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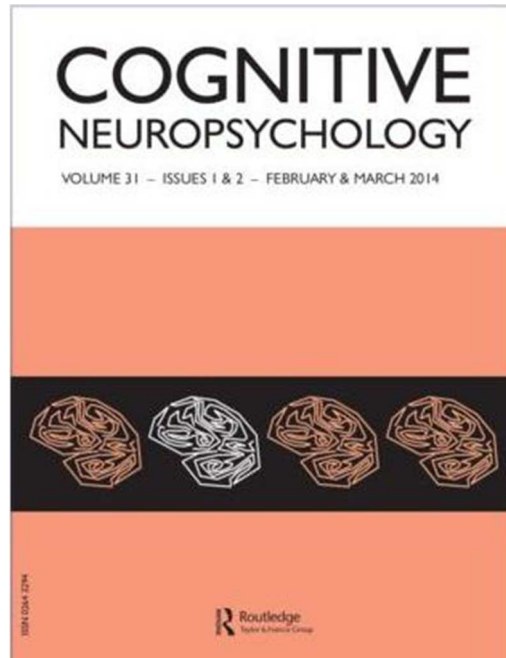
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Lesions to the Left Lateral Prefrontal Cortex Impair Decision Threshold Adjustment for Lexical Selection

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12 for Lexical Selection
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Abstract

Patients with lesions in the left prefrontal cortex (PFC) have been shown to be impaired in lexical selection, especially when interference between semantically-related alternatives is increased. To more deeply investigate which computational mechanisms may be impaired following left PFC damage due to stroke, a psychometric modeling approach is employed in which we assess the cognitive parameters of the patients from an evidence accumulation (sequential information sampling) modeling of their response data. We also compare the results to healthy speakers. Analysis of the cognitive parameters indicates that decision threshold adjustment is impaired for patients to appropriately handle the increased item difficulty that is introduced by semantic interference. Also, the modeling contributes to other topics in psycholinguistic theory, in which specific effects are observed on the cognitive parameters according to: item familiarization, and the opposing effects of priming (lower threshold) and semantic interference (lower drift) which are found to depend on repetition. These results are developed for the blocked-cyclic picture naming paradigm, in which pictures are presented within semantically-homogeneous (HOM) or heterogeneous (HET) blocks, and are repeated several times per block. Overall, the results are in agreement with a role of the left PFC in adjusting the decision threshold for lexical selection in language production.

Keywords: lexical selection, stroke patients, psychometric models for patient analysis, evidence accumulation, picture naming, prefrontal cortex

Lesions to the Left Lateral Prefrontal Cortex Impair Decision Threshold Adjustment for Lexical Selection

Introduction

Lexical selection may be broadly defined as the process of selecting a lexical target from a number of alternatives, such as when one names an object or a concept. Patients with chronic left prefrontal cortex (PFC) damage due to stroke often have lexical selection impairments. In particular, it has been proposed that these patients have difficulties in overcoming interference caused by semantically-related alternatives in lexical selection (e.g., Schnur et al., 2006, 2009; Riès et al., 2014). However, the mechanism by which the left PFC operates to help overcome interference between semantically-related alternatives is unclear. In this study, we use evidence accumulation modeling to investigate which computational mechanism may be impaired in left PFC patients when performing a lexical selection task.

The left PFC, and especially the left inferior frontal gyrus, have been proposed to play a role in resolving interference between semantically-related alternatives for lexical selection (e.g., Thompson-Schill et al., 1997, 1998; Schnur et al., 2006, 2009; Riès et al., 2014). This has been shown in both picture naming and verb generation tasks. For example in the blocked cyclic picture naming task (Damian et al., 2001), left PFC patients are particularly impaired in naming pictures when presented in semantically-homogeneous (HOM) versus heterogeneous (HET) blocks (e.g., Schnur et al., 2006, 2009). In this paradigm, pictures are repeated several times per block and performance is typically impaired in HOM versus HET blocks from the second presentation onwards, leading to a semantic interference effect. This semantic interference effect has been shown to be larger in left PFC patients compared to controls, which has been interpreted as being due to a greater difficulty for these patients to overcome interference between semantically-related alternatives.

Different mechanisms have been suggested to underlie the capacity of the left PFC in resolving this interference, including a “booster” mechanism helping to tease lexical representation apart (Oppenheim et al., 2010), a top-down mechanism allowing to bias the levels of activation of the task-relevant alternatives (Belke & Stielow, 2013), or more generally a proactive interference control mechanism proposed to operate across cognitive domains (Jonides & Nee, 2006; Kan & Thompson-Schill, 2004; Riès et al., 2014). However, additional developments are needed for the assessment of these mechanisms, as none of them have yet been integrated into quantitative models that can for example, rigorously analyze PFC patient data at the response time (RT) distributional level. In this paper, we demonstrate such a development: using evidence accumulation as a means to quantitatively analyze PFC patient data at this level of granularity, and so far it most closely provides a means to implement, or further research, a style of the “booster” mechanism suggested by Oppenheim et al. (2010, p.8) in PFC patient performance data.

This project builds upon recent work by Anders et al. (2015), who in the context of traditional psycholinguistic theories (e.g., see Chen & Mirman, 2012; Howard et al., 2006; Levelt et al., 1999; Oppenheim et al., 2010), advanced a theory that evidence accumulation may be an underlying component of the lexical selection process (see also Roelofs, 1992; Van Maanen & Van Rijn, 2007). Furthermore with experimental data, they newly demonstrate how the cognitive parameters of psychometric applications of evidence accumulation models can be used to progress psycholinguistic theory. Evidence accumulation (also known as sequential sampling) may be defined as an information processing paradigm for how behav-

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iors are selected in the context of time. Particularly, the paradigm postulates that behaviors, or processing choices across cognitive domains, are enabled through a sequential sampling of signal information (with noise at each sample), up until a sufficient triggering threshold is reached (Busemeyer & Townsend, 1992, 1993). Such sequential sampling approaches that are aimed to model signal interpretation, have experienced continued support since their beginning conception in the 1960s (Stone, 1960; Laming, 1968; Gerstein & Mandelbrot, 1964; Ratcliff, 1978) in both theoretical (e.g., simulation exploration) and real data applications of experimental psychology (e.g., Anders et al., 2016; Buckley & Gillman, 1974; Donkin & Van Maanen, 2014; Mulder et al., 2013; Ratcliff et al., 1999, 2004; Ratcliff & McKoon, 2008), as well as neuroscience research (e.g., Dehaene, 2008; Kelly & O'Connell, 2013; O'Connell et al., 2012; Mulder et al., 2014).

The evidence accumulation approach allows for a number of advantages. Specifically, the approach can provide cognitive psychometrics (Batchelder, 1998; Riefer et al., 2002) and quantitative modeling contributions for observed RT data that can be tied to existent psycholinguistic theory. Furthermore, the observed RT distributions can be simultaneously measured with the approach (e.g., see Anders et al., 2016), providing advancements over classical measurement (mean, s.d.) approaches (Luce, 1986; Balota & Yap, 2011). Evidence accumulation can be implemented to analyze lexical selection, by quantitatively modeling the appropriate experimental tasks that can elicit a minimally-biased (e.g., from language-neutral stimuli) lexical selection procedure, such as a sequential picture naming task (Cattell, 1886; Carroll & White, 1973; Oldfield & Wingfield, 1964) previously discussed.

In summary, Anders et al. (2015) provide the first application of evidence accumulation to the domain of lexical selection (in healthy speakers). Then in this study, we provide a first application of the approach to the domain of PFC patients performing lexical selection. Furthermore we examine replicability of the previous findings for healthy speakers. The paper is organized as follows. First, a description of the evidence accumulation paradigm and the psychometric model used to analyze the data is developed. Then the methods are outlined for the picture-naming experiment that contains the PFC patients and healthy speakers. Next, specifics are detailed about how the proposed cognitive-behavioral modeling is applied: fitting methods are outlined, and appropriate model fit is assessed, according to proper correspondence with the observed data. Then the model analysis results are interpreted in relation to the underlying cognitive mechanisms. Finally, the paper concludes with the general discussion.

Evidence Accumulation for Lexical Selection

In this section, we acquaint the reader with the paradigm of evidence accumulation, and specifically a model previously-proposed to analyze lexical selection in the context of picture-naming, the shifted Wald model (SWM, Anders et al., 2016; Folks & Chhikara, 1978; Heathcote, 2004; Luce, 1986; Ricciardi, 1977). The SWM, which involves only one accumulator with one threshold, and positive drift rates, could be considered a canonical, simplest case of racing accumulation that models the activation of the lexical target spoken, and not the alternatives (see Anders et al., 2015). Racing accumulation may hence consist of many SWM accumulators that are involved in a single trial (for each alternative), and the first that reaches the threshold is the lexical item activated. The race versions have been respectively proposed and simulated by LaBerge (1962) and Usher et al. (2002); however these approaches are currently not developed as quantitative data anal-

ysis techniques (e.g. psychometric models) in present lexical selection experiments, due to data limitations of insufficient observation numbers. Lastly, it is also worth noting that the accumulation process of the SWM involves nearly exactly the same kind of random-walk process as the popular drift diffusion model (or diffusion decision model, DDM, Ratcliff, 1978; Ratcliff & McKoon, 2008; Ratcliff et al., 1999), except there is only one absorbing threshold, and the drift rates are positive. While the DDM is appropriate for two-alternative forced choice paradigms, the SWM is appropriate for paradigms in which one characteristic response is observed across varying latencies (e.g., a picture's name).

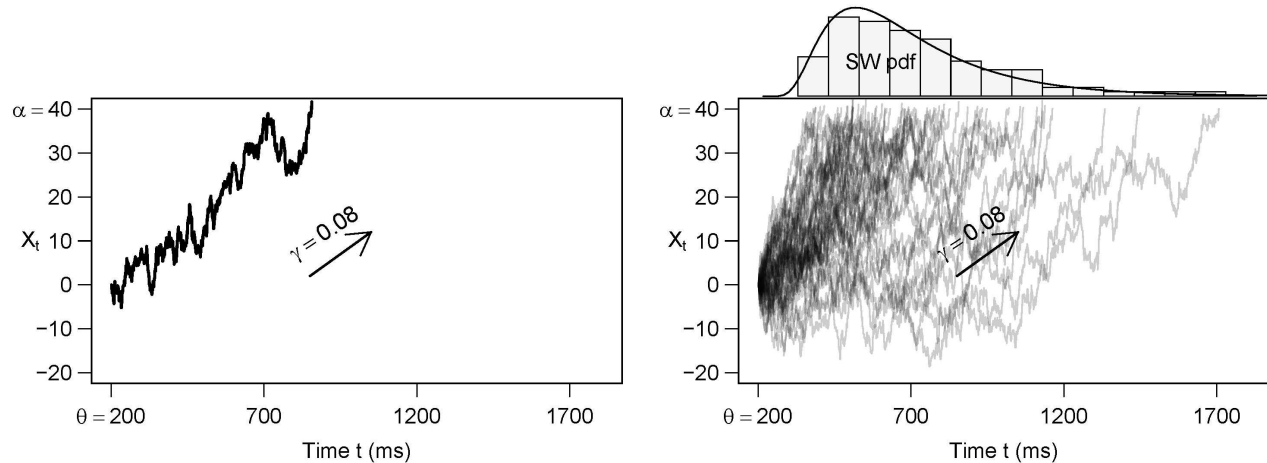


Figure 1: The SWM as a cognitive-behavioral model, describing the RT data in the context of a latent quantity (e.g. signal) accumulating to threshold, α , at rate, γ , where θ accounts for the time lapsed outside of (around) this process. Left, a single trial is modeled with the parameters. Right, many trials (e.g. an experimental design cell) are modeled with the same parameter values, and these ultimately form a SW distribution shaped with the same signal accumulation parameters.

An illustration of the SWM, and how it may be used as a simple cognitive-behavioral model for lexical target activation, is provided in Figure 1. In the left plot of the figure, a single trial is modeled. The fluctuating black line is a representation of evidence (or activation) that is accumulating over time for the lexical target, X_t ; note that the evidence begins at a value of 0, and increases (with noise) over time, until it hits the necessary threshold value, here a value of 40. The evidence accumulates at every time step (t , millisecond, ms) with sequential samples from a Gaussian signal distribution, with mean signal γ , and noise modeled by a standard deviation of 1.^{1,2} Upon reaching the threshold, the lexical target has been sufficiently activated, and the verbal response behavior is hence initiated. Parameter γ indicates the rate of activation accumulation, α is the value of the threshold needed to initiate the behavior, and θ is the time to perform the behavior, such as for response encoding to execution (here abbreviated TEA for time external to the accumulation process), and may also include time for perceptual

¹The standard deviation (SD) of 1 is used as a scaling parameter. For example if a value of $SD = 2$ is used when fitting data, all other parameters (γ, α, θ) would scale up, but be identically correlated to the parameters fit with $SD = 1$.

²The LATER model (Carpenter, 1981) is a variant that is typically used for visual saccade RT modeling. It accumulates evidence without noise ($SD = 0$), but rather samples the drift rate with noise from trial to trial. Its race extension is known as the extended LATER (Nakahara et al., 2006) or Linear Ballistic Accumulator (LBA, Brown & Heathcote, 2008).

processes.

In the right plot of Figure 1, many trials (e.g. a subject within an experimental design cell) are modeled with the same three parameters that simulated the single trial in the left plot. Note that all of these finishing times, of when the evidence accumulates to the necessary threshold, plus the TEA (θ)³, are the response times (RTs), and these are quantified directly by the probability density function of the SW distribution, also known as the three-parameter inverse Gaussian distribution. Then, quantitatively one can think of any given RT_j , $j \in 1 \dots N$, N being the number of RT observations, as having a probabilistic likelihood of arising based on the evidence accumulation parameters of the corresponding experimental design cell c in which RT_j resides, as

$$RT_j \sim \text{shifted Wald}(\gamma_c, \alpha_c, \theta_c), \quad (1)$$

in which each cell c may have various contributions based on experimental factors f , participant p , and item i . Hence in this kind of fitting approach (Anders et al., 2016), parameters are estimated per cell, as $\gamma_c, \alpha_c, \theta_c$, and these cells may vary according to such contributions. Each cell may be considered as corresponding to the right plot of Figure 1. The likelihood function of the SWM for a given RT_j in the context of an experimental design cell c is

$$f(RT_j | \gamma_c, \alpha_c, \theta_c) = \frac{\alpha_c}{\sqrt{2\pi(RT_j - \theta_c)^3}} \cdot \exp\left\{-\frac{[\alpha_c - \gamma_c(RT_j - \theta_c)]^2}{2(RT_j - \theta_c)}\right\}, \quad (2)$$

with expected value $\alpha_c/\gamma_c + \theta_c$, and variance α_c/γ_c^3 , for $RT_j \in (\theta_c, \infty)$ and $\gamma_c, \alpha_c, \theta_c > 0$.

Herein, each evidence accumulation parameter distinctly quantifies a unique RT distribution shape effect: γ for distribution tail length, α for variance around the mode, and θ for the distribution onset. Furthermore, it is important to note that each of these shape qualities can uniquely change an RT mean, as $E(RT) = \alpha_c/\gamma_c + \theta_c$ (see Figure 2 in Anders et al., 2016). Hence this kind of analysis goes beyond assessing the RT mean, which is not identified in the case of skewed distributions (like RT distributions). For example an RT mean can be greater due to a later distribution onset, a bigger tail, or more variance around the mode; two RT means can be equal (500 ms) but one distribution can have a larger tail and a faster onset $E(RT) = (10/.1) + 400 = 500$ ms, while the other a smaller tail and a later onset $E(RT) = (10/.2) + 450 = 500$ ms (the threshold, though equal in both cases as value 10 here, could also play a role in this issue). Furthermore, these opposing effects can be linked to distinct experimental predictors, and potentially disentangled. In the case of lexical selection experimental conditions (blocked-cyclic picture naming), which indeed has been proposed to have experimental predictors with opposing facilitatory and interference effects (e.g., semantic context and repetition, Navarrete et al., 2014; Oppenheim et al., 2010), then such a disentanglement provided by this modeling approach can be a notable advantage of the technique (see also Anders et al., 2015).

³For illustrative simplicity, here θ (TEA) is placed before the evidence accumulation begins (at $\theta = 200$ ms). However note that whether θ is placed here, or split around the actual accumulation process (e.g. accounting for both concept/visual recognition and response execution time), these options are quantified equally by the model likelihood function.

Lexical Target Activity versus Activity of Alternatives

The succinctness and tractability of the SWM, makes it a useful cognitive psychometric model for the empirical data that can inform psycholinguistic theory, and how experimental predictors (e.g., semantic context, repetition) may affect lexical activation dynamics. Furthermore, since the model does not require many observations, we can fit these predictors jointly, and also simultaneously account for between trial effects (e.g., lag), which can improve model fit validity (see Baayen, 2004; Baayen et al., 2008; Barr et al., 2013). Picture naming typically consists of correct responses among n -thousand possible alternatives, the correct responses having variability in latency. The model provides a sufficient, though simplistic account (activation rate, threshold, non-selection time) of the observed behavior (the lexical target), particularly via its accumulation dynamics. This kind of approach is compatible with research by Zandbelt et al. (2014), that demonstrate how focusing on the relevant, single accumulator among a large ensemble (e.g., thousands)—such as the behavior-triggering accumulator—can provide a sufficient cognitive-behavioral account of an observed response.

Though while we gain a suitable, tractable psychometric model through parameter succinctness (γ , α , θ), and through focusing on only the lexical target's accumulation, it is worthwhile to clarify that other neurologically-plausible dynamics (e.g., decay, inhibition—or competition) that may often be sourced by modeling the activation of lexical alternatives, can still be integrated in the fitted model parameter results (if present). That is, the SWM fit is *non-discriminating* in the sense that it does not force the presence nor absence of competition dynamics (e.g., by additional/constraining parameters), but will rather integrate these effects within the existing accumulation parameters $\{\gamma, \alpha, \theta\}$ that provide an account of the overall accumulation trends. For example, if inhibition from lexical alternatives occurs, depending on how these factors manifest in accumulation dynamics, this may reduce the overall drift rate fitted by the SWM, and/or alternatively, may increase the threshold needed—we note that psycholinguistic theories have different accounts in whether or not inhibition occurs, and how so (Dell, 1986; Mahon et al., 2007; Oppenheim et al., 2010; La Heij, 1988; Levelt et al., 1999; Roelofs, 2003)—these will be discussed later after our data modeling results.

We note that in contrast to having high tractability by modeling only the behavior-triggering accumulator, there exist multi-accumulator racing models that could simulate the n -thousand alternatives in lexical selection, and have added parameters that quantify decay/inhibition dynamics (e.g., Usher & McClelland, 2001; McMillen & Holmes, 2006). These models can resemble single-layer neural networks, and similarly, some also do not include motor/non-selection time parameters. However, these more complex models are currently too intractable or demanding of observation numbers to be fit as psychometric models along experimental predictors, even for $n \geq 4$ alternatives.⁴ However, it would be ideal if one could use these models as data measurement tools, and this is hence a desirable goal for future work.

Therefore, a primary difference between the SWM and these more complicated evidence accumulation models, is that it does not have additional parameters that will parse out these more specific dynamics separately (e.g., a repeated decay on

⁴See work by Miletic et al. (in review), where 10,000 or more trials are needed from a single subject, to measure inhibition effects in a 3 alternative modeling case (and without experimental manipulations): this analysis involved the Leaky Competing Accumulator (LCA) model and some of the most advanced fitting methods currently developed. However, it is a future goal to achieve psychometric applications of such models, either through bigger data and/or further optimized fitting methods.

drift, or by inhibition, a repeated reduction on drift, or increase in threshold) from the three standard accumulation dynamics parameters, $\{\gamma, \alpha, \theta\}$. Hence, to learn about the potential presence of competition (e.g., lateral inhibition) and its effects on the accumulation dynamics of lexical selection (drift rate, threshold, non-selection time) with the SWM (psychometrically), appropriately-planned experimental predictors may be used. For example, by holding target difficulty constant (e.g., easy), one could examine changes in $\{\gamma, \alpha, \theta\}$ for the lexical target between two experimental conditions, where one condition has another alternative(s) that is likely to be partially activated, but rarely selected. Fortunately in the case of lexical selection, observing unrelated versus shared semantic context conditions across trial repetitions, has been the corollary for this kind of analysis. Specifically, they have been the precedent experimental manipulations for examining effects of partial activation of alternatives, and if activation of these alternatives provides interference (Damian et al., 2001). Therefore we will utilize these kinds of manipulations to demonstrate how a psychometric modeling of these predictors may help us learn about the activation dynamics of the target, as well as how it may be influenced (e.g., inhibition) by non-target activations.

Experiment Methods

This study uses a subset of the data published in Riès et al. (2014) and only includes the data of the participants tested at UC Berkeley. This study differs from the previous work in that the analyses performed are of a completely different nature. To more deeply investigate how computational mechanisms may be impaired, or altered, in left PFC patients following damage due to stroke, herein a psychometric modeling approach is employed in which we assess the cognitive parameters of left PFC patients, which are obtained through a sequential information sampling (evidence accumulation) modeling analysis of their response data. Furthermore we compare the PFC patient results to healthy speakers in the experiment, assess replicability of previous analyses performed on healthy speakers (Anders et al., 2015), and interpret the findings in the context of current psycholinguistic theory.

Participants

The study was performed in agreement with the Declaration of Helsinki. All subjects gave informed consent approved by the University of California, Berkeley Committee for Protection of Human Subjects. The data of 9 PFC patients (5 females; mean age: 59, SD = 12.09 years old) and 14 healthy speakers (8 females; mean age: 66, SD = 8.79 years old, $t(13.41) = 1.50$, $p = .158$) matched in age, gender, and education were collected. Patients had on average 17 years of education (SD = 2.30) and controls had on average 16 years of education (SD = 1.74; $t(13.81) = -1.21$, $p = .245$).

All patients had left PFC lesions due to stroke caused by infarction of the left precentral branch of the middle cerebral artery (providing the major blood supply to the left PFC). All were tested on the neuropsychological tests (mentioned below) and for the current study at least 6 months post-stroke (chronic stage). Their lesions were delineated onto the MRICRO templates by a neurologist using input from T1, T2, and Flair scans acquired at least 6 months post-stroke on a Siemens Allegra 3.0 T MRI scanner. Of the 9 patients tested, 3 PFC patients (2 males) could not perform the experimental tasks adequately due to markedly poor performance: they either did not understand the instructions properly or their error rate on the experimental tasks was over 40% (mean score on Sequential Commands: 68/80, SD = 6.08, individual scores: 64, 65,

and 75; mean score for Spontaneous Speech: 18/20, $SD = 1.00$, individual scores: 18, 17, and 19, respectively). The data of these 3 left PFC patients were excluded from the analysis. The remaining 6 left PFC patients had a mean Spontaneous Speech score of 18.83/20 ($SD = 0.75$), reflecting overall good production abilities despite some articulation problems (two patients had a score of 18 reflecting a lack of detail in the picture description or in answering one of the questions). The mean Sequential Command score was of 74.9/80 ($SD = 8.88$ based on the 5 patients for which we had the Sequential Command score. The last patient had an overall Comprehension score of 9.8/10 reflecting good comprehension abilities). We note that 3 out of the 6 patients had a perfect score of 80, only one had a relatively low score of 59.5 and the other had a score of 75. The patient with the low comprehension score was nevertheless able to perform the naming task correctly. Thus the language production deficits of the left PFC patients included in the analysis were overall mild in nature allowing the patients to perform the task adequately. Lesion overlaps of the 6 patients included in the analyses is presented in Figure 2; and Table 1 provides by patient, information about total lesion volume and percent damage in and outside the left PFC. Particularly, the left PFC patient lesions were centered in both the inferior frontal gyrus and the middle frontal gyrus.

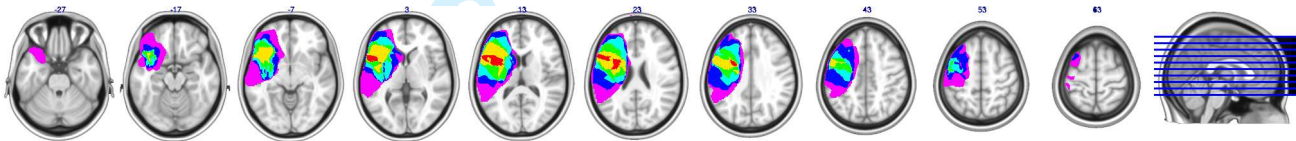


Figure 2: Lesion overlay of the 6 patients. The color coding indicates the amount of overlap between the different patients' lesions (red corresponds to 100% overlap and purple to 0% overlap).

	% Damage in PFC	% Damage outside the PFC			Total lesion volume (cm ³)
		Insula	Basal ganglia	Temporal lobe	
P1	82.85	9.45	2.47	5.23	134.02
P2	81.46	7.00	8.40	3.14	72.53
P3	78.68	6.42	6.63	8.27	187.29
P4	69.35	4.46	0.52	25.67	96.98
P5	44.87	6.99	3.83	44.31	167.00
P6	70.20	10.89	0.25	18.66	59.96
Average	71.24	7.54	3.68	17.55	119.63
Standard Deviation	14.10	2.29	15.68	16.37	51.62

Table 1: Percent damage in the PFC and outside the PFC (to the insula, basal ganglia or temporal lobe) derived from manual delimitation onto the MRICRO templates by a neurologist, and total lesion volume per patient included in the study.

Materials and design

As described in Riès et al. (2014), the stimuli were 252 line-drawings of common objects or animals selected from published collections (Snodgrass & Vanderwart, 1980; Bonin et al., 2003), the Internet, or constructed by us (mean name

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4 agreement: 91.25%, $SD = 8\%$). For purposes unrelated to the present study, participants also performed a Simon task
5 (Simon & Rudell, 1967) using the same stimuli as in the picture naming task. Therefore, the stimuli were colored in green
6 or purple presented on the left or on the right of the fixation cross. These dimensions were however not relevant here.
7 The stimuli were issued from 6 semantic categories (e.g. human dwellings, means of transportation). Each member (e.g.
8 house) was represented by 7 different items (e.g. 6 different houses: 5 used as experimental items, 1 for familiarization).
9 In the blocked cyclic naming task as generally used, pictures are repeated several times per block (5 or 6 most often; e.g.,
10 Damian et al., 2001; Maess et al., 2002). The semantic interference effect emerges only after the first presentation of the
11 stimuli. We reasoned that repeating the same pictures several times may cause the stimulus-response associations to be
12 over-learned. Therefore, we used different pictures to represent the same member of a category (e.g. different types of
13 houses) to minimize stimulus-response associations that could bypass lexical access. Importantly, the semantic interference
14 effect has been shown to extend to new items (Belke et al., 2005). We were therefore confident this design would give
15 us a reliable semantic interference effect. The items were presented in a pseudo-random order (using the software MIX;
16 Van Casteren & Davis, 2006) within each experimental run with the constraints that consecutive items were phonologically
17 unrelated (i.e., two pictures in a row never had the same initial phoneme) and items sharing the same name would be at least
18 3 trials away from one-another.
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27 28 *Procedure*

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30 The procedure was the same as described in Riès et al. (2013, 2014, 2015). We repeat relevant aspects for the current
31 study. Participants responded by making verbal answers to pictures presented on a computer screen situated 148 cm in
32 front of them while seated comfortably in a sound-attenuated dimly-lit environment. Stimuli were presented using Eprime
33 2.0 Professional (Psychology Software Tools, Inc., Pittsburgh, PA), allowing on-line recording of verbal responses. A trial
34 consisted of the following events: (1) a fixation point (“plus” sign presented at the center of the screen) for 500 ms; (2)
35 a picture for 2000 ms (3) a blank screen for 2000 ms. The following trial started automatically. As above-mentioned,
36 participants also performed a Simon task but did so in separate blocks. The order in which the tasks were performed was
37 counter-balanced across participants. In both tasks, participants had to add the possessive determiner “my” (e.g. “my
38 house”) before their answer. This was done to reduce variability in vocal onsets and because we also recorded EMG activity
39 of three facial articulators for unrelated purposes. The response-onset measure were the vocal-onsets. The task was split into
40 4 blocks of 108 trials each. Participants could take breaks whenever they needed and two built-in pauses were equally spaced
41 within each block. Participants had to respond as fast and as accurately as possible. Before the experiment, participants
42 were familiarized with the picture names.
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50 The results of the Simon task are not considered here. In the Simon task, participants were asked to say “my right” or
51 “my left” depending on the color of the picture while ignoring the side to which the picture was presented. Thus interference
52 is greater for a picture presented on the left of the fixation cross when the response to be given is “my right” and vice-versa.
53 The stimulus-response association rule (i.e., saying “my right” for a green picture and “my left” for a purple picture or
54 vice-versa) and the order in which the tasks were performed were counterbalanced across participants.
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Data post-processing and analysis

Response accuracy and verbal response times were measured offline using CheckVocal (Protopapas, 2007). Trials were excluded from the analysis if the participant did not respond, or produced any kind of verbal error: partial or complete production of incorrect words, omission of the pronoun “my”, verbal dysfluencies (e.g., stuttering, utterance repairs: 4%) and hesitations (e.g., if the experimenter perceived the production of the possessive pronoun to be abnormally lengthened or separated from the production of the noun by a pause). Finally in the main effects and interactions analyses, and also to provide results that translate to previous analyses (Anders et al., 2015), we follow the common practice to exclude the first occurrence of each stimulus (namely, repetition cycle 1) on each block (Ewald et al., 2012) in contexts of analyzing semantic interference, as the semantic interference effect can be absent or even reversed in the first presentation (e.g., Damian et al., 2001; Belke et al., 2005; Rahman & Melinger, 2007). However, we provide a supplementary analysis afterward that indeed examines the contrasts between repetition cycle 1 and subsequent repetitions (e.g., 2-3 and 4-6), with semantic context, in order to bridge our model-based results to more recent literature that involves the topic (e.g., Navarrete et al., 2014; Mahon et al., 2007; Rabovsky et al., 2016).

Application to Lexical Retrieval Patient Data

Fitting Approach

The fitting method utilized for the SWM combines techniques of maximum likelihood and deviance criterion minimization. In this approach (see Anders et al., 2016, for a tutorial and code), an independent SWM is fit for each unique experimental design cell (each possessing an RT distribution), in which main effects and interactions can be observed on the parameters of evidence accumulation. Note that for every design cell, the cognitive parameters: drift γ , threshold α , and TEA θ are estimated. The design cells consist of the possible combinations of the relevant factors of the task: semantic context, repetition, and lag between item repetition. Furthermore, we fit these cells by participant (20 total), in which six participants are PFC patients, and the other fourteen are healthy speakers. In order to provide comparison and replicability analysis of the modeling with the previous data researched (Anders et al., 2015), we similarly collapsed the repetition factor into two levels (2-3 and 4-6) and the lag factor into two levels (2-5 and 6-12). Then the result is $N = 20 \times 2 \times 2 \times 2 = 160$ experimental cells fit, each having an appropriate average cell size of 40 trials, with standard deviation (SD) of trials 10, and range (20,64). A supplementary analysis also compares semantic context effects of pictures in their first repetition (naming) with the other two repetition levels (2-3 and 4-6) for both healthy speakers and patients, resulting in $40 = 20 \times 2$ additional experimental cells (one lag level), with average cell size of 28 trials, standard deviation 6, and range (14,35). This totals in 200 experimental cells being fit by the model.

Finally before fitting, on each cell a very light processing of potential contaminant RTs (see Barnett & Lewis, 1994; Ratcliff & Tuerlinckx, 2002) was performed by an elimination criterion of below three or above six median absolute deviations (MADs, see Leys et al., 2013) from the cell RT median (preserving the long tail RT values), resulting in 6,506 trials for analysis out of the original 6,543 (0.5% of trials omitted). Then to fit the model, for each cell, maximum likelihood method of moment estimators were used to calculate the three SW parameters when a shape parameter is proposed (β , as in

Anders et al., 2016). Then searching across the near-entire range β , the optimal parameter set is selected according to the minimal difference between the data to model-predicted RT quantiles, by 100 equally-spaced quantile points in the range of (.01, .99).

Illustration of the Observed Response Time Distributions by Design Cell

The analysis approach hence consists of modeling the observed RT distributions corresponding to each experimental design cell (partitioned by subject as well). After the model is fit, values for the process parameters are obtained for each of these cells. Then a formal comparative analysis of these parameters (e.g. an ANOVA), can quantify the significant differences and/or interactions between the experimental conditions. Particularly, we obtain information on how these conditions modulate the dynamics of the underlying process modeled. In this section, we illustrate the observed RT distributions of the experimental design cells from sample subjects of each group: healthy speakers and left PFC patients.

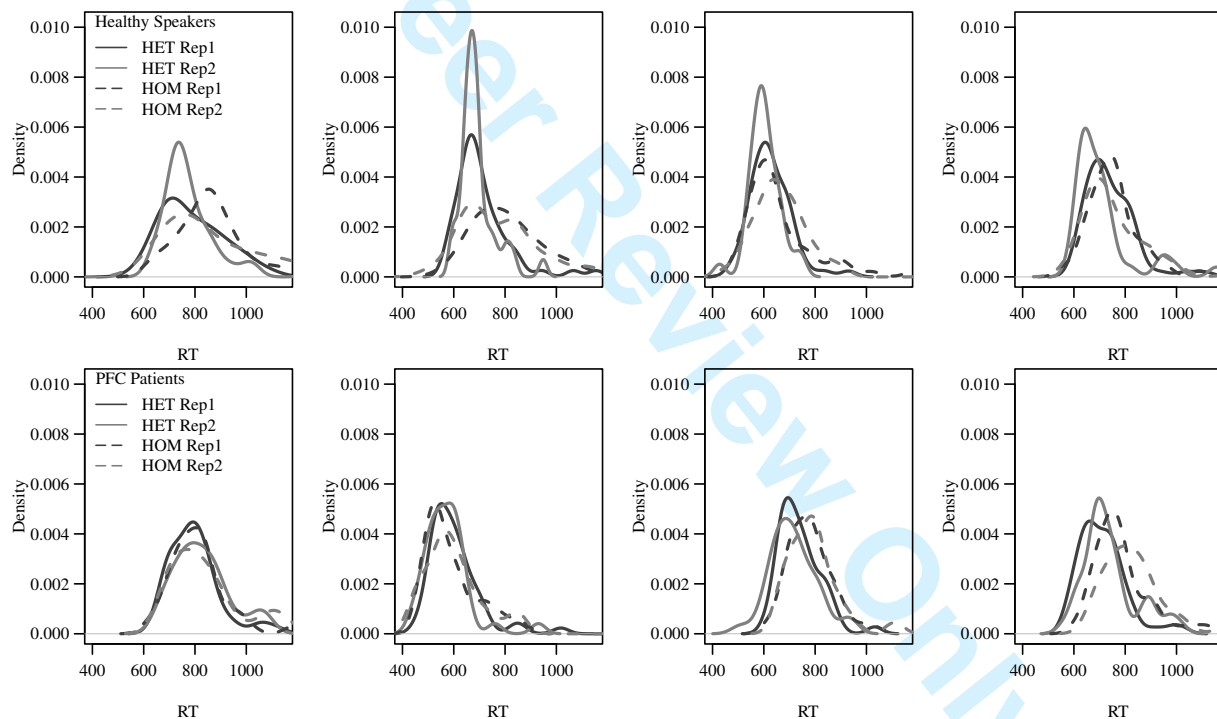


Figure 3: An illustration of the experimental cell distributions by subject that the SWM fits. The four distributions in each subject's plot respectively correspond to the HET/HOM conditions at repetition levels 1 (cycles 2-3) and 2 (cycles 4-6) for one lag level (>5).

The top row of Figure 3 provides the observed data distributions obtained from four example subjects of the healthy speakers group, and the bottom row provides the same for four subjects of the left PFC patient group. The four distributions in each subject's plot respectively correspond to the HET and HOM conditions at repetition levels 1 (cycles 2-3) and 2

(cycles 4-6) for a single lag level (>5).⁵ Keeping in mind that of the SWM parameters: γ quantifies the tail length, α quantifies the variance around the mode value, and θ quantifies the onset of the distribution, some elementary predictions can be made about what one might see when the model is fit to this data. For example, in comparing the healthy speakers versus patients (top and bottom rows), the healthy speakers clearly show an improvement in RTs at the second repetition of HET conditions by narrower distributions/higher modes, while the patients do not. This distribution effect would most likely correspond to a lower α value obtained in the model fit with increased HET repetitions for healthy speakers. Another observable difference between healthy speakers and patients exists in how the healthy speakers have notably slower RT distributions between the HET versus HOM conditions (by being wider around the mode), while the patients do not have this specific modulation in their distributions. This is another effect in which one could expect that the model fit will derive an α change (here increased) for HOM conditions in healthy speakers, and this kind of threshold adjustment would not be present in patients.

Through visually inspecting these by-cell observed RT distributions, these are just a few examples of speculations one could make about the results that will be obtained by the model fit. However from only eye-balling these plots, it is difficult to capture all such differences, and be right about them, especially when one is tasked to also consider changes in distribution tail and onset. This is especially the case since the unique cells provide for as many as 200 distributions total. Alternatively if one wanted to reduce these cells into a single distribution across subjects in order to make such conclusions, unfortunately this can blur the precision of the distributions (widen them) and can hence can mask or distort the underlying effects. In contrast, the proposed approach consists of applying a model to these distinct data cells, which will quantify each of these distribution features according to an objective function. Then comparison analyses of the posterior results will indicate what significant differences there are between the conditions and subject groups for the parameters. First before examining these posterior parameter results however, the following section provides important model fit diagnostics based on quantifying the fit error (observed distributions vs. the estimated distributions) at the specificity of all of the distribution deciles (e.g. 0.1, 0.2, ..., 0.9). This is done for every cell (200 total). These diagnostics hence provide a systematic method to assess quality of fit (and if the parameter results are appropriate to interpret).

Model Fit Checks

In this section, model fit diagnostics are examined to assess the degree to which the model appropriately fits the data, and hence if the parameter results may be appropriate to interpret. Then following a satisfactory fit, the next section reports the main parameter differences. Thirdly, an interpretation of these parameters and their relationships to psycholinguistic theory is provided.

The first plot in Figure 4 provides the quantile-quantile (QQ) matching of the deciles of the fit-simulated distributions against the observed distributions; it contains all 200 cells. The QQ plot may show overall trends in systematically misfitting quantiles of the distribution, as well as misfit outliers. It also gives an idea about the scale and range of the data. The

⁵Since plotting the four other distributions that correspond to the remaining lag level (<5) would considerably complicate comprehension of these illustrations, we do not plot them here.

importance of this check is to observe critically any curvatures in the plot, which is a strong sign of misfit. As one can see in the figure, there is no systematic curvature in the plot and the SWM performs systematically well on the data set, with minimal outliers.

The middle plot provides the distribution of standardized residuals for each of the nine deciles across the 200 cells fit. In this model fit check, one might optimally see a distinct ordering of decile residual distributions, due to the property that residual magnitude tends to correlate with RT data variance and magnitude (e.g., see Anders et al., 2016). In this application, such an ordering is present, and it is nicely shown that most standardized residuals are near 0.05 and below, similar to previous applications. Finally the third plot provides the sum standardized residual, Δ , by cell. One can see that there are not many large residual outliers, $\bar{\Delta} = 0.87$ is suitably low, and the correlation between the standardized residuals and the deviation of the data, $\rho_{\Delta\sigma} = -0.16$, is also satisfactorily low (see Anders et al., 2016). Thus the three model fit diagnostics demonstrate appropriate results.

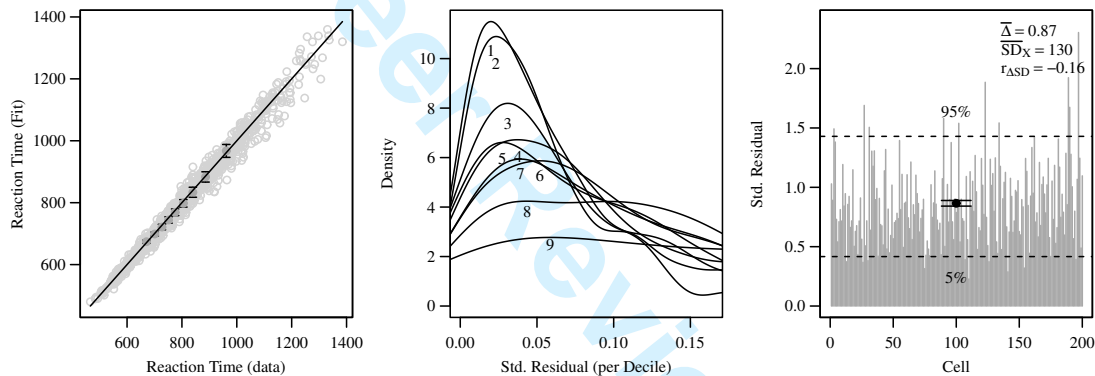


Figure 4: SWM fit to the observed data. The top-right plot shows the quantile-quantile match for the nine deciles (0.1 to 0.9) for each of the 200 cells. The bottom-right plot shows the distribution of residuals for each of the nine quantiles across the 200 cells; where the residual is the absolute difference (in ms) between the observed quantile and the model-predicted quantile. The left column provides mean parameter values with bars representing standard error of the mean ($N = 160$) grouped by experimental factor. From top to bottom are respectively the parameter values for activity accumulation rate, γ , inverse baseline activation (or threshold), α , and time external to activity accumulation (TEA), θ .

Results

In the model fit analysis of the 14 healthy speakers (left) and 6 left PFC patients (right) of Figure 5, the following results were observed. Firstly, overall group differences were shown to have marginal effects: the PFC patients were found to have overall higher accumulation rates γ (or excitability) than healthy speakers $t(104) = -1.8$, $p = 0.07$, which may be compensating for their overall slower non-accumulation delays θ (e.g., motor output) than healthy speakers $t(73) = -1.5$, $p = 0.12$. Then the most substantial effects were found along the experimental factors, as well as their interactions.

At the detail of the experimental manipulations, a main effect of semantic context (HET vs. HOM blocks) was found,

providing a decrease in the signal accumulation rate parameter, γ ($F_{\gamma}(1, 18) = 15.3, p = .001, \eta_p^2 = 0.46, \eta_G^2 = 0.080$)⁶, and an increase in the threshold parameter, α ($F_{\alpha}(1, 18) = 2.26, p = .15, \eta_p^2 = 0.11, \eta_G^2 = 0.012$) for HOM over HET, but no effect in external time θ ($F_{\theta}(1, 18) = 0.18, p = 0.69, \eta_p^2 = 0.01, \eta_G^2 = 0.000$). Critically, a significant interaction was discovered between groups regarding this α effect ($F_{\text{inter}\alpha}(1, 18) = 4.36, p = .04, \eta_p^2 = 0.21, \eta_G^2 = 0.026$), particularly the PFC patients were impaired to adjust their threshold appropriately according to semantic context. Hence this increase in threshold by HOM vs. HET blocks, occurs only in the healthy speakers ($t(110) = -2.2, p = 0.032$). Furthermore, a significant interaction was found between groups concerning θ ($F_{\text{inter}\theta}(1, 18) = 5.84, p = .03, \eta_p^2 = 0.24, \eta_G^2 = 0.011$), suggesting semantic interference (HOM) for the PFC patients also introduces added motor output difficulties.

In regard to repetition between levels 2:3 to 4:6, a main effect was also found, providing a decrease in signal criterion level, α ($F_{\alpha}(1, 18) = 4.05, p = .06, \eta_p^2 = 0.18, \eta_G^2 = 0.025$) with increased repetition, which could be interpreted as a residually higher starting activation for the lexical target by its repetition. Here, no significant interaction was found between groups in regard to repetition. However a significant interaction was evident for repetition crossed with semantic context, on accumulation rate γ ($F_{\text{inter}\gamma}(1, 18) = 7.32, p = .01, \eta_p^2 = 0.29, \eta_G^2 = 0.014$). Particularly semantic interference (HOM) provides decreased accumulation rate over repetition levels $t(73) = 2.1, p = 0.037$, see Figure 6 (Row 1, HOM 2:3 to 4:6). Thirdly, no significant effects were found in lag.

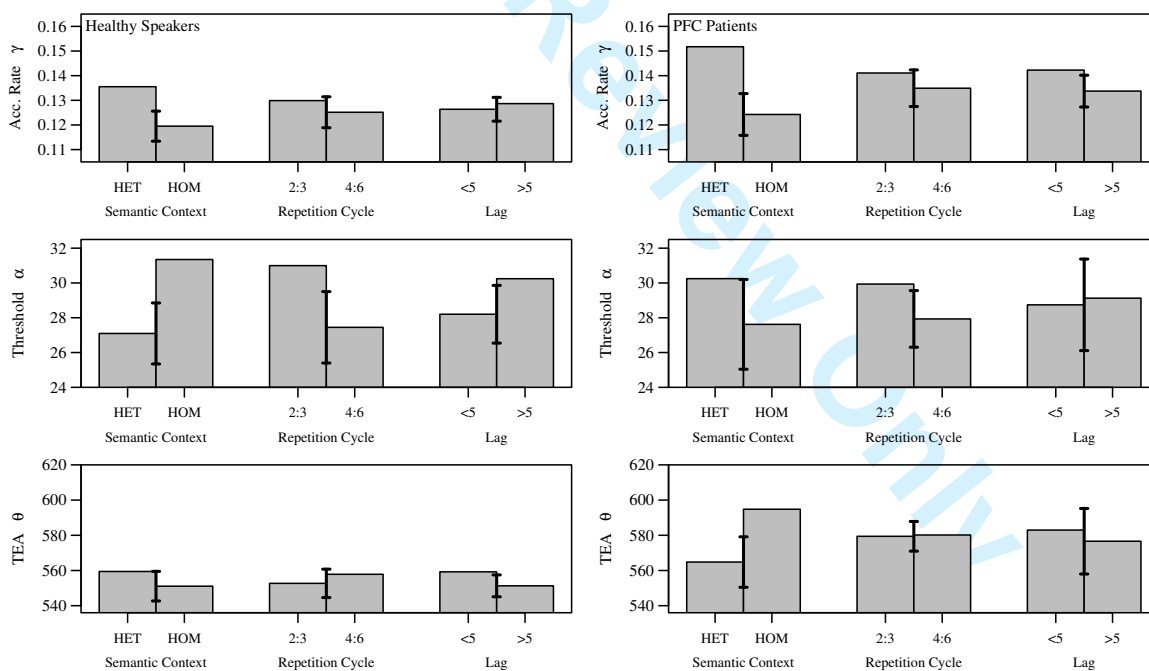


Figure 5: Left column: healthy speakers, right column: left PFC patients. The cognitive parameters from the model fit for each experimental condition: semantic context for semantically-unrelated conditions (HET) and semantically-related conditions (HOM), repetition level (2:3, 4:6), and lag (<5, >5).

⁶For an explanation of effect sizes η_p^2 and η_G^2 , see Bakeman (2005).

Supplementary Analysis with First Repetition (Naming) Level

In an ANOVA of the model parameters between the first repetition level to the next, crossed with semantic context and group, the model-based analysis accounts for the ‘known’ boost (or notable improvement) in RTs after the the first repetition, through the main effects of enhanced activation rate γ ($F_\gamma(1, 18) = 18.64, p < .001, \eta_p^2 = 0.51, \eta_G^2 = 0.107$) and reduced TEA θ ($F_\theta(1, 18) = 8.25, p = .01, \eta_p^2 = 0.31, \eta_G^2 = 0.029$), see Figure 6 (Rows 1 and 3). In contrast, a slowing in RTs occurs by increased threshold α ($F_\alpha(1, 18) = 3.58, p = .07, \eta_p^2 = 0.17, \eta_G^2 = 0.056$), up to a level that corresponds to relative condition difficulty (lower for HET vs. larger for HOM, see Healthy Speakers), as in Figure 6; while the LFC patients have increased threshold means (from 1 to 2:3) as well in HET/HOM, these levels do not adequately adjust for semantic context difficulty like in healthy speaker levels. Thirdly, no significant interactions were found. Finally, no significant pairwise differences were found between 1st repetitions in HOM versus HET.

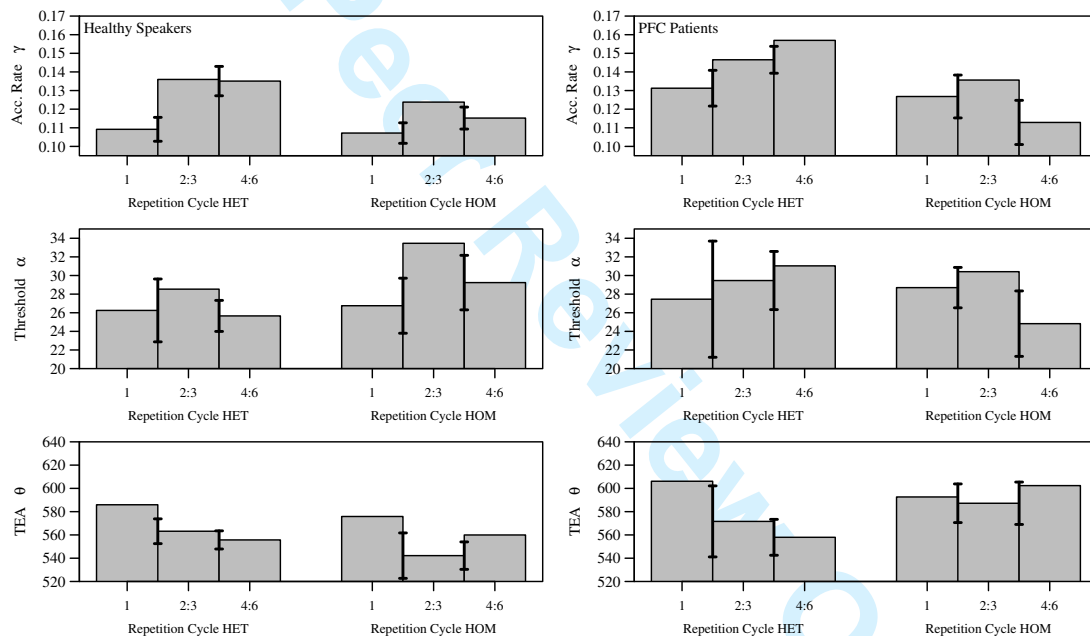


Figure 6: Left column: healthy speakers, right column: left PFC patients. A more detailed contrast provided for the semantically-unrelated conditions (HET) and semantically-related conditions (HOM) over each repetition level modeled (1, 2:3, 4:6). Used to further examine how the cognitive model parameters for HET/HOM adjust over repetition, and by subject group.

Summary

Replication Regarding the healthy speakers, we replicated the findings described by Anders et al. (2015): shared semantic context (HOM, or semantic interference) *reduces* the rate of lexical target activity accumulation (γ) and *increases* the amount of activation needed for the lexical target to be selected (α). Repetition decreases the amount of activation needed for the lexical target to be selected (α), and likewise, no significant effects were found in lag. Though similar to the previous study, a mean increase in threshold was observed with increased lag levels. We wonder if non-significance

here is an issue with power, and if improvements in experimental design that balance observation numbers per lag level, may improve analysis of this predictor and/or its interactions. Alternatively, we note that in previous analyses based on RT means, lag has not been found to have a significant effect.

Accounting for Differences Between PFC Patients and Healthy Speakers New findings in left PFC patient data are the following. With respect to semantic context, left PFC patients have an impairment from healthy speakers in appropriate threshold adjustment. Although the patients have a much more pronounced drop in evidence accumulation rate between semantic context conditions HET/HOM than healthy speakers, they do not differ significantly from healthy speakers in accumulation rate during semantic interference. Rather, they fail to up-adjust the threshold to deal with the increased difficulty that semantic interference provides, as in healthy speakers, and instead appear to need less activation to select the lexical target (threshold) α during semantic interference, but are then slowed by the interference at either a process before this decision process can take place, or during the post-selection stages (e.g. articulation), by θ .

Although parameter θ includes both pre- and post-selection delays, given the location of these patients' lesions, it may be reasonable to infer that the additional slowing in θ occurs post-selection (such as in articulation) for these patients. This is because their left PFC lesions also encompassed part of the premotor cortex, and in some cases, the insula. Several studies (e.g., Dronkers et al., 1996; Guenther et al., 2006) have discussed the relevant role of these regions in speech motor programming/functioning. Here (and as in, Kello et al., 2000; Fink et al., submitted), we are suggesting that these regions operating down-stream from the word selection process are affected by word retrieval difficulty, with the idea that processing difficulty in lexical selection can cascade into articulation difficulties.

Another notable difference involves that overall conditions, PFC patients have higher accumulation rates and non-accumulation times than healthy speakers. This tentatively suggests that PFC patients may compensate for such pre- and post lexical selection difficulties (increased θ) by exhibiting an increased overall excitability (γ) in the lexical network, which in combination with their impairment in appropriate threshold (α) modulation during semantic interference, could potentially account for their higher error rates. For example, Usher et al. (2002) demonstrate how in racing accumulation models, an increased threshold is needed to maintain accuracy levels when relevant alternative numbers are increased, which has also been replicated in fits to real data by Van Maanen et al. (2012); correspondingly, PFC patients do not appropriately increase threshold in the case of semantic context, which has more alternatives active, and hence according to this modeling framework, this finding predicts a higher error rate. Additional applications of this modeling (and advancements thereof) to patient data may further sharpen these inferences.

Disentangling Facilitatory and Interference Effects Through Different Dynamics in Lexical Activation Next, the significant interaction analyses between semantic context and repetition have revealed valuable findings for psycholinguistic theory. Navarrete et al. (2014) have argued that semantic interference (from shared semantic context, HOM) in cyclic naming depends on (and hence accumulates with) repetition. We have reproduced this result (in both healthy speakers and PFC patients), and furthermore we newly link this repetition-based interference with decreases in the lexical target activity

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4 accumulation rate, γ .⁷ Secondly, Navarrete et al. (2012) relate this accumulated interference not as through competition,
5 but by the negative effects of incremental learning: through naming semantically-related targets, that overcome the positive
6 learning effects, as forwarded by Oppenheim et al. (2007, 2010).
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9 While Oppenheim et al. (2010) consider two kinds of possible racing mechanisms, one competitive and one non-
10 competitive (the latter having a threshold that is not forced to be negatively-correlated to the booster accumulation rate),
11 Anders et al. (2015) showed that the non-competitive version is supported by their similar psychometric modeling of these
12 experimental data predictors in a different data set. Our work herein also supports/replicates these results of our previous
13 modeling analysis, and further *disentangles* how these opposing effects of learning may be occurring in the lexical activation
14 dynamics. That is, a deeper analysis of the cognitive parameters derived from the data here, shows how they are able to
15 disentangle the positive effects of learning (reduced thresholds, by repetition priming in general) from the negative effects
16 of learning (reduced activation rate, by weakening closely-related-but-incorrect targets by repetition priming in semantic
17 context).
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23 These results, that cumulative semantic interference occurs with repetition (by a reduction in lexical activation rate, γ)
24 are in contrast to the account by Belke & Stielow (2013), who propose that semantic interference does not accumulate with
25 repetition in the blocked naming paradigm (for young healthy speakers). This difference in conclusion may be attributed to
26 several reasons. First, Belke & Stielow's claim pertains only to young healthy speakers, in which our study involved older
27 healthy adults as well as patients, each of which demonstrated cumulative semantic interference effects—though we note
28 that Anders et al. (2015); Mulatti et al. (2014); Navarrete et al. (2014) have also observed the cumulative interference effect
29 for young healthy speakers in their experimental data as well. Secondly, Belke & Stielow's conclusions pertain to analyses
30 involving mean RT analyses. Since our response process (and RT distributional) modeling disentangles the priming effect
31 of repetition (quickening) from the semantic interference effect (slowing) on RTs, the methodology is in this respect less
32 prone to Type II errors than an analysis on RT means (see the final paragraph of the SWM explanation section). Hence,
33 more information on this debate might be yielded by re-analyzing Belke & Stielow's discussed data sets with this kind of
34 modeling, and seeing if similar opposing effects (facilitation and interference) occur on the RT means, which leaves room
35 for future work.
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43 ***Accounting for How a Network May be Calibrated, Adjusted for Difficulty*** The supplementary analysis across repeti-
44 tion levels demonstrates how a naming system (or lexical activation) can become quickly calibrated after the first repetition.
45 First, a commonly known result is a large boost (reduction) in RTs after the first repetition (Belke, 2013; Belke et al., 2005;
46 Navarrete et al., 2012, 2014). Here the model-based results specify that the dynamics of this boost relate to a quickly
47 calibrated network (e.g., through positive learning) by increased target activation rates γ and decreased external times θ
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52 ⁷Support for this claim is also evident in our previous analysis by Anders et al. (2015), which used a different data set. Though since the experimental
53 design therein included *three* shared semantic conditions and only *one* non-shared semantic condition, the conditions of shared semantic context had
54 greater influence over the main effects of other predictors (e.g., repetition). Hence it is likely that for this reason, the ANOVA did not retrieve a significant
55 context/repetition interaction of reduced γ over repetition in shared semantics, but rather a significant main effect of reduced γ simply over repetition, since
56 most conditions therein involved shared semantic context.
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(e.g., articulation preparation). Secondly, we show how the network is also simultaneously calibrated to the difficulty, in a way that slows RTs for increased selection caution by a magnitude relative to the difficulty level. For instance, while the initial threshold α states are nearly the same between HET/HOM conditions during the first naming (repetition), afterward the network adjusts the threshold according to the condition difficulty (e.g., by learned interference), such as increasing the threshold to be more cautious in HOM than in HET. This idea ties into work by Navarrete et al. (2014) who demonstrate how different “baselines” may be determined according to the presence or absence of semantic context within the first repetition, from which subsequent positive/negative incremental learning effects may act regularly.

Discussion

A number of current psycholinguistic theories consider lexical selection as a process related to selecting a lexical target from a number of lexical alternatives which each have varying activations (or signal supports), that are largely resultant of an initial stimulus recognition. How these activations develop into influencing the decision process of selecting the appropriate lexical target, can be described as a racing evidence accumulation process (e.g., see Anders et al., 2015). Herein we utilized data-derived (or psychometric) implementations of evidence accumulation modeling to provide a mechanistic account for lexical selection dynamics, as modulated by factors of semantic context and repetition cycles in the data, for the blocked-cyclic naming paradigm. We hence focus on studying the mechanics of the increased semantic interference effect observed in left PFC patients versus controls, through cognitive parameters that are estimated from the experimental data. Using this paradigm, our results suggest that it is the decision threshold adjustment that is specifically impaired in these patients. These results therefore shed light on the computational role of the left PFC in lexical selection.

A Cognitive Psychometric Modeling of Left PFC Patients and Healthy Speakers We hence provide a first psychometric evidence accumulation application to left PFC patients performing semantic (e.g., picture/concept) lexical retrieval. We do so by applying a simplest case model of racing noisy evidence accumulation (due to observed data size constraints), called the shifted Wald model (SWM), and examine the cognitive parameters along the experimental predictors (both main effects and interactions). We also apply the SWM to a control group of healthy speakers on the task, examining the replicability of the SWM parameters in a previous experimental study of only healthy speakers, by Anders et al. (2015).

Left PFC Patients The left PFC patients primarily differed from the healthy speakers in regard to semantic interference effects. While healthy speakers are slowed in both evidence accumulation rate (γ) and threshold (α) during semantic interference, the left PFC patients appear to show only a slowing effect in evidence accumulation rate (γ), and hence have an impairment in decision threshold adjustment (α). It may therefore be reasonable to speculate that for these patients, a module in the pre-frontal cortex may not be accumulating semantic interference anymore in a similar way as in healthy speakers. Furthermore, left PFC patients suffer semantic interference through difficulties (or modulations) of prior or posterior processes to lexical selection (e.g., lexical target articulation), as suggested by our finding of their slower non-accumulation function times (θ) during semantic interference contexts. A third finding is that the left PFC patients may be compensating for these slowings/impairments by exhibiting an increased overall excitability (or lexical target activation rate, γ) in the lexical network. This increased excitability, in combination with the other impairments, primarily the threshold

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4 modulation impairment of left PFC patients, may lead to increased error rates. For example, Usher et al. (2002) demonstrate
5 how in racing accumulation models, an increased threshold is needed to maintain accuracy levels when relevant alternative
6 numbers are increased, which has also been replicated in fits to real data by Van Maanen et al. (2012). Correspondingly,
7 PFC patients do not appropriately increase threshold in the case of semantic context, which has more alternatives active,
8 and hence this finding predicts a higher error rate.
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11 *Healthy Speakers* In the healthy speakers, the main effects in the previous experiment by Anders et al. (2015) were
12 replicated, and through deeper analyses here, notable interaction effects were newly revealed. For example, while repetition
13 itself provides a priming benefit in less activation needed (α) for the lexical target to be selected, it is the increasing semantic
14 interference that reduces the evidence accumulation rate (γ) with increased repetition. Furthermore, we provided a new
15 supplementary analysis that examined the first repetition (previously excluded) and its transition to subsequent repetitions.
16 The parameters for first repetitions were close (nearly equal) between unrelated and related contexts, however the large
17 boost in faster RTs occurring in subsequent repetitions (commonly observed in such experiments, see Belke, 2013; Belke
18 et al., 2005; Navarrete et al., 2012, 2014) were accounted for by the model with faster drift (γ) and TEA (θ) acquired
19 after the first repetition cycle. In contrast, subsequent repetitions also have a slowing effect: thresholds are heightened in
20 accordance to condition difficulty—this can relate to the kind of “baseline” adjustment for semantic context that Navarrete
21 et al. (2014) refer to. Furthermore, PFC patients had similar patterns as healthy speakers in calibration between the first
22 repetition to subsequent ones (see Figure 6), except fail to set a heightened threshold to account for difficulty, which is
23 compatible with the main results of the patient analysis.
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32 *Going Deeper into Modeling Left PFC Patient Errors* Although we were able to obtain psychometric indications for
33 why PFC patients may make more errors, unfortunately due to observation size limits, we were not able to fit the errors
34 themselves, and more deeply analyze them with the cognitive modeling. Hence an important development for future work,
35 is to collect enough observations to jointly fit the PFC patient errors as another contrast to compare, either as another mixture
36 (or factor) added onto the current modeling, or ideally with a racing evidence accumulation model, which can jointly model
37 multiple kinds of errors (e.g., articulation, or specific word alternatives, LaBerge, 1962; Usher et al., 2002). While full
38 racing models (e.g., quantifying $n \geq 4$ alternative accumulators) are technically not used as data measurement models for
39 experimental data (at least not currently), we can certainly learn from them instead as data-producing models for comparing
40 PFC patients versus healthy speakers and their errors. For example, one could examine simulated data for two groups, in one
41 model that adjusts threshold higher for semantic context (healthy speakers), and another group in which thresholds remain
42 the same (PFC patients). Alternatively, to maintain the data-derived cognitive modeling approach used herein, perhaps a
43 doubly-sized experiment with PFC patients may provide enough errors to fit them as another mixture/factor. However we
44 note that since many kinds of errors (articulation, studder, semantic versus phonological, etc. are possible, it is difficult
45 to obtain enough observations of any single error type, in order to study it with such modeling across the experimental
46 predictors. Ideally larger experimental studies with more patients, and more observations, should be performed to more
47 sharply develop these results.
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4 **Implications to Current Psycholinguistic Theory** One of our primary experimental findings here, that left PFC pa-
5 tients have threshold adjustment impairment during semantic interference contexts of lexical retrieval, builds upon current
6 interpretations for how lesions to the left PFC may impair word selection processes in language production (reported for
7 example, in Thompson-Schill et al., 1997, 1998; Schnur et al., 2006, 2009). In particular, our finding sits well with the
8 proposal that the left PFC, and especially the left inferior frontal gyrus (IFG), are involved in modulating a proactive in-
9 terference control mechanism, which has been considered to operate across cognitive domains (Kan & Thompson-Schill,
10 2004; Riès et al., 2014, 2015, see also Jonides & Nee, 2006). In this way, the adjustment of response threshold could be
11 operating in response to interference that arises from closely-related semantic contexts, and maintains a similar accuracy
12 rate despite greater task difficulty, through a cost of higher caution (slower RTs). However, if this mechanism is indeed
13 more domain-general than previously-believed, then it may be less specialized to just lexical networks than the mechanisms
14 proposed by Oppenheim et al. (2010) or Belke & Stielow (2013), though it is nonetheless informed of such interference
15 dynamics. For example the lower lexical activation rates, γ , probably do not scale up the threshold as in learned interference
16 (Oppenheim et al., 2010), since we observe how repetition priming instead scales down the threshold (in both HET and
17 HOM) while γ scales down only during semantic interference (HOM, see Figure 6).

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21 **Relationships to Broader Model-Based Neuroscience Literature** Our findings can also be related as being in agreement
22 to the broader neuroscience literature that also combine neurosciences and evidence accumulation, and similarly find the
23 PFC to be principally implicated in decision threshold adjustment—particularly in perceptual decision-making (Boehm
24 et al., 2014; Domenech & Dreher, 2010; Forstmann et al., 2008, 2010). These recent studies, which also discuss decision
25 threshold modulation by the PFC, are in turn similarly grounded by much earlier work. For example as described by Luria
26 (Chapter 7, 1997), one of the primary functions of the frontal cortex is to regulate mental activity, or cortical tone, depending
27 on the task to be accomplished; notably supported by the discovery of “expectancy waves,” or contingent negative variation,
28 initially found by Walter et al. in 1964. This specific electroencephalographic (EEG) potential, shown to originate from
29 the frontal lobes, arises in preparation for the processing of a task-relevant stimulus, and disappears when this stimulus
30 stops to be reinforced—with the interpretation that cortical activity is increased to facilitate the processing of task-relevant
31 information.

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Such previous studies, with evidence accumulation models similar to the one we used (e.g., the DDM, LBA), provide support that the frontal cortex, and especially the *medial* frontal cortex, are engaged in decision threshold adjustment in perceptual decision making (Boehm et al., 2014; Domenech & Dreher, 2010). In particular, trial-by-trial modulations of medial PFC activity (EEG theta power, 4-8 Hz) have been related to an increase in decision threshold adjustment as a function of conflict (Cavanagh et al., 2011). In our study, the patients tested generally did not have medial frontal damage. It is therefore possible that the distinction between the roles of the left lateral PFC and the medial PFC may be somewhat different in lexical selection compared to the more traditionally used two-forced choice tasks in cognitive control studies outside of language. After all, the choice made in language production and in picture naming (as opposed to two-choice perceptual decision) include a larger number of alternatives and the neuronal basis underlying this type of choice may be different. Another possibility may be linked to the connectivity patterns between the left lateral PFC and the medial PFC

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4 in the patients we studied. Indeed, given the damage to the lateral PFC, connections between these two regions were also
5 damaged. It is therefore possible that the deficit in decision threshold adjustment we observe may be a consequence of this
6 disruption. Further studies will need to be conducted to specify the precise frontal anatomical basis for decision threshold
7 adjustment in lexical selection for speech production.
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10 ***Data-Derived Sequential Sampling Models for Brain-Damaged Patient Cognition Study*** This study has provided an
11 important first step, for how evidence accumulation models may provide insight into brain-damaged patient cognition and
12 their observed data—here specifically on left PFC patients and their threshold-adjustment impairments in a multiple choice
13 lexical retrieval task (picture naming). Such a development for how evidence accumulation may be extended to patients
14 in general, is preceded by several previous works which have also demonstrated success in using quantitative model
15 parameters, rather than raw performance data comparisons, to account for aspects of patient cognition. For example, a
16 diffusion model study on low and high anxiety individuals in a two-alternative lexical decision task (White et al., 2010), also
17 found principal effects in threshold adjustment. There is also the notably-related work of the neural network model designs
18 by Dell et al. (2013), which use quantitative model parameters to handle well the accuracy patterns of aphasic patients in a
19 word-repetition task, and in tandem also draw relationships to neural structures. Hence an interesting development may be
20 to consider integrating elements of this proposed evidence accumulation approach into these kinds of neural network models
21 (see Anders et al., 2015), to further develop the networks into accounting for the RT distributions as well (see Oppenheim
22 et al., 2010, for a beginning development in accounting for mean RT with accuracy). The limiting factor for achieving such
23 advanced methodologies however, as well as the reliability of the patient cognition conclusions (as ours reported here), is
24 the problem of often working with so few patients or patient observations, and hence future work would do well to pursue
25 augmented numbers.
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28 ***Limitations and Possible Improvements for Future Work*** We propose therefore that the current results be used to
29 advance theory, and stimulate subsequent psychometric modeling applications to explore further replication. While we did
30 replicate previous results on healthy speakers, it is worthwhile to note how various modifications to the fitting approach
31 may be used in future work to sharpen results or improve reliability. Firstly the most straightforward method would be
32 to include more brain-lesioned PFC patient numbers. We note that our analysis involved only six left PFC patients, in
33 which power in the significance tests was more difficult to achieve. Secondly, in contrast to augmenting patient numbers,
34 one can perform more reliable (and advanced) modeling by augmenting observation numbers in the experimental design.
35 Specifically, increased observation numbers would allow jointly modeling additional factors in tandem, which will parse out
36 variance assigned to the experimental predictors that may actually be due to other factors (Barr et al., 2013). For instance,
37 our modeling here and by Anders et al. (2015), experienced better model fit along the experimental predictors, semantic
38 context and repetition, by parsing out (jointly modeling also) lag effects, also known as trial distance (see Barr et al., 2013).
39 Hence along this line, a modeling improvement for future work is to jointly model also the items (here 36) with the subjects
40 and experimental predictors (see Baayen, 2004; Baayen et al., 2008), though here it was not possible, as it would leave
41 near only 1 observation per experimental design cell, and 3 model parameters to fit it. Lastly, one should also note that the
42 strength of the conclusions provided by these data-derived cognitive models also depends on the quality of the model fit,
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for which there are a variety of estimation options (e.g. hierarchical Bayesian by likelihood functions, or quantile-residual minimization, Rouder & Lu, 2005; Brown & Heathcote, 2003), each with their respective strengths (e.g. how error can be constrained by grouping assumptions, Rouder et al., 2007) and weaknesses (e.g. to what extent outliers perturb model fit, Ratcliff & Tuerlinckx, 2002).

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