; Alexander & Brown, 2010). It is also seems that the response of the monitor to these events or processes that it is monitoring can be modulated by emotional arousal (Hajcak, McDonald and Simons, 2003a, 2003b; Hajcak, McDonald and Simons, 2004) or pharmacological agents (Zirnheld, Carroll, Kieffabé, O'Donnell, Shekhar, Hetrick, 2004; Ridderinkhof, 2002; Holroyd and Yeung, 2003).

1.5.1 Summary

The error-likelihood hypothesis provides evidence that the ACC activity during behavioral tasks is not necessarily being caused by conflicting activation or overt errors. The hypothesis shows that the ACC responds to the presentation of cues that have previously been associated with contexts in which errors occurred. Experimentally, Brown and Braver (2005) showed that similar trials in which there was no activation
conflict (a simple Go trial) can have varying degrees of ACC activity based on a color cue that was presented immediately prior to the trial onset. One of the color cues had been associated with contexts of high errors and the other was associated with low errors. The results from fMRI showed that in the identical Go trials in which there was no activation conflict, there was greater ACC activity on the trials that were preceded with the high error cue. The most critical aspect for this thesis is that Go trials that were preceded by the high error cue had slower reaction times compared to the exact same Go trials that were preceded by the low error cue. This provides direct evidence that anticipation has motor consequences.

The error-likelihood hypothesis can be directly applied to stuttering if instances of stuttering are viewed as performance errors. When stuttering (errors) occur, associations are formed between the stuttering and the context in which it occurred. In the future, when presented with those contextual cues, the ACC will respond to the association of increased error likelihood. If the response by the ACC is in some way capable of influencing the speech production system in a negative manner that increases the chance of stuttering, a cyclical pattern of increased stuttering can develop in which cues precipitate increased stuttering and increased stuttering (errors) strengthens the associations between errors and the contextual cues.

It is proposed in this thesis that the error likelihood hypotheses of monitoring provides an elegant biologically plausible explanation for how learned associations between stuttering (errors) and contextual cues can negatively influence speech production. It is further hypothesized that subjective ratings of stuttering expectancy to cues (words, sounds or situations) reflects a conscious awareness of underlying error-likelihood representations in the brain.
1.6 Statement of purpose

There is little known about the relationship between the expectancy of stuttering and actual stuttering. Is there a causal relationship between the expectations of stuttering and actual stuttering? What are the biological mechanisms if such a causal relationship does exist? The purpose of this thesis was to begin to understand these questions. By utilizing SAMI and the error likelihood hypothesis of monitoring (Brown and Braver, 2005) it was hypothesized that words that are rated with a high expectation of stuttering would have slower verbal response times compared to words with low ratings of stuttering expectation. To test this hypothesis, two studies were conducted. Two studies were conducted to test this hypothesis. Study one was a behavioral task that had two primary goals: 1) characterize the degree to which PWS anticipate stuttering and how accurately they can predict actual stuttering, 2) investigate the correlation between stuttering expectancy and the verbal response time in a word naming tasks. Study two was a series of simulations using a biological plausible neural network model of the monitoring system. The error rate in the model served as a proxy for the expectation of stuttering in study one. The goal of the simulations was to make a qualitative comparison of the behavioral results and the results from the neural network model.
Figure 2. Formalization of the SAMI framework

\[ A = \frac{1}{1 + e^{(p - m - c)}} \]

If \( A \leq .5 \) then the correct motor plan will not be fully activated within the time constraints necessary for fluent speech and stuttering occurs.

If \( A > .5 \) then the motor plan successfully initiated on time and fluent speech occurs.

- \( A \) = activation of correct motor plan at the end of the allotted for fluent speech to occur
- \( m \) = activation of the monitoring system (level of global inhibition)
- \( c \) = total amount of activation of incorrect motor plans
- \( p \) = amount of activation of the correct motor plan
Table 1. Data reproduced from Martin and Haroldson (1968).

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<th>Percent of All Words</th>
<th>Total Word Stuttered</th>
<th>Percent Stuttered</th>
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<td>56</td>
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CHAPTER 2: STUDY ONE

2.1 Methods

2.1.1 Participants

Participants were 8 adults who stutter (4 female), ranging in age from 18 to 41 years of age (mean=27.3; SD=7.3) and 8 age- and gender-matched adults who do not stutter (mean = 27.4; SD=7.4). The age matched pairs were all within 14 months in age. Demographic data for participants in this study are presented in table 2. All PWS were self-described to be stuttering, started stuttering in childhood and had no known neurological impairments. All of the PWS reported having received treatment for stuttering at one time in their life. Participants classified as PWS scored 13 and higher on the Stuttering Severity Instrument -3 (SSI-3; Riley, 1994) (mean SSI-3 = 24.88; SD=11.12). The PWS completed the Overall Assessment of the Speaker’s Experience of Stuttering (OASES) (Yaruss & Quesal, 2006). The OASES is a questionnaire that assesses the PWS in terms of their perceptions of their stuttering and a measure of the impact that stuttering has had on their lives. This study’s protocol was approved by the Institutional Review Board of the University of Iowa, Iowa City, Iowa. Each of the 16 participants read and then signed informed consent to participate in the present study.

All 16 participants were paid volunteers, with some recruited from the University of Iowa’s Wendell Johnson Speech and Hearing Clinic where they were receiving treatment for stuttering and others recruited from the areas surrounding Iowa City.

2.1.2 Anticipation of stuttering questionnaire

At the beginning of the first visit, the PWS responded to five questions on a laptop computer regarding their opinions about their anticipation of stuttering across different contexts. This is referred to as the Anticipation of Stuttering Questionnaire (ASQ). The questions were developed by the investigator of this study in order to get a
subjective perspective of the degree to which the PWS anticipate stuttering across a range of contexts. The questions are similar to questions asked in the Speech Situation Checklist (SSC) (Brutten, 1973), which uses a Likert scale to evaluate 51 speech situations the degree to which they elicit negative emotion and/or increased stuttering. Figure 3 shows the computer program that was used for data collection. Table 3 provides the five questions. The participants responded by using a mouse to slide a bar along a line that represented a continuum of how much they agreed with the statements. The far left of the continuum meant that they “strongly disagree” with statement and the far right extreme meant that they “strongly agree” with the statement. The placement of the slider equated to a continuous value from 0 to 100, with zero being “strongly disagree” and 100 being “strongly agree”.

2.1.3 Stimuli

A set of 50 words were used throughout this study to assess behavioral effects of stuttering expectancy. The words were selected using the English Lexicon Project (ELP) database (Balota, et. al., 2007). The ELP is a collection normative data from speeded naming tasks for over 40,000 words across 1200 subjects. Besides normative verbal response time data, the database also has lexical properties of the words to assist in stimuli selection to better test theories by reducing potential confounds in empirical studies. The goal of stimuli selection was to provide a set of 50 words that were similar to one another in verbal response time. This was accomplished by first filtering the ELP database such that only words that had a verbal response time z-score between -.5 and .5 were selected; this provided a set of 12,205 words. Next, in order to control for as many lexical and orthographic factors as possible, the words were further filtered to include only words that were 6 to 8 letters long, a word frequency log score from 2.75 to 4.25, and had 4 to 7 phonemes. This layer of filtering provided a set of 428 words. From this 428 words subset, a subset of 50 words were selected to fit into a 266 word passage that
was used for the oral reading task in this experiment (see appendix for the passage). Table 4 provides the list of 50 words that were selected as the final stimuli set. Table 5 provides the details of the lexical properties of the entire set. The word frequency measures were based on a corpus of words from movie and television shows called SUBTLEXus (Brysbaert and New, 2009).

2.1.4 Expectancy ratings

At the beginning of each visit, the PWS were asked to rate the degree to which they would expect to stutter on each of the 50 words in the stimuli set. The rating was very similar to how the participants responded to the five questions in the ASQ. The participants responded by using a mouse to slide a bar along a line that represented a continuum of how much they expect to stutter on the word. The far left of the continuum represented “no expectation of stuttering” on the word and the far right extreme represented “high expectation” of stuttering on the word. The placement of the slider equated to a continuous value from 0 to 100, with zero being “no expectation” and 100 being “high expectation”. The computer program in which the expectation ratings were recorded showed 5 of the 50 words from the stimuli set at a time. When the participants got finished rating the five on the screen, they clicked to the next page and rated the next five. When they finished all 10 pages they were finished. Figure 4 shows the screen from the first page of the computer program.

2.1.5 Reading task

A 266 word narrative was created that incorporated the 50 stimulus words. The appendix provides the entire narrative. At the first and second visits the PWS were asked to read the narrative 5 times in a row. Between the two visits the PWS read the passage 10 times, providing an opportunity to stutter on each of the 50 stimuli 10 times. The readings were audio and video recorded. The readings were analyzed for disfluencies on
the 50 words in the stimulus set. The purpose of the task was to collect a single stuttering score from 0 to 10 for each word in the stimulus set.

2.1.6 Immediate naming task

During the second visit for the PWS (but at the first and only visit for the PWNS) the participants engaged in a word naming task in which single words appeared on a computer screen and the participants were instructed to say the word as quickly as they can. The methodology of this task was based on Fushimi, Ijuin, Patterson and Tatsumi (1999). The stimuli words that were presented on the screen were the same 50 word stimulus set that were used throughout this study. Each of the 50 words were randomly presented four times for a total of 200 trials. Trials began with a mouse click by the participant after an inter-trial interval (ITI) of 1,500 ms. 1,500 ms after the click, a fixation point appeared in the center of the screen for 920 ms, after which time an audible tone occurred simultaneously with the visual presentation of the stimulus word. The word remained on the screen until the participant said the word or until 2,000 ms had elapsed. The participants were instructed to name each word that appeared on the screen as quickly as possible. Figure 5 provides a flowchart of the immediate task. During the experimental task, the audio was digitally recorded using a Sony voice recorder and the audio and video were recorded using a digital video recorder (Kodak PlaySport).

2.1.7 Delayed naming task

After the immediate naming task, the participants began the delayed naming task. The methodology of the delayed task was also based on Fushimi, Ijuin, Patterson and Tatsumi (1999). This task was very similar to the immediate naming task in that it also used the same 50 word stimulus set that was used throughout this study, and each word was presented four times in a random order for a total of two hundred trials. Trials began with a mouse click by the participant after an inter-trial interval (ITI) of 1,500 ms. The stimulus word appeared on the screen 1,500 ms after the mouse click that began the trial.
The word remained on the screen for a variable duration of 1,200, 1,360, 1,520 or 1,680 ms, during which time the participant was instructed to prepare but not speak the word. After the variable duration of word presentation, the word was replaced on the screen with a black square and accompanied by an audible tone, these cued the participant to initiate articulation of the word. Each word was presented with a delay of 1,200, 1,360, 1,520 and 1,680 ms. Figure 6 provides a flowchart of the delayed task. The variable delay was used to help prevent the participant’s anticipation of the articulation cue which may affect response time. Since all words were presented with the same delay times, any confounds for the effects of delay were controlled. During the delayed task, the audio and video were recorded similar to the immediate task.

The immediate and delayed naming tasks were both used because they measure different aspects of word naming. Immediate word naming tasks are assumed to involve the activation of orthographic representations, the translation of these in to a phonological code, and finally the conversion of the activated phonological representations into articulatory motor commands for speech production (Balota & Chumbley, 1985; Jescheniak & Levelt, 1994; Seidenberg, Petersen, MacDonald, & Plaut, 1996). In a delayed naming task, participants are instructed to prepare the pronunciation when each stimulus word arrives but then to wait until a response signal appears before initiating the response. Given a sufficient interval between the stimulus and the response signal, the phonological code is assumed to be fully generated during the delay. Response times in the delayed naming task are therefore considered to reflect the time required for processes subsequent to the phonological computation, namely initiation of articulation. A comparison of response time in the immediate and delayed naming tasks can thus control, to a large extent, for the influence of potentially confounding variables like the numbers of phonemes and differences between initial phonemes (Fushimi, Ijuin, Patterson and Tatsumi, 1999). By incorporating both of these tasks in this study, it
provided a better chance of understanding what specific processes or stages of production were being affected by stuttering expectancy.

2.1.8 Procedure

The PWS participated in two visits that took place between 5 to 10 days apart. At the first visit, after being consented, the PWS completed the 5 question ASQ. Next, they stayed at the computer and they rated the 50 stimuli words regarding their stuttering expectancy. Following the expectancy rating, they read the passage aloud 5 times while being audio and video recorded. Immediately after the readings, a 5 minute conversational speech sample was recorded to aid in assessing stuttering severity. Lastly, the PWS filled out the OASES and the Taylor-Johnson Temperament Analysis (Taylor & Morrison, 1996). The first visit lasted approximately one hour. At the beginning of the second visit, the PWS again rated the 50 words on the computer with regard to their stuttering expectancy. Following the ratings procedure, the PWS read the passage aloud five times in row while being audio and video recorded. Next, the experimental tasks were performed. The immediate task always preceded the delayed task. The participants sat comfortably in front of a laptop computer with a mouse and a microphone placed in front of them. Before each of the experimental tasks the participants performed ten practice trials that used stimuli words that were unrelated to the 50 stimulus set. The practice trials were not analyzed; these trials were used to ensure that the participants understood the instructions. The second visit for the PWS lasted approximately one hour.

The participants in the PWNS group were seen for one visit in which they participated in the experimental tasks (the immediate and delayed naming tasks). Their visit lasted approximately 45 minutes.
2.1.9 Data analysis

2.1.9.1 Stuttering identification

Fluency was determined by whether words were perceptually fluent, meaning there were no starter sounds, no repetitions or audible or inaudible prolongations. This was determined by both audio and video recordings. First the audio was used to identify lacks of continuity in the forward flow of speech. Words that were audibly perceived as stuttering were recorded as such. If there were any inappropriate hesitations, moments of silence, starter sounds or prolongations that were not easily identified as stuttering by the audio, the video was reviewed to make a definitive decision. This method was used to rate the conversational speech sample, the ten oral readings of the passage and during the analysis of the experimental task.

2.1.9.2 Reaction time data

The critical measurement from the immediate and delayed naming tasks was the verbal response time (reaction time). Using the digital audio recording, verbal response times were measured manually by displaying the waveform associated with each trial. Verbal response time was operationally defined as the duration of the interval between the onset of the tone and the first visual indication of speech initiation in the waveform. Manually measuring response time avoids the effects of nonspeech sounds and is more accurate than a voice-activated relay in detecting weak sound onsets like fricatives (Sakuma, Fushimi & Tatsumi, 1997). Trials were excluded from analysis if the words were pronounced incorrectly or if the subject said the word prior to the cue in the delayed task. In the immediate task, 15 trials were excluded due to mispronunciation of the word or errors (i.e. saying the wrong word) (13 by PWS and 2 by PWNS). In the delayed task 6 trials were excluded because the word was spoken prior to the cue (1 from PWS and 5 from PWNS). Trials were also excluded if the word was not spoken fluently. Fluency in the experimental tasks was similar to that used when the oral reading task was analyzed.
Only three subjects stuttered on words during the naming tasks, these were the three subjects with the highest SSI score. A total of 132 words were stuttered during the immediate task (subject 11001 had 28, subject 11007 had 100, and subject 12008 had 4). A total of 138 words were stuttered in the delayed task (subject 11001 had 9, subject 11007 had 127, and subject 12008 had 2).

Reaction time studies are typically used to investigate trait factors or knowledge. An example of a typical reaction time study would be one that investigates whether nouns are retrieved faster than verbs. One method of ensuring that the trait factors in question are truly being represented by the data, is to use a trimming method to exclude trials in which the reaction times were greater than two standard deviations away from the mean. This is a reasonable method based on the assumption that outliers are likely the result of state factors (e.g. a subjects fleeting lack of attention) that would erroneously skew the data. However, the current study was somewhat atypical in that the independent variable was not a trait characteristic (e.g. word class retrieval), but a subjective expectation of stuttering on words that was independent of properties of the words. The hypothesis was that the expectation of stuttering would induce a state response that would slow the production of the word, thus the expectation of stuttering would be positively correlated with reaction time. For the hypothesis to be tested it was essential that the reaction times across words (varying levels of expectation) not be trimmed based on standard deviations that were derived from the entire pool of words. The hypothesis was based on the assumption that some words will have slower reaction times. In the example study described above in which there is a hypothesis of differing reaction times between nouns and verbs, the data would be trimmed based on standard deviations derived from within the word class. If the data were trimmed based on all the words (nouns and verbs), true differences in reaction times may be lost because one word class may be trimmed more than the other. Due to the continuous nature of the independent variable (expectancy rating) in this study and the low number of trials per word, it was not reasonable to trim
the data based on standard deviations based on within independent variable categories. For this reason, the data in this study were not trimmed based on the typical two standard deviation method. Rather, a cutoff point was determined in which all trials in which the reaction time exceeded 1000 msec were excluded from data analysis. Based on this cut off criteria only 3 trials were excluded from the analysis in the immediate naming task (2 PWS and 1 PWNS) and 2 from the delayed naming task (1 PWS and 1 PWNS).

2.1.10 Statistical analysis

2.1.10.1 Data aggregation and normalization

Each PWS had a pooled stuttering expectancy rating (the average of the ratings from the first and second visit) for each word. Each subject also had two mean reaction times for each word, one from the immediate and from the delayed task. The mean reaction times were based on the valid trials the subject had for each word. Subject 11007 was the only person who had words in which there were no valid trials. For this subject there were 14 words in the immediate task and 17 words in the delayed task that were not used in the analysis.

One of the primary goals of the study was to determine whether the within subject variability of stuttering expectancy was correlated with the within subject variability of reaction time. In order to assess a PWS group effect of expectancy on reaction times it was necessary to normalize the expectancy and reaction time data so that all subjects were on the same scale. Two methods were used for normalizing the data. The first method was a delta approach in which the lowest score for each subject was used as a within subject baseline and this value was then subtracted from all other values within the subject, thus making differences in absolute scores irrelevant. These scores will be referred to as adjusted scores. However, a weakness of this approach is that there were significant differences in the range of reaction times and expectancy scores across subjects. A second method was used to try to reduce any confound of differences in
absolute ranges. In the second method, all scores were converted to within subject standard scores. The difference reaction time values (immediate – delayed) were not adjusted using the delta method because the within subject and word subtraction normalized the absolute time differences. However, the difference reaction times were standardized for the analysis that used standard scores. Results from analyses using both normalization methods are reported.

2.2.10.2 Statistical methods

Descriptive statistics were used to characterize the mean and standard deviation of the responses from the ASQ and the stuttering expectancy ratings for the 50 word stimulus set. Correlation analyses were the primary method for determine relationships between the expectancy rating and the reaction times from the experimental tasks. It was hypothesized that there would be a positive correlation between expectancy ratings between the two visits, implying a high degree of consistency within subjects with regard to the words that they expect to stutter upon. It was hypothesized that there would be a positive correlation between expectancy ratings and the number of times that the words were stuttered across the ten oral readings. It was also hypothesized that there would be a positive correlation between stuttering expectancy and reaction time on both the immediate and delayed task.

MANOVA analyses were conducted to determine between group differences in reaction time and the potential interaction of high/low expectancy words on the group differences. The reaction times from the immediate and delayed task, as well as the difference RT, were entered as the dependent variables. Group (PWS or PWNS) and expectancy level (high/low) were the fixed factors. It was hypothesized there would be a main effect for group, but the main effect would be driven by the a significant interaction effect of group and high/low word classification indicating that there was a group
difference only in the high stuttering expectancy words but not the low expectancy words.

2.2 Results

2.2.1 Stimulus validation

Significant effort was spent choosing stimulus words that would not differ in their verbal response time so that differences in reaction time within PWS could more likely be attributed to their stuttering expectation. To test the validity of this assumption, the pooled average reaction times on words within the PWNS were submitted to a MANOVA with reaction times from the immediate and delayed task as the dependent variable and words was the fixed factor. The results showed no significant main effect for words for the immediate \([F(49,350)=1.228, p=.152]\) and the delayed \([F(49,350)=.262, p>.999]\) reaction times. The lack of significance indicates that for fluent speakers there were no properties of the words that would make their verbal response time different from one another.

It was also important to investigate differences across words within the PWS group to ensure that within subject results were not influenced by properties of the word that were significant to PWS but not PWNS. A MANOVA was conducted with pooled average reaction times from the immediate and delayed task and the expectancy scores as the dependent variable and words was the fixed factor. Result showed that, as a group, there was no significant main effect for the immediate \([F(51,325)=.778, p=.862]\) and the delayed \([F(51,325)=.503, p=.998]\) reaction times. There was also not a significant main effect for expectancy score \([F(51,325)=1.384, p=.051]\). The main effect for expectancy score was close to significance, but given that there were no significant differences in reaction times across the PWS, it reduces that chances that differences in expectancy on words across the group would skew the effects of expectancy on reaction time.
2.2.2 Within subject results

2.2.2.1 Stuttering expectancy and stuttering consistency

Table 6 provides information regarding the PWS stuttering severity, their 5 responses to the ASQ, and descriptive statistics about the expectancy rating for the 50 words. At both visit 1 and visit 2 the PWS rated their stuttering expectancy for each word. It was hypothesized that ratings across the visits would be significantly correlated. A correlation analysis showed a significant relationship between the ratings \[ r(400)=.741, \ p<.001 \]. Figure 7 plots the relationship.

It was hypothesized that there would be a correlation between expectancy scores and the number of times the word was actually stuttered across the ten oral reading of the passage that contained the 50 stimuli words. Only three of the eight PWS produced stuttering while reading the text aloud. These were the subjects with the three highest SSI scores. Given that the five subjects with the lowest SSI scores produced no stuttering, two correlation analyses were conducted. One analysis was conducted using only the three subjects who produced stuttering during the oral reading. Another analysis was conducted using all eight PWS. The analysis using only the three PWS who presented with stuttering in the oral reading showed a significant correlation between the total number of stuttered words and the absolute expectancy scores \[ r(150)=.374, \ p<.001 \], the adjusted expectancy scores \[ r(150)=.381, \ p<.001 \] and the standard expectancy scores \[ r(150)=.383, \ p<.001 \]. The analysis using all eight PWS showed a significant correlation between the total number of stuttered words and the absolute expectancy scores \[ r(400)=.248, \ p<.001 \], the adjusted expectancy scores \[ r(400)=.281, \ p<.001 \] and the standard expectancy scores \[ r(400)=.189, \ p<.001 \]. The relationship between total number of stuttered words and expectancy across all PWS is plotted in figure 8. These results provide evidence that PWS who show more severe stuttering were able to predict with a fair degree of accuracy the words that they were going to stutter.
upon, even when there are several minutes between the expectation rating and the actual stuttering.

### 2.2.2.2 Effects of stuttering expectancy on reaction time

There was a significant correlation between expectancy scores and immediate reaction time using both the adjusted scores \([r(386)=.248, p<.001]\) and the standard scores \([r(386)=.170, p=.001]\). The delayed reaction times were not significantly correlated with expectancy scores using either the adjusted scores \([r(384)=.036, p=.483]\) or the standard scores \([r(384)=.065, p=.201]\). There was a significant correlation between expectancy scores and the difference reaction time using the adjusted scores \([r(377)=.162, p=.002]\) but not the standard scores \([r(377)=.033, p=.526]\). Figure 10 is a scatter plot of the relationship between the immediate reaction times and expectancy scores using the adjusted values. The adjusted scores were specifically used in the figure because the relative difference of the absolute reaction times most closely maps to the output from the neural network simulations.

### 2.2.3 Interactions with stuttering severity

#### 2.2.3.1 Stuttering severity and ASQ

Analyses were conducted to determine whether any of the responses on the ASQ were correlated with stuttering severity (SSI). There was a significant correlation between SSI and the response to question 4, “I believe that anticipating that I am going to stutter increases the likelihood that I will stutter” \([r(8)=.909, p=.002]\). Figure 10 displays a plot this relationship. There was also a significant correlation between SSI and the response to question 3, “There are certain sounds that I anticipate stuttering on more than other sounds” \([r(8)=.837, p=.01]\). The correlation between SSI and the response to question 2, “I consistently anticipate stuttering on particular words” was close to being significant \([r(8)=.635, p=.09]\).
2.2.3.2 Stuttering severity and the effect of stuttering expectancy on reaction time

There was a significant correlation between normalized expectancy scores and normalized reaction times for the immediate task using both relative absolute differences and standardized values. Due to the statistically significant effects across normalization strategies, a post-hoc correlation analysis was conducted between SSI and the within subject correlation coefficient (r-value) of stuttering expectancy and the immediate task reaction time. This analysis yielded a significant result \[ r(8)=.814, p=.014 \]. This relationship is plotted in figure 11.

2.2.3.3 Stuttering severity and absolute reaction time

A MANOVA was conducted to determine whether there were differences in absolute reaction times across the PWS. The dependent variables were the pooled reaction times from the immediate and delayed tasks and the fixed factor was subject. There was a significant main effect for subject for both the immediate \[ F(7,369)=139.393, p<.001 \] and delayed reaction times \[ F(7,369)=145.437, p<.001 \]. Typical post hoc analyses were not conducted because the specific subject comparisons were not relevant. What was relevant was whether the differences in reaction times were related to stuttering severity. A correlation analysis was conducted on the average reaction time for each subject and SSI score. There was no significant correlation for immediate \[ r(8)=.598, p=.117 \] or delayed \[ r(8)=.454, p=.258 \] reaction time.

2.2.4 Between group comparisons

Of interest was whether there was a group difference in absolute reaction time between PWS and PWNS in the word naming tasks. In addition, it was hypothesized that the magnitude of group difference would interact with the degree of stuttering expectation, and that there would be a larger group difference for the high expectancy compared to low expectancy words. To determine high and low expectancy words, standard scores were created within each PWS based on the expectancy ratings. Words that had a
standard score above 0 were high expectancy words and words with scores less than 0 were low expectancy words. Since PWNS did not generate expectancy scores, in order to compare high and low expectations across groups each PWNS was matched to a PWS based on age and gender. The words for each PWNS were given a high/low classification based on the high/low classification assigned by their matched PWS to those words. This made the comparisons comparable such that both groups had the same words in the high/low category.

The group means and standard deviations for the three reaction times are represented in figure 12. In the immediate task, the mean for PWS was 439 msec (standard deviation of 68 msec) and the mean for PWNS was 419 msec (standard deviation of 63 msec). In the delayed task, the mean for PWS was 282 msec (standard deviation of 102 msec) and the mean for PWNS was 267 msec (standard deviation of 92 msec). For the difference reaction time, mean for PWS was 157 msec (standard deviation of 68 msec) and the mean for PWNS was 153 msec (standard deviation of 73 msec).

A univariant general linear model (GLM) analysis was performed on the pooled reaction times to determine main effect for group and high/low distinction, as well as the interaction between group and high/low classification. The dependent variable was reaction time and the two fixed factors were subject group (PWS and PWNS) and high/low category. A separate analysis was conducted for the immediate, delayed and difference (immediate-delayed) reaction times.

The results of the GLM analysis for immediate reaction time showed a significant main effect for group \([F(1,782)=38.275, p<.001, \text{partial eta square} = .047]\), but no significant main effect for high/low classification \([F(1,782)=2.334, p=.127, \text{partial eta square} = .003]\). The was no significant interaction between group and high/low classification \([F(1,782)=3.323, p=.069, \text{partial eta square} = .004]\).

The GLM analysis for the delayed reaction time showed a significant main effect for group \([F(1,780)=158.852, p<.001, \text{partial eta square} = .169]\), but there was no
significant main effect for high/low classification [$F(1,780)=3.705, p=.055$, partial eta square = .005]. The interaction between group and high/low classification was not significant [$F(1,780)=.004, p=.948$, partial eta square < .001].

The results of the GLM analysis for the difference (immediate-delay) reaction time showed a significant main effect for group [$F(1,773)=123.689, p<.001$, partial eta square = .138], there was no significant main effect for high/low classification [$F(1,773)=.690, p=.406$, partial eta square = .001]. The interaction between group and high/low classification was not significant [$F(1,773)=2.401, p=.122$, partial eta square = .003].

Figure 3. Image of the screen where the Anticipation of Stuttering Questionnaire is filled out.
Figure 4. Image of the first screen where the expectation of stuttering ratings were entered for each word in the stimulus set.
Figure 5. Flowchart of the each trial in the immediate word naming task.
Figure 6. Flowchart of the each trial in the delayed word naming task.

Click mouse to begin next trial

Beginning of trial

Blank screen for 1,500 ms

Stimulus word is present on screen for either 1,200, 1,360, 1,520 or 1,680 ms. The participant is instructed to silently prepare to say the word

divorced

A black square is presented on the screen concurrently with an audible tone. These cue the participant to say the word as quickly as possible.

Blank screen for 1,500 ms. This is the inter-trial interval. After this the next trial begins.
Figure 7. Scatterplot of the relationship between stuttering expectancy scores for words at visit 1 and visit 2. Each data point is single word from one PWS. There were a total 400 data points in this plot, from 8 PWS across 50 words.
Figure 8. The scatterplot represents the relationship between stuttering expectancy scores and the number of times that word was stuttered across the 10 readings of the passage. These data are from all eight PWS.
Figure 9. Scatterplot of the adjusted stuttering expectancy scores for words and the adjusted reaction time from the immediate task. Each data point is a single word from one PWS.
Figure 10. Scatterplot of the relationship between SSI and the response to question 5 of the ASQ. The question regarded their belief that the anticipation of stuttering makes stuttering more likely to occur.
Figure 11. This is a scatterplot of the relationship between SSI and the r-value correlation coefficient from correlations that were conducted within each PWS regarding the relationship between their expectation of stuttering on words and the reaction time for those words in the immediate task. Each data point in this plot is from a single PWS.
Figure 12. Summary of group average reaction times from the immediate and delayed task, along with the difference between the immediate and delayed reaction time.
Table 2. Demographic information about the PWS and their matched PWNS.

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<th>PWS Subject ID</th>
<th>PWS Age</th>
<th>PWS Gender</th>
<th>SSI Score</th>
<th>SSI Severity</th>
<th>OASES Score</th>
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Table 3. List of questions from the anticipation of stuttering questionnaire.

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<td>I sometimes anticipate that I will stutter on words before I say them</td>
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<tr>
<td>2</td>
<td>I consistently anticipate stuttering on particular words</td>
</tr>
<tr>
<td>3</td>
<td>There are certain sounds that I anticipate stuttering on more than other sounds</td>
</tr>
<tr>
<td>4</td>
<td>I anticipate stuttering more when speaking to certain people or in certain situations</td>
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<tr>
<td>5</td>
<td>I believe that anticipating that I am going to stutter increases the likelihood that I will stutter</td>
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Table 4. List of the 50 stimulus word set with lexical properties from English Lexicon Project (ELP) database (Balota, et. al., 2007).

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Table 5. Summary of lexical properties of the 50 word stimulus set.

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Table 6. Data regarding the PWS stuttering severity, their responses to the 5 ASQ and summary data of their expectancy ratings of the 50 word stimulus set.

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<th>ASQ Q-3</th>
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<th>ASQ Q-5</th>
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<th>Minimum Word Expectancy</th>
<th>Maximum Word Expectancy</th>
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<td>100</td>
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</table>

Note: Standard deviations for the average word expectancies are in parentheses.
CHAPTER 3: STUDY TWO

3.1 Methods

3.1.2 Overview of the neural network model

The error likelihood hypothesis and the neural network model (Brown and Braver, 2005) were briefly discussed in the literature review, but a more detailed overview of the model is provided here. The goal of the original model was to test whether the ACC responds to learned error likelihood associations to contextual cues. This was accomplished within a go/change paradigm in which a subject saw an arrow on the screen and he was told to push the button that corresponded to the direction that the arrow was pointing. There were two types of trials, go trials and change trials. In go trials only the initial arrow was presented and the subject had little difficulty pushing the correct button. In change trials, a second arrow was presented above the first arrow following a short delay, and the subjects had to respond with the button that corresponded to the direction of the second arrow that was presented. The change trials were more difficult because the subject had to adjust their ongoing motor plan in order to respond appropriately to the change signal. Manipulations to the delay at which the change signal was presented, varied the chance of an error (responding to the initial arrow direction) because the longer the delay the more likely that the motor plan for initial arrow response would be activated and the less likely that the activation of the change signal would override the ongoing plan. What was critical about this particular Go/Change paradigm was every trial was preceded with one of two color cues: either white or blue. One of the colors always preceded the High Error Change trials and the other color always preceded the Low Error Change trials. The High Error Go and Low Error Go trials were exactly the same because both had no change signal, the only difference was that the High Error Go trial was preceded by the High Error color cue and the Low Error Go trial was preceded by the Low Error Cue. The purpose of color cues was to associate one of the color cues with
more errors so that in the Go trials the ACC activity could be compared across the High Error and Low Error Go trials. Both the model simulations and the fMRI data confirmed that there was increased ACC activity only in the Go trials that were preceded by the High Error color cue. This confirmed that one of the factors that activate the ACC was the error likelihood.

3.1.3 Model architecture

The original computational neural model was developed by Brown and Braver (2005) using the RNS++ simulation framework that employs a set of rate-coded model neurons whose activities and synaptic efficacies form a nonlinear dynamical system. The model consists of several connected modules. Figure 13 provides a schematic of the model’s architecture. A broad overview of the model and simulations are provided in the next two sections. For greater technical details see the supplementary material associated with Brown & Braver (2005).

3.1.3.1 Input layer

In the model there were four input layer units. One High Error Go unit, one High Error Change unit, one Low Error Go unit and one Low Error change unit. The High Error Go and Low Error Go units had excitatory connections with the Go response unit. The High Error Change and Low Error Change units had excitatory connections with the Change response unit. All four input units had excitatory connections with all of the 35 ACC units. The weights between the input layer and the response layer were fixed. However, the weights between the input layer and ACC layer were not fixed. The weight matrix connecting these two layers was where the error likelihood representations resided. Details regarding how and when these weights were modified is provided below.
3.1.3.2 ACC layer

The ACC layer consisted of a 2 dimensional 5x7 unit grid, providing 35 total ACC units. Excitation of the nearest neighbor helped to preserve topographic ordering during the learning process, much like in a self-organizing map. Inhibitory interneurons provided pooled lateral inhibition among ACC units. The ACC output was thresholded for each ACC cell, this implemented a kind of threshold sum detector. The numerical result of the threshold operation (subsequently summed across all ACC cells) was used to excite the control signal unit. By thresholding the ACC units, the control signal unit received very little activation unless there was sufficient activity across the ACC layer.

3.1.3.3 Control signal unit

The control signal was a single unit that when activated by the thresholded ACC output, provided a persistent control signal that globally inhibited the response layer units, thus slowing the initiation of motor plan.

3.1.3.4 Output layer

There were two output (response) units, a Go unit and a Change unit. These units were excited by their respective input units and were globally inhibited by the control signal unit. A “response” was made when one of the two units (Go or Change) exceeded a fixed threshold of activation level of .5.

3.1.3.5 Error signal unit

There was a single error signal unit that was activated if the “response” from the output layer was incorrect, an error. For example, if in a change trial the Go unit was activated to the .5 threshold before the activation of the Change response unit reached the threshold. When an error occurred, the error signal unit was activated with a level of 1.0. If the response for a trial was correct, the error signal activation was zero. The error
signal unit determined whether or not the weights between the input units and the ACC units were changed. In trials with errors, the weights between the input units that were activated and the ACC units that were activated were strengthened. This established the representations of the error likelihood based on the input cues. In trials in which there was no error, the weights between the active inputs and the active ACC units were not strengthened.

3.1.4 Trial details

There were four different trial types that were used within each simulation. Each trial type differed in terms of which input units were activated and the timing of the activation. Below, the details for each trial type are provided, along with a short narrative that describes the activation flow across the layers of the model during each trial type. It is important to note the model did not have explicit units for the color cues. The High Error Go and Low Error Go units served as the color cues since the high Error Go unit was active in all high Error trials and the Low Error Go unit was active in all Low Error trials.

3.1.4.1 High error – change trial

The trial began with the High Error Go input unit being activated at a level of .94 and remained active for the duration of the trial. The activation of the High Error Go input unit sent excitatory input to the Go response output unit. At each time step the activation level of the Go response unit moved closer to the .5 activation threshold at which time it would win out and be the selected response for that trial. However, the High Error Change input unit was also activated at a level of .94 after a delay (in the original model the delay was 328 msec) and began to send excitatory input to the Change response output unit. In order to give the Change unit an opportunity to beat the Go response to the .5 threshold, the weights between the Change input unit and the Change response unit were significantly stronger than the weights between the Go input unit and
the Go response unit, thus when the Change input unit activated the Change response unit it more strongly activated the Change response unit than the Go input unit activated the Go response unit. Essentially what happened was a foot race between the Go and Change response units to see which unit would reach the .5 threshold first. By varying the delay time by which the Change input was activated, the likelihood that the Change response would overtake the Go response was manipulated. If the delay was short, there was less of a chance of an error. If the delay was long, there was a greater chance of an error.

At the same time that the active High Error input units were exciting the output units, they were also exciting the ACC units based their respective connection weights. The stronger the connection weights between the High Error Input units and the ACC, the greater the activation of the ACC. If the activation level of any of the ACC units exceeded a threshold, excitatory input was sent to the Control Signal unit which then exerted global inhibitory inputs to both of the response units.

If the Go response unit beat the Change response unit to the .5 threshold, the trial was an error and the Error Signal unit became activated at a level of 1.0. If the error signal became activated, the weights between the High Error input units and the ACC units were strengthened in proportion to the amount of activity of each of the ACC units. If the trial was not an error, meaning that the Change response unit won, the Error Signal unit had an activation of zero and no weights were changed between the High Error input units and the ACC units.

In the original model, the Change Signal Delay in this trial was 328 msec which yielded an error rate of approximately 50%. This means that 50% of the High Error Change trials strengthened the connection weights between the High Error input units and the ACC units. The salient dependent variable of the Change trials was the error rate.
3.1.4.2 Low error – change trial

This trial was exactly the same as the High Error change trials except instead of the High Error input units being activated it was the Low Error input units. There was also a difference in the Change Signal Delay. The delay in these trials was significantly shorter than the High Error Change trials, this increased the likelihood that the Change response unit would overtake the Go response unit and become activated above threshold first.

In the original model, the Change Signal Delay in this trial was 124 msec which yielded an error rate of approximately 4%. This means that only 4% of the Low Error Change trials strengthened the connection weights between the Low Error input units and the ACC units.

3.1.4.3 High error – go trial

The trial began with the High Error Go input unit being activated at a level of .94 and it remained active for the duration of the trial. The activation of the High Error Go input unit sent excitatory input to the Go response output unit. At each time step the activation level of the Go response unit moved closer to the .5 activation threshold at which time it would win out to be the selected response for that trial. During the Go trials the Change input unit was never activated, which means that the Change response unit was not activated. Since the Go response unit was the only response unit being activated it always crossed threshold first, yielding no possibility of errors in this trial. Hence, no weights were changed between the input units and the ACC units.

At the same time that the active High Error input units were exciting the output units, they were also exciting the ACC units based their respective connection weights. The stronger the connection weights were between the High Error Go input unit and the ACC, the greater the activation of the ACC. If the activation level of any of the ACC
units exceeded a threshold, excitatory input was sent to the Control Signal unit which then exerted global inhibitory inputs to both of the response units. The inhibitory input served to slow the response selection (reaction time). The salient dependent variable of the Go trials was the reaction time.

3.1.4.4 Low error – go trial

This trial was exactly the same as the High Error Go trial except instead of the High Error Go input unit being activated it was the Low Error Go input unit. Another difference between the High Error Go and Low Error Go trials was amount of inhibition that was exerted from the Control Signal unit and this was dependent on the difference in the error rates between the High Error Change and Low Error Change trials. If the error rate of the High Error Change trials was higher than the error rate of the Low Error change trials there was greater inhibition from the Control Signal unit because the weights between the High Error Go unit and the ACC units were stronger than the weights from the Low Error Go unit and the ACC units.

3.1.5 Simulations

The goal was to collect mean reaction time data from the High Error Go trials within simulations that varied in their error rates on the High Error Change trials. 50 simulations were run in which the Change Signal Delay in the High Error Change trials was systematically varied from 100 – 700 msec. in 12 msec increments. The delay time of the change signal for the Low Error cue was held constant at 124 msec. Each simulation ran for 300 epochs, and each epoch was four trials, for a total of 1200 trials in each simulation.

The dependent variable was the reaction time from the High Error Go trials. The independent variable was the error rate from the High Error Change trials. The hypothesis was that simulations with greater error likelihood (error rate) in the high Error change trials would be positively correlated with reaction times in the High Error Go
trials. This hypothesis was based on the assumption that the greater association there was between the High Error cue and actual errors, a greater response would be elicited in the ACC upon the presentation of the high Error Cue. The increased activation of the ACC would elicit inhibitory activation of the motor response via the Control Signal, and the inhibitory activation would then lead to slower reaction times.

3.1.6 Statistical analysis

For each simulation the error rate (percent of errors committed) across the High Error Stop trials and the average reaction time from the High Error Go trials was calculated. Also, an average activation level from the 35 ACC units was recorded from the High Error Go trials. For each of the fifty simulations there was a single error rate, a single average reaction time and a single average ACC activation level. The hypotheses were that across the simulations the average reaction time and ACC activity would increase as error rates increased. To test these hypotheses a correlation was conducted with error rate and reaction time and ACC activity.

3.2 Results

3.2.1 Correlation of error rate, RT and ACC activity

The simulations yielded a range of errors rates from 4.9% to 96.9% and average reaction times from 530 to 569 msec. Table 7 provides the error rate, reaction time and ACC activation level for each simulation. The hypothesis was that across the simulations the average reaction time would be correlated with error rate. A correlation analysis showed a significant relationship between these values \[ r(50) = .334, p = .018 \]. This confirms that the model is able to simulate behavioral differences in reaction time and their relationship to error likelihood. This relationship is plotted in figure 14. Although the primary hypothesis of this simulation was regarding the behavioral data, the model is also capable of simulating neural activation of the ACC. In order to test the relationship
between activation of the 35 ACC units in the model, the average activation of the ACC units were averaged across the High Error Go trials in a similar manner as the reaction times, such that there was a single average ACC activation value for each of the fifty simulations. Across simulations there was a range of average ACC activation values from .0434 to .0973 (these are arbitrary units). A correlation analysis showed a significant relationship between error rate and ACC activation \([r(50)=.992, p<.001]\). This relationship is plotted in figure 15. Besides both being significant, the correlation between error rate and ACC activity was considerably higher than the correlation between error rate and the behavioral results. Across the simulations the ACC activity had a two fold increase while the reaction time showed modest increases as a function of error rate. This implies that the activity of the ACC was quite sensitive to error likelihood while the control processes that were triggered by the increases ACC activity had a subtle, but significant, effect on behavior.

3.2.2 Qualitative comparison of simulations and experimental results

There was no qualitative analysis comparing the results from simulations with the behavioral results from the current study. Such an analysis was hampered by lack equivalence between the error rate from the simulation and the subjective stuttering expectancy scores provided in the current study. However, if expectancy scores from the current study and the error rate from the model are viewed as qualitatively comparable, the scatter plots from figure 9 and figure 14 show close similarities in both the slope and relative change in reaction time.
Figure 13. Diagram of the architecture of the ACC Error-Likelihood Model from Brown & Braver (2005).
Figure 14. Scatterplot of the adjusted reaction time and the error rate from the simulations. Each data point is from one of the 50 simulations.
Figure 15. Scatterplot of the activity from the ACC units and the error rate from the simulations. Each data point is from one of the 50 simulations.
Table 7. Summary of results from each of the 50 neural network simulations.

<table>
<thead>
<tr>
<th>Simulation</th>
<th>Error Rate (percent)</th>
<th>Average Reaction Time from High Error Go Trials (msec)</th>
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CHAPTER 4: DISCUSSION

There were four key findings: 1) Across the group of PWS, there was a positive correlation between stuttering expectancy and verbal response time in the immediate naming task. 2) The degree to which stuttering expectancy was correlated with response time was found to be positively correlated with stuttering severity. 3) There was a correlation between stuttering severity and the belief that expecting to stutter increases the likelihood stuttering will occur. 4) The results from the simulations were qualitatively comparable to the results of the behavioral task.

4.1 Study One

4.1.1 ASQ and stuttering consistency

This study found that, as a group, PWS anticipated that they were going to stutter prior to speaking across several situations and contexts. These results are in agreement with experimental studies and data from clinical assessments that measure the degree of anticipation across contexts (Brutten, 1973; Johnson, Darley, & Spriesterbach, 1963). One of the more well developed clinical assessments of stuttering anticipation is the Speech Situation Checklist (SSC) (Brutten, 1973), which uses a Likert scale to evaluate 51 speech situations the degree to which they elicit negative emotion and/or increased stuttering. Studies using the SSC have shown similar results as those found in this study: both the results of this study and the SSC found that there were greater levels of stuttering anticipation as a function of stuttering severity (Bakker, 1995).

One of the more interesting finding of the current study was the strong correlation between stuttering severity and the response to ASQ question 4 regarding the belief that the anticipation of stuttering increases the likelihood that stuttering will occur. The essence of ASQ question 4 is the same as the primary research question of this thesis: Does the anticipation of stuttering increase the likelihood that stuttering will occur?
From the subjective perspective of the PWS in this study it appears that the answer depends on the stuttering severity. The more severe PWS strongly agreed with the statement while the less severe PWS agreed to a much lesser degree.

The data from the number of disfluencies across the reading passages matched well with the responses to ASQ question 4. The three most severe PWS who had some the highest responses to ASQ question 4, are the only PWS to stutter during the oral readings. Across those three PWS, there was a significant correlation between the stuttering expectancy score and the number of times the word was stuttered. These results are in agreement with the results from Martin and Haroldson (1967) which provided the motivation for incorporating the oral reading task in this study. They found that the words that had the highest expectancy ratings were stuttered at a much higher proportion compared to the words with low expectancy ratings. It is important to note that Martin and Haroldson (1967) specifically stated that the participants in their study were only severe stutterers. This is in agreement with the findings from this study that more severe PWS are more accurate at predicting future stuttering. It is also important to note that the result from this study, like the results of Martin and Haroldson (1967), showed that many of the words with high expectancy ratings were never stuttered and many of the words with low expectancy ratings were stuttered multiple times. This highlights that fact that there is not a one-to-one relationship between the expectations of stuttering and actual stuttering. However, in severe PWS there is a significant correlation.

These results show that PWS do, at least in some situations, anticipate stuttering. More severe PWS anticipate stuttering to a higher degree than less severe PWS and the more severe PWS more strongly believe that the anticipation increases that likelihood that stuttering will occur. What these results do not tell us is whether the anticipation of stuttering actually does increase its frequency or whether the more severe PWS anticipate stuttering more because they actually do stutter more. The question regarding the nature
of the relationship between anticipation and stuttering has been discussed for decades and several hypotheses (Brutten and Shoemaker, 1967; Johnson, 1959, Bloodstein, 1960) have been proposed but none have been successfully confirmed or refuted.

4.1.2 Influence of stuttering expectancy on reaction time

The major finding of this thesis is that there was a significant correlation between the stuttering expectancy scores and the relative change in verbal response time (reaction time) in the immediate naming task across the group of PWS. Another major finding was that the degree to which expectancy of stuttering was correlated with reaction time varied as a function of stuttering severity. More severe PWS had reaction times that were more strongly correlated with their expectancy scores compared to the less severe PWS.

This study is the first to utilize a within-subject design to investigate differences in fluent speech production based on the expectancy of stuttering. It is critical to point out that only fluent speech production was evaluated in these tasks. By observing differences in fluent speech production, this the first study to show that the expectation of stuttering is capable of influencing the speech production process. If fluency is viewed on a continuum from highly stable fluent speech on one extreme to highly variable and unstable disfluent speech on the other, these results provide evidence that by influencing the timely initiation of speech, the expectation of stuttering is capable of creating increased variability that may push speech toward the more unstable and disfluent edge of the continuum.

This study sheds light on the old question regarding the nature of the relationship between expectations of stuttering and actual stuttering. These results provide evidence that the expectation of stuttering does influence speech production and may play a causal role in the frequency and predictability of stuttering. If indeed, the anticipation of stuttering was driven purely by association with past stuttering but did not play a causal role in the behavior, there should be no correlation between stuttering expectancy and
dependent variables associated with fluent speech production. The results showing the speech production of more severe PWS is affected more by stuttering expectancy provides even further evidence that the expectation of stuttering may be a causal contributor to breakdown in fluency.

The significant correlations between stuttering expectancy scores and reaction time were seen in the immediate task but not in the delayed naming task. The purpose of incorporating both the immediate naming task and the delayed naming task was to gain a better understanding of what processes during speech production are being affected by stuttering expectancy. Immediate word naming tasks are assumed to involve the activation of orthographic representations, the translation of these into a phonological code, and finally the conversion of the activated phonological representations into articulatory motor commands for speech production (Balota & Chumbley, 1985; Jescheniak & Levelt, 1994; Seidenberg, Petersen, MacDonald, & Plaut, 1996). Response times in the delayed naming task are considered to reflect the time required for processes subsequent to the phonological computation, namely initiation of articulation. A comparison of response time in the immediate and delayed naming tasks can thus control, to a large extent, for the influence of potentially confounding variables like the numbers of phonemes and differences between initial phonemes (Fushimi, Ijuin, Patterson and Tatsumi, 1999).

Based on the psycholinguistic processes that each of the tasks are supposed to reflect, the results of this study are interpreted to mean that the expectation of stuttering does not affect the initiation of the speech motor plan given that the articulatory motor plans are already formulated and ready for execution. This interpretation is based on lack of correlation between expectancy scores and the response time in the delayed task. Using the standard scores, there was a significant also a correlation between the expectancy scores and the RT difference value which is the time difference between the immediate and delayed task. Given that the RT difference value is supposed to reflect a
more precise value of the planning phase because it reduces confounds relating to the
differences in articulation, it can be concluded that expectancy of stuttering most likely
affected one of the following processes: the activation of orthographic representations,
the translation of these in to a phonological code, and finally the conversion of the
activated phonological representations into articulatory motor commands. Future studies
should use different experimental techniques to further hone in on what specific
processes are most affected by stuttering expectancy.

4.1.3 Between group differences

The purpose of including PWNS in the study was twofold. First, it was
hypothesized that there would be an interaction effect in which the reaction times of PWS
would differ from the PWNS in a manner that interacted with the expectancy ratings of
the words from the PWS. The second reason to include the PWNS was to validate the
stimuli that were used in the study to ensure that they were equivalent in reaction times.

The hypothesis that there would be an interaction between the reaction times
differences between groups and the expectancy ratings was not confirmed. The
differences in reaction times across the groups were consistent across words. There was
however a significant main effect for group showing that PWS had significantly slower
reaction times in the immediate and delayed task. The PWS also had significantly lower
difference values when subtracting the delayed reaction times from the immediate
reaction times. This is interpreted to mean that the PWS have slower response times in
general, and compared to the PWNS in the delayed task, the PWS were less able to take
advantage of the delay between word presentation and the onset of the cue to prepare the
articulatory motor plans for execution. These results add to long list of studies that have
shown evidence that PWS have slower reaction times across several modalities (for a
review see Bloodstein, 1995).
The second reason to include the PWNS group was to validate the stimulus word set that was used throughout this study. The goal was to have a set of fifty words that were controlled in terms of their lexical properties so that their reaction times were as similar as possible. The analysis showed that there was no significant difference in the reaction times across words in the PWNS. It was critical that across normally fluent speakers the words had similar reaction times so that within subject variability in reaction time in the PWS could be attributed to stuttering expectancy and not lexical properties of the words.

4.2 Study Two

4.2.1 Neural network simulation

In this study a neural network model of performance monitoring in the ACC (Brown and Braver, 2005) was used to test whether the results from simulations of the effects of error likelihood on reaction time would match the behavioral data from this study. The simulations were conducted in a well established and tested model that is capable of replicating real-time behavioral data and neural activation data from fMRI. This study consisted of a series of fifty simulations in which manipulations were made so that the error rates across the simulations varied along a continuum from 5% to 97%. Analyses showed that both the reaction time and ACC activity were significantly correlated with the error rate. In order to qualitatively compare the results of the simulations with that of the behavioral data, it was necessary to make the assumption that the stuttering expectancy ratings for the words were equivalent to the error rates from the simulations. Providing this assumption, a qualitative comparison showed a good fit between the results of the model simulations and the behavioral data, both in terms of correlation coefficient and the absolute change in reaction time across the error rates. The relatively close fit between the absolute changes in reaction times was surprising
given that the timing of the model was fit to behavioral data from a Go/Change button press task.

The qualitative fit of the data provides evidence that the effects of stuttering expectancy on verbal response time are the result of activation of the ACC and subsequent control processes that globally inhibit motor execution. Several fMRI studies have shown over activation of the ACC during speech production in PWS compared to PWNS (Braun, et al. 1997; DeNil & Bosshardt, 2001; DeNil, Kroll & Houle, 2001; Fox et al., 1996; 2000; Wu et al., 1995). However, the tasks were not specifically investigating differences within-subject across words of varying levels of stuttering expectancy. A study that investigated changes in neural activity before and after stuttering therapy found ACC activity was significantly reduced in PWS who decreased their stuttering severity due to successful therapy (DeNil & Kroll, 2001a; 2001b). These findings were interpreted by the authors as evidence of an increased level of automaticity during speech production, reduced anticipatory needs to scan words for potential stuttering, increased emphasis of online self-monitoring, and optimized sequencing and timing of articulatory, phonatory, and respiratory movements gained through fluency shaping therapy. The data from this study suggests that their interpretation that reduced anticipatory scanning of words for potential stuttering, may be accurate considering that our results show that PWS who are less severe report less anticipation of stuttering.

The results of the model showed that the behavioral effects of error likelihood are considerably more subtle compared to the changes in ACC activity. As stated in the results, across the simulations the ACC activity had a two fold increase while the reaction time showed modest increases as a function of error rate. This implies that the activity of the ACC is quite sensitive to error likelihood while the control processes that are
triggered by the increases ACC activity have a subtle, but significant, effect on behavior. Considering that this study found significant correlations between stuttering expectancy and reaction time, it provides strong support that if the ACC is involved the effects of stuttering expectancy, results from a similar study to the current one that utilizes fMRI should find robust correlations between ACC activity and stuttering expectancy.

4.2.2 Future neural network simulations

It has been hypothesized that the anticipation of stuttering is a developmental process (Bloodstein, 1960; Brutten and Shoemaker, 1967; Johnson, 1959). The neural network model that was used in this study can also be used to investigate potential interactions between factors to gain an understanding of how they contribute to the development of stuttering expectancy. Future simulations can be run in which manipulations of the model parameters can be made to simulate individual differences in temperament and/or environmental factors. For example, Brown and Braver (2007) revised their account of the effects that error likelihood has on activating the ACC in order to account for individual differences that have been found to be present in people’s ability to show an error likelihood response (Nieuwenhuis, Schweizer, Mars, Botvinick, Hajcak, 2007). They hypothesized that that ACC activity reflects the amount of risk of an error for a given context. Risk is defined as a function of both error likelihood and the severity of the consequences of an error. In order to instantiate this revised hypothesis in the computational model they manipulated the magnitude of the error signal strength which is a contributing factor in changing the weights between the ACC and the context input units. Large consequences, like losing a lot of money when an error occurs, equated to a greater magnitude of an error signal in the model. Conversely, losing a small amount of money equated to a smaller magnitude error signal. Brown and Braver (2007) conducted an experiment in which they used a slightly modified version of the
go/change paradigm described above. They modified the experiment by introducing monetary rewards after each trial where the amount of money depended on whether the trial was correct and what color the cue was that preceded the trial. In this modified experiment there were four color cues instead of two in the previous experiment. The colors were paired with the four possible combinations of high and low error likelihood and consequence magnitude (high or low). When this paradigm was instantiated in the computational model the output predicted that ACC activity would have a linear relationship with the product of the error likelihood and the expected consequence of an error. The results of the simulation were compared to fMRI data from real subjects and the results were found to be highly correlated.

What was striking about the study just described was that the activation of the ACC was strongly influenced by individual differences in risk aversion. The participants of the study filled out the DOSPERT survey (Weber, Blais, & Betz, 2002) which is a domain specific risk taking inventory. The DOSPERT scale reflecting gambling-likelihood was negatively correlated with error-likelihood effects. It was hypothesized by Brown and Braver that temperamental factors, like risk aversion can influence the magnitude of error evaluation. Since error evaluation (which is equated to error magnitude) is one of the factors that influences connection weight changes between contextual cues and the ACC, temperamental factors can indirectly impact the rate at which associations are made between errors and contextual cues given that these associations are strengthened as a function of error signal magnitude. This relates to stuttering in that temperamental factors may play a role in the error evaluation of stuttering. This notion fits well with speculation that children who stutter are more at risk of developing chronic persistent stuttering if they have particular temperamental profiles. One of those profiles is behaviorally inhibited (Oyler, 1995), which has been associated with a decreased willingness to take social risks (Kagan, Reznick & Snidman, 1987). If a child who stutters is not risk averse they may outgrow the disorder more easily because
they may have less of a negative reaction to the stuttering (smaller magnitude of error signal) and hence not develop the associations between stuttering and contextual cues that may lead to the precipitation of the disorder.

4.3 Interpretation of the results within the SAMI framework

The SAMI framework explains the interaction of factors that contribute to the moment of stuttering. The framework splits the contributing factors into two general categories: the factors that modulate the speech production system and the factors that modulate the monitoring system. There is an explicit assumption that the neural substrate is the ACC. When sufficiently activated, the ACC initiates global inhibitory processes on the premotor cortex that slows the initiation of motor plans. It also explicitly hypothesized that the neural substrates of the inhibitory processes involves what has been coined the “braking network” (Aron, et al., 2007), which consists of the ACC, preSMA, and STN.

The aim of this study was to test whether the anticipation of stuttering affects fluent speech production. The SAMI framework was utilized to aid in designing the study and it was used to make specific hypotheses. According to the formalization within SAMI, if the factors that modulate the speech production system are held constant, there should be an observable relationship between factors that activate the monitoring system and the timing parameters of motor plan initiation. Given this general hypothesis from SAMI, coupled with the neural network model that instantiates the error likelihood hypothesis (Brown and Braver, 2005), the specific hypothesis of this study was tested regarding the influence of stuttering expectancy on the initiation of fluent speech. The statistically significant results from the behavioral study, along with the qualitative fit between the behavioral data and the simulations, also suggests that the formalization of
the neural substrates proposed in SAMI, and the assumptions regarding them, are highly probable.

The SAMI framework was presented in this paper as a biologically based and quantitative alternative to the current multi-factorial models of stuttering that acknowledge the range of factors that modulate stuttering but lack the specificity to make hypotheses regarding the interaction of factors and their effects on speech production. The scope of SAMI is confined to formalizing the interaction between the monitoring system (non-linguistic factors) and the speech production system (linguistic factors). As such, it does not provide details regarding the processes within the speech production system that lead to activation of the correct and competing motor plans. There are several models of stuttering that delineate and formalize speech and language processes to a sufficient degree that specific hypotheses can be made regarding how these factors contribute to stuttering (Bohland, Bullock & Guenther, 2010; Howell, 2004; Postma & Kolk, 1993). For example, the EXPLAN model of stuttering (Howell, 2004) separates planning (PLAN) and execution (EX) as independent processes that reflect the linguistic and motor levels, respectively. Similar to SAMI, the EXPLAN model accounts for stuttering moments as a lack of synchrony between the planning of linguistic units and their timely execution. An integrated model that couples the basic tenets of SAMI with those of EXPLAN would likely provide a more robust account of the linguistic and non-linguistic factors that contribute to such a deficit in the temporal integration of speech planning and execution. Further, such an integrated model may provide a better fit for the hypotheses generated in this thesis, and the results from the behavioral task used in this study.

The neural network modeling and the SAMI framework provide a strong partnership for the investigation of stuttering at different levels of analysis. SAMI provides a more general conceptual view of the disorder that can assist in making predictions regarding potential interactions between factors. SAMI can then be used to
guide more in depth investigations using the neural network modeling and experimental methods. For example, SAMI proposes that the interaction between subtle deficits in the speech production system and factors that modulate the monitoring system are what ultimately contribute to the development and persistence of stuttering. In order to further investigate the biological plausibility of this interaction, future development of a neural network model is necessary that combines a model of the monitoring system (like the one used in this study) with current models of speech production like the GODIVA model (Bohland, Bullock & Guenther, 2010). This would allow for more specific simulations that test the interaction of deficits in the speech production system with individual differences in non-speech factors (i.e. temperament or environment). Such a model would also allow for more detailed hypotheses regarding the temporal coordination between speech motor acts (initiation of phonemes within and across words) as opposed to being restricted to the initiation of the first phoneme that was tested in this study.

4.4 Limitations of study

This study had several limitations that should be addressed in future studies. The first issue relates to the reliance on verbal response time (VRT) to measure the behavioral consequences of the anticipation of stuttering. The decision to use VRT was based on previous studies that investigated the effects of error-likelihood on reaction time (Brown & Braver, 2005). These studies led to the expectation that VRT would be correlated with increased error-likelihood (as measured by stuttering expectancy). As such, the statistically significant results from the current study could be interpreted to reflect a known effect of error-likelihood on reaction time, but not necessarily a novel test of a hypothesis based on the SAMI framework. The SAMI framework hypothesizes that non-linguistic factors (like stuttering expectancy) are capable of interfering with the timely execution of speech units necessary for fluent speech production. Although speech initiation time is one measure of motor execution, future studies should investigate the
correlation between non-linguistic factors and within-word temporal acoustic measures (e.g. voice onset time or frication duration) to determine if such factors influence the temporal coordination of speech production behaviors during syllable and word production. Results from such studies may provide evidence that not only do non-linguistic factors influence the initiation of speech, but also effect the continuous stream of motor planning and execution that is necessary for fluent speech. Based on SAMI, the global motor inhibition from increased activation of the monitoring system should be positively correlated with the temporal duration of acoustic measures within syllables and words.

A second limitation of this study was the use of an adaptation paradigm to measure the total number of stuttered words across readings. Adaptation occurs when a PWS shows a reduction in the frequency of speech disfluency across consecutive oral readings or recitations of the same material. The employment of an adaptation paradigm in study 1 may have contributed to the lack of stuttering during the oral reading from five of the eight PWS. Future studies should avoid having PWS read the same passage consecutively if the goal is to measure the frequency of stuttering across a closed word set. One solution would be to create five different passages that include the words in the stimulus set and have the PWS read the different passages consecutively. A third limitation of this study was the number of subjects. Future studies should include more subjects, particularly more PWS who are severe and very severe. An increased number of subjects would provide increased power for assessing the effect of stuttering expectancy on speech production, but perhaps more importantly it would provide more convincing evidence that these effects interact with stuttering severity.

4.5 Conclusions

This paper is significant for the following reasons. First, the results of this study add to the extant literature showing that PWS anticipate stuttering and this is the first
study to show a relationship between stuttering severity and the subjective belief that the anticipation of stuttering increases the likelihood of stuttering, inferring a causal relationship between expecting to stuttering and actually stuttering. Second, this is the first study to use a within-subject design to show that subjective levels of stuttering expectancy are correlated with behavioral differences in fluent speech production. This provides the empirical evidence that stuttering expectancy may be a causal contributor to the frequency of stuttering. Third, results from simulations that were run in a biologically plausible neural network model were qualitatively similar to the behavioral results of this study. This provides preliminary evidence that the effects of stuttering expectancy on speech production may be the result of increased activation of the ACC and subsequent imitation of inhibitory processes. The results of the model provide specific hypotheses regarding neural activation that can be empirically tested using fMRI. Lastly, this paper presented that SAMI framework which guided the development of the studies in this paper. The purpose of SAMI is to provide a framework in which hypotheses can be made regarding the interactions of factors that contribute to the variability of stuttering. The results of the studies in this paper support the formalization and the biological substrates that are described in the SAMI framework, providing initial validation that this is a useful framework for inspiring future studies.
APPENDIX

Charlie’s parents are divorced and he lives with his mother. They live in the north where the weather requires a jacket all year round. That’s why it was so exciting when his dad invited him to join him aboard a cruise ship over summer vacation. Charlie loved to travel and has always dreamed of fishing in the ocean. He was grateful to have the chance to finally spend time with his dad. However, Charlie became silent when his dad told him that he was also bringing Connie. When his dad and Connie got engaged, Charlie promised his mother that he would not support his father marrying Connie. Although he thinks his father and Connie make a good couple, he believes it’s terrible that he is forced into a position of taking sides. Knowing that his mom is not selfish and she would understand, Charlie did eventually decide to accept the trip that his father offered. When Charlie headed to his mother’s room to tell her about his decision to go on the cruise, he hoped she would forgive him. He planned the most charming and innocent defense that he could imagine. Once he started his speech he began to realize how foolish he was worrying about his mother’s response. She was more than understanding and received the news with open arms. She told Charlie that he was her clever little prince and she loved him to pieces. Charlie could not stop smiling because he had the greatest mom in the entire world. He immediately called the agency to ask them to send the tickets.


