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Title

Activation in Right Dorsolateral Prefrontal Cortex Underlies Stuttering Anticipation
(Abbreviated title: Stuttering and Anticipation)

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Conflict of Interest

The authors do not report competing interests.

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Abstract

People who stutter learn to anticipate many of their overt stuttering events. Despite the critical role of anticipation, particularly how responses to anticipation shape stuttering behaviors, the neural bases associated with anticipation are unknown. We used a novel approach to identify anticipated and unanticipated words in 22 adult stutterers, which were produced in a delayed-response task while hemodynamic activity was measured using functional near infrared spectroscopy (fNIRS). Twenty-two control participants were included such that each individualized set of anticipated and unanticipated words was produced by one stutterer and one control participant. We conducted an analysis on the right dorsolateral prefrontal cortex (R-DLPFC) based on converging lines of evidence from the stuttering and cognitive control literatures. We also assessed connectivity between the R-DLPFC and right supramarginal gyrus (R-SMG), two key nodes of the frontoparietal network (FPN), to assess the role of cognitive control, and particularly error-likelihood monitoring, in stuttering anticipation. All analyses focused on the five-second anticipation phase preceding the go signal to produce speech. The results indicate that anticipated words are associated with elevated activation in the R-DLPFC, and that compared to non-stutterers, stutterers exhibit greater activity in the R-DLPFC, irrespective of anticipation. Further, anticipated words are associated with reduced connectivity between the R-DLPFC and R-SMG. These findings highlight the potential roles of the R-DLPFC and the greater FPN as a neural substrate of stuttering anticipation. The results also support previous accounts of error-likelihood monitoring and action-stopping in stuttering anticipation. Overall, this work offers numerous directions for future research with clinical implications for targeted neuromodulation.

Keywords: stuttering; anticipation; disfluency; fNIRS; frontoparietal network; error-likelihood monitoring; action-stopping.

Introduction

Stuttering is a complex neurodevelopmental communication disorder that often negatively impacts social, emotional, and professional opportunities for more than 50 million adults worldwide. The disorder manifests itself to listeners as intermittent interruptions in speech production including part-syllable repetitions and audible and inaudible prolongations of sounds. However, these behaviors do not always accompany stuttering events because most, if not all, stutterers develop the remarkable ability to anticipate stuttering. As a result, they can alter their speech plan prior to execution by, for example, avoiding, stalling, or using a speaking strategy (e.g., pull-out, easy onset) (Jackson et al., 2015, 2019). This potential discrepancy between the internal experience of the speaker (i.e., sensing upcoming speech breakdown) and how stuttering manifests itself to the listener (i.e., as something out of the ordinary) has important clinical implications because how individuals respond to anticipation shapes their communicative experiences. Behavioral and qualitative investigations of anticipation, especially in recent years, have improved our understanding of the phenomenon, but it is critical to augment this evidence with a neural account of stuttering anticipation, especially given the covert nature of anticipation. The purpose of this study was to initiate a brain-based understanding of stuttering anticipation by linking neural activation to self-reported anticipation and subsequent stuttering behaviors.

The Anticipation of Stuttering

Stuttering anticipation refers to the sense or prescience that upcoming speech will be stuttered, should the speaker execute their speech plan as originally intended without alterations (Jackson et al., 2015; Wingate, 1975). Anticipation occurs on a temporal continuum from a longer-term or looming sense of impending stuttering to a shorter-term immediate sense of upcoming stuttering. For example, a speaker may anticipate a word months in advance of saying it (a student

knowing at the beginning of the semester that they have to say a certain word in a presentation at the end of the semester); minutes or seconds before (when they are about to introduce themselves); or immediately before executing speech. Anticipation is driven by error-likelihood monitoring whereby the speaker learns associations between "errors" (i.e., stuttered utterances) and listener reactions or other environmental consequences, thereby learning to predict the occurrences of these errors (Arenas, 2012, 2017; Garcia-Barrera & Davidow, 2015). While adult stutterers, as a group, predict stuttering with high accuracy in experimental settings (greater than 90% accuracy; Knott et al., 1937; Milisen, 1938; Van Riper, 1936), there is a range in which speakers report anticipating stuttering, from, "sometimes" to "always" (Jackson et al., 2015). Anticipation is a relatively stable feature such that *anticipated* or *feared* words are stuttered in experiments even three months after they are identified by participants (Mersov et al., 2018). Arguably most important in the speaker's experience is how they learn or choose to respond to anticipation, whether by avoiding, approaching, or implementing physical speaking strategies that prevent stuttering from coming to the surface (Jackson et al., 2015, 2019). In this way, responding to anticipation is mediated by cognitive control.

Cognitive Control

Cognitive control refers to the ability to orchestrate brain functions to complete a given task or reach a certain goal (Miller, 2000). Cognitive control encompasses planning, initiating and inhibiting actions or tasks, and being flexible and vigilant to tasks in response to environmental demands (Niendam et al., 2012). All of these processes are involved in responding to stuttering anticipation. For example, when a stutterer knows that they are going to stutter, they must initiate (or choose not to initiate) a response which may include avoidance or using a speaking strategy (Jackson et al., 2015, 2019); they may inhibit responses due to fear of negative reactions from the

listener; they must be flexible with the challenge at hand (i.e., not being able to say what they want to say when they want to say it); and they must remain vigilant to their goal (i.e., producing speech). While numerous studies have examined cognitive control in children and adults who stutter (for review, see Anderson & Ofoe, 2019), no studies have assessed the relationship between cognitive control and anticipation directly.

A Potential Neural Substrate of Stuttering Anticipation

While anticipation is pervasive in the stuttering experience and contributes significantly to the negative impact on quality of life for stutterers (Jackson et al., 2015; Tichenor & Yaruss, 2019), the neural underpinnings of anticipation and related cognitive control processes are unknown. Neurofunctional investigations of stuttering have instead focused on the speech motor network, revealing atypical activation in left perisylvian and motor areas along the arcuate and superior longitudinal fasciculus, and their homologous regions in the right hemisphere, as well as atypical activity in basal ganglia and cerebellum (Braun et al., 1997; S.-E. Chang et al., 2011; De Nil et al., 2000; Fox et al., 2000; Kell et al., 2009; Neef et al., 2016; Neumann et al., 2004; Preibisch et al., 2003; Toyomura et al., 2018). Some of these studies reported significant findings outside of the speech motor network in areas related to cognitive control, even though these areas were not the focus of those investigations. For example, it is widely known that the right dorsolateral prefrontal cortex (R-DLPFC) plays a critical role in cognitive control processes (Koechlin et al., 2003; MacDonald et al., 2000; Miller, 2000; Ridderinkhof et al., 2004). Stutterers exhibit elevated activation in the R-DLPFC (Kell et al., 2009; Lu et al., 2009; Neef et al., 2018), and treatment temporarily reduces activity in R-DLPFC while reducing stuttering symptoms (De Nil et al., 2004; Neumann et al., 2005). Kell et al. (2009) also found that stutterers who reported recovering from stuttering without treatment did not show elevated activation in R-DLPFC, suggesting that these

patterns reflect compensatory efforts not learned in therapy (e.g., avoiding, stalling, or using other self-learned speaking strategies). However, because anticipation and the R-DLPFC were not the focus of these studies, the potential relevance of the R-DLPFC to stuttering anticipation, and to stuttering more broadly, is unknown.

We focus primarily on the R-DLPFC in this study, but other regions and networks are likely recruited during anticipation and responding to anticipation. Modern imaging and computational approaches, including task-induced and task-free connectivity analysis, have identified multiple non-overlapping and distributed brain networks that underlie cognitive control, including the fronto-parietal network (FPN), salience network, cingulo-opercular network, and dorsal and ventral attention networks, as well as others (D'Esposito & Postle, 2002; Menon & D'Esposito, 2021; Niendam et al., 2012). The FPN, which includes the R-DLPFC and supramarginal gyrus (R-SMG) (Menon & D'Esposito, 2021), is particularly relevant because it co-activates with multiple other networks (e.g., salience network, cingulo-opercular network) to carry out the diverse processes associated with cognitive control (Marek & Dosenbach, 2018). The FPN initiates and flexibly modulates interactions between the salience and cinguo-opercular networks (Marek & Dosenbach, 2018), which both include the anterior cingulate cortex (ACC). ACC underlies error-likelihood monitoring (Brown & Braver, 2005): DLPFC co-activates with ACC, whereby ACC underlies the detection of errors in response to unintended outcomes and generates error signals, and DLPFC holds task-relevant information in working memory and initiates subsequent actions (Alexander & Brown, 2015; Holroyd & Yeung, 2012). It is reasonable to propose that the ACC underlies stuttering anticipation – the recognition of the breakdown or "glitch" in speech-language planning – and reasonable to predict that the R-DLPFC underlies

initiating a response to this breakdown[1]. Given the strong bidirectional connections within the FPN (Goldman-Rakic, 1988; Menon & D'Esposito, 2021; Mesulam, 1998), particularly between R-DLPFC and R-SMG, it is also reasonable to hypothesize that anticipation destabilizes these connections, resulting in altered connectivity.

The current study examined the relationship between R-DLPFC and stuttering anticipation, which may clarify the significance of previous and seemingly incidental findings of elevated activation in R-DLPFC in stutterers (e.g., Kell et al., 2009; Neef et al., 2018). In a first visit, we used a clinical interview to determine individual-specific anticipated and unanticipated words (Jackson et al., 2020). In a second visit, which occurred between three and 10 days after the first visit, we used functional near-infrared spectroscopy (fNIRS) to measure cortical activation immediately prior to participants producing the anticipated and unanticipated words in a delayed-response task. We focused on superficial cortical structures in part due to imaging depth restrictions associated with fNIRS. While we were not able to measure activation from deeper structures such as ACC, fNIRS offered several advantages compared to other techniques (e.g., fMRI, EEG, MEG), including 1) robustness to speech movement artifact and 2) allowing participants to produce speech while they sat upright and across from a communicative partner, which increased the likelihood of anticipation. fNIRS has also been validated as a tool to measure DLPFC activation associated with anticipation (Vassena et al., 2019). A matched control group was included to test whether stutterers recruit R-DLPFC differently than non-stutterers. Each

[1]While it was not possible in the current design to disentangle anticipation and responding to anticipation, the reader should be aware of these somewhat distinct but overlapping processes. We conceive of anticipation as an event – the point in time at which the speaker becomes aware that should they proceed as planned, they will overtly stutter – and responding to anticipation as the constellation of processes, involving the cognitive control system, that underlies how a speaker chooses to proceed in light of the knowledge that they are likely about to stutter. In the current design and given the hemodynamic lag associated with fNIRS, it was not possible to disentangle anticipation and responding to anticipation, and thus, the current study examined the processes involved in anticipation generally. Techniques with better temporal resolution than fNIRS (i.e., MEG/EEG) are more suited to disentangle these two process.

anticipated and unanticipated word list was produced by a stutterer and control speaker. We conducted a region of interest (ROI) analysis of R-DLPFC and hypothesized that 1) anticipation would be associated with greater activation in R-DLPFC, reflecting cognitive control processes associated with responding to anticipation, and 2) stutterers would exhibit greater activation than control speakers in R-DLPFC during this same time period. We also assessed functional connectivity to test whether anticipation was associated with reduced intrinsic connectivity within the FPN, specifically between R-DLPFC and R-SMG.

Materials and Methods

This study was approved by the Institutional Review Boards at New York University and Yale University. Consent was obtained for all participants in accordance with the Declaration of Helsinki.

Participants

Twenty-seven adult stutterers were recruited through the first author's clinical network, mass emails distributed by *Friends: The National Association of Young People Who Stutter* and the *National Stuttering Association*, and word of mouth. After the fNIRS screening (see below), 22 stutterers (9 female; mean age = 31.9, SD = 9.1; three left-handed) and 22 control speakers participated in the study (10 female; mean = 27.4, SD = 8.0; three left-handed). Control participants were recruited after the stuttering participants so that they could be matched for age, gender, and stimuli (see below). Male-to-female ratio was lower than what is typical observed in the stuttering literature (59% vs. ~75-80%). All participants were between the ages of 18 and 50, reported that American English was their primary language (multilingual acceptable as long as English was learned during early childhood [younger than 6 years of age]), and reported negative histories of neurological, speech-language, psychological, learning, and hearing impairment. All but two

stutterers were right-handed. Participant characteristics, including age, gender, treatment history, and extent score from the *Stuttering Anticipation Scale* are included in Table 1.

INSERT TABLE 1

First Visit (Stutterers Only)

The first visit comprised the 1) stuttering assessment, 2) clinical interview to determine participant-specific stimuli, and 3) screening for fNIRS. Only the stuttering group participated in two visits; the fNIRS screening for the control group took place on the same day as fNIRS testing.

Diagnostic Assessment

The stuttering group was identical to that in Jackson et al. (2020). That study validated the clinical interview used here, and the only overlapping data between that study and the current study are the speech classification data (stuttered/ambiguous/fluent, interrater reliability). Stuttering was diagnosed by the first author, an American Speech-Language-Hearing Association certified speech-language pathologist (SLP) with more than 10 years of expertise in stuttering intervention. All stuttering participants (1) self-reported as a person who stutters; and (2) exhibited three or more stuttering-like disfluencies (Yairi & Ambrose, 1992) with temporally aligned physical concomitants (e.g., eye blinking, head movements) during a 5-10 minute conversation. Participants also completed the Stuttering Anticipation Scale (SAS) (Jackson et al., 2018), which provided self-report ratings of the extent of anticipation based on a 0-100 (never-always) visual analog scale ("How much do you anticipate stuttering?").

Clinical Interview

The clinical interview was described previously in Jackson et al. (2020), and will be

described briefly here. Interviews were conducted at New York University and Yale University, and for two participants remotely. The purpose of the interview was to identify 10 words for which the participants anticipated that they would stutter (hence: anticipated words) and 10 words for which they did not anticipate that they would stutter (hence: unanticipated words), resulting in individual lists for each participant. This approach extends previous methods to identify anticipated/unanticipated words (Bowers et al., 2012; Mersov et al., 2018; Wymbs et al., 2013), by including clinical inference (e.g., asking participants whether stuttered words/sounds not immediately identified by participants as anticipated or "feared" words/sounds should be included as anticipated words), as well as using counseling techniques to create an environment in which participants were comfortable to identify/reveal feared words. The words were used to create the stimuli for fNIRS testing, consisting of short questions or sentence completions that would require the participant to produce the words. Anticipated and unanticipated words were matched for length (number of syllables). Each stutterer was matched with a control participant who produced the same set of words.

fNIRS Screening

The goal of the fNIRS screening was to determine whether a reliable hemodynamic signal could be acquired from each participant. This is because factors such as bone density and skull thickness weaken fNIRS signals (Krall & Dawson-Hughes, 1993; Okada & Delpy, 2003). Screening fNIRS participants limits acquisition of invalid data (Zhang et al., 2017). The screening consisted of a finger tapping task during which participants were required to tap their fingers in an alternating pattern for 15 s then rest for 15 s, for a total of 3 minutes (right hand). Typically, this task elicits a robust response in left motor/premotor areas. Here, a response was determined to be reliable if there was sufficient separation between HbO and HbR signals in any channel in left

motor/premotor areas based on visual inspection of the event-triggered average. Importantly, potential participants were excluded before the study began. That is, no participants were excluded from the study after their signals were determined to be reliable. Twenty-seven stutterers were screened, and 22 participated in the study; twenty-eight controls were screened, and 22 participated in the study.

Second Visit

The second visit (for the stuttering group) occurred between 3 and 10 days after the first visit, and included fNIRS testing in the Brain Function Laboratory at Yale. Participant-specific stimuli were created between the first and second visit based on the anticipated and unanticipated word lists established during the clinical interview (first visit). Each word list was used to create the stimuli for one stuttering and one control speaker. Stimuli included simple questions or sentence completions that required one-word responses (e.g., "You can fly in an _____" → airplane, "What month comes after June?" → July). All stimulus questions were approximately between 1-3 s. Participants were exposed to the stimuli before the experiment to minimize the potential impact of language retrieval and formulation processes (i.e., they became familiar with the questions and answers prior to the experiment).

Figure 1 depicts the task timeline. The question was presented auditorily while participants viewed a cross on the monitor. Verbal responses were delayed by 5 s. Participants responded when the screen turned green. Participants were asked to look straight ahead and to try to remain still. The paradigm included interactive and alone conditions, but the condition contrast was not the focus of the current study. The two conditions were pooled in all analyses. During the interactive

INSERT FIGURE 1

condition, participants responded to questions asked by the examiner who was seated directly across from the participant and in full view. Participants were instructed to look at the examiner while responding (e.g., "…make sure you're looking at me when you respond…"). The experimenter was given a cue on a separate monitor (unseen by the participant) just before the go signal was presented, allowing him to look at the participants when they responded. During the alone condition, participants responded to pre-recorded stimuli, the same questions as asked by the examiner as described above, while alone in the testing room. Questions during the interactive condition were matched, to the best of the examiner's ability, to the pre-recorded questions in terms of duration and prosody. The paradigm included a total of 80 trials: 4 interactive runs, 4 alone runs; each run included 10 words. Each word was produced 4 times.

Data acquisition

Behavioral

Speech data were acquired via acoustic and video recordings. Acoustic signals were recorded using a head-mounted microphone with a pop-screen filter set at the same fixed distance for each participant. Video was captured using a Logitech c920 HD 1080p video camera mounted on the participant's monitor.

Neural

Data collection methods have been described previously (e.g., Hirsch et al., 2018, 2021), and are also described here. Hemodynamic signals were acquired using an 80-fiber continuous-wave fNIRS system (Shimadzu LABNIRS, Kyoto, Japan) with a temporal resolution of 27 ms. Forty emitters and forty detectors were arranged in a 134-channel layout covering bilateral frontal, temporal, parietal, and occipital lobes. Depending on the size of the participant's head, caps with optode distances of either 2.75 cm or 3 cm were used. Three wavelengths of light (780, 805, and

830 nm) were delivered by each LABNIRS emitter. Absorption was converted to concentration changes for deoxyhemoglobin (HbR) and oxyhemoglobin (HbO) using the Beer-Lambert Law (Matcher et al., 1995). After the experiment, anatomical locations of optodes were determined based on standard head landmarks (inion, nasion, top center [Cz], and left and right tragi) using a Patriot 3D Digitizer (Polhemus, Colchester, VT) and linear transform techniques (Eggebrecht et al., 2012; Ferradal et al., 2014; Okamoto & Dan, 2005). MNI coordinates for the channels were obtained using NIRS-SPM (Ye et al., 2009) in MATLAB (Mathworks, Natick, MA), and corresponding anatomical locations were determined for each channel. See Supplementary Table 1 for group median coordinates, atlas-based probabilities, and anatomical regions for each channel; see Supplementary Figure 1 for a visual representation. Channels were clustered into anatomical regions based on shared anatomy. The average number of channels per region was 2.69 ± 1.40.

Data Processing

Behavioral

Errors, which comprised non-productions or incorrect productions due to participants forgetting answers or producing erroneous speech, were not included in any analyses (Table 2). Reaction time was calculated as the time between the go signal (i.e., the green screen) and speech onset as defined by the first articulatory movement or accessory behavior (Table 3). Articulatory onset was marked as the first articulatory movement based on visual inspection using Davinci Resolve (Black Magic Design, Australia), which allowed for frame-by-frame scanning (29.97 frames per second) of the recordings of participants' faces. Interrater reliability between the first author and a SLP blind to the study yielded a Cohen's weighted kappa of .89 ($p < .05$), indicating strong agreement (McHugh, 2012). We used articulatory onset because 1) inaudible sound prolongations (blocks) typically included observable movement such as posturing, and 2) it

appeared that for some participants, video and audio were not synchronized due to technical error. While determining neural correlates of stuttered speech was not the primary goal of this study, we include a comparison of stuttered and fluent speech for completeness. A three-point rating system was used to classify stuttering response type: "0" indicated unambiguous fluency; "1" indicated ambiguity (unclear whether stuttered or fluent); and "2" indicated unambiguous stuttering (Jackson et al., 2020).

Neural

Data processing was similar to that previously reported (Hirsch et al., 2017, 2018; Zhang et al., 2017). Baseline drift was removed using a NIRS-SPM detrending procedure. Global components were removed using a principal component analysis spatial filter (Zhang et al., 2016), which is comparable to using short-source channels (Noah et al., 2021). Channels were rejected if the root mean square (RMS) of the raw signal was 10 times greater than the group mean RMS, which resulted in a mean of 1.48 of 134 (1.1%) channels per participant rejected. Little to no motion artifact was observed in the data, based on visual inspection, likely because participants were instructed to remain still during the task. This is typical for compliant adult participants (Noah et al., 2021). Further, there was no speech movement observed during the anticipation phase. HbR and HbO signals were acquired. The fNIRS datasets for each subject were reshaped into 3-D volume images for the general linear model (GLM) analysis using SPM8 (Wellcome Trust, London, UK). Beta value coordinates were converted to standard MNI space using NIRS-SPM (Ye et al., 2009). Contrast images were rendered on a standardized MNI brain template using a p-value threshold of .05 and cluster size threshold of 50 voxels, for visual representation. Anatomical

locations of peak voxel activity were identified using the Brodmann area Talairach atlas (Lancaster et al., 2000).

Statistical Analysis

Behavioral

To examine reaction time and stuttering response type (stuttered/ambiguous/fluent), linear mixed effects models were fit using the lme4 (Bates et al., 2014) and ImerTest packages (Kuznetsova et al., 2017) in R (R Core Team, 2014). The MuMIn package (Barton, 2020) was used to calculate estimated R^2 for model fit. For reaction time, the model included word type (anticipated/unanticipated), word length (number of syllables), trial, and stuttering response type as fixed factors, and participant as a random factor to account for expected variation due to individual differences. Wilcoxon rank sum tests were also used to assess reaction time differences for stuttered, ambiguous, and fluent speech. For stuttering response type, the model included word type, word length, and trial as fixed factors, and participant as a random factor.

Neural

All fNIRS analyses followed standard voxel-wise GLM techniques (Friston et al., 1994, 1995) adapted for fNIRS (e.g., Descorbeth et al., 2020; Hirsch et al., 2021). Analyses targeted the anticipation phase, i.e., the five-second time window between the end of the question and the go cue (see Figure 1). All included voxels were within 2 cm from the cortical surface. The primary ROI analyses focused on the R-DLPFC; secondary ROI analyses examined activation in the R-IFG and R-preSMA. Connectivity between the R-DLPFC and R-SMG was also assessed.

ROI

The mask for R-DLPFC was created by generating a 10-millimeter sphere using the MarsBar toolbox (Brett et al., 2002) and xyz coordinates [50 26 38] from Kell et al. (2009). Five

ROI analyses of R-DLPFC were conducted. (1) Anticipated vs. Unanticipated: Activation associated with anticipated vs. unanticipated words was compared by using SPM8 to convolve a 5-second block regressor during the anticipation phase, with a standard hemodynamic response function (HRF) that was fitted to the data. The first-level analysis yielded two beta values for each participant in each run (i.e., 10 trials per run), for anticipated and unanticipated words (five anticipated, five unanticipated). The results of the second-level contrast of anticipated vs. unanticipated words were projected onto a standardized MNI template image using SPM ($p<.05$ and cluster size of >50 voxels, uncorrected). Note that the "whole-brain" image was only used to test the ROIs, though whole-brain results for this analysis, and all analyses below, are included as supplementary material. Significance for the ROI analysis was tested using a one-tailed t-test in SPM ($p<.05$) by determining overlap between the second-level image (anticipated vs. unanticipated) and the mask. Two additional analyses were conducted to test whether the R-DLPFC ROI was significantly activated during the anticipation phase for anticipated and unanticipated words (separately). The second-level results, which compared activation for both anticipated and unanticipated words to rest, were projected onto the same MNI template, and the ROI analyses were carried out as described above. (2) Stuttered vs. Fluent: To compare activation related to response type within stutterers (stuttered, ambiguous, fluent), the regressor was modulated in height (0 = fluent, 1 = ambiguous, 2 = stuttered) in the first level analysis. The assumption was that stuttered speech yielded greater (or lesser) activity than ambiguous responses, which yielded greater (or lesser) activity than fluent responses, effectively providing a "contrast" of stuttered and fluent speech. Beta values were compared to zero using a one-tailed t-test in SPM. The ROI analysis was conducted as described above. (3) Stutterers vs. Controls: The ROI group level comparison was similar to the anticipated vs. unanticipated contrast described above, except

for the second-level contrast. The 5-second block regressor was convolved with the HRF, irrespective of whether the word was anticipated. Results of the second-level contrast for stutterers vs. controls were projected onto a standardized MNI template image, and the ROI analysis was carried out as described above. (4) Task vs. Rest (controls only): We also conducted an analysis of the controls only, during the same 5-second window. The purpose of this analysis was to determine whether the R-DLPFC ROI was activated prior to speech execution, which could indicate whether R-DLPFC is involved in speech motor planning in unimpaired speakers. The second-level results, which compared activation of the controls to rest, were projected onto the same MNI template, and the ROI analysis was carried out as described above. (5) Interactive vs. Alone: Finally, we compared activation between the interactive and alone conditions to justify our decision to exclude "condition" as a factor from all of the models. Confirmation of the null hypothesis would suggest that condition is not contributing to the anticipated and unanticipated responses.

We completed two additional ROI analyses. First, we used two control ROIs to confirm that our findings in R-DLPFC were specific to stuttering anticipation, and not, for example, general to stuttering or due to systemic artifact. These included L-DLPFC, the homologue of the R-DLPFC ROI, and R-precentral gyrus (preCG) from Belyk et al. (2017), the most recent activation-likelihood meta-analysis of state stuttering (i.e., stuttered versus fluent). ROIs were 10 mm spheres with centroids [-50 26 38] for L-DLPFC and [54 -14 34] for R-preCG. Second, we tested two key superficial cortical nodes of the action-stopping network (i.e., right inferior frontal gyrus [R-IFG] and right pre-supplementary motor [R-preSMA]), as it has been proposed that action-stopping is associated with stuttering anticipation (Arenas, 2017; Hannah & Aron, 2021; Neef et al., 2018). Centroid coordinates were obtained from recent meta-analyses of stuttering: [46 23 -5] for R-IFG,

included as a "state" finding in Belyk et al. (2015); and [15 13 59] for R-preSMA, included as a "trait" finding in Budde et al. (2014). Trait findings were used for R-preSMA because state findings were not reported in either meta-analysis. The coordinates were used to create 10 mm ROI spheres.

Functional Connectivity

Psychophysiological Interaction (PPI) analysis (Friston et al., 1994, 2003) was used to examine functional connectivity between R-DLPFC and R-SMG. PPI computations were performed on residual components of the modeled task (anticipated vs. unanticipated), after the GLM was effectively removed. Significant correlations are thought to reflect dynamic neural coupling, though not necessarily related to the task. PPI analysis was conducted using the gPPI toolbox (McLaren et al., 2012) with SPM8. The PPI analysis can be described by the following equations:

$$Y_k = H(x_a), \quad (1)$$

$$Y_i = \left[H\left(x_a * \ g_p\right)\right] * \beta_i + \left[H\left(g_p\right)\right] * \beta_p + Y_k * \beta_k + \ e_i, \quad (2)$$

in which the HRF is represented by *H* and *H(x)* is the convolution of signal *X* with kernel *H*. The demeaned time course is represented by g_p where 1 is task time and -1 is rest. β_i is the PPI beta value, whereas β_p is the beta values for the task, β_k is the beta value of the time course of the seed, and Y_k represents the fNIRS data collected at the seed region. Here, *k* is the functionally-defined cluster for the seed region based on the GLM. x_a represents the estimated neural activity for the seed region; the residual error is represented by e_i. R-DLPFC had ROI coordinates [50 26 38] and R-SMG coordinates were determined using the atlas from NIRS-SPM (Rorden & Brett, 2000) and creating a 10 mm sphere which was projected onto the brain surface. Each area was used both as a seed and a target, resulting in two comparisons in total. Two-tailed t-tests were used to compare

residual activity for anticipated versus unanticipated words, and the Holm-Bonferroni method was used to correct for familywise error rates.

Results

Behavioral

Two linear mixed effects models (not included in Jackson et al. (2020)) were fit to examine reaction time and response type. Mean reaction time across all stuttering participants was 348.27 ms (SD = 132.34 ms) (Table 2). Reaction time was significantly impacted by word type

INSERT TABLE 2

(anticipated/unanticipated), such that anticipated words had longer reaction times ($\hat{\beta}$ = 11.45, t = 2.13, $p < .05$). Reaction time was also impacted by trial ($\hat{\beta}$ = -.34, t = -2.91, $p < .01$), such that reaction time decreased as the experiment progressed. Reaction time was not impacted by word length (syllables) ($\hat{\beta}$ = -.07, t = -.73, $p > .05$) or stuttering response type ($\hat{\beta}$ = -5.45, t = -1.54, p > .05), indicating that reaction time for stuttered trials was not significantly longer than that for fluent trials. R^2 for the reaction time model was 0.24. In addition, posthoc Wilcoxon rank sum tests did not reveal differences in reaction time between stuttered and ambiguous trials (W = 203, p > .10), ambiguous and fluent trials (W = 190, p >.10), or stuttered and fluent trials (W = 152, p > .10). Interrater reliability for reaction time between the first author and a SLP blind to the study yielded a Cohen's weighted kappa of .79 (p < .05), indicating moderate to strong agreement (McHugh, 2012).

Table 3 shows the amounts of stuttered, ambiguous, and fluent trials for each participant.

INSERT TABLE 3

For all trials, 43.6% were stuttered (2), 43.3% were fluent (0), and 13.1% were ambiguous (1), or not unambiguously stuttered or fluent (reported in Jackson et al., 2019; see Table 3). Interrater reliability between the first author and an SLP with 8 years of experience (blind to the study) yielded a Cohen's weighted kappa of .85 ($p < .05$), indicating high agreement. The remaining response type data were not reported in Jackson et al. (2020) 53.9% of anticipated words were unambiguously stuttered and 33.4% of unanticipated words were unambiguously stuttered. 35.4% of anticipated words were unambiguously fluent whereas 51.2% of unanticipated words were unambiguously fluent. In addition, 10.7% of anticipated words were ambiguous and 15.41% of unanticipated words were ambiguous. It is important to note that words characterized as ambiguous using the Jackson et al. (2020) approach would most likely have been categorized as fluent with a standard binary stuttered/fluent distinction that is most commonly applied clinically. There was more stuttering for anticipated than unanticipated words, as expected ($\hat{\beta}$ = -.36, t = -9.76, $p < .001$). There was also more stuttering for longer than shorter words ($\hat{\beta}$ = .11, $t = 5.84$, $p < .001$) and for earlier than later trials ($\hat{\beta}$ = -.004, $t = -4.42$, $p < .001$), reflecting a reduction in stuttering over the course of the experiment. R^2 for the stuttering response type model was 0.38. Pearson's correlation test indicated that stuttering rate, expressed as the percentage of trials stuttered for each participant, and extent of anticipation for each participant (i.e., SAS extent score) were not related (t = .47, $p > .10$).

Neural

ROI

(1) Anticipated vs. Unanticipated: The primary contrast compared activation in the R-

DLPFC mask associated with anticipated vs. unanticipated words. Anticipated words were associated with greater activation than unanticipated words ($p = .0217$) based on HbR, but not HbO ($p > .05$). Fig. 2 includes the ROI illustration (A) and event-related averages for anticipated vs. unanticipated words, for HbO (B) and HbR (C), respectively. The HbR difference between

INSERT FIGURE 2

event-related averages at 5 seconds (i.e., the end of the anticipation phase) for anticipated vs. unanticipated words approached significance ($t = 1.67$, $p = .0567$), though for HbO, signals were similar ($t = .57$, $p > .05$). Note that the remaining analyses in this section focus on HbR due to these null HbO results, and also evidence suggesting that HbR 1) is more strongly correlated to the blood oxygen level dependent signal (Boas et al., 2004, 2014; Ferrari & Quaresima, 2012); 2) has greater spatial specificity than HbO (Noah et al., 2021; Zhang et al., 2016); 3) has been validated for speech-language tasks (Zhang et al., 2017); and 4) is less sensitive to systemic effects (e.g., heart rate, breathing, bloodflow) than HbO (Franceschini et al., 2003; Kirilina et al., 2012; Santosa et al., 2019; Scholkmann et al., 2013; Tachtsidis & Scholkmann, 2016; Zhang et al., 2016). Still, Supplementary Figure 2 and Table 2 include the uncorrected whole-brain results for both chromophores.

The two within-group analyses that compared activation to rest provided some support that anticipated words are associated with increased activation ($p = .02$), whereas there was no change for unanticipated words ($p = .82$). Thus, it appears that prior to execution, anticipated words recruited the R-DLPFC ROI, whereas unanticipated words did not. Lastly, the correlation between reaction time and anticipation approached significance ($r = .38$, $p = .08$), indicating that for

anticipated words, reaction time may increase as activation increases (Figure 3).

INSERT FIGURE 3

(2) Stuttered vs. Fluent: Activation in the R-DLPFC mask, using the same five-second time window during the anticipation phase, was also greater for stuttered vs. fluent trials (p=.0039). See Supplementary Figure 3 and Table 3 for uncorrected whole-brain results for HbR as well as HbO. Given that anticipation is strongly associated with stuttering (above), it is possible that the previous results for the anticipated vs. unanticipated contrast may be in part due to atypical planning associated with stuttering, that is not necessarily related to anticipation. It was not possible to compare activation associated with *anticipated/stuttered*, *anticipated/fluent*, *unanticipated/stuttered*, and *unanticipated/fluent*, due to the unbalanced distribution of stuttering within participants (see Table 3) and the relatively limited number of trials. Importantly, however, 33.4% of unanticipated words were unambiguously stuttered, and 35.4% of anticipated words were unambiguously fluent (35.4%). (3) Stutterers vs. Controls: Activation in the R-DLPFC mask, during the anticipation phase, was greater for stutterers compared to control speakers (p=.0442), irrespective of anticipation and stuttering. See Supplementary Figure 4 and Table 4 for uncorrected whole-brain results for HbR and HbO. (4) Task vs. Rest (controls only): We did not find evidence that R-DLPFC was significantly activated during the "anticipation" phase in the control group (p=.1764). This may indicate that the area in the R-DLPFC mask was not recruited for speech planning, which is in line with previous accounts of speech motor control that do not attribute speech motor function to R-DLPFC. (5) Interactive vs. Alone: Condition was not a variable of interest in the current study, but it was important to confirm that condition (interactive vs. alone) did not contribute to R-DLPFC over-activation. A significant difference between the interactive

and alone conditions was not observed (p=.4826).

Significant differences in activation were not observed for anticipated vs. unanticipated words for the two control ROIs: L-DLPFC (p = .99); R-preCG (p = .95). The lack of significant differences in these areas provides evidence that the differences observed in the R-DLPFC ROI were due to the contrast (anticipated vs. unanticipated) and not to general differences associated with the stuttering brain or systemic artifact. In addition, significant activation differences were not observed between anticipated vs. unanticipated words in R-IFG (p = .75) or R-preSMA (p = .39).

Functional Connectivity

Functional connectivity between the R-DLPFC and R-SMG was assessed using each node as a seed and target, and Holm-Bonferroni correction was applied for the two comparisons. Compared to unanticipated words, anticipated words were associated with lower intrinsic connectivity when R-DLPFC was the seed and R-SMG was the target (t=-2.89 p=0.01), and also when R-SMG was the seed and R-DLPFC was the target (t=-2.06 p=0.05). This reduction in functional connectivity between the R-DLPFC and R-SMG for anticipated relative to unanticipated words is taken as evidence of involvement of the FPN in stuttering anticipation.

Discussion

That stutterers anticipate overt stuttering events is well known, but the neural substrates of anticipation had not been studied previously. In this study, we used a novel, clinically inspired approach to identify anticipated and unanticipated words in a relatively large sample of adults who stutter. The words were produced in a delayed-response paradigm while neural signals were recorded with fNIRS. We identified R-DLPFC as a neural substrate of stuttering anticipation. A connectivity analysis was also conducted to explore whether the FPN, specifically the R-DLPFC

and R-SMG, is associated with stuttering anticipation. Results are discussed in the context of theoretical accounts of stuttering anticipation, error-likelihood monitoring, and action-stopping. Our findings and potential limitations, as well as possible clinical implications, are also discussed in the following sections.

Right Dorsolateral Prefrontal Cortex Underlies Stuttering Anticipation

The primary hypothesis was confirmed—anticipated words are associated with greater pre-execution activation in the R-DLPFC, compared to unanticipated words. This means that the production of words previously identified by participants as being difficult or likely to be stuttered, up-regulates activation in this area. It was also shown that stutterers exhibit greater activation than non-stutterers during this same time period, irrespective of anticipation and stuttering, and while anticipated words elicited activation in the R-DLPFC ROI in stutterers, unanticipated words of stutterers, and all words produced by controls, did not elicit activation in this area. Further, anticipated words were associated with longer reaction times, and there was some indication that as activation in R-DLPFC increased, so did reaction time (for anticipated words only). This extra time may be due to speakers delaying speech onset until the word can appear fluent to listeners, or "letting the stuttering pass," which could be a function of the R-DLPFC. Our results provide some clarification of Kell et al. (2009) who found that stutterers exhibit greater activation than controls in the R-DLPFC, but that after therapy this is not the case, suggesting that therapy down-regulates an overactive R-DLPFC. Kell et al. (2009) also found that stutterers who reportedly "recovered" from stuttering after early childhood exhibited similar activation compared to controls in the R-DLPFC, suggesting that elevated activation in the R-DLPFC is a maladaptive response to stuttering. Our results suggest that the Kell et al. (2009) result was due to stuttering anticipation. It is important to highlight that we focused on longer- (vs. shorter-) term anticipation in this study,

as stimuli were identified between 3 and 10 days prior to the fNIRS experiment. While it is possible that the neural processes for longer- and shorter-term anticipation overlap, many words identified as anticipated before the experiment were not stuttered during the experiment, indicating that they may not have been anticipated either, suggesting that there may be differences in the underlying processes related to longer- vs. shorter-term anticipation. Future studies can attempt to disentangle these types of anticipation to provide clarity on the timescales associated with anticipation.

Stuttered speech was also associated with elevated activation in R-DLPFC, calling into question whether atypical activation in this area was due to anticipation, or aberrant planning associated with stuttering. Given our novel approach, it was not possible to completely differentiate processes associated with anticipation and aberrant planning, because we did not have enough power to make comparisons between *anticipated-stuttered*, *anticipated-fluent*, *unanticipated-stuttered*, *unanticipated-fluent* speech. fNIRS, like fMRI, relies on the slow hemodynamic response, which requires longer trials. With 80 trials already, each lasting at least 20 seconds, the experiment lasted approximately 30 minutes, which pushed the comfort threshold for participants. Together with the unpredictability of stuttering, this resulted in too few trials for the aforementioned analysis. However, there is reason to believe that activation differences were in fact driven by anticipation. First, a significant portion of anticipated/unanticipated words were fluent/stuttered, respectively. 33% of anticipated words were unambiguously fluent, whereas 33% of unanticipated words were unambiguously stuttered. Second, it is likely that anticipation was present throughout the five-second anticipation phase, whereas it is unlikely that speech planning processes would comprise the entire five-second anticipation phase. Third, we did not find evidence that non-stutterers activate R-DLPFC during the anticipation phase, suggesting that R-DLPFC does not play a primary role in speech planning. This is consistent with work on speech

motor control or production models that do not include DLPFC as a part of the speech motor network (e.g., Forseth et al., 2021; Guenther, 2016; Sörös et al., 2006). Future work can tease these processes apart by obtaining enough trials to make the necessary statistical comparisons. It should also be noted, however, that due to the dynamic nature of both anticipation and stuttering, it may not be possible to completely differentiate these processes, especially because speech execution, or the possibility of it, is required to elicit anticipation in speakers.

Error-Likelihood Monitoring

Anticipation is likely driven by error-likelihood monitoring, which refers to the ability to predict errors based on prior experience making those errors. In their original account, Brown and Braver (2005) showed that ACC learns to predict the likelihood of errors, generating a "warning signal" to heighten readiness or initiate cognitive control in response to predicted errors. Arenas (2012, 2017) and Garcia-Barrera and Davidow (2015) extended the error-likelihood account to stuttering, whereby associative learning is the basis for anticipation. The speaker learns that some words/sounds are difficult, and when the speaker next says these words/sounds, they are primed to respond to upcoming stuttering. DLPFC works in concert with ACC to detect and respond to anticipated errors, such that ACC underlies the detection of errors in response to unintended outcomes, and subsequently generates error signals, whereas DLPFC generates representations of these errors including holding task-relevant information in working memory, and initiating subsequent actions (Alexander & Brown, 2015; Botvinick et al., 2001; Donoso et al., 2014; Holroyd & Yeung, 2012; Kolling et al., 2012). Interestingly, while Brown and Braver (2005) primarily focused on ACC, they also found error-likelihood effects in the R-DLPFC and R-SMG, which were less connected functionally, for anticipated vs. unanticipated words in the current study. Thus, anticipation may destabilize the FPN, potentially reducing speakers' control in

responding to anticipation. We propose a model that extends the Arenas and Garcia-Barrera and Davidow accounts to the right FPN (see Figure 4). The ACC detects the error (i.e., anticipates) and subsequently generates an error signal which is sent to the R-DLPFC, which coupled with R-SMG, holds this information in working memory and initiates a response. Ultimately, we could not measure ACC due to imaging depth restrictions associated with fNIRS. Future work can test this proposal using fMRI.

INSERT FIGURE 4

Action-stopping

A related interpretation involves the action-stopping network. Action-stopping has been studied primarily in the lab using the stop-signal task, in which participants are required to stop an initiated response when a signal occurs. Aron and colleagues (Aron, 2011; Wessel & Aron, 2017) proposed a prefrontal-basal ganglia-thalamocortical model of action-stopping in which prefrontal areas (R-IFG, R-preSMA) receive sensory information and subsequently produce a stop command, which is then transmitted to the subthalamic nucleus. The subthalamic nucleus then relays this information to the globus pallidus interna of the basal ganglia, which then inhibits the excitatory drive to motor cortex (i.e., to stop the action) (Aron, 2011; Wessel & Aron, 2017). It has been proposed that this mechanism underlies stuttering by inducing a global inhibition response (over-suppression) that impedes the execution of successive motor programs (Arenas, 2017; Aron et al., 2014; Neef et al., 2018). Our results did not support this hypothesis—we did not find evidence of heightened activity in R-IFG or R-preSMA. This may be because the global inhibition account attempts to explain stuttering events at a speech motor control level, whereas anticipation occurs at a cognitive control level.

Disentangling reactive and proactive control, two forms of action-stopping, may provide

clarity regarding the distinction between speech motor and cognitive control process associated with stuttering. Reactive control is stimulus-driven and habitual or automatic, whereas proactive control is prospective and goal-directed (Hannah & Aron, 2021). Reactive control is supported by the hyperdirect cortico-subthalamic-pallidal-thalamo-cortical pathway, and includes superficial cortical structures, the R-IFG and R-preSMA (Jahanshahi et al., 2015). This pathway is likely associated with the global inhibition that has been proposed to prevent the execution of successive motor programs (e.g., Arenas, 2017; Aron et al., 2014; Neef et al., 2018), i.e., to cause or trigger stuttering events. Proactive control, on the other hand, is regulated by the indirect fronto-striato-pallido-thalamo-cortical pathway, which includes the R-DLPFC, R-SMG, caudate, and thalamus (A. Chang et al., 2017; Chikazoe et al., 2009; Jahanshahi et al., 2015; Jahfari et al., 2010; Marek & Dosenbach, 2018; Smittenaar et al., 2013). Neef et al. (2018) proposed that proactive control may underlie responses to stuttering anticipation, which is supported by the current findings of over-activation in the R-DLPFC and reduced connectivity between R-DLPFC and R-SMG. Thus, while the indirect and hyperdirect pathways share neural circuitry, the hyperdirect pathway may underlie global suppression to prevent the succession of speech gestures (i.e., the stuttering event), whereas the indirect pathway seems to be related to how the speaker responds to *knowing* that the stuttering event is going to happen. The temporal dynamics associated with fNIRS as well as the slow hemodynamic response makes fNIRS a sub-optimal tool to test this hypothesis. Future studies could use fMRI and MEG to tease apart the specific spatial and temporal contributions (respectively) of both pathways, as well as interactions with the error monitoring system (including the ACC), to the manifestation of overt stuttering events and the speaker's anticipation of them.

Clinical Implications

The current work may inform neuromodulation techniques which have recently been

applied to stuttering in clinical trials. Chesters et al. (2018) tested the impact of anodal transcranial direct current stimulation (tDCS) of left inferior frontal cortex on overt severity in 30 adult stutterers. They found that the treatment significantly reduced overt stuttering severity at a 1-week follow-up assessment, and at 6 weeks for reading but not conversational speech (Chesters et al., 2018). Garnett et al.(2019) tested the impact of anodal tDCS on overt severity in 14 adult stutterers. They did not find significant effects on overt stuttering severity, though they found that the atypically strong association between overt severity and right thalamocortical activity was attenuated, especially in severe stutterers. It may be that the modest effects reported to date are due to the focus on single areas, specifically in the speech network. Stuttering is not simply a speech motor control problem. Our data show that the cognitive and sensorimotor processes that underlie anticipation and subsequent overt stuttering elicit elevated activation in R-DLPFC, as well as potentially other areas within the FPN. Down-regulating R-DLPFC using tDCS and concurrently up-regulating, for example, left speech-language areas (premotor cortex/inferior frontal gyrus), which are typically under-active in speakers who stutter, could be the basis for clinical trials of the effects of tDCS in stuttering therapy.

Behaviorally, the anticipation of stuttering allows stutterers to disguise stuttering, such that there will be a discrepancy between what listeners hear or see and what speakers experience. While anticipating stuttering, the speaker may experience anxiety, fear, shame, or other cognitive responses, but the listener may not be privy to this information, creating misunderstanding between the speaker and the listener which could lead to negative listener perceptions of stuttering. For example, a listener might judge a speaker for looking "nervous" or for not being intelligent because they do not respond in a timely manner (as perceived by the listener). Understanding how the brain functions during anticipation could mitigate this discrepancy through increased understanding of

639 the phenomenon and public awareness. This could also improve how speech-language pathologists
640 (SLPs) work with people who stutter, particularly with respect to helping clients develop adaptive
641 responses to anticipation. Developing such adaptive responses during therapy can be challenging,
642 primarily because of the unobservable or "hidden" nature of anticipation. A brain-based
643 understanding of anticipation may provide an entry point to begin discussing anticipation with
644 their clients.

645 **Limitations**

646 One limitation of this study is that the hypothesized effect in R-DLPFC – activation greater
647 for anticipated vs. unanticipated words – was only evident based on the HbR signal, not HbO. For
648 other motor tasks (e.g., finger tapping), effects are expected to be present for both chromophores.
649 However, it is important to highlight that there is evidence that HbR may be a more reliable signal
650 for speech tasks. For example, HbR is more strongly correlated to the blood oxygen level
651 dependent signal compared to HbO (Boas et al., 2004, 2014; Ferrari & Quaresima, 2012), has
652 greater spatial specificity than HbO (Noah et al., 2021; Zhang et al., 2016), and has been validated
653 for speech-language tasks (Zhang et al., 2017). Further, HbR is less sensitive to systemic effects
654 (e.g., heart rate, breathing, bloodflow) than HbO (Franceschini et al., 2003; Kirilina et al., 2012;
655 Santosa et al., 2019; Scholkmann et al., 2013; Tachtsidis & Scholkmann, 2016; Zhang et al., 2016),
656 which may be relevant to stuttering because stuttering speakers may experience elevated
657 anxiety/heart rate prior to speaking tasks. Neither anxiety nor autonomic data were collected in the
658 current study, which may have contributed to activation in R-DLPFC. Future studies could
659 examine the impact of systemic effects on HbO and HbR, particularly for populations that may
660 exhibit greater systemic effects such as anxiety. This would allow researchers to both determine
661 whether systemic effects differentially impact HbO/HbR, and also differentiate between

anticipation and anxiety, to the extent that the latter is even possible.

Another limitation was that the design did not allow us to differentiate the awareness of upcoming stuttering, with the speaker's response to the knowledge that upcoming speech will be stuttered (should they initiate their speech plan as intended). Thus, anticipation and *responding* to anticipation are conflated. Future work can address this limitation, for example, by testing the model of anticipation proposed herein, which postulates that the initial awareness of upcoming speech breakdown may occur in ACC, whereas the response to anticipation may be supported by R-DLPFC and the broader FPN. A third limitation is that anticipation was not measured during the actual experiment (e.g., by using a button press), and therefore there was no indication of the extent to which participants anticipated, if they anticipated at all. There were several reasons for this. First, we did not want to increase the length of the experiment, which was already nearing the participant comfort threshold. Identifying whether anticipation occurred could be difficult for some, if not all, participants, which would require additional time. Second, the act of identifying anticipation may alter neural responses. If identification comes before the trial, the act of identifying itself could alter the neural response. If identification occurs after the trial, responses may be biased based on whether the trial was overtly stuttered. One compromise could be to ask participants to identify the extent of anticipation for all words, that were previously identified as anticipated, just prior to the experiment. Participants could be presented with stimuli as they would during the actual experiment, which may help with identifying whether a word would be anticipated.

Conclusion

This study determined that the R-DLPFC is a likely neural substrate of stuttering anticipation, and also that anticipation may be associated with reduced connectivity within the

right hemisphere FPN. The results support accounts of error-likelihood monitoring and action-stopping and their association with stuttering events. Future investigations will benefit from adapting the current paradigm for use with fMRI and MEG to determine the relationships between error-likelihood monitoring, action-stopping, and stuttering, and whether functional connectivity within the FPN and related networks (salience, cingulo-opercular) is weaker for anticipated words. It will also be critical to study children who stutter so that the developmental bases of anticipation can be determined. Finally, results from this study may inform clinical trials that test the efficacy of neuromodulation in stuttering therapy, particularly by focusing not only on speech motor control networks, but also cognitive control and related networks.

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Figure

Table 1. Participant data. Treatment history descriptions written as reported by participants. TS = Trials Stuttered; SAS = Stuttering Anticipation Scale, extent score out of 100, 0 = "never," 100 = "always." M = mean; SD = standard deviation.

ID	Age	Gender	Treatment history	%TS	SAS
1	29	F	On and off in elementary school	43%	50
2	35	F	7 years	36%	100
3	23	M	2 weeks (intensive program)	0%	76
4	34	M	6 years	0%	79
5	26	F	3 years	87%	95
6	48	F	Roughly 2 years	80%	87
7	21	M	8 years	27%	90
8	23	M	2.5 years	23%	77
9	29	M	9 months	45%	80
10	34	M	2 years	98%	80
11	37	F	Off and on for years	23%	75
12	39	F	12-15 years	76%	86
13	23	F	6+ years	93%	89
14	47	F	None	30%	98
15	42	M	8 years	15%	90
16	18	M	10 years, on and off	70%	75
17	30	M	approx. 10 years	51%	76
18	29	M	about 7 years	25%	67
19	39	F	about 6 months	55%	76
20	25	M	About 7 years (on and off)	21%	70
21	22	M	None	55%	77
22	50	F	3 months	15%	99
M	32			40%	81
SD	9			30%	12

Figure 1. Task timeline. The question/sentence completion was presented auditorily. The anticipation period was five seconds; participants looked at a fixation cross during this time. The green screen signaled participants to produce the word. Participants again looked at the cross during the rest period.

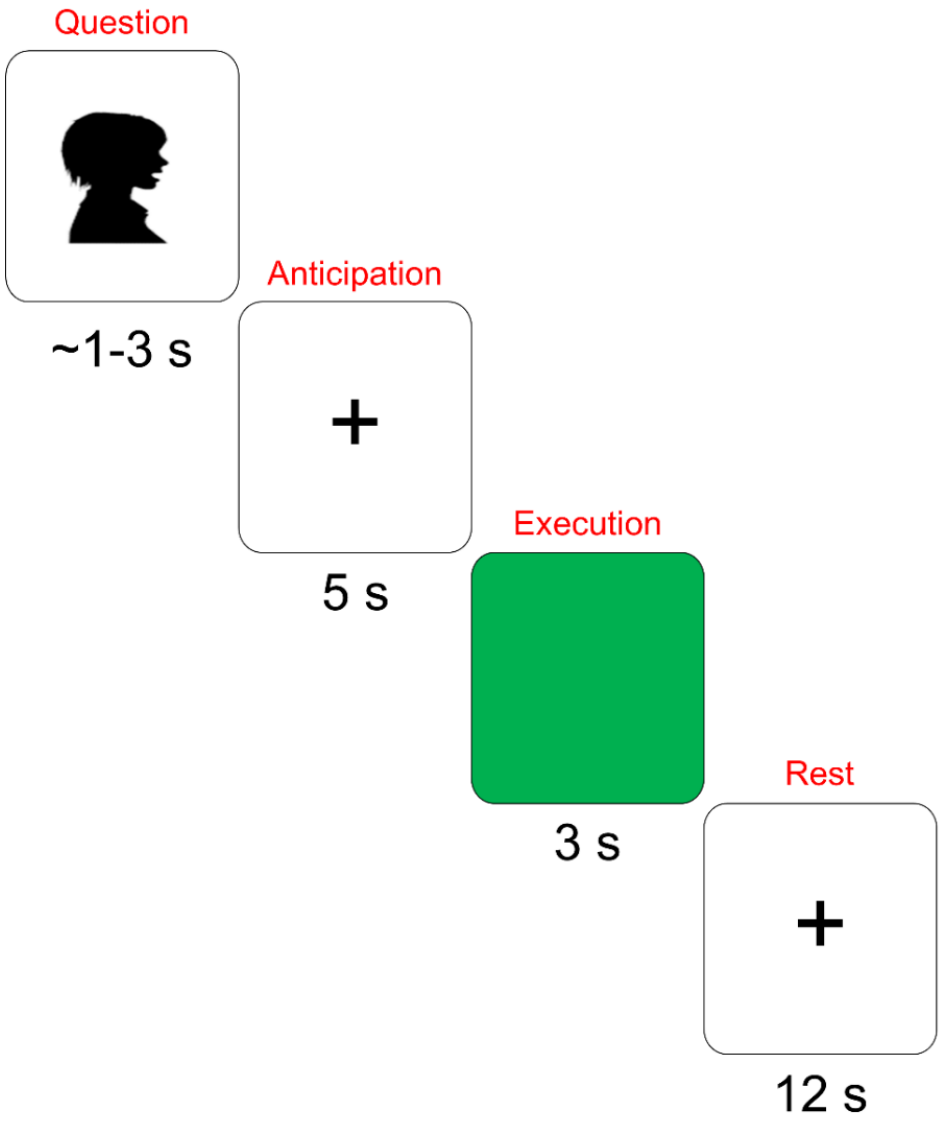


Table 2. Reaction time means and standard deviations, as determined by first movement. ID = participant; SD = standard deviation.

ID	Fluent	SD	Ambiguous	SD	Stuttered	SD
1	352.14	103.71	286.03	69.08	297.39	108.48
2	362.68	115.64	307.75	101.19	368.22	97.08
3	398.99	126.61	300.33	168.84		
4	311.76	120.47	467.18	47.19		
5						
6	386.14	90.07	517.24	50.61	458.53	109.47
7	341.00	187.38	406.51	132.49	272.23	87.77
8	310.22	72.77	296.29	60.60	300.33	62.43
9	370.68	109.21	433.81	129.24	324.43	104.50
10					282.50	81.94
11	302.33	75.50	361.51	114.57	367.07	144.78
12	333.70	100.11	305.89	64.95	262.28	94.69
13	360.40	148.11	300.33	75.91	303.49	69.47
14	480.53	147.94	524.39	195.26	560.34	154.65
15	327.57	115.33	322.58	70.79	294.77	111.89
16	290.32	99.55	340.85	68.24	282.73	69.12
17	281.56	87.84	302.18	84.48	330.44	140.16
18	421.30	132.68	446.17	208.43	361.51	134.58
19	515.72	122.92	517.24	306.75	489.97	134.20
20	323.34	76.61	260.29	27.92	288.55	110.64
21	304.64	160.21	133.48	47.19	285.78	97.98
22	362.01	125.17	310.76	147.09	367.07	95.85
	358.90		352.49		336.10	

Table 3. Stuttering response type by participant, including percentages. ID = participant.

ID	Trials	Errors	Unambiguously fluent (0)	%	Ambiguous (1)	%	Unambiguously stuttered (2)	%	Anticipated words that were unambiguously stuttered	%
1	80	1	38	49%	7	9%	34	43%	26	65%
2	80	4	38	53%	9	11%	29	36%	25	63%
3	80	4	70	93%	6	8%	0	0%	0	0%
4	80	3	75	98%	2	3%	0	0%	0	0%
5	60	1	2	4%	5	6%	52	87%	29	97%
6	70	3	7	10%	6	8%	54	80%	30	89%
7	70	1	38	49%	12	15%	19	27%	17	49%
8	80	1	27	35%	34	43%	18	23%	11	28%
9	80	0	37	46%	7	9%	36	45%	24	60%
10	80	2	0	3%	0	0%	78	98%	40	100%
11	80	0	50	63%	12	15%	18	23%	7	18%
12	80	1	7	9%	12	15%	60	76%	30	78%
13	80	0	5	6%	1	1%	74	93%	38	95%
14	80	1	47	60%	8	10%	24	30%	20	50%
15	80	9	49	73%	10	13%	12	15%	11	28%
16	80	0	10	13%	14	18%	56	70%	34	85%
17	80	0	18	23%	21	26%	41	51%	24	60%
18	80	1	30	39%	29	36%	20	25%	11	28%
19	80	4	33	43%	2	3%	41	55%	33	90%
20	80	0	58	73%	5	6%	17	21%	10	25%
21	80	5	33	41%	2	4%	40	55%	24	68%
22	60	2	33	43%	16	21%	9	15%	6	20%
			736		222		742		458	
	% across dataset		43.30%		13.10%		43.60%		53.88%	

Table 3. Stuttering response type (cont.)

ID	Unanticipated words that were unambiguously stuttered	%	Anticipated words that were unambiguously fluent	%	Unanticipated words that were unambiguously fluent	%	Anticipated words that were ambiguous	%	Unanticipated words that were ambiguous	%
1	8	20%	13	33%	25	65%	1	3%	6	15%
2	4	10%	10	30%	28	75%	3	8%	6	15%
3	0	0%	38	98%	32	88%	1	3%	5	13%
4	0	0%	38	98%	37	98%	1	3%	1	3%
5	23	77%	0	3%	2	7%	0	0%	5	17%
6	24	71%	1	3%	6	20%	3	9%	3	9%
7	2	6%	11	34%	27	77%	6	17%	6	17%
8	7	18%	13	35%	14	35%	15	38%	19	48%
9	12	30%	13	33%	24	60%	3	8%	4	10%
10	38	95%	0	0%	0	5%	0	0%	0	0%
11	11	28%	27	68%	23	58%	6	15%	6	15%
12	30	75%	0	0%	7	18%	9	23%	3	8%
13	36	90%	2	5%	3	8%	0	0%	1	3%
14	4	10%	16	40%	31	80%	4	10%	4	10%
15	1	3%	20	58%	29	88%	6	15%	4	10%
16	22	55%	2	5%	8	20%	4	10%	10	25%
17	17	43%	8	20%	10	25%	8	20%	13	33%
18	9	23%	15	38%	15	40%	14	35%	15	38%
19	8	20%	3	10%	30	75%	0	0%	2	5%
20	7	18%	28	70%	30	75%	2	5%	3	8%

21	16	43%	13	33%	20	50%	0	0%	2	8%
22	3	10%	19	63%	14	50%	5	17%	11	40%
	284		301		435		91		131	
	33.41%		35.41%		51.18%		10.71%		15.41%	

Figure 2A-C. Region of interest analysis. (A) ROI mask for right dorsolateral prefrontal cortex (R-DLPFC) [58 26 38]. A 10- millimeter sphere was created using the MarsBar toolbox (Brett et al., 2002) and the xjView toolbox (https:// www.alivelearn.net/xjview). The image was then projected onto the template using BrainNet Viewer (Xia, M.,Wang, J., & He,Y. (2013). BrainNet Viewer: a network visualization tool for human brain connectomics. PloS one, 8(7), e68910). (B-C) Event-related averages of activity for the stuttering group within the R-DLPFC ROI for anticipated vs. unanticipated words, for Oxyhemoglobin (HbO) and DeOxyhemoglobin (HbR), respectively. 0 is the beginning of the anticipation phase, and 30 seconds is shown because the hemodynamic response could feasibly last this long. HbR signals prior to sign reversal such that decreasing values reflect increases in activation strength. Shading represents standard error of the mean.

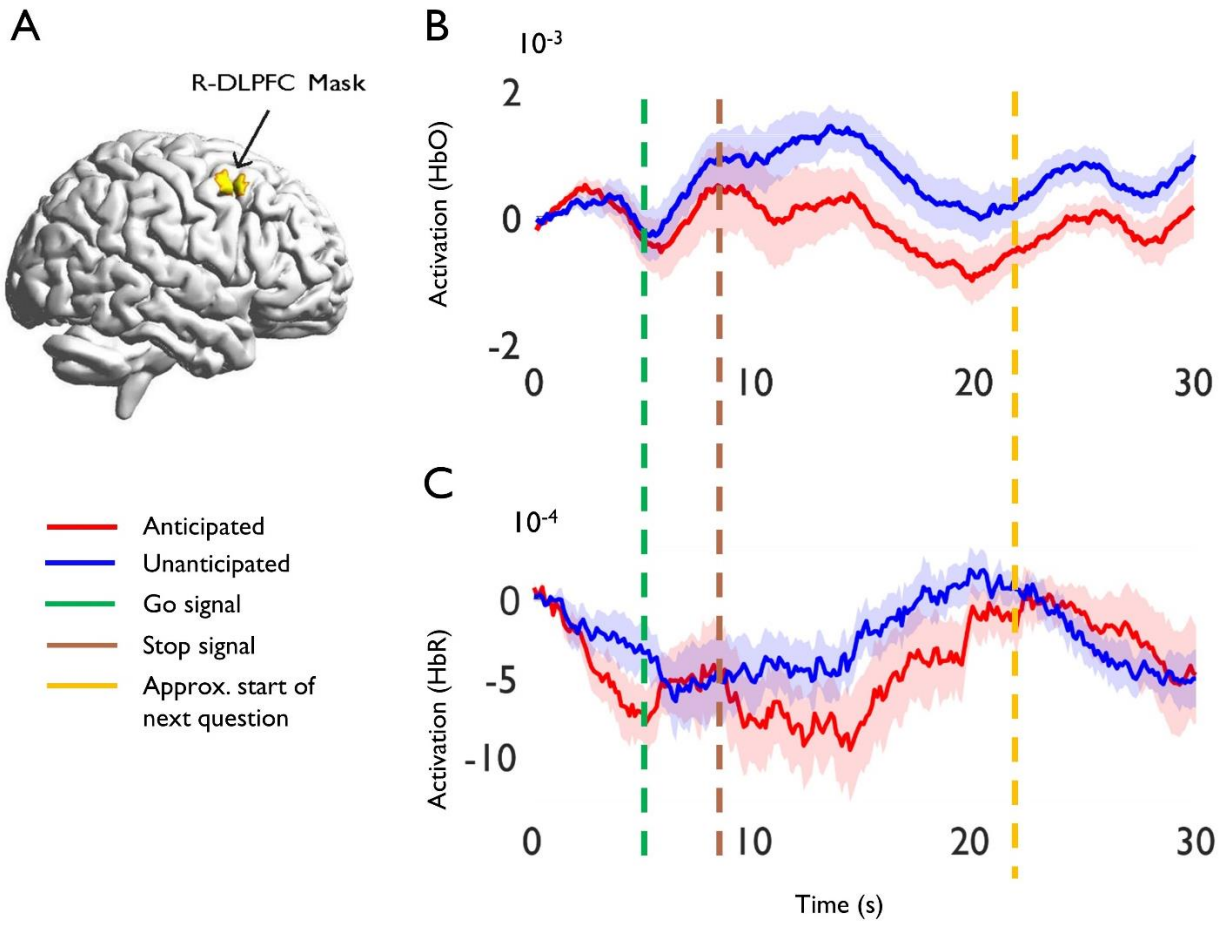


Figure 3. Correlation between activation and reaction time (ms) in R-DLPFC (approached significance. Deoxyhemoglobin (HbR) signals prior to sign reversal such that decreasing values reflect increases in activation strength.

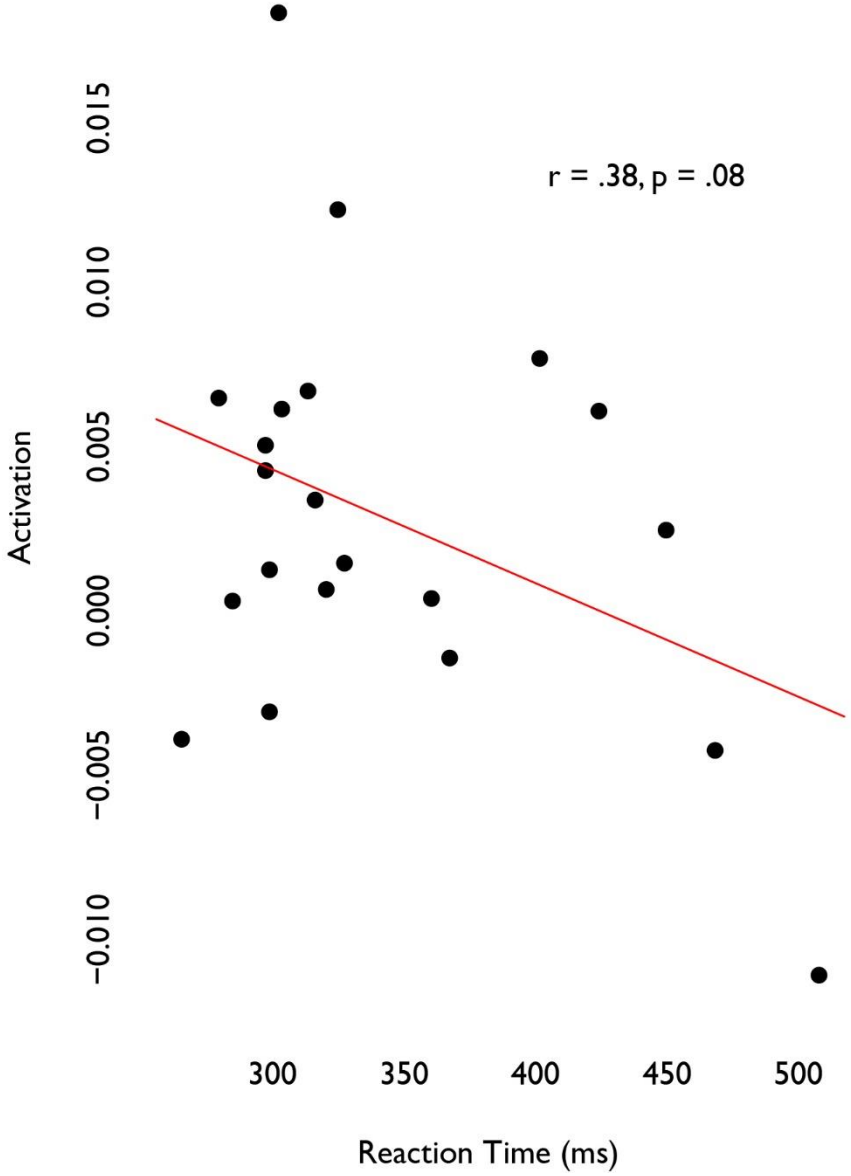
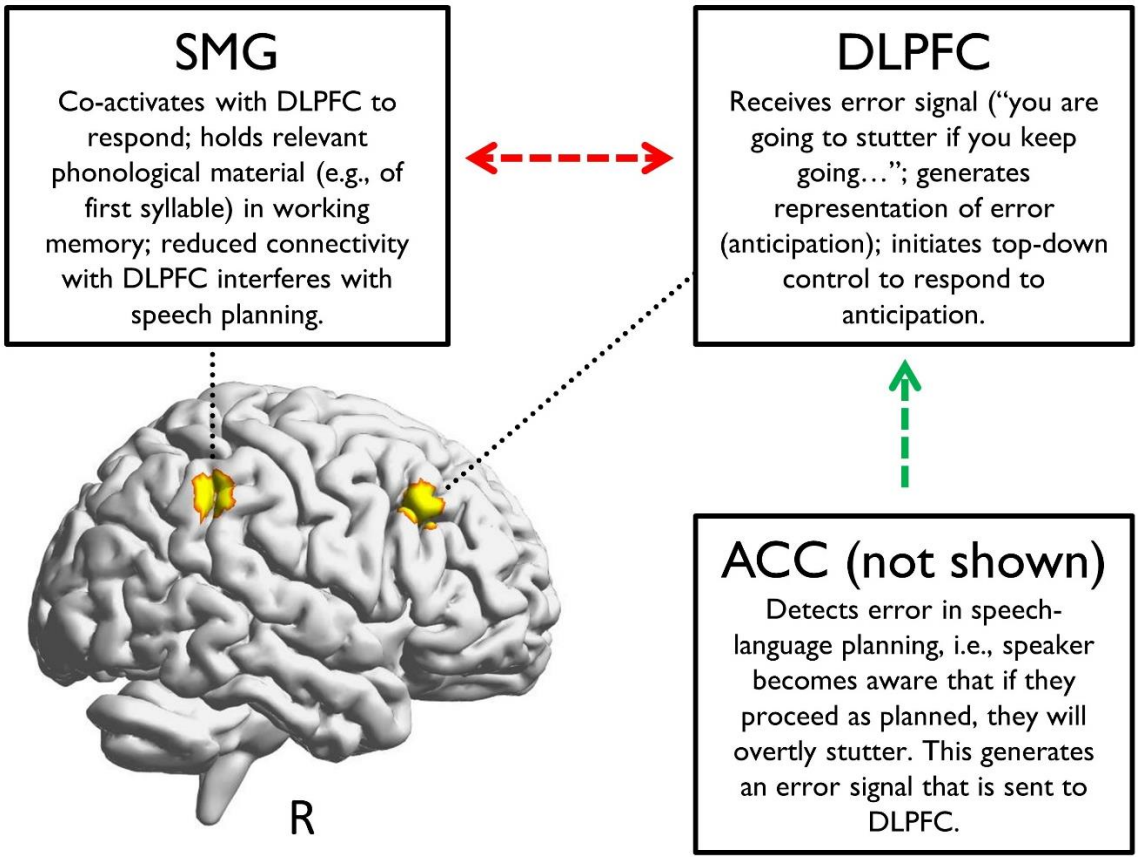
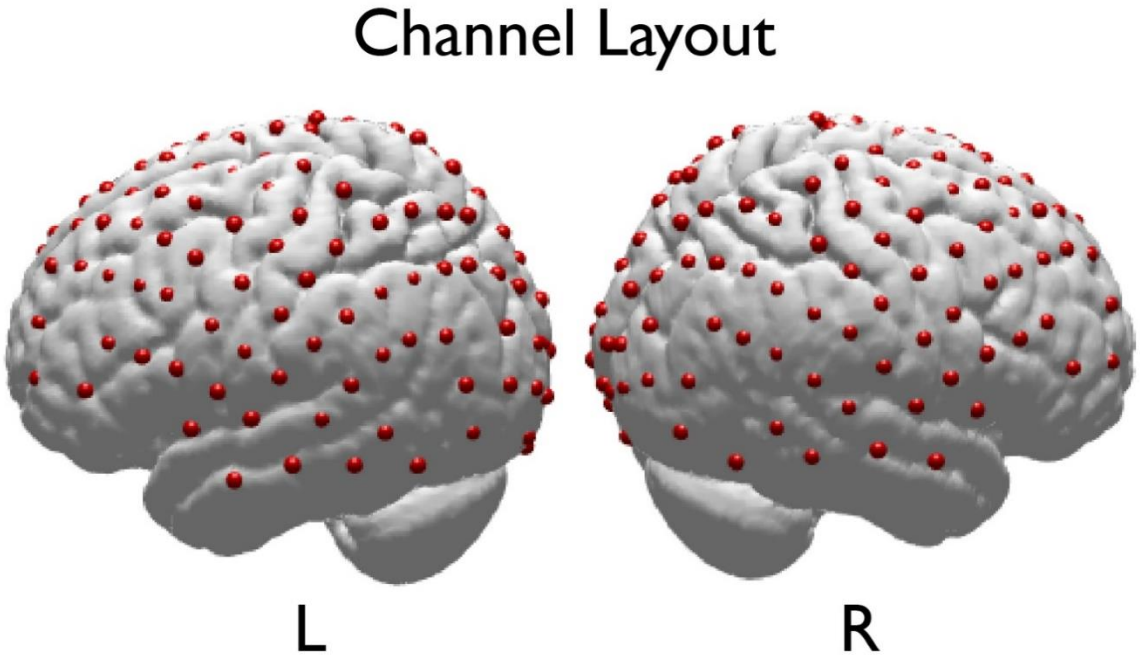


Figure 4. A model of stuttering anticipation. Anticipation develops via associative learning. Memory traces of associations between overtly stuttered utterances and environmental/listener reactions form. ACC detects upcoming stuttering via error-likelihood monitoring, and signals R-DLPFC which initiates cognitive control to respond to anticipation, thereby elevating activation in R-DLPFC. R-DLPFC initiates activation within the broader FPN to support the response. Atypical connection between R-DLPFC and areas within the FPN, such as SMG, impedes the speaker's ability to adaptively respond to anticipation. ACC – anterior cingulate cortex; R-DLPFC = right dorsolateral prefrontal cortex; FPN = frontoparietal control network; R-SMG = right supramarginal gyrus.



SUPPLEMENTARY MATERIAL

Supplementary Figure 1. Right (R) and left (L) hemispheres of rendered brains illustrate average locations (red circles) for the 134 channels per participant. Montreal Neurological Institute (MNI) coordinates were determined by digitizing the locations of the optodes in relation to the 10–20 system based on conventional landmarks. See Supplementary Table 1 for group median coordinates, anatomical regions, and atlas-based probabilities for each channel.



Supplementary Table 1. Group median coordinates, atlas-based probabilities, Brodmann's areas, and anatomical regions for each channel. Montreal Neurological Institute (MNI) coordinates for each of the 134 channels per participant were determined by digitizing the locations of the optodes in relation to the 10-20 system based on conventional landmarks.

Channel number	MNI coordinates			Probability	BA	Anatomical region
	X	Y	Z			
1	-37	61	9	0.97	10	Frontopolar area
				0.03	46	Dorsolateral prefrontal cortex
2	38	63	10	1	10	Frontopolar area
3	-26	62	24	0.96	10	Frontopolar area
				0.04	9	Dorsolateral prefrontal cortex
4	26	64	26	0.94	10	Frontopolar area
				0.06	9	Dorsolateral prefrontal cortex
5	-13	58	39	0.83	9	Dorsolateral prefrontal cortex
				0.14	10	Frontopolar area
				0.03	8	Includes Frontal eye fields
6	13	59	39	0.81	9	Dorsolateral prefrontal cortex
				0.17	10	Frontopolar area
				0.02	8	Includes Frontal eye fields
7	-23	48	44	0.54	9	Dorsolateral prefrontal cortex
				0.46	8	Includes Frontal eye fields
8	-1	51	48	0.7	8	Includes Frontal eye fields
				0.3	9	Dorsolateral prefrontal cortex
9	22	50	46	0.58	8	Includes Frontal eye fields
				0.42	9	Dorsolateral prefrontal cortex
10	-49	37	29	0.88	46	Dorsolateral prefrontal cortex
				0.12	9	Dorsolateral prefrontal cortex
11	-33	38	46	0.7	8	Includes Frontal eye fields
				0.3	9	Dorsolateral prefrontal cortex
12	-12	41	55	0.98	8	Includes Frontal eye fields
				0.02	6	Pre-Motor and Supplementary Motor Cortex
13	12	42	56	0.97	8	Includes Frontal eye fields
				0.03	6	Pre-Motor and Supplementary Motor Cortex
14	33	40	47	0.77	8	Includes Frontal eye fields
				0.23	9	Dorsolateral prefrontal cortex
15	50	39	32	0.66	46	Dorsolateral prefrontal cortex
				0.34	9	Dorsolateral prefrontal cortex
				0.01	10	Frontopolar area
16	-55	25	27	0.39	46	Dorsolateral prefrontal cortex

				0.32	9	Dorsolateral prefrontal cortex
				0.29	45	pars triangularis Broca's area
				0	44	pars opercularis
17	-43	26	47	0.82	8	Includes Frontal eye fields
				0.18	9	Dorsolateral prefrontal cortex
18	-22	29	59	0.6	8	Includes Frontal eye fields
				0.4	6	Pre-Motor and Supplementary Motor Cortex
19	-1	33	60	0.52	8	Includes Frontal eye fields
				0.48	6	Pre-Motor and Supplementary Motor Cortex
20	22	32	59	0.6	8	Includes Frontal eye fields
				0.4	6	Pre-Motor and Supplementary Motor Cortex
21	43	29	49	0.87	8	Includes Frontal eye fields
				0.13	9	Dorsolateral prefrontal cortex
22	56	29	30	0.54	46	Dorsolateral prefrontal cortex
				0.35	9	Dorsolateral prefrontal cortex
				0.11	45	pars triangularis Broca's area
23	-62	8	5	0.5	22	Superior Temporal Gyrus
				0.3	44	pars opercularis
				0.17	6	Pre-Motor and Supplementary Motor Cortex
				0.03	45	pars triangularis Broca's area
24	-60	13	26	0.51	9	Dorsolateral prefrontal cortex
				0.22	45	pars triangularis Broca's area
				0.2	44	pars opercularis
				0.07	6	Pre-Motor and Supplementary Motor Cortex
25	-49	17	47	0.64	8	Includes Frontal eye fields
				0.2	9	Dorsolateral prefrontal cortex
				0.15	6	Pre-Motor and Supplementary Motor Cortex
26	-35	19	61	0.52	8	Includes Frontal eye fields
				0.48	6	Pre-Motor and Supplementary Motor Cortex
27	-14	22	67	1	6	PreMotor and Supplementary Motor Cortex
28	13	23	67	1	6	PreMotor and Supplementary Motor Cortex
29	34	21	61	0.52	6	PreMotor and Supplementary Motor Cortex
				0.48	8	Includes Frontal eye fields
30	50	19	49	0.74	8	Includes Frontal eye fields
				0.14	9	Dorsolateral prefrontal cortex

				0.12	6	Pre-Motor and Supplementary Motor Cortex
31	62	17	28	0.6	9	Dorsolateral prefrontal cortex
				0.27	45	pars triangularis Broca's area
				0.08	44	pars opercularis
				0.04	46	Dorsolateral prefrontal cortex
32	62	13	8	0.43	44	pars opercularis; part of Broca's area
				0.23	45	pars triangularis Broca's area
				0.22	22	Superior Temporal Gyrus
				0.08	6	Pre-Motor and Supplementary Motor Cortex
				0.04	47	Inferior prefrontal gyrus
33	-68	-7	-11	1	21	Middle Temporal gyrus
34	-65	-1	20	0.58	6	PreMotor and Supplementary Motor Cortex
				0.17	43	Subcentral area
				0.08	22	Superior Temporal Gyrus
				0.08	4	Primary Motor Cortex
				0.06	44	pars opercularis
				0.03	9	Dorsolateral prefrontal cortex
				0.01	45	pars triangularis Broca's area
35	-59	2	41	0.86	6	PreMotor and Supplementary Motor Cortex
				0.11	9	Dorsolateral prefrontal cortex
				0.03	8	Includes Frontal eye fields
36	-44	9	59	0.87	6	PreMotor and Supplementary Motor Cortex
				0.13	8	Includes Frontal eye fields
37	-24	10	69	1	6	PreMotor and Supplementary Motor Cortex
38	-2	11	71	1	6	PreMotor and Supplementary Motor Cortex
39	23	11	70	1	6	PreMotor and Supplementary Motor Cortex
40	45	9	59	0.82	6	PreMotor and Supplementary Motor Cortex
				0.18	8	Includes Frontal eye fields
41	60	5	43	0.68	6	PreMotor and Supplementary Motor Cortex
				0.21	9	Dorsolateral prefrontal cortex
				0.11	8	Includes Frontal eye fields
42	67	4	22	0.62	6	PreMotor and Supplementary Motor Cortex
				0.13	9	Dorsolateral prefrontal cortex
				0.1	44	pars opercularis
				0.07	45	pars triangularis Broca's area
				0.06	4	Primary Motor Cortex
				0.02	43	Subcentral area
43	68	-2	-8	0.85	21	Middle Temporal gyrus
				0.15	22	Superior Temporal Gyrus
44	-68	-12	12	0.35	42	Primary and Auditory Association Cortex
				0.33	43	Subcentral area

				0.3	22	Superior Temporal Gyrus
				0.02	40	Supramarginal gyrus part of Wernicke's area
				0	4	Primary Motor Cortex
45	-65	-10	35	0.64	6	PreMotor and Supplementary Motor Cortex
				0.15	3	Primary Somatosensory Cortex
				0.11	4	Primary Motor Cortex
				0.09	1	Primary Somatosensory Cortex
				0.01	2	Primary Somatosensory Cortex
46	-54	-8	55	0.58	6	PreMotor and Supplementary Motor Cortex
				0.27	3	Primary Somatosensory Cortex
				0.14	4	Primary Motor Cortex
				0.01	1	Primary Somatosensory Cortex
47	-35	-4	67	1	6	PreMotor and Supplementary Motor Cortex
48	-14	-2	75	1	6	PreMotor and Supplementary Motor Cortex
49	13	-1	75	1	6	PreMotor and Supplementary Motor Cortex
50	34	-3	68	1	6	PreMotor and Supplementary Motor Cortex
51	54	-5	55	0.72	6	PreMotor and Supplementary Motor Cortex
				0.14	3	Primary Somatosensory Cortex
				0.13	4	Primary Motor Cortex
52	67	-6	37	0.96	6	PreMotor and Supplementary Motor Cortex
				0.03	4	Primary Motor Cortex
				0.01	3	Primary Somatosensory Cortex
53	70	-9	15	0.38	43	Subcentral area
				0.25	22	Superior Temporal Gyrus
				0.22	42	Primary and Auditory Association Cortex
				0.08	6	Pre-Motor and Supplementary Motor Cortex
				0.07	4	Primary Motor Cortex
				0	40	Supramarginal gyrus part of Wernicke's area
54	-71	-26	-3	0.7	21	Middle Temporal gyrus
				0.22	22	Superior Temporal Gyrus
				0.08	42	Primary and Auditory Association Cortex
55	-68	-22	28	0.35	40	Supramarginal gyrus part of Wernicke's area
				0.24	2	Primary Somatosensory Cortex
				0.18	1	Primary Somatosensory Cortex
				0.11	43	Subcentral area
				0.08	3	Primary Somatosensory Cortex
				0.02	6	Pre-Motor and Supplementary Motor Cortex
				0.02	42	Primary and Auditory Association Cortex

56	-62	-22	48	0.33	2	Primary Somatosensory Cortex
				0.25	1	Primary Somatosensory Cortex
				0.19	6	Pre-Motor and Supplementary Motor Cortex
				0.14	3	Primary Somatosensory Cortex
				0.08	4	Primary Motor Cortex
57	-46	-19	65	0.47	3	Primary Somatosensory Cortex
				0.25	6	Pre-Motor and Supplementary Motor Cortex
				0.15	4	Primary Motor Cortex
				0.13	1	Primary Somatosensory Cortex
				0	2	Primary Somatosensory Cortex
58	-24	-15	75	0.97	6	PreMotor and Supplementary Motor Cortex
				0.03	4	Primary Motor Cortex
59	-2	-12	75	1	6	PreMotor and Supplementary Motor Cortex
60	24	-13	75	1	6	PreMotor and Supplementary Motor Cortex
61	46	-16	65	0.46	6	PreMotor and Supplementary Motor Cortex
				0.32	3	Primary Somatosensory Cortex
				0.19	4	Primary Motor Cortex
				0.02	1	Primary Somatosensory Cortex
62	63	-20	49	0.26	1	Primary Somatosensory Cortex
				0.22	3	Primary Somatosensory Cortex
				0.22	2	Primary Somatosensory Cortex
				0.21	6	Pre-Motor and Supplementary Motor Cortex
				0.09	4	Primary Motor Cortex
				0.01	40	Supramarginal gyrus part of Wernicke's area
63	70	-20	30	0.26	40	Supramarginal gyrus part of Wernicke's area
				0.21	2	Primary Somatosensory Cortex
				0.2	1	Primary Somatosensory Cortex
				0.16	3	Primary Somatosensory Cortex
				0.07	6	Pre-Motor and Supplementary Motor Cortex
				0.07	43	Subcentral area
				0.01	4	Primary Motor Cortex
64	73	-22	0	0.42	21	Middle Temporal gyrus
				0.4	22	Superior Temporal Gyrus
				0.18	42	Primary and Auditory Association Cortex
65	-69	-36	14	0.75	22	Superior Temporal Gyrus
				0.21	42	Primary and Auditory Association Cortex

				0.04	40	Supramarginal gyrus part of Wernicke's area
66	-66	-33	40	0.78	40	Supramarginal gyrus part of Wernicke's area
				0.16	2	Primary Somatosensory Cortex
				0.06	1	Primary Somatosensory Cortex
67	-54	-29	57	0.41	2	Primary Somatosensory Cortex
				0.37	40	Supramarginal gyrus part of Wernicke's area
				0.2	1	Primary Somatosensory Cortex
				0.02	3	Primary Somatosensory Cortex
68	-37	-26	72	0.33	6	PreMotor and Supplementary Motor Cortex
				0.3	3	Primary Somatosensory Cortex
				0.3	4	Primary Motor Cortex
				0.07	1	Primary Somatosensory Cortex
				0.01	2	Primary Somatosensory Cortex
69	-14	-25	79	0.64	6	PreMotor and Supplementary Motor Cortex
				0.34	4	Primary Motor Cortex
				0.02	3	Primary Somatosensory Cortex
70	14	-25	79	0.72	6	PreMotor and Supplementary Motor Cortex
				0.28	4	Primary Motor Cortex
				0	3	Primary Somatosensory Cortex
71	36	-25	73	0.43	4	Primary Motor Cortex
				0.38	6	Pre-Motor and Supplementary Motor Cortex
				0.18	3	Primary Somatosensory Cortex
				0.02	1	Primary Somatosensory Cortex
72	55	-29	59	0.35	40	Supramarginal gyrus part of Wernicke's area
				0.3	2	Primary Somatosensory Cortex
				0.24	1	Primary Somatosensory Cortex
				0.1	3	Primary Somatosensory Cortex
73	68	-31	42	0.62	40	Supramarginal gyrus part of Wernicke's area
				0.19	1	Primary Somatosensory Cortex
				0.16	2	Primary Somatosensory Cortex
				0.02	3	Primary Somatosensory Cortex
				0.01	4	Primary Motor Cortex
74	71	-32	16	0.42	22	Superior Temporal Gyrus
				0.36	42	Primary and Auditory Association Cortex
				0.22	40	Supramarginal gyrus part of Wernicke's area
75	-69	-47	-3	0.76	21	Middle Temporal gyrus

				0.19	22	Superior Temporal Gyrus
				0.05	37	Fusiform gyrus
76	-67	-45	26	0.76	40	Supramarginal gyrus part of Wernicke's area
				0.24	22	Superior Temporal Gyrus
77	-61	-43	47	1	40	Supramarginal gyrus part of Wernicke's area
78	-45	-38	65	0.37	40	Supramarginal gyrus part of Wernicke's area
				0.32	2	Primary Somatosensory Cortex
				0.18	1	Primary Somatosensory Cortex
				0.11	5	Somatosensory Association Cortex
				0.03	3	Primary Somatosensory Cortex
79	-23	-39	75	0.4	3	Primary Somatosensory Cortex
				0.3	5	Somatosensory Association Cortex
				0.16	2	Primary Somatosensory Cortex
				0.1	4	Primary Motor Cortex
				0.03	1	Primary Somatosensory Cortex
80	24	-37	77	0.41	3	Primary Somatosensory Cortex
				0.28	4	Primary Motor Cortex
				0.16	5	Somatosensory Association Cortex
				0.13	2	Primary Somatosensory Cortex
				0.02	1	Primary Somatosensory Cortex
81	46	-38	65	0.3	2	Primary Somatosensory Cortex
				0.3	40	Supramarginal gyrus part of Wernicke's area
				0.17	1	Primary Somatosensory Cortex
				0.13	5	Somatosensory Association Cortex
				0.1	3	Primary Somatosensory Cortex
82	63	-42	48	1	40	Supramarginal gyrus part of Wernicke's area
83	69	-43	26	0.75	40	Supramarginal gyrus part of Wernicke's area
				0.24	22	Superior Temporal Gyrus
				0.01	42	Primary and Auditory Association Cortex
84	71	-44	-1	0.68	21	Middle Temporal gyrus
				0.28	22	Superior Temporal Gyrus
				0.04	37	Fusiform gyrus
85	-66	-56	8	0.56	21	Middle Temporal gyrus
				0.38	22	Superior Temporal Gyrus
				0.03	39	Angular gyrus
				0.03	37	Fusiform gyrus

86	-62	-55	35	0.83	40	Supramarginal gyrus part of Wernicke's area
				0.17	39	Angular gyrus
87	-53	-53	53	1	40	Supramarginal gyrus part of Wernicke's area
88	-35	-52	70	0.49	5	Somatosensory Association Cortex
				0.41	7	Somatosensory Association Cortex
				0.08	40	Supramarginal gyrus part of Wernicke's area
				0.02	2	Primary Somatosensory Cortex
89	35	-51	70	0.49	5	Somatosensory Association Cortex
				0.46	7	Somatosensory Association Cortex
				0.03	2	Primary Somatosensory Cortex
				0.02	40	Supramarginal gyrus part of Wernicke's area
90	55	-53	54	1	40	Supramarginal gyrus part of Wernicke's area
91	63	-55	34	0.85	40	Supramarginal gyrus part of Wernicke's area
				0.15	39	Angular gyrus
92	66	-55	8	0.51	21	Middle Temporal gyrus
				0.4	22	Superior Temporal Gyrus
				0.05	39	Angular gyrus
				0.04	37	Fusiform gyrus
93	-60	-67	-6	0.61	37	Fusiform gyrus
				0.33	19	V3
				0.06	21	Middle Temporal gyrus
94	-57	-64	38	0.56	39	Angular gyrus; part of Wernicke's area
				0.44	40	Supramarginal gyrus part of Wernicke's area
95	-45	-64	54	0.54	40	Supramarginal gyrus part of Wernicke's area
				0.44	7	Somatosensory Association Cortex
				0.02	39	Angular gyrus
96	-26	-62	70	0.99	7	Somatosensory Association Cortex
				0.01	5	Somatosensory Association Cortex
97	26	-59	72	0.96	7	Somatosensory Association Cortex
				0.04	5	Somatosensory Association Cortex
98	46	-61	57	0.51	7	Somatosensory Association Cortex
				0.49	40	Supramarginal gyrus part of Wernicke's area
99	57	-65	38	0.67	39	Angular gyrus; part of Wernicke's area

				0.33	40	Supramarginal gyrus part of Wernicke's area
100	59	-68	-7	0.5	37	Fusiform gyrus
				0.49	19	V3
				0.01	21	Middle Temporal gyrus
101	-50	-75	37	0.9	39	Angular gyrus; part of Wernicke's area
				0.1	19	V3
102	-36	-73	54	0.96	7	Somatosensory Association Cortex
				0.04	19	V3
103	-15	-72	65	1	7	Somatosensory Association Cortex
104	16	-72	67	1	7	Somatosensory Association Cortex
105	34	-71	55	1	7	Somatosensory Association Cortex
106	49	-74	39	0.64	39	Angular gyrus; part of Wernicke's area
				0.3	19	V3
				0.05	7	Somatosensory Association Cortex
				0	40	Supramarginal gyrus part of Wernicke's area
107	-25	-81	51	0.82	7	Somatosensory Association Cortex
				0.18	19	V3
108	2	-78	56	1	7	Somatosensory Association Cortex
109	26	-79	52	0.94	7	Somatosensory Association Cortex
				0.06	19	V3
110	-14	-89	43	0.78	19	V3
				0.22	7	Somatosensory Association Cortex
111	17	-88	43	0.75	19	V3
				0.25	7	Somatosensory Association Cortex
112	-24	-93	28	0.97	19	V3
				0.03	18	Visual Association Cortex (V2)
113	1	-95	32	0.99	19	V3
				0.01	18	Visual Association Cortex (V2)
114	26	-92	30	0.99	19	V3
				0.01	18	Visual Association Cortex (V2)
115	-35	-95	12	0.57	19	V3
				0.43	18	Visual Association Cortex (V2)
116	-14	-102	18	0.74	18	Visual Association Cortex (V2)
				0.26	19	V3
117	17	-101	19	0.62	18	Visual Association Cortex (V2)
				0.38	19	V3
118	34	-94	13	0.67	19	V3
				0.33	18	Visual Association Cortex (V2)
119	-23	-102	6	0.85	18	Visual Association Cortex (V2)
				0.15	19	V3
120	-1	-102	11	1	18	Visual Association Cortex (V2)

121	24	-100	7	0.8	18	Visual Association Cortex (V2)
				0.2	19	V3
122	-15	-105	-1	0.95	18	Visual Association Cortex (V2)
				0.05	17	Primary Visual Cortex (V1)
123	16	-103	1	0.97	18	Visual Association Cortex (V2)
				0.03	17	Primary Visual Cortex (V1)
124	-24	-100	-11	0.76	18	Visual Association Cortex (V2)
				0.24	17	Primary Visual Cortex (V1)
125	-5	-103	-5	0.8	18	Visual Association Cortex (V2)
				0.2	17	Primary Visual Cortex (V1)
126	23	-99	-12	0.66	18	Visual Association Cortex (V2)
				0.34	17	Primary Visual Cortex (V1)
127	-17	-99	-18	0.56	17	Primary Visual Cortex (V1)
				0.44	18	Visual Association Cortex (V2)
128	15	-97	-17	0.53	17	Primary Visual Cortex (V1)
				0.47	18	Visual Association Cortex (V2)
129	-50	44	12	0.79	46	Dorsolateral prefrontal cortex
				0.12	10	Frontopolar area
				0.09	45	pars triangularis Broca's area
130	51	48	15	0.72	46	Dorsolateral prefrontal cortex
				0.27	10	Frontopolar area
				0	45	pars triangularis Broca's area
131	-48	-84	1	0.77	19	V3
				0.19	18	Visual Association Cortex (V2)
				0.04	37	Fusiform gyrus
132	48	-85	0	0.72	19	V3
				0.28	18	Visual Association Cortex (V2)
133	-49	-82	19	0.67	19	V3
				0.33	39	Angular gyrus
134	48	-82	19	0.82	19	V3
				0.18	39	Angular gyrus
				0.01	18	Visual Association Cortex (V2)

Supplementary Table 2. Whole-brain results for anticipated > unanticipated (anticipation phase) (stutterers only) (deoxyhemoglobin [HbR]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Supramarginal Gyrus		L	-62	-44	44	935	2.70	.0067	
Supramarginal Gyrus	40	L							.97
Visual Association Cortex (V3)		L	-44	-86	8	272	-3.04	.0031	
Visual Association Cortex (V3)	19	L							.74
Visual Association Cortex (V2)	18	L							.24
Visual Association Cortex (V2)		R	40	-74	28	246	-2.22	.0189	
Visual Association Cortex (V2)	18	R							.58
Visual Association Cortex (V3)	19	R							.42
Visual Association Cortex (V3)		R	40	-90	14	245	3.67	.0007	
Visual Association Cortex (V3)	19	R							.81
Visual Association Cortex (V2)	18	R							.18
Somatosensory Association Cortex		L	-8	-80	52	220	-2.40	.0128	
Somatosensory Association Cortex	7	L							.84
Visual Association Cortex (V3)	19	L							.16
Premotor/Supplementary Motor		R	14	-6	70	215	-2.28	.0167	
Premotor/Supplementary Motor	6	R							1.0
Dorsolateral Prefrontal Cortex		R	52	20	40	174	2.97	.0037	
Dorsolateral Prefrontal Cortex	9	R							.54
Frontal Eye Fields	8	R							.34
Frontopolar area		L	-20	48	24	128	-2.44	.0117	
Frontopolar area	10	L							.60
Dorsolateral Prefrontal Cortex	9	L							.40
Inferior prefrontal gyrus		L	-46	30	4	84	-2.16	.0214	
Inferior prefrontal gyrus	47	L							.48
Pars Triangularis (Broca's area)	45	L							.34
Dorsolateral Prefrontal Cortex	46	L							.16
Premotor/Supplementary Motor		R	54	-12	54	69	2.83	.0050	
Premotor/Supplementary Motor	6	R							.50
Primary Somatosensory Cortex	3	R							.23
Primary Motor Cortex	4	R							.11
Primary Somatosensory Cortex	1	R							.10
Premotor/Supplementary Motor		L	-42	8	60	66	2.75	.0059	
Premotor/Supplementary Motor	6	L							.82
Frontal Eye Fields	8	L							.18
Superior Temporal Gyrus		R	68	-40	4	63	2.64	.0077	
Superior Temporal Gyrus		R							.53
Middle Temporal Gyrus		R							.42
Supramarginal Gyrus		R	56	-36	52	61	2.70	.0067	
Supramarginal Gyrus	40	R							.72
Primary Somatosensory Cortex	2	R							.20

Supplementary Table 3. Whole-brain results for anticipated > unanticipated (anticipation phase) (stutterers only) (oxyhemoglobin [HbO). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Dorsolateral Prefrontal Cortex		L	-40	20	38	2528	-3.99	.0003	
Dorsolateral Prefrontal Cortex	9	L							.52
Frontal Eye Fields	8	L							.46
Dorsolateral Prefrontal Cortex		R	44	44	20	410	-3.62	.0008	
Dorsolateral Prefrontal Cortex	46	R							.63
Frontopolar area	10	R							.34
Premotor/Supplementary Motor		L	-36	6	60	265	4.10	.0003	
Premotor/Supplementary Motor	6	L							.90
Fusiform gyrus		R	60	-52	-16	121	2.26	.0174	
Fusiform Gyrus		R							.61
Inferior Temporal		R							.28
Middle Temporal Gyrus		R							.10
Premotor/Supplementary Motor		R	62	2	38	120	-2.97	.0037	
Premotor/Supplementary Motor	6	R							.72
Dorsolateral Prefrontal Cortex	9	R							.24
Dorsolateral Prefrontal Cortex		L	-22	52	38	105	2.91	.0042	
Dorsolateral Prefrontal Cortex	9	L							.63
Frontal Eye Fields	8	L							.20
Frontopolar area	10	L							.17
Premotor/Supplementary Motor		R	6	32	62	95	3.18	.0023	
Premotor/Supplementary Motor	6	R							.56
Frontal Eye Fields	8	R							.44
Visual Association Cortex (V3)		R	50	-66	-12	76	-2.29	.0161	
Visual Association Cortex (V3)	19	R							.64
Fusiform Gyrus	37	R							.28
Somatosensory Association Cortex		R	4	-84	50	75	2.60	.0084	
Somatosensory Association Cortex	7	R							.68
Visual Association Cortex (V3)	19	R							.32
Premotor/Supplementary Motor		L	-58	-16	46	74	-2.36	.0140	
Premotor/Supplementary Motor	6	L							.41
Primary Somatosensory Cortex	3	L							.20
Primary Somatosensory Cortex	1	L							.16
Primary Somatosensory Cortex	2	L							.15
Somatosensory Association Cortex		L	-22	-52	68	71	2.15	.0216	
Somatosensory Association Cortex	7	L							.66
Somatosensory Association Cortex	5	L							.31
Visual Association Cortex (V2)		R	12	-94	-14	60	-2.33	.0149	
Visual Association Cortex (V2)	18	R							.65
Primary Visual Cortex	17	R							.35
Premotor/Supplementary Motor		L	-2	-30	78	57	2.88	.0045	
Premotor/Supplementary Motor	6	L							.65
Primary Motor Cortex	4	L							.26
Visual Association Cortex (V2)		L	-24	-88	-22	53	2.08	.0249	
Visual Association Cortex (V2)	18	L							.84
Primary Visual Cortex	17	R							.16

Supplementary Table 4. Whole-brain results for stutterers > controls analysis (anticipation phase) (deoxyhemoglobin [HbR]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Frontal Eye Fields		R	26	22	54	6,016	3.41	.0007	
Frontal Eye Fields	8	R							.51
Premotor/Supplementary Motor	6	R							.49
V3		R	36	-82	8	1,074	-2.86	.0033	
Visual Association Cortex (V3)	19	R							.80
Visual Association Cortex (V2)	18	R							.20
Middle Temporal Gyrus		L	-64	-18	-10	425	2.57	.0069	
Middle Temporal Gyrus	21	L							.83
Somatosensory Association Cortex		L	-16	-56	74	396	2.75	.0044	
Somatosensory Association Cortex	7	L							.72
Somatosensory Association Cortex	5	L							.24
Visual Association Cortex (V3)		R	46	-74	-16	124	-2.45	.0093	
Visual Association Cortex (V3)	19	R							.65
Visual Association Cortex (V2)	18	R							.35
Supramarginal Gyrus		L	-52	-66	46	106	-2.23	.0157	
Supramarginal Gyrus	40	L							.46
Angular Gyrus	39	L							.37
Somatosensory Association Cortex	7	L							.13
Middle Temporal Gyrus		L	-68	-48	-8	103	2.68	.0052	
Middle Temporal Gyrus	21	L							.58
Fusiform Gyrus	37	L							.23
Inferior Temporal Gyrus	20	L							.12
Somatosensory Association Cortex		R	36	-70	56	70	2.97	.0025	
Somatosensory Association Cortex	7	R							.94

Supplementary Table 5. Whole-brain results for stutterers > controls analysis (anticipation phase) (oxyhemoglobin [HbO]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Somatosensory Association Cortex		R	18	-80	54	1,729	2.68	.0053	
Somatosensory Association Cortex	7	R							.87
Visual Association Cortex (V3)	19	R							.13
Premotor/Supplementary Motor		R	60	-2	42	800	-3.35	.0009	
Premotor/Supplementary Motor	6	R							.81
Inferior Temporal gyrus		R	60	-32	-20	428	2.13	.0194	
Inferior Temporal gyrus	20	R							.75
Middle Temporal gyrus	21	R							.24
Premotor/Supplementary Motor		L	-60	-20	46	390	-3.56	.0005	
Premotor/Supplementary Motor	6	L							.26
Primary Somatosensory Cortex	2	L							.23
Primary Somatosensory Cortex	3	L							.17
Primary Somatosensory Cortex	1	L							.16
Supramarginal Gyrus	40	L							.12
Frontopolar area		R	42	56	14	185	3.00	.0022	
Frontopolar area	10	R							.82
Dorsolateral Prefrontal Cortex	46	R							.18
Fusiform Gyrus		L	-58	-66	-12	147	-2.56	.0071	
Fusiform Gyrus	37	L							.48
Visual Association Cortex (V3)	19	L							.41
Visual Association Cortex (V2)		R	14	-94	10	90	2.35	.0117	
Visual Association Cortex (V2)	18	R							.91
Premotor/Supplementary Motor		L	-28	16	54	88	2.56	.0071	
Premotor/Supplementary Motor	6	L							.55
Frontal Eye Fields	8	L							.45
Angular Gyrus		L	-62	-62	12	84	-2.45	.0093	
Angular Gyrus	39	L							.26
Superior Temporal Gyrus	22	L							.25
Middle Temporal Gyrus	21	L							.24
Visual Association Cortex (V3)	19	L							.12
Fusiform Gyrus	37	L							.11
Dorsolateral Prefrontal Cortex		R	22	52	32	83	-2.10	.0211	
Dorsolateral Prefrontal Cortex	9	R							.64
Frontopolar area	10	R							.32
Visual Association Cortex (V2)		R	20	-102	-4	79	2.22	.0158	
Visual Association Cortex (V2)	18	R							.85
Primary Visual Cortex (V1)	17	R							.15
Fusiform Gyrus		R	60	-62	-14	62	-2.53	.0075	
Fusiform Gyrus	37	R							.54
Visual Association Cortex (V3)	19	R							.30
Inferior Temporal gyrus	20	R							.13

Supplementary Table 6. Whole-brain results for stuttered > fluent analysis (anticipation phase) (stutterers only) (deoxyhemoglobin [HbR]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Visual Association Cortex (V3)		L	-30	-86	22	1,383	3.12	.0026	
Visual Association Cortex (V3)	19	L							.99
Frontal Eye Fields		R	50	16	44	1,034	4.48	.0001	
Frontal Eye Fields	8	R							.41
Dorsolateral Prefrontal Cortex	9	R							.38
Pre-motor and Supplementary	6	R							.21
Pre-motor and Supplementary		L	-10	-32	76	308	-2.44	.0118	
Pre-motor and Supplementary	6	L							.43
Primary Motor Cortex	4	L							.30
Primary Somatosensory Cortex	3	L							.20
Superior Temporal Gyrus		R	68	-46	6	211	-3.11	.0027	
Superior Temporal Gyrus	22	R							.57
Middle Temporal Gyrus	21	R							.39
Superior Temporal Gyrus		L	-64	-56	14	169	-2.34	.0147	
Superior Temporal Gyrus	22	L							.42
Middle Temporal Gyrus	21	L							.23
Angular Gyrus	39	L							.17
Supramarginal Gyrus	40	L							.11
Supramarginal Gyrus		R	64	-38	40	136	2.69	.0068	
Supramarginal Gyrus	40	R							.88
Visual Association Cortex (V2)		L	-30	-86	.28	133	2.34	.0146	
Visual Association Cortex (V2)	18	L							1.0
Pre-motor and Supplementary		L	-22	22	64	86	3.50	.0010	
Pre-motor and Supplementary	6	L							.76
Frontal Eye Fields	8	L							.24

Supplementary Table 7. Whole-brain results for stuttered > fluent analysis (anticipation phase) (stutterers only) (oxyhemoglobin [HbO]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Somatosensory Association Cortex		R	28	-42	70	4,310	4.67	.0001	
Somatosensory Association Cortex	5	R							.33
Primary Somatosensory Cortex	3	R							.23
Somatosensory Association Cortex	7	R							.17
Primary Somatosensory Cortex	2	R							.14
Pre-motor and Supplementary		R	66	-16	38	965	-2.87	.0046	
Pre-motor and Supplementary	6	R							.40
Primary Somatosensory cortex	1	R							.17
Primary Somatosensory Cortex	2	R							.14
Primary Somatosensory Cortex	3	R							.14
Middle Temporal gyrus		L	-60	-18	-14	573	2.51	.0103	
Middle Temporal gyrus	21	L							.81
Inferior Temporal gyrus	20	L							.19
Frontal Eye Fields		L	-8	44	54	341	-2.45	.0116	
Frontal Eye Fields	8	L							.90
Pre-motor and Supplementary		L	-40	4	62	304	-3.42	.0013	
Pre-motor and Supplementary	6	L							.95
Dorsolateral Prefrontal Cortex		L	-26	36	34	193	-2.26	.0174	
Dorsolateral Prefrontal Cortex		L							.66
Frontal Eye Fields		L							.34
Frontopolar area		R	26	64	42	161	-2.46	.0113	
Frontopolar area	10	R							1.0
Visual Association Cortex (V2)		R	8	-102	-6	154	-2.93	.0040	
Visual Association Cortex (V2)	18	R							.83
Primary Visual Cortex (V1)	17	R							.17
Frontopolar area		R	34	58	10	153	-2.12	.0229	
Frontopolar area	10	R							.98
Dorsolateral Prefrontal Cortex		R	46	36	22	63	-1.96	.0316	
Dorsolateral Prefrontal Cortex	46	R							.84
Dorsolateral Prefrontal Cortex		L	-54	15	40	57	-3.39	.0014	
Dorsolateral Prefrontal Cortex	9	L							.52
Pre-motor and Supplementary	6	L							.24
Frontal Eye Fields	8	L							.22
Frontal Eye Fields		R	32	34	52	51	2.51	.0102	
Frontal Eye Fields	8	R							.89

Figure

Table 1. Participant data. Treatment history descriptions written as reported by participants. TS = Trials Stuttered; SAS = Stuttering Anticipation Scale, extent score out of 100, 0 = "never," 100 = "always." M = mean; SD = standard deviation.

ID	Age	Gender	Treatment history	%TS	SAS
1	29	F	On and off in elementary school	43%	50
2	35	F	7 years	36%	100
3	23	M	2 weeks (intensive program)	0%	76
4	34	M	6 years	0%	79
5	26	F	3 years	87%	95
6	48	F	Roughly 2 years	80%	87
7	21	M	8 years	27%	90
8	23	M	2.5 years	23%	77
9	29	M	9 months	45%	80
10	34	M	2 years	98%	80
11	37	F	Off and on for years	23%	75
12	39	F	12-15 years	76%	86
13	23	F	6+ years	93%	89
14	47	F	None	30%	98
15	42	M	8 years	15%	90
16	18	M	10 years, on and off	70%	75
17	30	M	approx. 10 years	51%	76
18	29	M	about 7 years	25%	67
19	39	F	about 6 months	55%	76
20	25	M	About 7 years (on and off)	21%	70
21	22	M	None	55%	77
22	50	F	3 months	15%	99
M	32			40%	81
SD	9			30%	12

Figure 1. Task timeline. The question/sentence completion was presented auditorily. The anticipation period was five seconds; participants looked at a fixation cross during this time. The green screen signaled participants to produce the word. Participants again looked at the cross during the rest period.

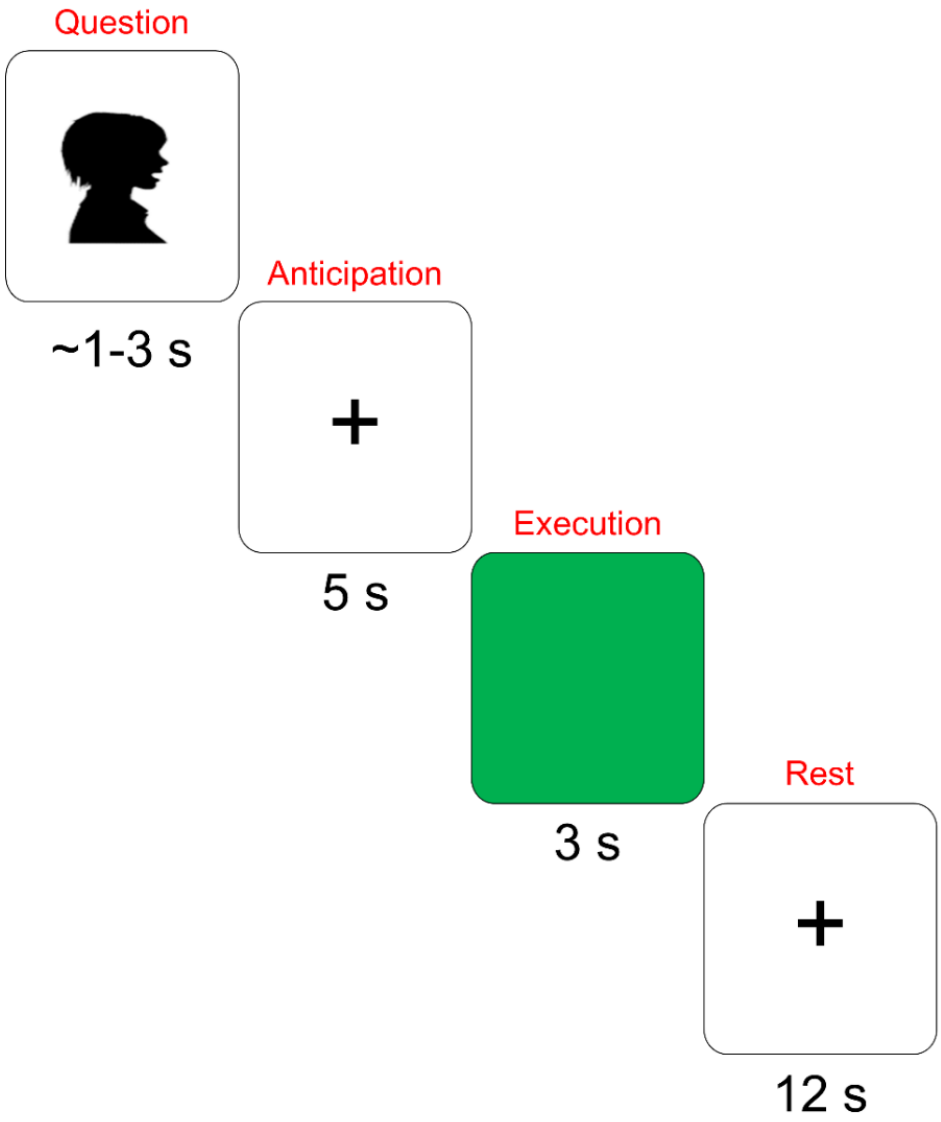


Table 2. Reaction time means and standard deviations, as determined by first movement. ID = participant; SD = standard deviation.

ID	Fluent	SD	Ambiguous	SD	Stuttered	SD
1	352.14	103.71	286.03	69.08	297.39	108.48
2	362.68	115.64	307.75	101.19	368.22	97.08
3	398.99	126.61	300.33	168.84		
4	311.76	120.47	467.18	47.19		
5						
6	386.14	90.07	517.24	50.61	458.53	109.47
7	341.00	187.38	406.51	132.49	272.23	87.77
8	310.22	72.77	296.29	60.60	300.33	62.43
9	370.68	109.21	433.81	129.24	324.43	104.50
10					282.50	81.94
11	302.33	75.50	361.51	114.57	367.07	144.78
12	333.70	100.11	305.89	64.95	262.28	94.69
13	360.40	148.11	300.33	75.91	303.49	69.47
14	480.53	147.94	524.39	195.26	560.34	154.65
15	327.57	115.33	322.58	70.79	294.77	111.89
16	290.32	99.55	340.85	68.24	282.73	69.12
17	281.56	87.84	302.18	84.48	330.44	140.16
18	421.30	132.68	446.17	208.43	361.51	134.58
19	515.72	122.92	517.24	306.75	489.97	134.20
20	323.34	76.61	260.29	27.92	288.55	110.64
21	304.64	160.21	133.48	47.19	285.78	97.98
22	362.01	125.17	310.76	147.09	367.07	95.85
	358.90		352.49		336.10	

Table 3. Stuttering response type by participant, including percentages. ID = participant.

ID	Trials	Errors	Unambiguously fluent (0)	%	Ambiguous (1)	%	Unambiguously stuttered (2)	%	Anticipated words that were unambiguously stuttered	%
1	80	1	38	49%	7	9%	34	43%	26	65%
2	80	4	38	53%	9	11%	29	36%	25	63%
3	80	4	70	93%	6	8%	0	0%	0	0%
4	80	3	75	98%	2	3%	0	0%	0	0%
5	60	1	2	4%	5	6%	52	87%	29	97%
6	70	3	7	10%	6	8%	54	80%	30	89%
7	70	1	38	49%	12	15%	19	27%	17	49%
8	80	1	27	35%	34	43%	18	23%	11	28%
9	80	0	37	46%	7	9%	36	45%	24	60%
10	80	2	0	3%	0	0%	78	98%	40	100%
11	80	0	50	63%	12	15%	18	23%	7	18%
12	80	1	7	9%	12	15%	60	76%	30	78%
13	80	0	5	6%	1	1%	74	93%	38	95%
14	80	1	47	60%	8	10%	24	30%	20	50%
15	80	9	49	73%	10	13%	12	15%	11	28%
16	80	0	10	13%	14	18%	56	70%	34	85%
17	80	0	18	23%	21	26%	41	51%	24	60%
18	80	1	30	39%	29	36%	20	25%	11	28%
19	80	4	33	43%	2	3%	41	55%	33	90%
20	80	0	58	73%	5	6%	17	21%	10	25%
21	80	5	33	41%	2	4%	40	55%	24	68%
22	60	2	33	43%	16	21%	9	15%	6	20%
			736		222		742		458	
	% across dataset		43.30%		13.10%		43.60%		53.88%	

Table 3. Stuttering response type (cont.)

ID	Unanticipated words that were unambiguously stuttered	%	Anticipated words that were unambiguously fluent	%	Unanticipated words that were unambiguously fluent	%	Anticipated words that were ambiguous	%	Unanticipated words that were ambiguous	%
1	8	20%	13	33%	25	65%	1	3%	6	15%
2	4	10%	10	30%	28	75%	3	8%	6	15%
3	0	0%	38	98%	32	88%	1	3%	5	13%
4	0	0%	38	98%	37	98%	1	3%	1	3%
5	23	77%	0	3%	2	7%	0	0%	5	17%
6	24	71%	1	3%	6	20%	3	9%	3	9%
7	2	6%	11	34%	27	77%	6	17%	6	17%
8	7	18%	13	35%	14	35%	15	38%	19	48%
9	12	30%	13	33%	24	60%	3	8%	4	10%
10	38	95%	0	0%	0	5%	0	0%	0	0%
11	11	28%	27	68%	23	58%	6	15%	6	15%
12	30	75%	0	0%	7	18%	9	23%	3	8%
13	36	90%	2	5%	3	8%	0	0%	1	3%
14	4	10%	16	40%	31	80%	4	10%	4	10%
15	1	3%	20	58%	29	88%	6	15%	4	10%
16	22	55%	2	5%	8	20%	4	10%	10	25%
17	17	43%	8	20%	10	25%	8	20%	13	33%
18	9	23%	15	38%	15	40%	14	35%	15	38%
19	8	20%	3	10%	30	75%	0	0%	2	5%
20	7	18%	28	70%	30	75%	2	5%	3	8%

21	16	43%	13	33%	20	50%	0	0%	2	8%
22	3	10%	19	63%	14	50%	5	17%	11	40%
	284		301		435		91		131	
	33.41%		35.41%		51.18%		10.71%		15.41%	

Figure 2A-C. Region of interest analysis. (A) ROI mask for right dorsolateral prefrontal cortex (R-DLPFC) [58 26 38]. A 10- millimeter sphere was created using the MarsBar toolbox (Brett et al., 2002) and the xjView toolbox (https:// www.alivelearn.net/xjview). The image was then projected onto the template using BrainNet Viewer (Xia, M.,Wang, J., & He,Y. (2013). BrainNet Viewer: a network visualization tool for human brain connectomics. PloS one, 8(7), e68910). (B-C) Event-related averages of activity for the stuttering group within the R-DLPFC ROI for anticipated vs. unanticipated words, for Oxyhemoglobin (HbO) and DeOxyhemoglobin (HbR), respectively. 0 is the beginning of the anticipation phase, and 30 seconds is shown because the hemodynamic response could feasibly last this long. HbR signals prior to sign reversal such that decreasing values reflect increases in activation strength. Shading represents standard error of the mean.

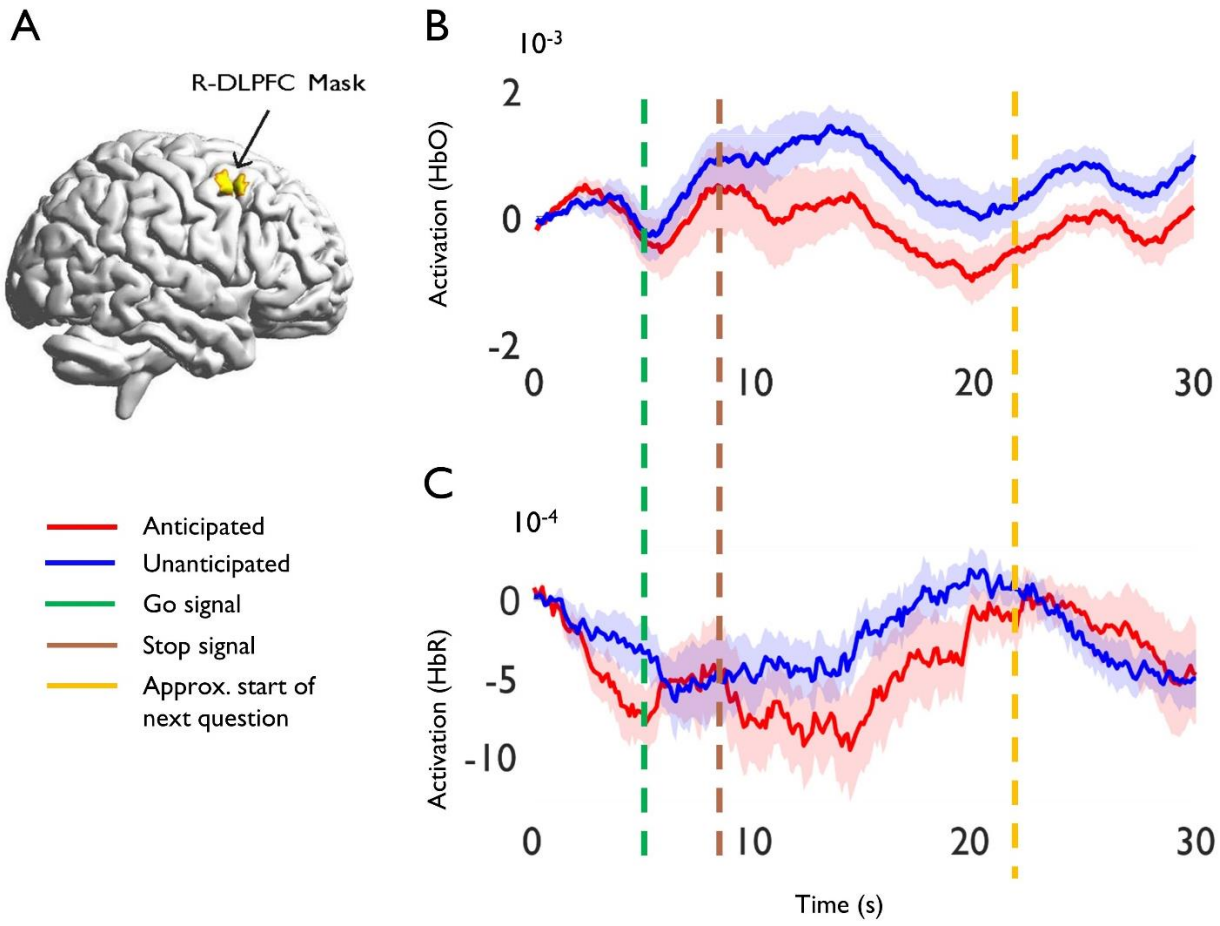


Figure 3. Correlation between activation and reaction time (ms) in R-DLPFC (approached significance. Deoxyhemoglobin (HbR) signals prior to sign reversal such that decreasing values reflect increases in activation strength.

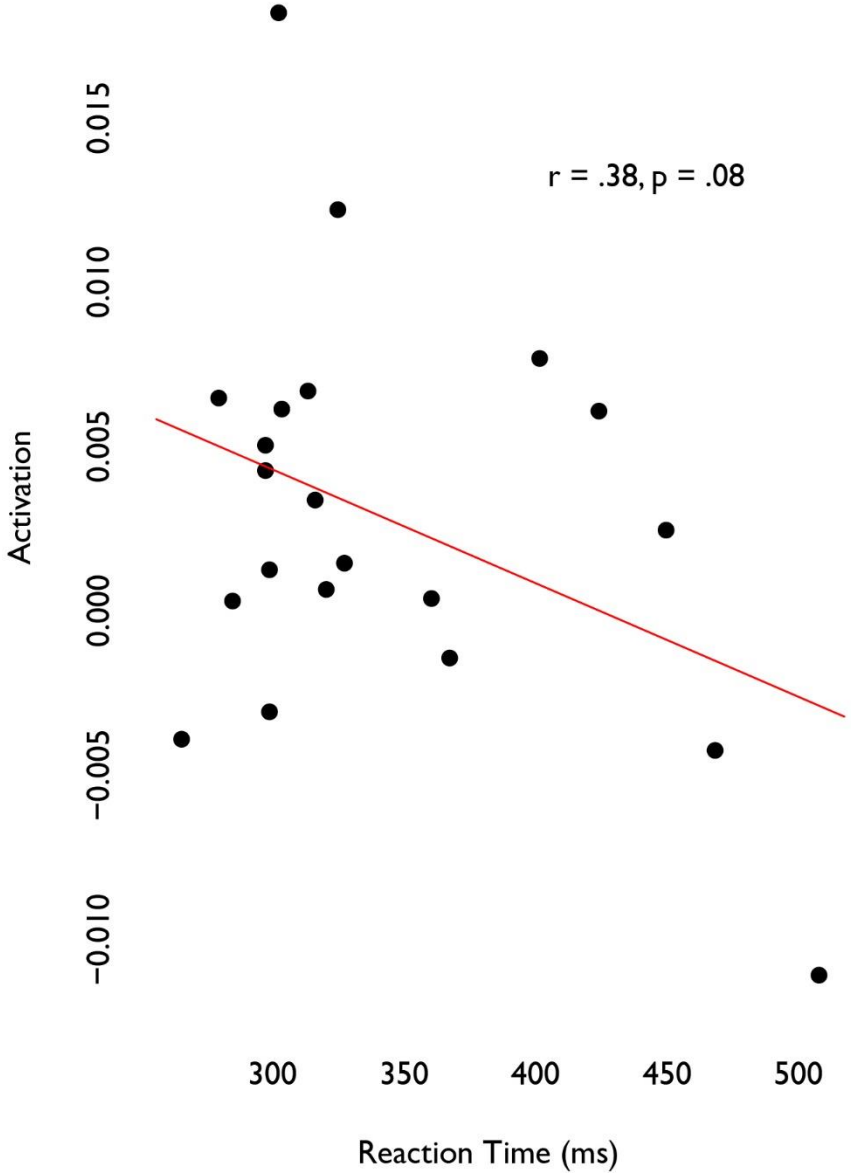
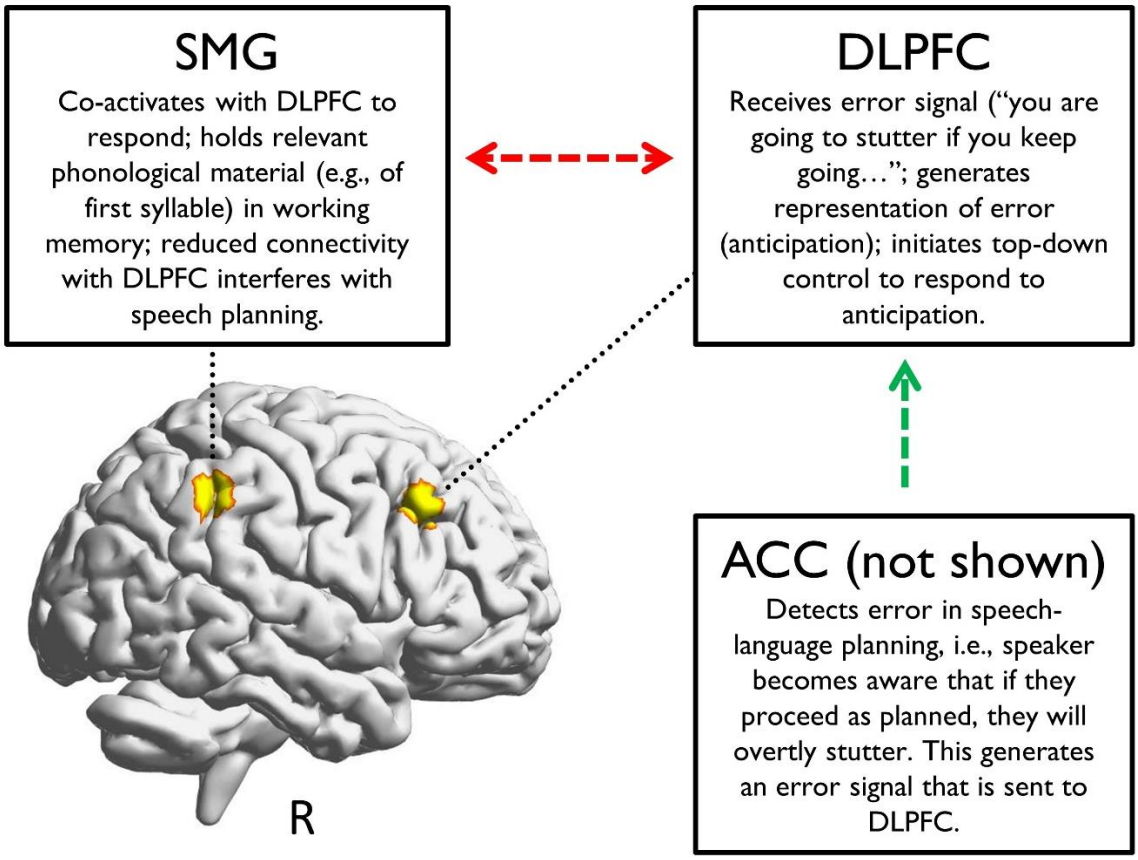
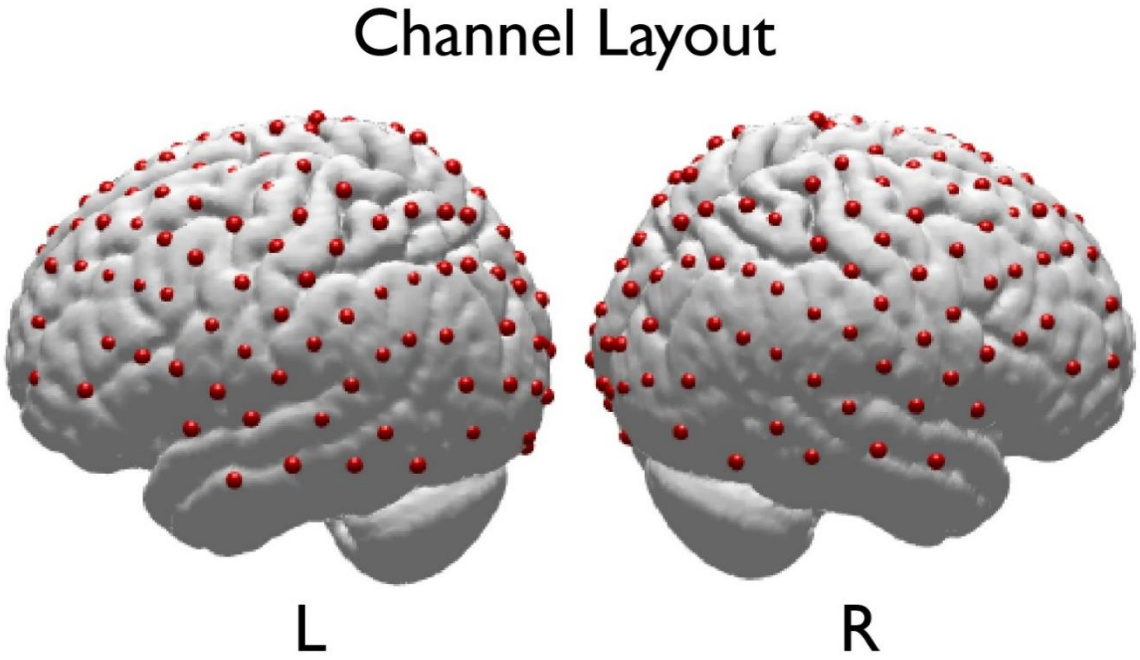


Figure 4. A model of stuttering anticipation. Anticipation develops via associative learning. Memory traces of associations between overtly stuttered utterances and environmental/listener reactions form. ACC detects upcoming stuttering via error-likelihood monitoring, and signals R-DLPFC which initiates cognitive control to respond to anticipation, thereby elevating activation in R-DLPFC. R-DLPFC initiates activation within the broader FPN to support the response. Atypical connection between R-DLPFC and areas within the FPN, such as SMG, impedes the speaker's ability to adaptively respond to anticipation. ACC – anterior cingulate cortex; R-DLPFC = right dorsolateral prefrontal cortex; FPN = frontoparietal control network; R-SMG = right supramarginal gyrus.



SUPPLEMENTARY MATERIAL

Supplementary Figure 1. Right (R) and left (L) hemispheres of rendered brains illustrate average locations (red circles) for the 134 channels per participant. Montreal Neurological Institute (MNI) coordinates were determined by digitizing the locations of the optodes in relation to the 10–20 system based on conventional landmarks. See Supplementary Table 1 for group median coordinates, anatomical regions, and atlas-based probabilities for each channel.



Supplementary Table 1. Group median coordinates, atlas-based probabilities, Brodmann's areas, and anatomical regions for each channel. Montreal Neurological Institute (MNI) coordinates for each of the 134 channels per participant were determined by digitizing the locations of the optodes in relation to the 10-20 system based on conventional landmarks.

Channel number	MNI coordinates			Probability	BA	Anatomical region
	X	Y	Z			
1	-37	61	9	0.97	10	Frontopolar area
				0.03	46	Dorsolateral prefrontal cortex
2	38	63	10	1	10	Frontopolar area
3	-26	62	24	0.96	10	Frontopolar area
				0.04	9	Dorsolateral prefrontal cortex
4	26	64	26	0.94	10	Frontopolar area
				0.06	9	Dorsolateral prefrontal cortex
5	-13	58	39	0.83	9	Dorsolateral prefrontal cortex
				0.14	10	Frontopolar area
				0.03	8	Includes Frontal eye fields
6	13	59	39	0.81	9	Dorsolateral prefrontal cortex
				0.17	10	Frontopolar area
				0.02	8	Includes Frontal eye fields
7	-23	48	44	0.54	9	Dorsolateral prefrontal cortex
				0.46	8	Includes Frontal eye fields
8	-1	51	48	0.7	8	Includes Frontal eye fields
				0.3	9	Dorsolateral prefrontal cortex
9	22	50	46	0.58	8	Includes Frontal eye fields
				0.42	9	Dorsolateral prefrontal cortex
10	-49	37	29	0.88	46	Dorsolateral prefrontal cortex
				0.12	9	Dorsolateral prefrontal cortex
11	-33	38	46	0.7	8	Includes Frontal eye fields
				0.3	9	Dorsolateral prefrontal cortex
12	-12	41	55	0.98	8	Includes Frontal eye fields
				0.02	6	Pre-Motor and Supplementary Motor Cortex
13	12	42	56	0.97	8	Includes Frontal eye fields
				0.03	6	Pre-Motor and Supplementary Motor Cortex
14	33	40	47	0.77	8	Includes Frontal eye fields
				0.23	9	Dorsolateral prefrontal cortex
15	50	39	32	0.66	46	Dorsolateral prefrontal cortex
				0.34	9	Dorsolateral prefrontal cortex
				0.01	10	Frontopolar area
16	-55	25	27	0.39	46	Dorsolateral prefrontal cortex

				0.32	9	Dorsolateral prefrontal cortex
				0.29	45	pars triangularis Broca's area
				0	44	pars opercularis
17	-43	26	47	0.82	8	Includes Frontal eye fields
				0.18	9	Dorsolateral prefrontal cortex
18	-22	29	59	0.6	8	Includes Frontal eye fields
				0.4	6	Pre-Motor and Supplementary Motor Cortex
19	-1	33	60	0.52	8	Includes Frontal eye fields
				0.48	6	Pre-Motor and Supplementary Motor Cortex
20	22	32	59	0.6	8	Includes Frontal eye fields
				0.4	6	Pre-Motor and Supplementary Motor Cortex
21	43	29	49	0.87	8	Includes Frontal eye fields
				0.13	9	Dorsolateral prefrontal cortex
22	56	29	30	0.54	46	Dorsolateral prefrontal cortex
				0.35	9	Dorsolateral prefrontal cortex
				0.11	45	pars triangularis Broca's area
23	-62	8	5	0.5	22	Superior Temporal Gyrus
				0.3	44	pars opercularis
				0.17	6	Pre-Motor and Supplementary Motor Cortex
				0.03	45	pars triangularis Broca's area
24	-60	13	26	0.51	9	Dorsolateral prefrontal cortex
				0.22	45	pars triangularis Broca's area
				0.2	44	pars opercularis
				0.07	6	Pre-Motor and Supplementary Motor Cortex
25	-49	17	47	0.64	8	Includes Frontal eye fields
				0.2	9	Dorsolateral prefrontal cortex
				0.15	6	Pre-Motor and Supplementary Motor Cortex
26	-35	19	61	0.52	8	Includes Frontal eye fields
				0.48	6	Pre-Motor and Supplementary Motor Cortex
27	-14	22	67	1	6	PreMotor and Supplementary Motor Cortex
28	13	23	67	1	6	PreMotor and Supplementary Motor Cortex
29	34	21	61	0.52	6	PreMotor and Supplementary Motor Cortex
				0.48	8	Includes Frontal eye fields
30	50	19	49	0.74	8	Includes Frontal eye fields
				0.14	9	Dorsolateral prefrontal cortex

				0.12	6	Pre-Motor and Supplementary Motor Cortex
31	62	17	28	0.6	9	Dorsolateral prefrontal cortex
				0.27	45	pars triangularis Broca's area
				0.08	44	pars opercularis
				0.04	46	Dorsolateral prefrontal cortex
32	62	13	8	0.43	44	pars opercularis; part of Broca's area
				0.23	45	pars triangularis Broca's area
				0.22	22	Superior Temporal Gyrus
				0.08	6	Pre-Motor and Supplementary Motor Cortex
				0.04	47	Inferior prefrontal gyrus
33	-68	-7	-11	1	21	Middle Temporal gyrus
34	-65	-1	20	0.58	6	PreMotor and Supplementary Motor Cortex
				0.17	43	Subcentral area
				0.08	22	Superior Temporal Gyrus
				0.08	4	Primary Motor Cortex
				0.06	44	pars opercularis
				0.03	9	Dorsolateral prefrontal cortex
				0.01	45	pars triangularis Broca's area
35	-59	2	41	0.86	6	PreMotor and Supplementary Motor Cortex
				0.11	9	Dorsolateral prefrontal cortex
				0.03	8	Includes Frontal eye fields
36	-44	9	59	0.87	6	PreMotor and Supplementary Motor Cortex
				0.13	8	Includes Frontal eye fields
37	-24	10	69	1	6	PreMotor and Supplementary Motor Cortex
38	-2	11	71	1	6	PreMotor and Supplementary Motor Cortex
39	23	11	70	1	6	PreMotor and Supplementary Motor Cortex
40	45	9	59	0.82	6	PreMotor and Supplementary Motor Cortex
				0.18	8	Includes Frontal eye fields
41	60	5	43	0.68	6	PreMotor and Supplementary Motor Cortex
				0.21	9	Dorsolateral prefrontal cortex
				0.11	8	Includes Frontal eye fields
42	67	4	22	0.62	6	PreMotor and Supplementary Motor Cortex
				0.13	9	Dorsolateral prefrontal cortex
				0.1	44	pars opercularis
				0.07	45	pars triangularis Broca's area
				0.06	4	Primary Motor Cortex
				0.02	43	Subcentral area
43	68	-2	-8	0.85	21	Middle Temporal gyrus
				0.15	22	Superior Temporal Gyrus
44	-68	-12	12	0.35	42	Primary and Auditory Association Cortex
				0.33	43	Subcentral area

				0.3	22	Superior Temporal Gyrus
				0.02	40	Supramarginal gyrus part of Wernicke's area
				0	4	Primary Motor Cortex
45	-65	-10	35	0.64	6	PreMotor and Supplementary Motor Cortex
				0.15	3	Primary Somatosensory Cortex
				0.11	4	Primary Motor Cortex
				0.09	1	Primary Somatosensory Cortex
				0.01	2	Primary Somatosensory Cortex
46	-54	-8	55	0.58	6	PreMotor and Supplementary Motor Cortex
				0.27	3	Primary Somatosensory Cortex
				0.14	4	Primary Motor Cortex
				0.01	1	Primary Somatosensory Cortex
47	-35	-4	67	1	6	PreMotor and Supplementary Motor Cortex
48	-14	-2	75	1	6	PreMotor and Supplementary Motor Cortex
49	13	-1	75	1	6	PreMotor and Supplementary Motor Cortex
50	34	-3	68	1	6	PreMotor and Supplementary Motor Cortex
51	54	-5	55	0.72	6	PreMotor and Supplementary Motor Cortex
				0.14	3	Primary Somatosensory Cortex
				0.13	4	Primary Motor Cortex
52	67	-6	37	0.96	6	PreMotor and Supplementary Motor Cortex
				0.03	4	Primary Motor Cortex
				0.01	3	Primary Somatosensory Cortex
53	70	-9	15	0.38	43	Subcentral area
				0.25	22	Superior Temporal Gyrus
				0.22	42	Primary and Auditory Association Cortex
				0.08	6	Pre-Motor and Supplementary Motor Cortex
				0.07	4	Primary Motor Cortex
				0	40	Supramarginal gyrus part of Wernicke's area
54	-71	-26	-3	0.7	21	Middle Temporal gyrus
				0.22	22	Superior Temporal Gyrus
				0.08	42	Primary and Auditory Association Cortex
55	-68	-22	28	0.35	40	Supramarginal gyrus part of Wernicke's area
				0.24	2	Primary Somatosensory Cortex
				0.18	1	Primary Somatosensory Cortex
				0.11	43	Subcentral area
				0.08	3	Primary Somatosensory Cortex
				0.02	6	Pre-Motor and Supplementary Motor Cortex
				0.02	42	Primary and Auditory Association Cortex

56	-62	-22	48	0.33	2	Primary Somatosensory Cortex
				0.25	1	Primary Somatosensory Cortex
				0.19	6	Pre-Motor and Supplementary Motor Cortex
				0.14	3	Primary Somatosensory Cortex
				0.08	4	Primary Motor Cortex
57	-46	-19	65	0.47	3	Primary Somatosensory Cortex
				0.25	6	Pre-Motor and Supplementary Motor Cortex
				0.15	4	Primary Motor Cortex
				0.13	1	Primary Somatosensory Cortex
				0	2	Primary Somatosensory Cortex
58	-24	-15	75	0.97	6	PreMotor and Supplementary Motor Cortex
				0.03	4	Primary Motor Cortex
59	-2	-12	75	1	6	PreMotor and Supplementary Motor Cortex
60	24	-13	75	1	6	PreMotor and Supplementary Motor Cortex
61	46	-16	65	0.46	6	PreMotor and Supplementary Motor Cortex
				0.32	3	Primary Somatosensory Cortex
				0.19	4	Primary Motor Cortex
				0.02	1	Primary Somatosensory Cortex
62	63	-20	49	0.26	1	Primary Somatosensory Cortex
				0.22	3	Primary Somatosensory Cortex
				0.22	2	Primary Somatosensory Cortex
				0.21	6	Pre-Motor and Supplementary Motor Cortex
				0.09	4	Primary Motor Cortex
				0.01	40	Supramarginal gyrus part of Wernicke's area
63	70	-20	30	0.26	40	Supramarginal gyrus part of Wernicke's area
				0.21	2	Primary Somatosensory Cortex
				0.2	1	Primary Somatosensory Cortex
				0.16	3	Primary Somatosensory Cortex
				0.07	6	Pre-Motor and Supplementary Motor Cortex
				0.07	43	Subcentral area
				0.01	4	Primary Motor Cortex
64	73	-22	0	0.42	21	Middle Temporal gyrus
				0.4	22	Superior Temporal Gyrus
				0.18	42	Primary and Auditory Association Cortex
65	-69	-36	14	0.75	22	Superior Temporal Gyrus
				0.21	42	Primary and Auditory Association Cortex

				0.04	40	Supramarginal gyrus part of Wernicke's area
66	-66	-33	40	0.78	40	Supramarginal gyrus part of Wernicke's area
				0.16	2	Primary Somatosensory Cortex
				0.06	1	Primary Somatosensory Cortex
67	-54	-29	57	0.41	2	Primary Somatosensory Cortex
				0.37	40	Supramarginal gyrus part of Wernicke's area
				0.2	1	Primary Somatosensory Cortex
				0.02	3	Primary Somatosensory Cortex
68	-37	-26	72	0.33	6	PreMotor and Supplementary Motor Cortex
				0.3	3	Primary Somatosensory Cortex
				0.3	4	Primary Motor Cortex
				0.07	1	Primary Somatosensory Cortex
				0.01	2	Primary Somatosensory Cortex
69	-14	-25	79	0.64	6	PreMotor and Supplementary Motor Cortex
				0.34	4	Primary Motor Cortex
				0.02	3	Primary Somatosensory Cortex
70	14	-25	79	0.72	6	PreMotor and Supplementary Motor Cortex
				0.28	4	Primary Motor Cortex
				0	3	Primary Somatosensory Cortex
71	36	-25	73	0.43	4	Primary Motor Cortex
				0.38	6	Pre-Motor and Supplementary Motor Cortex
				0.18	3	Primary Somatosensory Cortex
				0.02	1	Primary Somatosensory Cortex
72	55	-29	59	0.35	40	Supramarginal gyrus part of Wernicke's area
				0.3	2	Primary Somatosensory Cortex
				0.24	1	Primary Somatosensory Cortex
				0.1	3	Primary Somatosensory Cortex
73	68	-31	42	0.62	40	Supramarginal gyrus part of Wernicke's area
				0.19	1	Primary Somatosensory Cortex
				0.16	2	Primary Somatosensory Cortex
				0.02	3	Primary Somatosensory Cortex
				0.01	4	Primary Motor Cortex
74	71	-32	16	0.42	22	Superior Temporal Gyrus
				0.36	42	Primary and Auditory Association Cortex
				0.22	40	Supramarginal gyrus part of Wernicke's area
75	-69	-47	-3	0.76	21	Middle Temporal gyrus

				0.19	22	Superior Temporal Gyrus
				0.05	37	Fusiform gyrus
76	-67	-45	26	0.76	40	Supramarginal gyrus part of Wernicke's area
				0.24	22	Superior Temporal Gyrus
77	-61	-43	47	1	40	Supramarginal gyrus part of Wernicke's area
78	-45	-38	65	0.37	40	Supramarginal gyrus part of Wernicke's area
				0.32	2	Primary Somatosensory Cortex
				0.18	1	Primary Somatosensory Cortex
				0.11	5	Somatosensory Association Cortex
				0.03	3	Primary Somatosensory Cortex
79	-23	-39	75	0.4	3	Primary Somatosensory Cortex
				0.3	5	Somatosensory Association Cortex
				0.16	2	Primary Somatosensory Cortex
				0.1	4	Primary Motor Cortex
				0.03	1	Primary Somatosensory Cortex
80	24	-37	77	0.41	3	Primary Somatosensory Cortex
				0.28	4	Primary Motor Cortex
				0.16	5	Somatosensory Association Cortex
				0.13	2	Primary Somatosensory Cortex
				0.02	1	Primary Somatosensory Cortex
81	46	-38	65	0.3	2	Primary Somatosensory Cortex
				0.3	40	Supramarginal gyrus part of Wernicke's area
				0.17	1	Primary Somatosensory Cortex
				0.13	5	Somatosensory Association Cortex
				0.1	3	Primary Somatosensory Cortex
82	63	-42	48	1	40	Supramarginal gyrus part of Wernicke's area
83	69	-43	26	0.75	40	Supramarginal gyrus part of Wernicke's area
				0.24	22	Superior Temporal Gyrus
				0.01	42	Primary and Auditory Association Cortex
84	71	-44	-1	0.68	21	Middle Temporal gyrus
				0.28	22	Superior Temporal Gyrus
				0.04	37	Fusiform gyrus
85	-66	-56	8	0.56	21	Middle Temporal gyrus
				0.38	22	Superior Temporal Gyrus
				0.03	39	Angular gyrus
				0.03	37	Fusiform gyrus

86	-62	-55	35	0.83	40	Supramarginal gyrus part of Wernicke's area
				0.17	39	Angular gyrus
87	-53	-53	53	1	40	Supramarginal gyrus part of Wernicke's area
88	-35	-52	70	0.49	5	Somatosensory Association Cortex
				0.41	7	Somatosensory Association Cortex
				0.08	40	Supramarginal gyrus part of Wernicke's area
				0.02	2	Primary Somatosensory Cortex
89	35	-51	70	0.49	5	Somatosensory Association Cortex
				0.46	7	Somatosensory Association Cortex
				0.03	2	Primary Somatosensory Cortex
				0.02	40	Supramarginal gyrus part of Wernicke's area
90	55	-53	54	1	40	Supramarginal gyrus part of Wernicke's area
91	63	-55	34	0.85	40	Supramarginal gyrus part of Wernicke's area
				0.15	39	Angular gyrus
92	66	-55	8	0.51	21	Middle Temporal gyrus
				0.4	22	Superior Temporal Gyrus
				0.05	39	Angular gyrus
				0.04	37	Fusiform gyrus
93	-60	-67	-6	0.61	37	Fusiform gyrus
				0.33	19	V3
				0.06	21	Middle Temporal gyrus
94	-57	-64	38	0.56	39	Angular gyrus; part of Wernicke's area
				0.44	40	Supramarginal gyrus part of Wernicke's area
95	-45	-64	54	0.54	40	Supramarginal gyrus part of Wernicke's area
				0.44	7	Somatosensory Association Cortex
				0.02	39	Angular gyrus
96	-26	-62	70	0.99	7	Somatosensory Association Cortex
				0.01	5	Somatosensory Association Cortex
97	26	-59	72	0.96	7	Somatosensory Association Cortex
				0.04	5	Somatosensory Association Cortex
98	46	-61	57	0.51	7	Somatosensory Association Cortex
				0.49	40	Supramarginal gyrus part of Wernicke's area
99	57	-65	38	0.67	39	Angular gyrus; part of Wernicke's area

				0.33	40	Supramarginal gyrus part of Wernicke's area
100	59	-68	-7	0.5	37	Fusiform gyrus
				0.49	19	V3
				0.01	21	Middle Temporal gyrus
101	-50	-75	37	0.9	39	Angular gyrus; part of Wernicke's area
				0.1	19	V3
102	-36	-73	54	0.96	7	Somatosensory Association Cortex
				0.04	19	V3
103	-15	-72	65	1	7	Somatosensory Association Cortex
104	16	-72	67	1	7	Somatosensory Association Cortex
105	34	-71	55	1	7	Somatosensory Association Cortex
106	49	-74	39	0.64	39	Angular gyrus; part of Wernicke's area
				0.3	19	V3
				0.05	7	Somatosensory Association Cortex
				0	40	Supramarginal gyrus part of Wernicke's area
107	-25	-81	51	0.82	7	Somatosensory Association Cortex
				0.18	19	V3
108	2	-78	56	1	7	Somatosensory Association Cortex
109	26	-79	52	0.94	7	Somatosensory Association Cortex
				0.06	19	V3
110	-14	-89	43	0.78	19	V3
				0.22	7	Somatosensory Association Cortex
111	17	-88	43	0.75	19	V3
				0.25	7	Somatosensory Association Cortex
112	-24	-93	28	0.97	19	V3
				0.03	18	Visual Association Cortex (V2)
113	1	-95	32	0.99	19	V3
				0.01	18	Visual Association Cortex (V2)
114	26	-92	30	0.99	19	V3
				0.01	18	Visual Association Cortex (V2)
115	-35	-95	12	0.57	19	V3
				0.43	18	Visual Association Cortex (V2)
116	-14	-102	18	0.74	18	Visual Association Cortex (V2)
				0.26	19	V3
117	17	-101	19	0.62	18	Visual Association Cortex (V2)
				0.38	19	V3
118	34	-94	13	0.67	19	V3
				0.33	18	Visual Association Cortex (V2)
119	-23	-102	6	0.85	18	Visual Association Cortex (V2)
				0.15	19	V3
120	-1	-102	11	1	18	Visual Association Cortex (V2)

121	24	-100	7	0.8	18	Visual Association Cortex (V2)
				0.2	19	V3
122	-15	-105	-1	0.95	18	Visual Association Cortex (V2)
				0.05	17	Primary Visual Cortex (V1)
123	16	-103	1	0.97	18	Visual Association Cortex (V2)
				0.03	17	Primary Visual Cortex (V1)
124	-24	-100	-11	0.76	18	Visual Association Cortex (V2)
				0.24	17	Primary Visual Cortex (V1)
125	-5	-103	-5	0.8	18	Visual Association Cortex (V2)
				0.2	17	Primary Visual Cortex (V1)
126	23	-99	-12	0.66	18	Visual Association Cortex (V2)
				0.34	17	Primary Visual Cortex (V1)
127	-17	-99	-18	0.56	17	Primary Visual Cortex (V1)
				0.44	18	Visual Association Cortex (V2)
128	15	-97	-17	0.53	17	Primary Visual Cortex (V1)
				0.47	18	Visual Association Cortex (V2)
129	-50	44	12	0.79	46	Dorsolateral prefrontal cortex
				0.12	10	Frontopolar area
				0.09	45	pars triangularis Broca's area
130	51	48	15	0.72	46	Dorsolateral prefrontal cortex
				0.27	10	Frontopolar area
				0	45	pars triangularis Broca's area
131	-48	-84	1	0.77	19	V3
				0.19	18	Visual Association Cortex (V2)
				0.04	37	Fusiform gyrus
132	48	-85	0	0.72	19	V3
				0.28	18	Visual Association Cortex (V2)
133	-49	-82	19	0.67	19	V3
				0.33	39	Angular gyrus
134	48	-82	19	0.82	19	V3
				0.18	39	Angular gyrus
				0.01	18	Visual Association Cortex (V2)

Supplementary Table 2. Whole-brain results for anticipated > unanticipated (anticipation phase) (stutterers only) (deoxyhemoglobin [HbR]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Supramarginal Gyrus		L	-62	-44	44	935	2.70	.0067	
Supramarginal Gyrus	40	L							.97
Visual Association Cortex (V3)		L	-44	-86	8	272	-3.04	.0031	
Visual Association Cortex (V3)	19	L							.74
Visual Association Cortex (V2)	18	L							.24
Visual Association Cortex (V2)		R	40	-74	28	246	-2.22	.0189	
Visual Association Cortex (V2)	18	R							.58
Visual Association Cortex (V3)	19	R							.42
Visual Association Cortex (V3)		R	40	-90	14	245	3.67	.0007	
Visual Association Cortex (V3)	19	R							.81
Visual Association Cortex (V2)	18	R							.18
Somatosensory Association Cortex		L	-8	-80	52	220	-2.40	.0128	
Somatosensory Association Cortex	7	L							.84
Visual Association Cortex (V3)	19	L							.16
Premotor/Supplementary Motor		R	14	-6	70	215	-2.28	.0167	
Premotor/Supplementary Motor	6	R							1.0
Dorsolateral Prefrontal Cortex		R	52	20	40	174	2.97	.0037	
Dorsolateral Prefrontal Cortex	9	R							.54
Frontal Eye Fields	8	R							.34
Frontopolar area		L	-20	48	24	128	-2.44	.0117	
Frontopolar area	10	L							.60
Dorsolateral Prefrontal Cortex	9	L							.40
Inferior prefrontal gyrus		L	-46	30	4	84	-2.16	.0214	
Inferior prefrontal gyrus	47	L							.48
Pars Triangularis (Broca's area)	45	L							.34
Dorsolateral Prefrontal Cortex	46	L							.16
Premotor/Supplementary Motor		R	54	-12	54	69	2.83	.0050	
Premotor/Supplementary Motor	6	R							.50
Primary Somatosensory Cortex	3	R							.23
Primary Motor Cortex	4	R							.11
Primary Somatosensory Cortex	1	R							.10
Premotor/Supplementary Motor		L	-42	8	60	66	2.75	.0059	
Premotor/Supplementary Motor	6	L							.82
Frontal Eye Fields	8	L							.18
Superior Temporal Gyrus		R	68	-40	4	63	2.64	.0077	
Superior Temporal Gyrus		R							.53
Middle Temporal Gyrus		R							.42
Supramarginal Gyrus		R	56	-36	52	61	2.70	.0067	
Supramarginal Gyrus	40	R							.72
Primary Somatosensory Cortex	2	R							.20

Supplementary Table 3. Whole-brain results for anticipated > unanticipated (anticipation phase) (stutterers only) (oxyhemoglobin [HbO). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Dorsolateral Prefrontal Cortex		L	-40	20	38	2528	-3.99	.0003	
Dorsolateral Prefrontal Cortex	9	L							.52
Frontal Eye Fields	8	L							.46
Dorsolateral Prefrontal Cortex		R	44	44	20	410	-3.62	.0008	
Dorsolateral Prefrontal Cortex	46	R							.63
Frontopolar area	10	R							.34
Premotor/Supplementary Motor		L	-36	6	60	265	4.10	.0003	
Premotor/Supplementary Motor	6	L							.90
Fusiform gyrus		R	60	-52	-16	121	2.26	.0174	
Fusiform Gyrus		R							.61
Inferior Temporal		R							.28
Middle Temporal Gyrus		R							.10
Premotor/Supplementary Motor		R	62	2	38	120	-2.97	.0037	
Premotor/Supplementary Motor	6	R							.72
Dorsolateral Prefrontal Cortex	9	R							.24
Dorsolateral Prefrontal Cortex		L	-22	52	38	105	2.91	.0042	
Dorsolateral Prefrontal Cortex	9	L							.63
Frontal Eye Fields	8	L							.20
Frontopolar area	10	L							.17
Premotor/Supplementary Motor		R	6	32	62	95	3.18	.0023	
Premotor/Supplementary Motor	6	R							.56
Frontal Eye Fields	8	R							.44
Visual Association Cortex (V3)		R	50	-66	-12	76	-2.29	.0161	
Visual Association Cortex (V3)	19	R							.64
Fusiform Gyrus	37	R							.28
Somatosensory Association Cortex		R	4	-84	50	75	2.60	.0084	
Somatosensory Association Cortex	7	R							.68
Visual Association Cortex (V3)	19	R							.32
Premotor/Supplementary Motor		L	-58	-16	46	74	-2.36	.0140	
Premotor/Supplementary Motor	6	L							.41
Primary Somatosensory Cortex	3	L							.20
Primary Somatosensory Cortex	1	L							.16
Primary Somatosensory Cortex	2	L							.15
Somatosensory Association Cortex		L	-22	-52	68	71	2.15	.0216	
Somatosensory Association Cortex	7	L							.66
Somatosensory Association Cortex	5	L							.31
Visual Association Cortex (V2)		R	12	-94	-14	60	-2.33	.0149	
Visual Association Cortex (V2)	18	R							.65
Primary Visual Cortex	17	R							.35
Premotor/Supplementary Motor		L	-2	-30	78	57	2.88	.0045	
Premotor/Supplementary Motor	6	L							.65
Primary Motor Cortex	4	L							.26
Visual Association Cortex (V2)		L	-24	-88	-22	53	2.08	.0249	
Visual Association Cortex (V2)	18	L							.84
Primary Visual Cortex	17	R							.16

Supplementary Table 4. Whole-brain results for stutterers > controls analysis (anticipation phase) (deoxyhemoglobin [HbR]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Frontal Eye Fields		R	26	22	54	6,016	3.41	.0007	
Frontal Eye Fields	8	R							.51
Premotor/Supplementary Motor	6	R							.49
V3		R	36	-82	8	1,074	-2.86	.0033	
Visual Association Cortex (V3)	19	R							.80
Visual Association Cortex (V2)	18	R							.20
Middle Temporal Gyrus		L	-64	-18	-10	425	2.57	.0069	
Middle Temporal Gyrus	21	L							.83
Somatosensory Association Cortex		L	-16	-56	74	396	2.75	.0044	
Somatosensory Association Cortex	7	L							.72
Somatosensory Association Cortex	5	L							.24
Visual Association Cortex (V3)		R	46	-74	-16	124	-2.45	.0093	
Visual Association Cortex (V3)	19	R							.65
Visual Association Cortex (V2)	18	R							.35
Supramarginal Gyrus		L	-52	-66	46	106	-2.23	.0157	
Supramarginal Gyrus	40	L							.46
Angular Gyrus	39	L							.37
Somatosensory Association Cortex	7	L							.13
Middle Temporal Gyrus		L	-68	-48	-8	103	2.68	.0052	
Middle Temporal Gyrus	21	L							.58
Fusiform Gyrus	37	L							.23
Inferior Temporal Gyrus	20	L							.12
Somatosensory Association Cortex		R	36	-70	56	70	2.97	.0025	
Somatosensory Association Cortex	7	R							.94

Supplementary Table 5. Whole-brain results for stutterers > controls analysis (anticipation phase) (oxyhemoglobin [HbO]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Somatosensory Association Cortex		R	18	-80	54	1,729	2.68	.0053	
Somatosensory Association Cortex	7	R							.87
Visual Association Cortex (V3)	19	R							.13
Premotor/Supplementary Motor		R	60	-2	42	800	-3.35	.0009	
Premotor/Supplementary Motor	6	R							.81
Inferior Temporal gyrus		R	60	-32	-20	428	2.13	.0194	
Inferior Temporal gyrus	20	R							.75
Middle Temporal gyrus	21	R							.24
Premotor/Supplementary Motor		L	-60	-20	46	390	-3.56	.0005	
Premotor/Supplementary Motor	6	L							.26
Primary Somatosensory Cortex	2	L							.23
Primary Somatosensory Cortex	3	L							.17
Primary Somatosensory Cortex	1	L							.16
Supramarginal Gyrus	40	L							.12
Frontopolar area		R	42	56	14	185	3.00	.0022	
Frontopolar area	10	R							.82
Dorsolateral Prefrontal Cortex	46	R							.18
Fusiform Gyrus		L	-58	-66	-12	147	-2.56	.0071	
Fusiform Gyrus	37	L							.48
Visual Association Cortex (V3)	19	L							.41
Visual Association Cortex (V2)		R	14	-94	10	90	2.35	.0117	
Visual Association Cortex (V2)	18	R							.91
Premotor/Supplementary Motor		L	-28	16	54	88	2.56	.0071	
Premotor/Supplementary Motor	6	L							.55
Frontal Eye Fields	8	L							.45
Angular Gyrus		L	-62	-62	12	84	-2.45	.0093	
Angular Gyrus	39	L							.26
Superior Temporal Gyrus	22	L							.25
Middle Temporal Gyrus	21	L							.24
Visual Association Cortex (V3)	19	L							.12
Fusiform Gyrus	37	L							.11
Dorsolateral Prefrontal Cortex		R	22	52	32	83	-2.10	.0211	
Dorsolateral Prefrontal Cortex	9	R							.64
Frontopolar area	10	R							.32
Visual Association Cortex (V2)		R	20	-102	-4	79	2.22	.0158	
Visual Association Cortex (V2)	18	R							.85
Primary Visual Cortex (V1)	17	R							.15
Fusiform Gyrus		R	60	-62	-14	62	-2.53	.0075	
Fusiform Gyrus	37	R							.54
Visual Association Cortex (V3)	19	R							.30
Inferior Temporal gyrus	20	R							.13

Supplementary Table 6. Whole-brain results for stuttered > fluent analysis (anticipation phase) (stutterers only) (deoxyhemoglobin [HbR]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Visual Association Cortex (V3)		L	-30	-86	22	1,383	3.12	.0026	
Visual Association Cortex (V3)	19	L							.99
Frontal Eye Fields		R	50	16	44	1,034	4.48	.0001	
Frontal Eye Fields	8	R							.41
Dorsolateral Prefrontal Cortex	9	R							.38
Pre-motor and Supplementary	6	R							.21
Pre-motor and Supplementary		L	-10	-32	76	308	-2.44	.0118	
Pre-motor and Supplementary	6	L							.43
Primary Motor Cortex	4	L							.30
Primary Somatosensory Cortex	3	L							.20
Superior Temporal Gyrus		R	68	-46	6	211	-3.11	.0027	
Superior Temporal Gyrus	22	R							.57
Middle Temporal Gyrus	21	R							.39
Superior Temporal Gyrus		L	-64	-56	14	169	-2.34	.0147	
Superior Temporal Gyrus	22	L							.42
Middle Temporal Gyrus	21	L							.23
Angular Gyrus	39	L							.17
Supramarginal Gyrus	40	L							.11
Supramarginal Gyrus		R	64	-38	40	136	2.69	.0068	
Supramarginal Gyrus	40	R							.88
Visual Association Cortex (V2)		L	-30	-86	.28	133	2.34	.0146	
Visual Association Cortex (V2)	18	L							1.0
Pre-motor and Supplementary		L	-22	22	64	86	3.50	.0010	
Pre-motor and Supplementary	6	L							.76
Frontal Eye Fields	8	L							.24

Supplementary Table 7. Whole-brain results for stuttered > fluent analysis (anticipation phase) (stutterers only) (oxyhemoglobin [HbO]). Regions on first line indicate locations of peak within cluster; indented lines represent other regions within cluster (Prob = probability, or the relative contributions of each region within the cluster). Positive t-values indicate anticipated > unanticipated; negative t-values indicate anticipated < unanticipated. Hemi = hemisphere; MNI = Montreal Neurological Institute.

Region	Brodmann Area	Hemi	Peak MNI			Voxels	t	p	Prob
			x	y	z				
Somatosensory Association Cortex		R	28	-42	70	4,310	4.67	.0001	
Somatosensory Association Cortex	5	R							.33
Primary Somatosensory Cortex	3	R							.23
Somatosensory Association Cortex	7	R							.17
Primary Somatosensory Cortex	2	R							.14
Pre-motor and Supplementary		R	66	-16	38	965	-2.87	.0046	
Pre-motor and Supplementary	6	R							.40
Primary Somatosensory cortex	1	R							.17
Primary Somatosensory Cortex	2	R							.14
Primary Somatosensory Cortex	3	R							.14
Middle Temporal gyrus		L	-60	-18	-14	573	2.51	.0103	
Middle Temporal gyrus	21	L							.81
Inferior Temporal gyrus	20	L							.19
Frontal Eye Fields		L	-8	44	54	341	-2.45	.0116	
Frontal Eye Fields	8	L							.90
Pre-motor and Supplementary		L	-40	4	62	304	-3.42	.0013	
Pre-motor and Supplementary	6	L							.95
Dorsolateral Prefrontal Cortex		L	-26	36	34	193	-2.26	.0174	
Dorsolateral Prefrontal Cortex		L							.66
Frontal Eye Fields		L							.34
Frontopolar area		R	26	64	42	161	-2.46	.0113	
Frontopolar area	10	R							1.0
Visual Association Cortex (V2)		R	8	-102	-6	154	-2.93	.0040	
Visual Association Cortex (V2)	18	R							.83
Primary Visual Cortex (V1)	17	R							.17
Frontopolar area		R	34	58	10	153	-2.12	.0229	
Frontopolar area	10	R							.98
Dorsolateral Prefrontal Cortex		R	46	36	22	63	-1.96	.0316	
Dorsolateral Prefrontal Cortex	46	R							.84
Dorsolateral Prefrontal Cortex		L	-54	15	40	57	-3.39	.0014	
Dorsolateral Prefrontal Cortex	9	L							.52
Pre-motor and Supplementary	6	L							.24
Frontal Eye Fields	8	L							.22
Frontal Eye Fields		R	32	34	52	51	2.51	.0102	
Frontal Eye Fields	8	R							.89

Figure1

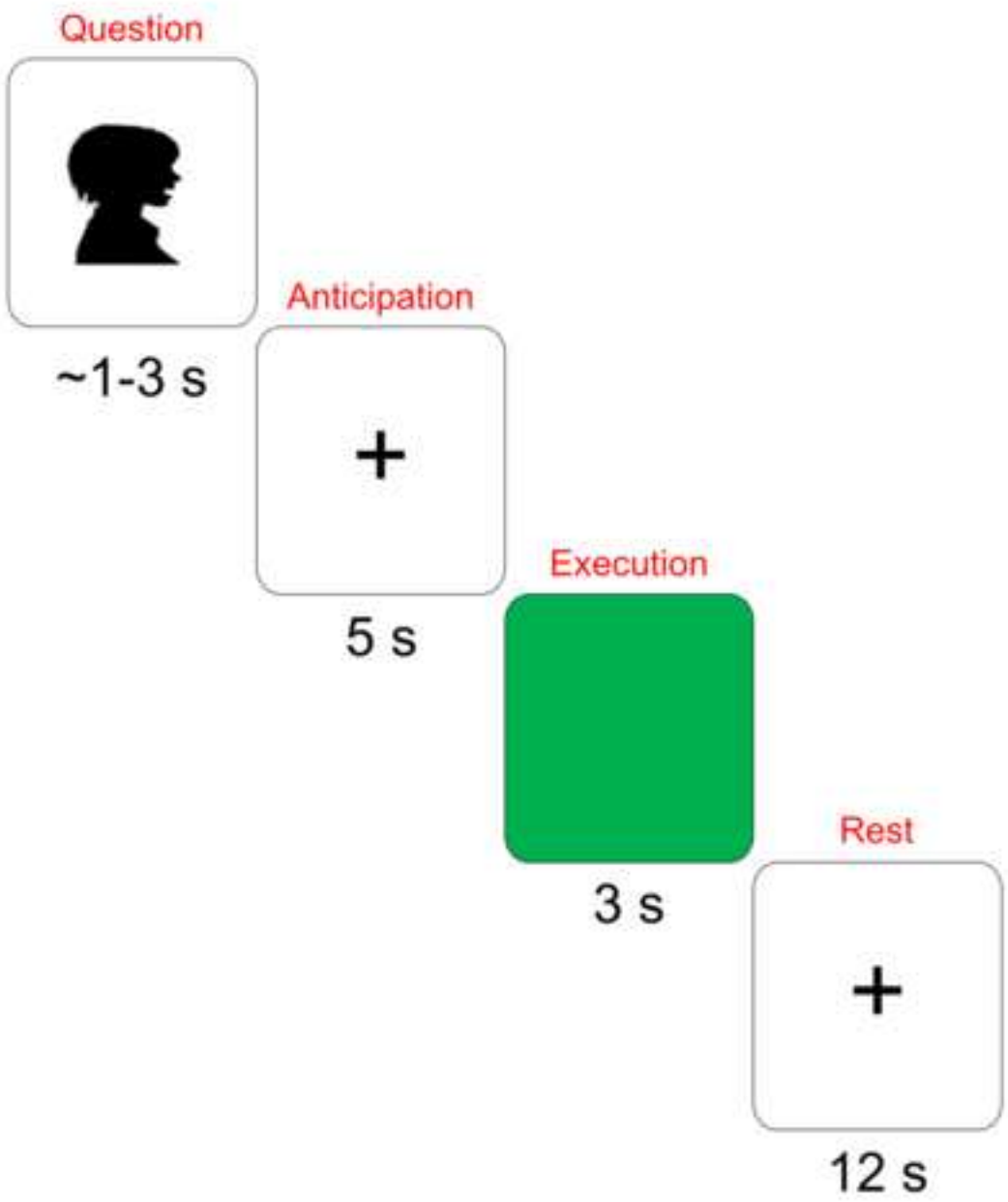


Figure2

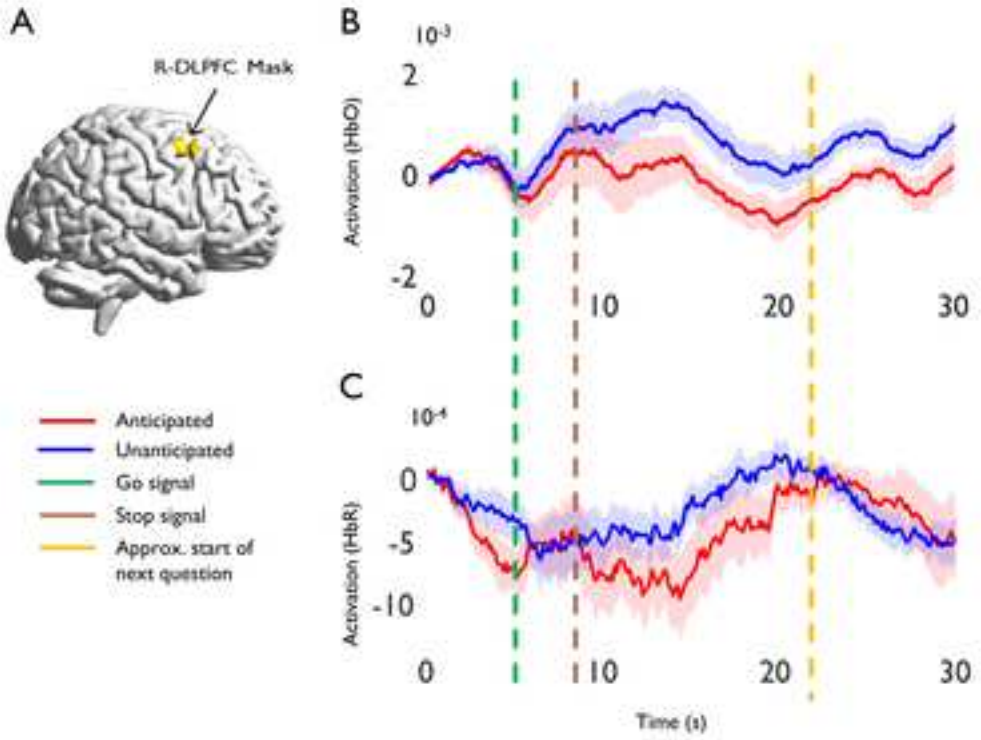


Figure3

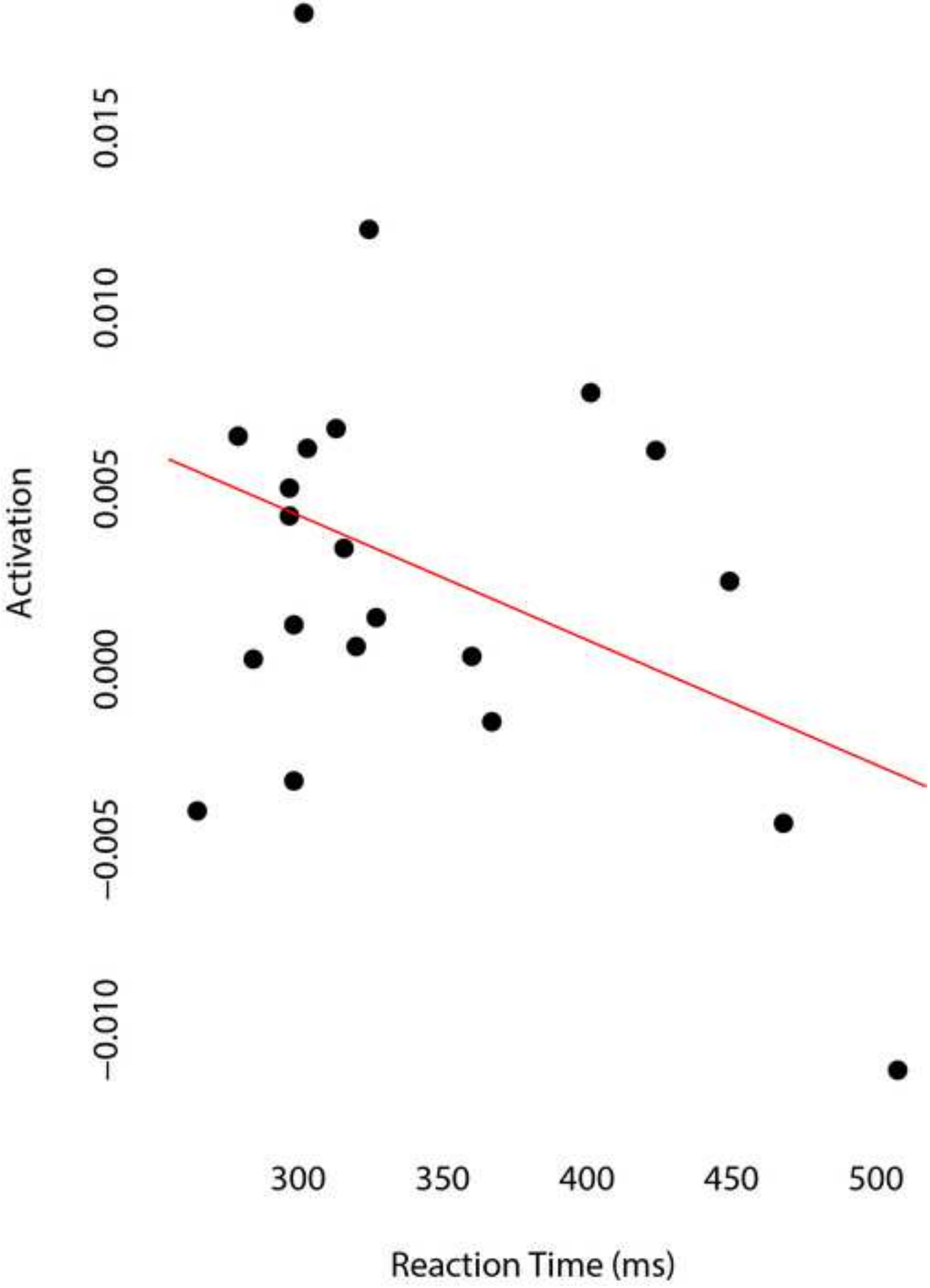


Figure4