

What, me saccade?

Abstract

Basically there is the vwp and it is used as a proxy for word recognition. This use follows from allopenna 1996, in which he showed the the proportion of fixations to different referents matches what would be expected with TRACE (after suitable transformation, of course). This resulted in over two decades of vwp use for such purposes. In 2021, mcmurray asked if that curve really was what we thought it was. Through an analysis of generating hypotheses of increasing (but still relatively minimal) complexity via simulation, bob showed that even in cases of moderate complexity, we were not able to positively recover the generating curve responsible for eye movements. why was this, and what are the implications for understanding these curves? In this paper, we revisit the 2021 princess bride paper and offer an explanation for the demonstrated bias of the simulations. from this, we propose a new method for using vwp data to estimate underlying activation curves. finally, we top this all off by comparing the proposed method to allopenna 1996 in a head-to-head, single elimination match up, a battle suitable for mount olympus itself in which two ultimate theories fight to a bloody and violent death to see which has been ordained by god to reign as total champion in the hearts and minds of language psychologists the world over.

Notes

Also not sure where to include information on the linking hypothesis

Sections that are more “narrative” are less fleshed out, including introduction. Citations are hard coded but no bib created yet. Some plots need to be completely redone. There is also some meta-narrative that is partially intended for the reader but mostly intended for the author

1 Introduction

Spoken words create analog signals that are processed by the brain in real time. That is, as the spoken word unfolds, a cohort of possible resolutions are considered until the target word is recognized. The degree to

which a particular candidate word is recognized is known as activation. An important part of this process involves not only correctly identifying the word but also eliminating competitors. For example, we might consider a discrete unfolding of the word “elephant” as “el-e-phant”. At the onset of “el”, a listener may activate a cohort of potential resolutions such as “elephant”, “electricity”, or “elder”, all of which may be considered competitors. With the subsequent “el-e”, words consistent with the received signal, such as “elephant” and “electricity” remain active competitors, while incompatible words, such as “elder”, are eliminated. Such is a rough description of this process, continuing until the ambiguity is resolved and a single word remains.

Our interest is in measuring the degree of activation of a target, relative to competitors. Activation, however, is not measured directly, and we instead rely on what can be observed with eyetracking data, collected in the context of the Visual World Paradigm (VWP) (Tannenhaus 1995)[?]. In the last few years, researchers have begun to reexamine some of the underlying assumptions associated with the VWP, calling into question the validity of current methods. We present here a brief history of the VWP and an examination of contemporary concerns. We further address some of these directly, presenting a different method for relating eyetracking data to word activation. We demonstrate how this is resolved and conclude by comparing our new method to the original impetus for the current. This paragraph needs work but it mostly covers the gist of what I am trying to convey, namely we are about to go from history → current stuff → proposal and comparison → back to beginning.

I addressed above “contemporary concerns” but I could elaborate on the more fully here. In particular, this paper is motivated by the realization that the linking hypothesis is implicitly xyz with a few contenders (none of which implemented) and then Bob pretty much showing that the leading implicit assumption is “patently false”.

2 A brief history

We begin with a brief history to give context to later discussion. In particular, we will consider one of the leading theoretical models in speech perception, TRACE, followed by the introduction of the leading experimental paradigm, the VWP. We examine empirical evidence for the relation between these, and relevant theoretical advancements that have been made. Topics here are presented only briefly and limited to those directly relevant to the present work. For a fuller discussion of the history and uses of vwp, use google. (Or Huettig 2011b? Haven’t read, found this citation in Magnuson)

An outline of the presentation (for internal use only):

1. TRACE in 1986 along with connectionist model of language

2. VWP by Tannenhaus 1995
3. VWP + TRACE, allopenna 1996
4. As far as I can tell, it's Bob's 2010 paper that was first to
 - (a) Look at individual differences in word recognition (relevant for the "group distribution of curves" hypothesis and
 - (b) Introduce parametric forms to be fit to the data (the assumption we continue to run with), or at very least, introduce ones that are interpretable

I don't really like any of these paragraphs and each needs to be fleshed in more detail, but its mostly expository and not mission critical.

TRACE How speech is perceived and understood has been a subject of much debate for a significant portion of psycholinguistic's history. Starting in the 1980s and persisting today, many researchers subscribe to what is known as the connectionist model of speech perception. Briefly, this model posits that speech perception is best understood as a hierarchical dynamical system in which aspects of the model are either self reinforcing or self inhibiting with feedforward and feedback mechanisms. For example, hearing the phoneme \h\ as in "hit" will "feedforward", cognitively activating words that begin with the \h\ sound. These activated words then "feedback" to the phoneme letter, inhibiting activation for competing phonemes such as \b\ or \t\. In 1986, McClelland and Elman introduced the TRACE¹ model implementing theoretical considerations into a computer model [?]. Maybe useful here to discuss activation, sigmoid shape, etc.,

VWP To briefly illustrate, the VWP is an experimental design in which participants undergo a series of trials to identify a spoken word. Typically, each trial has a single target word, along with multiple competitors. The target word is spoken, and participants are asked to identify and select an image on screen associated with the spoken word. Eye movements and fixations are recorded as this process unfolds, with the location of the participants' eyes serving as proxy for which words/images are being considered.

Relating TRACE to VWP It was against simulated TRACE data that Allopenna (1998) found a tractable way of analyzing eye tracking data [?]. By coding the period of a fixation as a 0 or 1, depending on the referent and taking the average of fixations towards a referent at each time point, Allopenna was able to create a "fixation proportion" curve that largely reflected the shape and competitive dynamics of word activation suggested by TRACE. Since this publication, it has been this transformation of the data that has guided the last 25 years of VWP research. Could include here an illustration of VWP. also a lot of other various details from allopenna

¹TRACE doesn't stand for anything – the name is a reference to "the trace", a network structure for dynamically processing things in memory

Parametric Methods and Individual Curves While there have most certainly been advancements to the use of the VWP for speech perception and recognition (and expanded into related domains, such as sentence processing and characterizing language disorders (according to Bob)), we limit ourselves here to one in particular. In 2010, McMurray et al expanded the domain of the VWP by introducing emphasis on individual differences in participant activation curves. Two aspects of this paper are relevant here. First, although they were not the first to introduce non-linear functions to be fit to observed data, they did introduce a number of important parametric functions in use today, namely the four (or five) parameter logistic and the double-gauss (asymmetrical gauss). Second, which I suppose was also introduced by Mirman (2008) to some degree (though I have not read it yet) is specifying individual subject curves across participants. This has been critical in that:

1. The parameters of the functions describe interpretable properties
2. This made the idea of distributions of parameters for a particular group a relevant construct

This also served as the impetus for investigating group differences in word activation through the use of bootstrapped differences in time series (Oleson 2017) and the subsequent development of the `bdots` software in R for analyzing such differences. (A history of exploring differences in group curves can be found in (Seedorff 2018 `bdots` paper)).

This brings us to the current day, where the state of things is such that TRACE-validated VWP data is widely used to measure word recognition by collecting data on individual subjects and fitting to them non-linear parametric curves with interpretable parameters. Context in hand, we are now able to introduce some of the main characters of our story, specifically how data in the VWP is understood and used.

linking hypothesis? not sure if this is a section worth elaborating on. more interesting narrative, but relevant inasmuch as our conclusions are adjacent to the conversations regarding linking hypotheses. some minor inclusion of that here may make for an interesting call back/tie in when i bring up the saccade and its proximity to the construct (activation) in question, which I note I haven't fully addressed yet. Specifically, the fact that there is some construct in question that we are trying to measure through physiological response.

3 Where we are now

Context in hand, we are ready to introduce some of the characters of our story. this includes the finer points of the vwp, eye tracking data, and how allopena's introduction ties in with bob's parametric

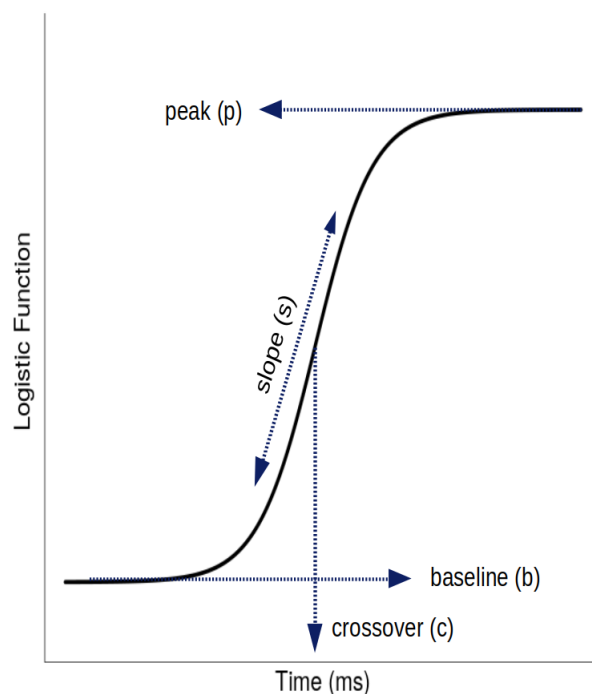


Figure 1: An illustration of the four-parameter logistic and its associated parameters. also might be premature to include here since i haven't even shown the logistic or detailed its case, only its existence. Still, ill leave this up so that the image can be moved around if needed. What i would do in this caption otherwise is explain each of the parameters.

3.1 anatomy of eye movements

There are three components of eye movements with which we are concerned with here. The first two, saccades and fixations, are associated with physical mechanics of eye movements; the third, oculomotor delay, is a phenomenon related to the association between cognitive activation and physiological response. We will briefly introduce each of these topics here.

Saccades and fixations: Rather than acting in a continuous sweeping motion, as our perceived vision might suggest, our eyes themselves move about in a series of short, ballistic movements, followed by brief periods of stagnation. These, respectively, are the saccades and fixations.

The short ballistic movements are known as saccades, periods of between 20ms-60ms (source? more accurate times?) in which the eye is in motion and during which time we are effectively blind. Once in motion, saccades have no ability to change their intended destination. Following the movement itself is a period of stillness known as a fixation, itself made up of a necessary refraction period from the saccade (time?)

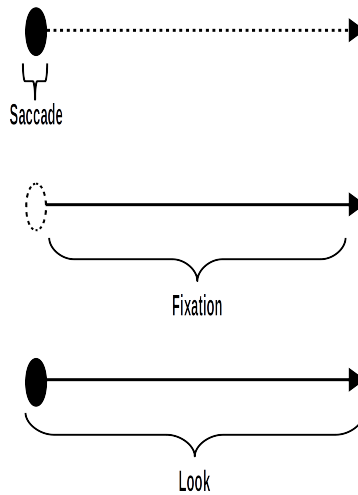


Figure 2: this obviously looks terrible here, need to redo image from odg file

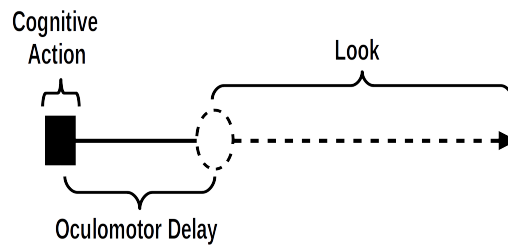


Figure 3: this also could probably be reformatted or made bigger

followed by a period of voluntary fixation; the typical duration of a fixation is (some length). Together, an initiating saccade and its subsequent fixation is known colloquially as a “look”. See Figure 2.

Oculomotor delay: While the physiological responses are what we can measure, they themselves are not what we are interested in. Rather, we are interested in determining word activation, itself governing the cognitive mechanism facilitating the movements in the eyes. It’s suspected/stated/known (source?) that upon finishing a particular saccade, the mind is already anticipating where it will move next. What is relevant for our purpose here, however, is that the period of oculomotor delay is random, resulting in biased observations between what we are able to measure and what we are interested in discovering. How this phenomenon relates to saccades and fixations is demonstrated in Figure 3.

Alternatively, there is a full figure i could use here:

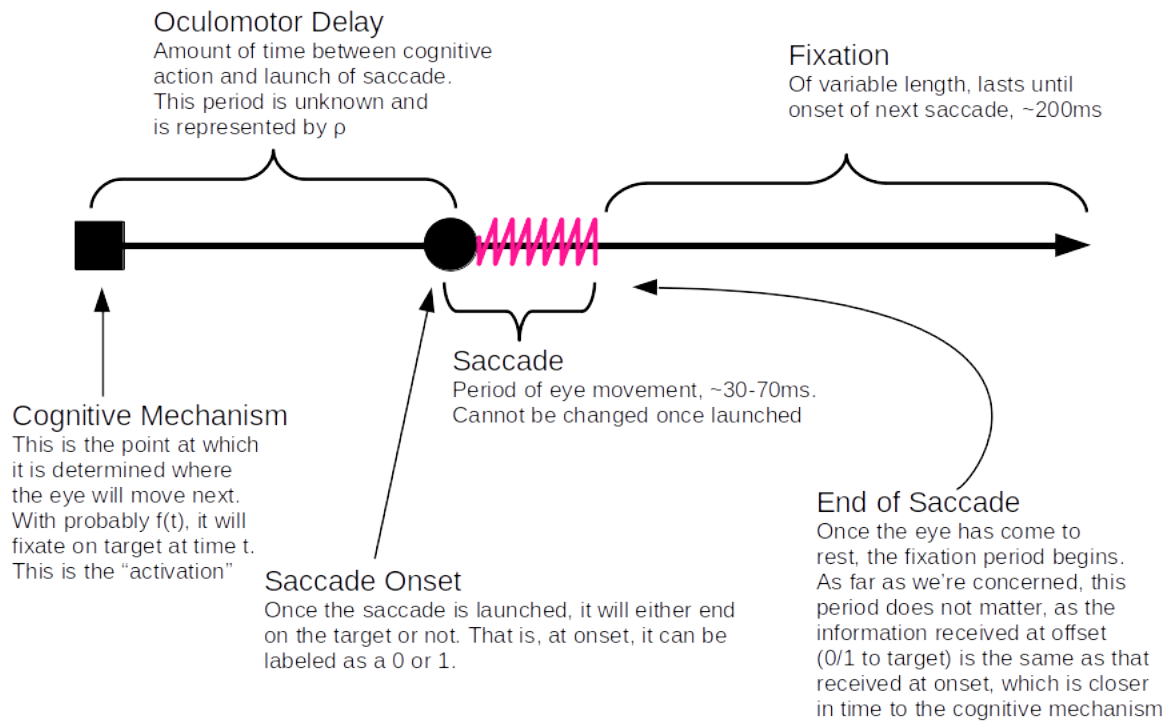


Figure 4: This figure actually doesn't look too bad, but may be better when articulating how saccades measured and why (also includes info on $f(t)$, ρ , etc., so maybe we will present this later around the time of simulation

(thinking out loud here) The organization on this one is a little up in the air. It’s interesting to include and will be one of the things we discuss in more detail. it seems a bit premature to bring in the whole idea of ρ *here* when that might be better suited to preparation for the simulation/comparison section.

3.2 VWP data

technically this kind of includes the “proportion methd” too, not sure if i should break up or how exactly to treat this

We now consider how the aforementioned mechanics relate to the VWP. In a typical instantiation of the VWP, a participant is asked to complete a series of trials, during each of which they are presented with a number of competing images on screen (typically four). A verbal cue is given, and the participants are asked to select the image corresponding to the spoken word.

An individual trial of the VWP may be short, lasting anywhere from 1000ms to 2500ms before the correct image is selected. Prior to this, the participants eyes scan the environment, considering images as potential candidates to the spoken word. As this process unfolds, a snapshot of the eye is taken at a series of discrete steps (typically every 4ms) indicating where on the screen the participant is fixated. While there is evidence of cognition happening behind the scenes in a continuous fashion (spivey, mouse trials), an individual trial of the vwp may contain no more than four to eight total “looks” before the correct image is clicked, resulting in a paucity of data in any given trial.

To create a visual summary of this process aggregated over all of the trials, a la Allopena, a “proportion of fixations” curve is created, aggregating at each discrete timepoint the average of indicators indicating that a participant is fixated on a particular image. A resulting curve is created for each of the competing categories (target, cohort, rhyme, unrelated), creating an empirical estimate of the activation curve, $f_{\theta}(t)$. See Figure 5. Mathematically, it looks like this:

$$y_{it} = \frac{1}{J} \sum z_{ijt} \tag{1}$$

where z_{ijt} is an indicator $\{0, 1\}$ for subject i in trial j at time t and such that

$$f_{\theta}(t) \equiv y_t. \tag{2}$$

Nonlinear parametric curves can be fit to the observed y_t as is done in the `bdots` package in R (see chapter 1).

Here, I’ve copied and pasted what I had about this elsewhere. There are bits and pieces I like from each,

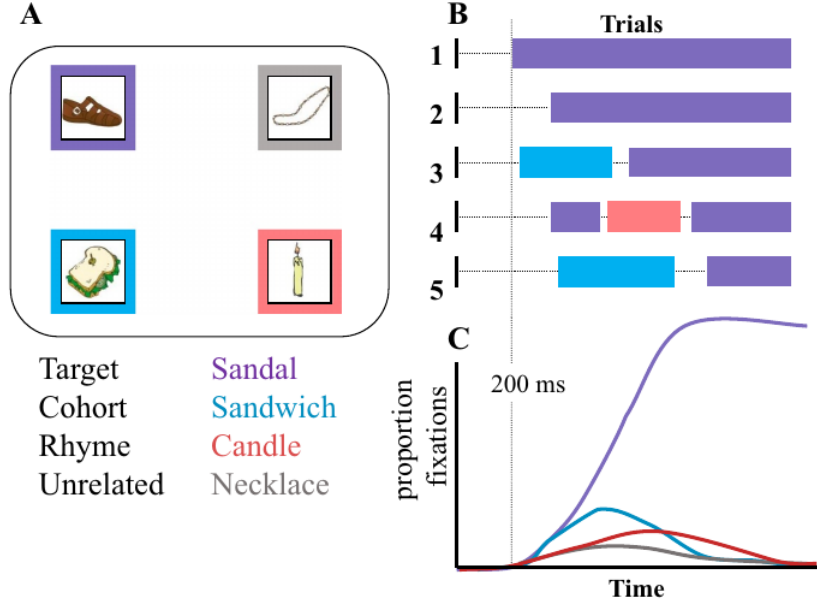


Figure 5: This is screenshoted from Bob’s princess bridge paper. I would like to reconstruct a similar illustration here as it does a great job illustrating the point. *However*, this section as it stands may make more sense elaborated elsewhere, in particular where I give a mathematical treatment to what the “fixation curve” is

so for now going to leave them both and make them “do it” and have babies later.

One method employed to use this data involves measuring intervals of fixation to a target over a series of trials. For each trial j and time point t (typically sampled at intervals of 4ms, i.e., $t = 0, 4, \dots, 2000$), we collect a sample of z_{jt} , an indicator of whether a participant is fixated on the target object at that point in time. Averaging over the collection of trials, we construct an estimate of $f_{\theta}(t)$,

$$y_t = \frac{1}{J} \sum_j z_{jt}.$$

In other words, it is implicitly assumed that the trajectory of the eye follows the trajectory of activation, where the average proportion of fixations at a particular time is a direct estimate of activation. As each individual trial is only made up of a few ballistic movements, the aggregation across trials allows for these otherwise discrete measurements to more closely represent a continuous curve. Curve fitting methods, such as those employed by ‘bdots’, are then used to construct estimates of function parameters fitted to this curve.

[this really belongs with a compare-and-contrast section following saccades. primary benefit of this....over what?] One of the primary benefits of this method is that it captures the duration of fixations, with longer times being associated with stronger activations. This becomes important when differentiating fixations associated with searching patterns (i.e., what images exist on screen?) against those associated with consideration (is this the image I’ve just heard?). A shortcoming, however, is that it conflates two distinct types

of data, generated via different mechanisms, the fixation and saccade.

4 where we are going

ok so now what

4.1 princess bride paper

From the abstract of this paper: “All theoretical and statistical approaches make the tacit assumption that the time course of fixations is closely related to the underlying activation in the system. However, given the serial nature of fixations and their long refractory period, it is unclear how closely the observed dynamics of the fixation curves are actually coupled to the underlying dynamics of activation.”

This is a critical statement to be made, and in a sense, it ties into the general idea of the linking hypothesis presented in Magnuson. I make no attempts here to challenge or otherwise present a drastic new linking hypothesis. Rather, I take assumption made here: an outline (because i love enumerated lists) of the events, in order, is as follows:

1. Some measure of activation is occurring in the mind as a consequence of audio stimuli
2. Through some indeterminate mechanism (linking hypothesis), this level of activation governs the mechanics of eye movements, specifically the direction/location of saccades and duration of fixation
3. The resulting mechanism governing *saccades*, namely, where to look *at a specific period in time* follows a sigmoid curve, here specifically the four-parameter logistic

n.b., bob refers to these as “fixation curve”

In 2022, McMurray brought into question the validity of a standard VWP analysis, and a more thorough treatment of his presented arguments is warranted but for the time being counts as narrative and so the elaboration will wait. For now, we will present those elements that are crucial for understanding the direction of the methodology to be presented.

In short, the question that is being gotten at is: are we able to recover the underlying dynamics of the system in question (activation) in light of the “nature of the fixation record as a stochastic series of discrete and fairly long lasting physiologically constrained events?” In short, the answer is no. McMurray notes that the typical, unspoken assumption implicit in VWP studies is the “high frequency sampling” (HFS) assumption, which states that the underlying activation at some time determines the probability of fixation. He then goes on to note that this is “patently” untrue and is nothing more than a polite fiction.

Nonetheless, it is useful to compare the relationship of the underlying dynamics (as we will elaborate upon further, a generating function) with the observed data in the context of the HFS, relative to other, more complex assumptions. This is done through a series of simulations, each with their own set of stochastic mechanics determining eye movements and fixations. In all cases, however, it is assumed that there exists an underlying generating function that, at any particular time, is responsible for dictating some aspect of a subsequent fixation. We will start with an overview of the general algorithm for an individual subject, followed by a brief summary of each of the simulations.

Algorithm:

1. A set of generating parameters for the four-parameter logistic is drawn from an empirically determined distribution. This curve, $f_{\theta}(t)$, is treated as the probability of fixating on a target at time t
2. After a random offset start time, t_0 , a binomial random event is drawn determining the probability of fixating on the target, $p \sim \text{Bin}(f_{\theta}(t_1))$
3. After this initial draw, a fixation occurs for a period of time:
 - (a) Under the HFS assumption, this period is instantaneous – that is, whatever the time, t is also the probability of fixation
 - (b) Under the FBS assumption, the length of the fixation draws from a gamma distribution, ending at time t_2
 - (c) Under the FBS+T assumption, again a random length fixation is drawn from a gamma distribution, but with a higher mean value if the fixation is drawn to the target
4. idk im describing this weird – maybe come back to this list later

what I can do instead is offer a brief written summary of each of the methods.

High Frequency Sampling (HFS) The underlying activation of the word *is* the probability of fixating at a particular time.

Frequency Based Sampling (FBS) The FBS assumption differs from that of the HFS assumption in that the observed data is gathered from a period of fixations of random duration. Once each fixation is “drawn”, the subject remains fixated on a particular object for the full length of the fixation. The next fixation’s location is determined at the *onset* of the previous fixation. In particular, this simulation assumes that immediately once a fixation is made, the subject begins preparing to launch their next saccade

Frequency Based Sampling + Target (FBS+T) This simulation is identical to the previous with the exception that duration of fixations to the target, while still random, follow a different distribution than fixations to non-targets, with longer durations afforded to target fixations to account for “information gathering behavior”.

Naturally, as the complexity of the assumptions increased, so did the observed bias in recovering the parameters of the generating function.

At any rate, I'm getting to caught up in the particulars when what I really want or need to say is quite simple. It comes down to this: *the only observed behavior governed by the generating curve is the saccade when launched.*

Saccades The entirety of the bias resulting from FBS and FBS+T we the consequences of two facts, or put differently, there are two sources of bias that we need to consider:

1. We were “observing” data points $\{0,1\}$ at any time t without having observed any behavior from the generating curve at that time (not sure what to call this, added observation bias?)
2. When we did sample directly from the curve at fixation onset, we were actually sampling from the onset of the previous fixation (delay bias)

The solution to this, then, is to simply use the saccade data, or only collect as $\{0,1\}$ the instance at which a fixation occurs, discarding the rest. While this does not address the delay bias, it does remove a significant amount of bias from the added observations. One obvious shortcoming is that it dismisses all “information gathering behavior” that could otherwise be gleaned from the duration of fixations. To what effect this or other enhancements may have on the efficiency of this data are yet to be seen, but at very least it offers a more clearly defensible relationship between the observed data and the generating function.

The idea of information gathering behavior is a useful one, but it assumes a linear relationship between the length of time of the fixation and strength of activation. However, one might suspect that after a period of necessary refraction, each subsequent period of time gives exponentially more weight to the argument of activation. A potential consequence of this is that an indication of fixation 50ms following the launch of a saccade may convey different information than the indication of a fixation still present 300ms following a saccade, despite the fact that these are recorded equally as $\{0,1\}$. That is, under the present system, rather than indicating the gathering of more information, longer fixations simply increase both the bias due to added observation *and* the amount of bias on account of delay (as the subsequent fixation will have been determined further removed from its occurrence when following a longer fixation). On the other hand, a mechanism for recording information-gathering behavior may be more readily implemented in a saccade-style method whereby each saccade is weighted by the length of its subsequent activation, for example.

From here, we will describe the proposed saccade method in more detail, compare the results of using saccade data against what was found in mcmurray, see that it largely resolves the problems, and then ask the natural question: how does this stack up against what allopenna found?

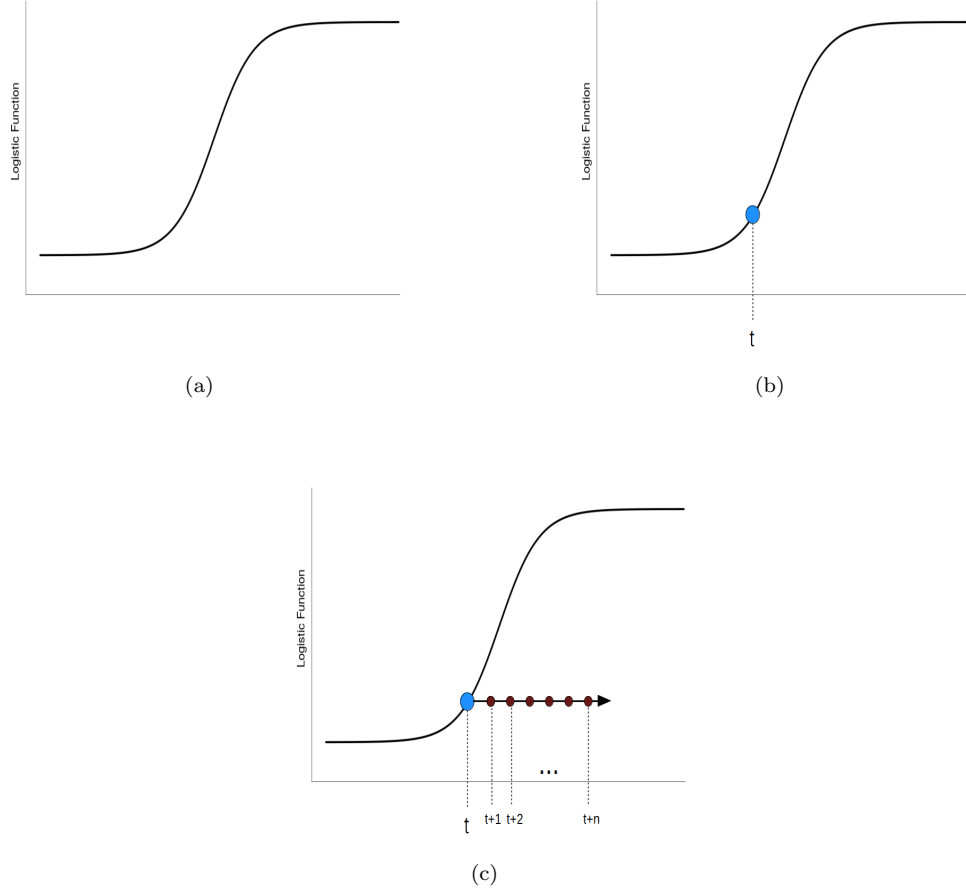


Figure 6: These illustrations can all be made larger (they were made for slides in an image editing program), but they illustrate the main point. **(a.)** here we see an example of a generating logistic function **(b.)** at some time, t , a saccade is launched (in the algorithm, a binomial is drawn with probability $\text{Bin}(f_\theta(t))$) **(c.)** at subsequent times, $t+1, \dots, t+n$, we are recording “observed” data, adding to the proportion of fixations at each time but without having gathered any additional observed data at $f_\theta(t+1), \dots, f_\theta(t+n)$, thus inflating (or in the case of a monotonically increasing function like the logistic, deflating) the true probability.

Also note somewhere that we are primarily limiting discussion to the logistic here. A brief treatise on the asymmetric gaussian is given in the appendix

4.2 Saccade method

If we are to consider eyetracking data samples from some probabilistic curve, it becomes necessary to differentiate between the two types. A saccade launched at some time, t , can be considered a sample from a data-generating mechanism at t . The duration of time between a given saccade and the one following follows a different mechanism altogether. By clearly delineating the mechanism from which we are sampling, we are able to reduce observed bias in the reconstruction of the activation curve.

In light of this, and in contrast to the fixation method, we propose estimating the activation curve with the saccade data alone. The primary benefit of this is two-fold. First, as suggested above, by decoupling two different types of data we are able to be more precise in what it is we are sampling. Second, as explained in the previous section, we are removing the added observation bias.

An important difference between these two methods is in the structure of the data itself. Whereas the former collects an array of data, with an observation for each time point in each trial, the saccade method is sparse, with the observed data indicating the outcome of the saccade, as well as the time observed. It is best represented as a set of ordered pairs, $\mathcal{S} = \{(s_j, t_j)\}$, with j indexing each of the observed saccades, and with

$$s_j \sim \text{Bern}(f_\theta(t_j)). \quad (3)$$

A value of $s_j = 1$ indicates a saccade resulting in a fixation on the target.

As with the proportion method, the observed data can be used as input for `bdots` to construct estimates of generating parameters.

5 Simulations Against Princess Bride

Here we now replicate the results of the princess bride paper, though with a few adjustments in light of the previous discussion. In particular, we noted that the two types of bias presented in the original simulations were added observation bias and delay bias. The first, added observation, we are addressing with the proposed saccade method. As to the second, we first elaborate here with a brief discussion of the delay bias and how it may also related to oculomotor delay.

In the original princess bride paper, FBS and FBS+T were only differentiated by the amount of additional

bias introduced in FBS+T. Specifically, by drawing fixations to the target from a gamma distribution with a larger mean, we were both increasing the amount of added observation and delay bias, both consequence of the longer fixation period. Understanding that these differ in degree rather than kind, we collapse them into a single construct here, as we will elaborate on shortly.

We also make adjustments to how we deal with oculomotor delay. As was shown in the simulations with the HFS assumption, a fixed oculomotor delay simply resulted in a horizontal shift of the estimated function, having otherwise no impact on the *shape* of the function. In contrast, added observation and delay bias both drastically impact the final shape. Further, in typical instances in which we are using the VWP, we are more frequently concerned with the relative difference between two curves rather than the curves themselves. The magnitude and relative location of such differences will be preserved under a horizontal shift, having ultimately little consequence on the resulting analysis.

In light of this, we will seek to combine the functional impact of oculomotor delay with the impact of more complex eye behavior in the following way: we recognize that with the exception of the HFS assumption (which is not considered here), any observation at time t will have been prompted at some time previously (that is, drawn from the activation curve). The length of this delay will be denoted $\rho(t)$. In the case of the HFS assumption, for example, this simply would have been $\rho(t) = 200ms$. For the FBS and FBS+T case, it would have been $\rho(t) = 200ms + \text{length of previous fixation}$. As such, we can reduce the conditions under which we compare the fixation and saccade methods to two scenarios:

1. $\rho(t)$ is a constant function, including zero
2. $\rho(t)$ is a random variable, independent of the value of t_j

As such, we will not be including as a part of this the original 200ms oculomotor delay.

As in the princess bride paper, we will let $f_\theta(t)$ be a four parameter logistic, representing of generating or activation curve. Understanding that what we observe at time t was drawn from this function at time $t - \rho(t)$, we will differentiate the underlying activation curve the the observed data,

$$g_\theta(t) = f_\theta(t - \rho(t)) \tag{4}$$

Each simulation will be conducted with $N = 300$ trials, sampled from the same data generating function for each, with the attempted recovery of the generating curve done using the `bdots` package.

Note somewhere: this and all other code used available on my github

5.1 Simulations

Funny it seems that all this shit is on my home computer. even then, its been long enough now that i don't quite remember. probably a worthwhile endeavor to just rerun those simulations since i know far more definitively now why they work and what i want them to do

things that i will include in each section

1. plots of generating and estimated curves (3 curves total, no shift)
2. histogram of parameter estimates against each (\leftarrow and actually he plots bias against these bad boys not pars themselves)
3. mean integrated squared error or. R^2
4. fun little quips

So reading it a bit more closely, here's the rub. he does this for 1000 subjects. only one set of histograms, showing bias for each, nbd. however, he doesn't have just *one* generating curve against which he compares – he does this for all, and most of his analysis is on the parameters. What we will do look at representative collection of them, have a table of MISE and R^2 (or whatever) and then have an average for fixation method and saccade method, so 3 by 9 table or whatever. i do not intend on replicating all of this distributino of parameter business because i quite simply do not care.

5.1.1 Fixed Delay

Since horizontal shifts don't matter (as evidenced by HFS simulation and common sense) we let this be zero to reduce complexity. Doing so also removes by design the delay observation bias, showing how added observation bias is what fucks things up

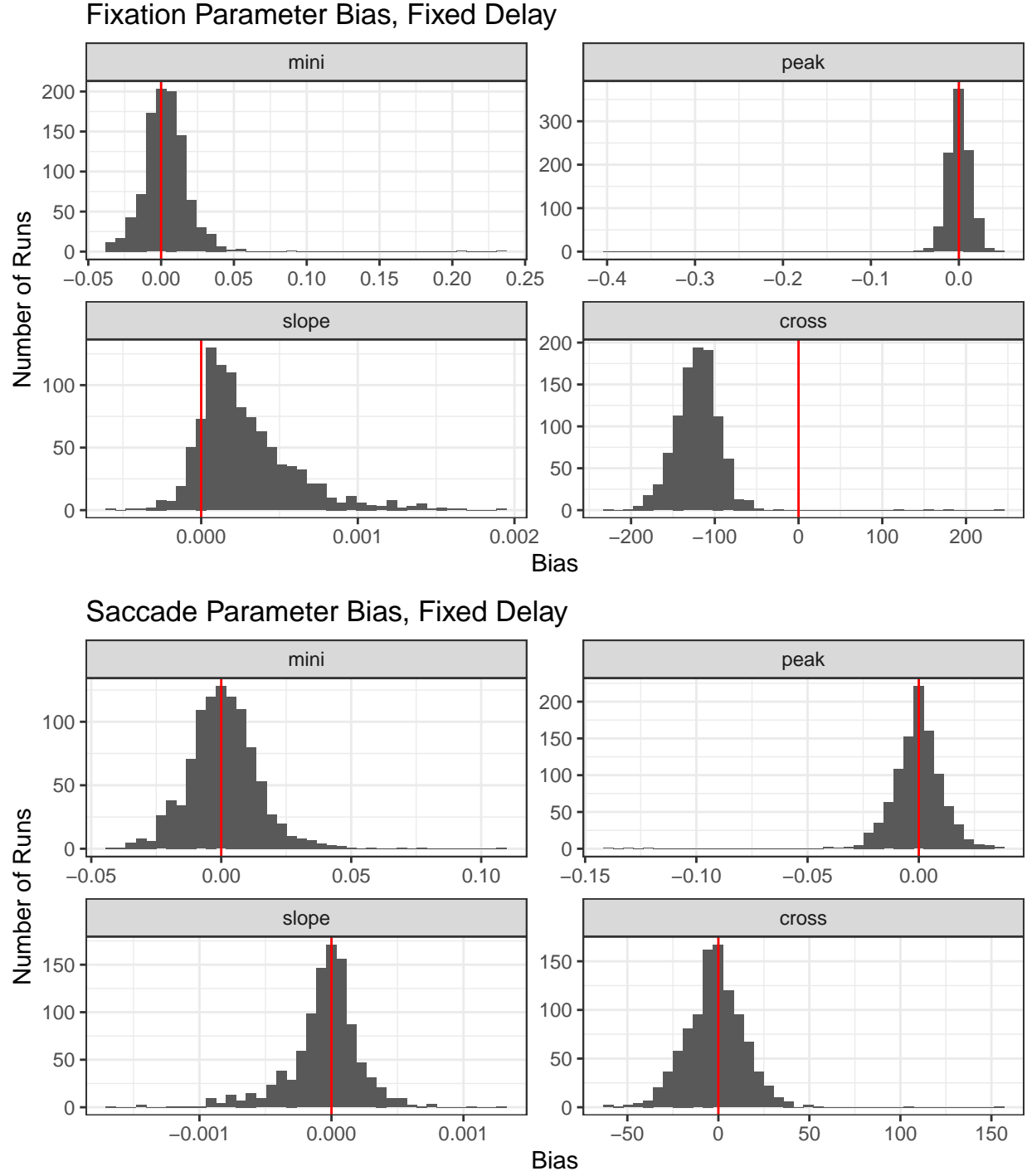


Figure 7: Distribution of parameter bias for fixation and saccade methods under fixed-delay simulation. The bias induced in the fixation method is all a consequence of the added observation bias. Somewhere note these are TRUE PARS - FIT PARS. We see evidence that added observation bias has the effect of “pulling” the curve forward, resulting in later cross over and less steep curves

blahkja ls

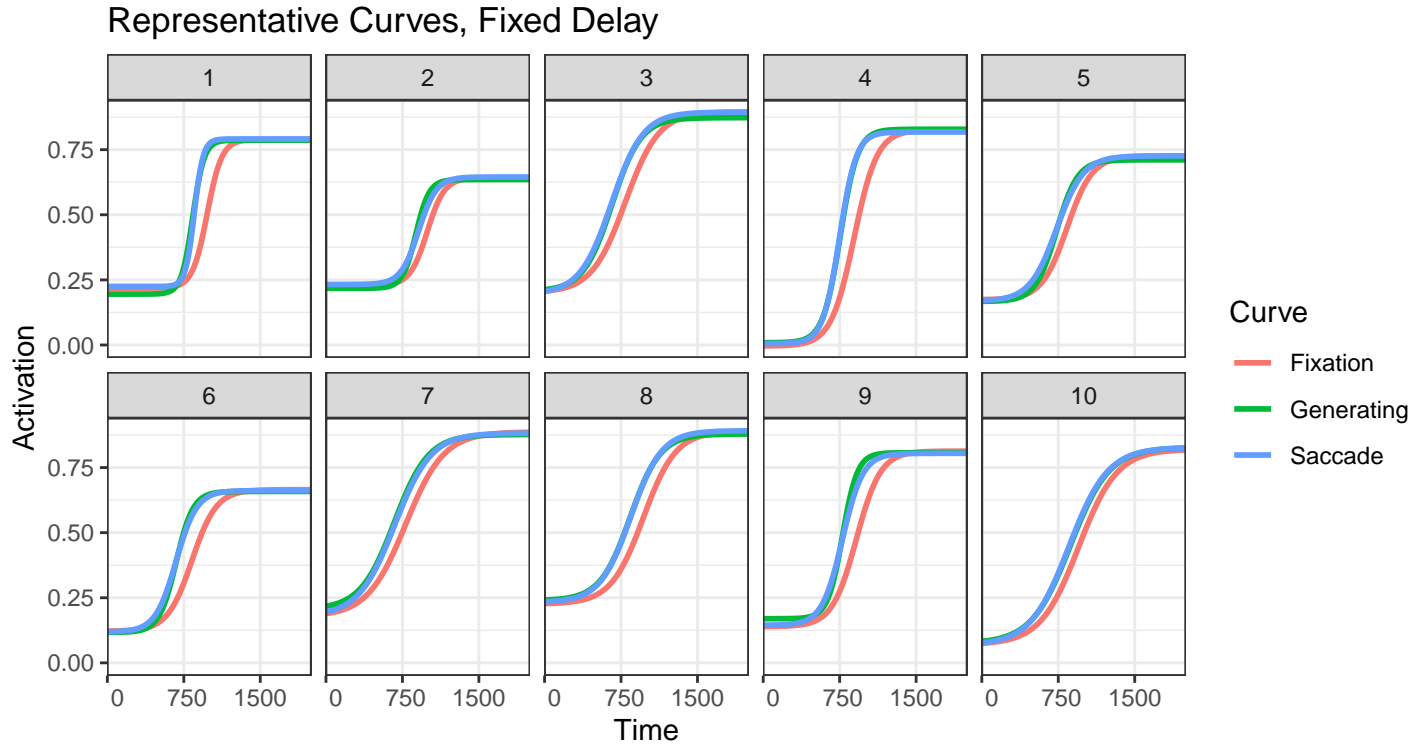


Figure 8: Representative collection of fixed-delay curve, including the generating function, as well as estimated curves from fitting data using fixation and saccade methods

5.1.2 Random Delay

This is the kicker. We will just add a random delay for each time, combining fbs and $fbs+t$. i forget the exact details, also why i should just go back and rerun things.

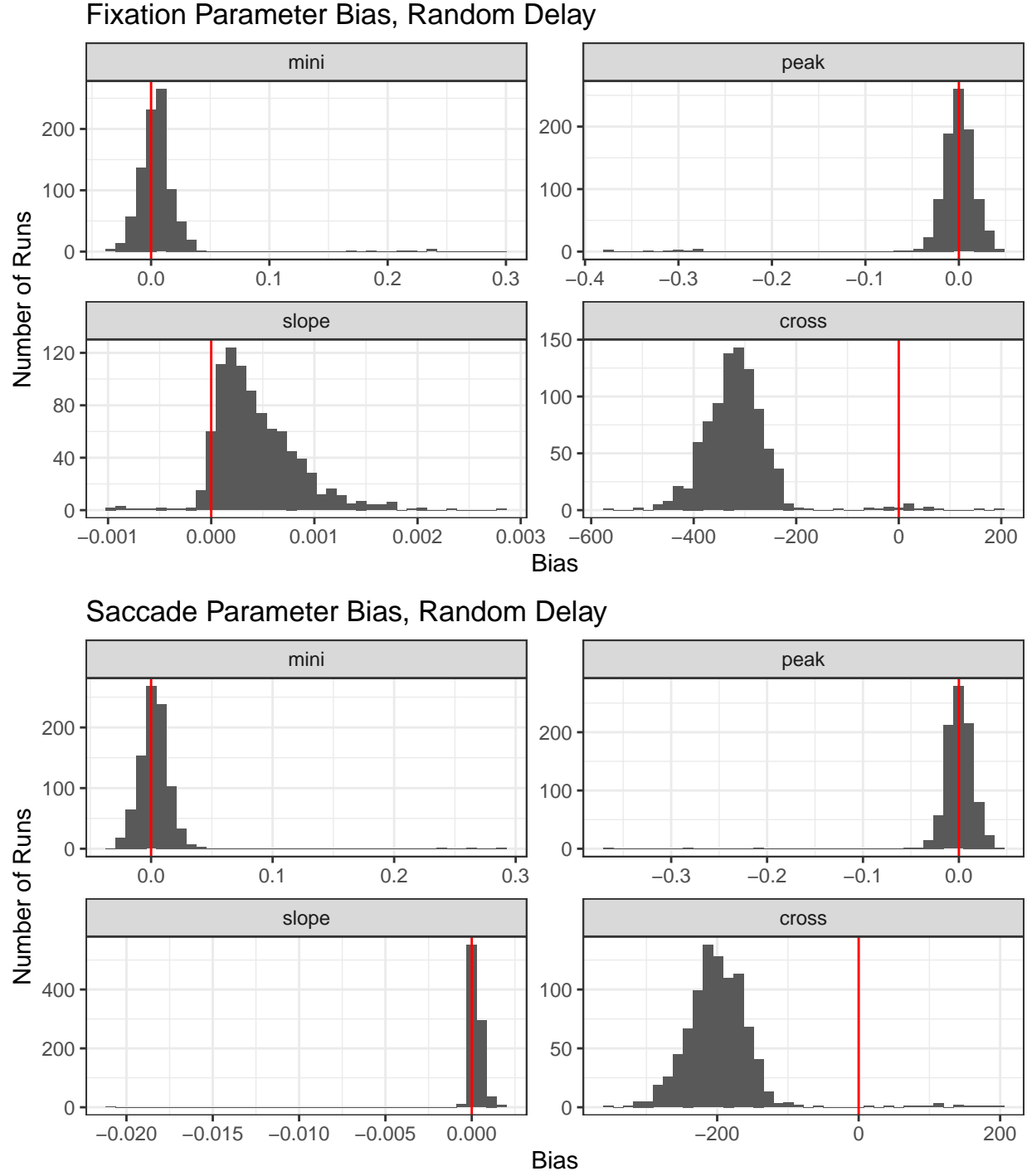


Figure 9: Distribution of parameter bias for fixation and saccade methods under random-delay simulation. The bias induced in the fixation method is all a consequence of the added observation bias AND delay bias, which has consequence of further shifting crossover parameter underestimating slope, but now with saccade too. Somewhere note these are TRUE PARS - FIT PARS. We see evidence that added observation bias has the effect of “pulling” the curve forward, resulting in later cross over and less steep curves

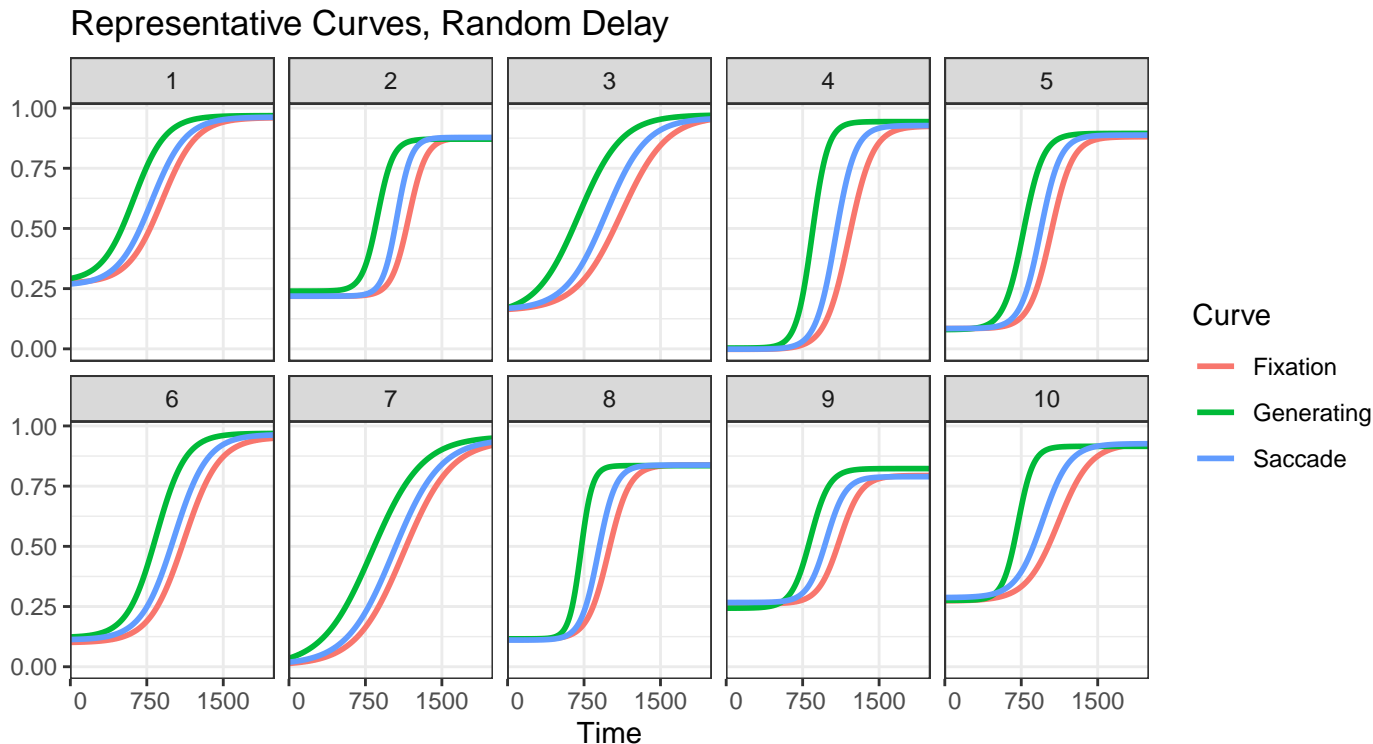


Figure 10: Representative collection of random-delay curve, including the generating function, as well as estimated curves from fitting data using fixation and saccade methods

See though I could have all of the results together here or in individual sections. I think it makes more sense to have them together for an “overall” sort of discussion thing

Curve	Delay	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Fixation	Fixed	1.95	8.18	11.40	13.28	15.98	215.67
Saccade	Fixed	0.01	0.16	0.32	0.52	0.56	78.22
Fixation	Random	20.25	50.95	68.60	73.08	90.92	192.56
Saccade	Random	5.74	21.42	29.29	33.40	40.63	185.79

Table 1: Summary of mean integrated squared error of the fits with their generating curves

6 Compare with TRACE

This section needs to move to the prominent spot since its the meat of my argument Here is a snippet I stole from elsewhere (that i also wrote):

In Allopenna (1998), they say ”... A time course analysis of the proportion of fixations on the target object (current referent), cohort competitor, and unrelated objects suggested that this measure would be

extremely sensitive to the uptake of information during the lexical process. *Furthermore, the shapes of the functions suggested that they could be closely mapped onto activation levels*...*We also put forth an explicit account of the mapping between activation levels simulated by the TRACE model and fixation probabilities.*”

That is, it seems like the connection between fixations and activation was first posited due to the similarities in shape of the functions with TRACE models. (“linking hypothesis” – also, see magnuson)

Actually, this entire paper gives an explicit transformation from what was predicted in TRACE to what should be predicted as proportion/probability of fixation

6.1 Collecting TRACE data

My main concern in this entire endeavor is that the TRACE data never baselines at 0, pretty much no matter what. I can change luce choice temperature, I can change whether or not i use empirical peak/baseline for scaling factor, it doesn’t matter. i never get near 0. Going back to email where we discussed issues with scaling factor, there was this line:

When I implement those things, I get a smooth function that goes up from .0002 at $\text{maxact} = -.2$ to .739 at $\text{maxct} = .55$.

I was able to replicate these numbers. The issue is on pg 23 of the SLI paper, it looks like the plot for the model is the scaling factor, not luce choice transform times the scaling factor

6.2 TRACE Data

In McMurray (2011), a range of simulated TRACE data was collected across the space of hyperparameters, each simulation containing 14 trials, each with a Target, Cohort, Rhyme, and Unrelated object (Appendix B. in Bob’s paper). Here, we included only TRACE data associated with the default parameters specified in jTRACE (magnuson) and found the average TRACE activation across frames/cycles for each object (target, cohort, etc.,).

As has been discussed elsewhere (Allopena, Magnuson, Bob), TRACE outputs a set of activations relative to all of the words included in its lexicon, and what is needed is a linking function that is able to return a simple mapping of TRACE activations to an estimated probability. We follow the steps given in the appendix of McMurray (2011), which we briefly outline here.

First is an implementation of the Luce Choice Rule (from somewhere) which dictates that, when considering the probability of fixation of a candidate word, we need only consider the probability of fixation

relative to the other candidates present rather than our entire lexicon. This means that in the context of the VWP, our probability of fixation would be relative to the (usually three) other objects on the screen, rather than the thousands of words with which we may be familiar.

Really, though, does this have to be done at all? Can I not simply point to bob's paper and be like, look, this is the trace data and this has all the data on the subjects. here we are just going to look at the results because nothing else matters or is relevant. Or this can be in the appendix

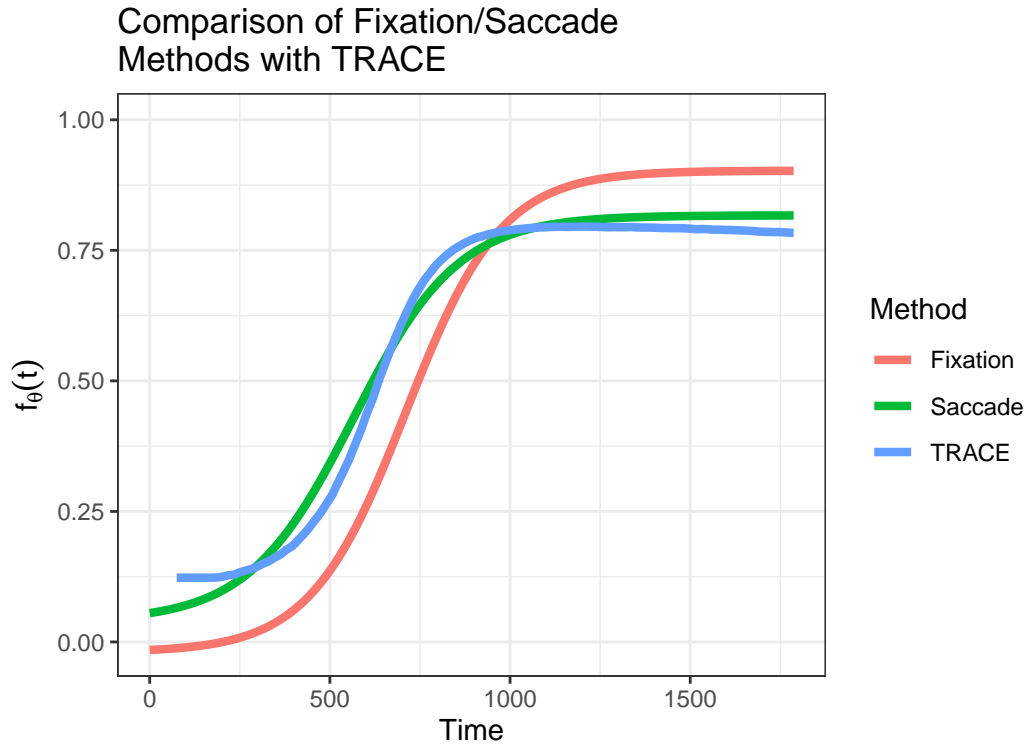


Figure 11: this is just the mean value of the curve parameters. also only includes NH subjects. I feel like confidence intervals would make this chart look messy so might just instead include table of mean integrated squared error using approxfun since trace only has 108 data points and need a function to integrate in R

Actually, can have two tables here and see which makes sense. We can look at the boring as normal mean vs trace and see what shakes OR we can find MISE of each curve against trace predictions. lets do that. 30 Subjects retained after removing shitty fittys

Method	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Fixation	0.01440949	0.01613453	0.01695755	0.01714883	0.01807178	0.02283428
Saccade	0.00000315	0.00065379	0.00333943	0.00451492	0.00762961	0.01847311

Table 2: Summary of mean integrated squared error of 30 NH subjects fit with `bdots` against TRACE, fit with both fixation and saccade method

7 Discussion

what have we learned?

Here I think are some of the main takeaways. First Bob showed that even under moderate assumptions, the fixation method is unable to recover unbiased parameter estimates for the generating function. Here, we examined the sources of this bias and demonstrated that by removing the period of fixation from the observed data, we are better able to estimate the generating curve.

Of course, the conclusions drawn from this rest on the tacit assumption that there is some parameteric generating function mediating the relationship between word activation and physiological behavior. By no means do we seek to argue for this either – this lies in the domain of the linking hypothesis, a.k.a someone else’s problem. However, the content of the arguments made is worth consideration. In particular, putting names to the two types of bias observed almost certainly have parallels in the empirical world: the oculomotor delay (delay bias) being a known phenomenon, and the added observation bias being tautologically true under moderate assumptions about the linking hypothesis. At very least, there is the question of the linear relationship between fixation length and activation, casting doubt on the validity of treating indicators of fixation equally at the beginning of a fixation period (and especially during the refraction period of a fixation) as those at the end. Treating only the saccades as observations removes this issue and is more defensible from a theoretical/statistical perspective. To what degree the proposed saccade method is representative of the true state of nature is up for debate.

Insomuch as it relates to the fixation method, it is worth recalling the proportion of fixation method itself was never (I think) argued for from the ground up. That is, its validity and subsequent adoption was a consequence of its agreement with the predictions of the TRACE model. To this end, we have shown that even with agreement to TRACE being the guiding principle, the saccade method shows greater fidelity to what would be predicted, even after accounting for researcher degrees of freedom.

[I need to reread magnuson before using such strong language here] The conclusions that we draw from this are twofold. Even under moderate assumptions regarding the linking hypothesis, the fixation method contains at least one source of bias by conflating two very distinct types of data. Really, that’s the only main conclusion, that and saccade method is cool. I said twofold above because twofold is a cool thing to have in a concluding paragraph and im pretty sure that onefold isn’t a word, and even if it is it isn’t as neat of a word as twofold.

something to have somewhere re decoupling of saccade and fixation and infromation gaterhing. somewhere presetned idea of weighting saccades but the info gathering itself may be implicit, whereby longer fixations simply increase propensity of movements to target, etc., removing need to control for it explicitly. Idk im

not really going to touch linking hypothesis stuff because it would involve me pretending to know a lot more about this than i do.

8 limitations

probably good idea to keep running list of these all in one place

1. linking hypothesis/cognition curve
2. trace parameters maybe/general degrees of freedom
3. only evidenced on logistic, though for practical not theoretical reasons
4. adding parametric form (necessity for saccade method)
5. oculomotor delay, where to discuss

9 appendices

Here I am just including more or less random sections that either do not have a definite place yet in the main body of the paper, are part of what might be considered future work, or truly are things that belong in the appendix

Appendix A

Here, I want to cover a handful of things:

1. Where did this trace data come from (bob's paper)
2. How could it be reconstructed in trace (parameters)
3. What words were used
4. How did I get it into acceptable format for me
5. Luce choice rule, time transformation, scaling term

We validate our arguments above using a fortuituous study by McMurray (2010) in which subject data was collected to be analyzed against a collection of hyperparameters in TRACE testing a number of theoretical constructs in language impairment, including sensory and phonological confusion, vocabulary size, etc.,

presenting us with both a collection of empirically collected data from the subjects, as well as accompanying TRACE data, simulated with words used in the empirical portion of the study. Using this, we will be able to examine the relationship between empirically collected look data, saccade data, and validating TRACE data.

I don't want to in the paper go through the entire thing of how i manipulated either trace or the empirical data. i might only mention that for a brief section in considered the implications of adjusting for the response time – i saw that bob did do this in his in sort of a weird way, cutting off each person at their own median so that half of the observed data would be truncated, half of it would not be truncated. i dont remember the exact reason for this but it seemed fine. not what i did though what i did was more standard.

There is a question if i should not limit things to only starttimes that occur before 1780, which i believe is the total number of cycles. of course, having that be the end vs another would have impacts on peak, slope etc. researcher degrees of freedom, hooray!

Appendix B

Here, I can discuss parallel results when I adjust for reaction time when gathering observations

Appendix C

Oculomotor Delay We begin with an assumption that the curve of interest can be represented parametrically. For example, the four parameter logistic, defined as

$$f(t|\theta) = \frac{h - b}{1 + \exp\left(4 \cdot \frac{s}{h-b}(x - t)\right)} + b,$$

is often used to describe the trajectory of probability of a subject launching a saccade and fixating on the target location while simultaneously used as a proxy for word activation. To illustrate, a subject with the depicted fixation curve may initiate a saccade beginning at time $t = 970$, with a probability of $p = 0.5$ of subsequently resting on the target:

[insert image]

Mentioned previously, this same parametric curve is used in both the proportion and saccade methods.

As saccades are easily gathered from available eyetracking data, we are, in principle, able to collect samples directly from this curve. This goal is complicated, however, by oculomotor delay. That is, an observed saccade at t_j is likely a sample from the fixation curve $f_\theta(t)$ at some point prior to t_j . The degree

to which this delay occurs, as well as the between and within subject variability of this delay, is a matter of active investigation. Most generally, we may consider an observation s_j at time t_j to be distributed

$$s_j \sim \text{Bern}[f_\theta(t_j - \rho(t_j))],$$

where $\rho(t)$ represents oculomotor delay. As written, we may consider circumstances in which:

1. $\rho(t)$ is a constant function (including 0)
2. $\rho(t)$ is a random variable, independent of the value of t_j
3. $\rho(t)$ is a random variable, dependent on t_j and possibly other aspects of the trial

To differentiate between the underlying data-generating mechanism and what is observed, we let

$$g_\theta(t) = f_\theta(t - \rho(t)),$$

where $g_\theta(t)$ is what is *observed* at time t . A saccade planned at $t = 300\text{ms}$ with an oculomotor delay of $\rho = 200\text{ms}$ will be observed at $t = 500\text{ms}$. That is, $g_\theta(500) = f_\theta(500 - 200) = f_\theta(300)$.

At present, it is common under the proportion method to account for this delay via a 200ms shift of the entire constructed proportion curve. We will propose instead a method whereby each saccade may be shifted individually and less homogeneously. Reasons and implications for this will be presented in the next section.

We now consider a variety of scenarios for oculomotor delay and the subsequent impacts on the recovery of the underlying fixation curve from the observed data.

There is also this from elsewhere that i didn't delete:

—

The simulation was conducted using a fixed oculomotor delay of $\rho = 200\text{ms}$. Although the resulting recovered curve is biased, this bias simply results in a horizontal shift, $g(t) = f(t - \rho)$. This is especially relevant in a situation in which we are interested in comparing the data generating curve between two groups.

For example, one method of analyzing VWP data (which inspired the ‘bdots’ package) was to determine on which intervals $I = \cup_k I_k$ two data generating curves were statistically different. Suppose, for simplicity, that there is an interval $I = [t_1, t_2]$ on which the difference between two curves, $f(t|\theta_1) - f(t|\theta_2)$, is statistically significant. Given that we observe $g_i(t) = f(t - \rho|\theta_i)$, we would simply find that a significant difference occurs at $I + \{\rho\} = [t_1 + \rho, t_2 + \rho]$, a horizontal shift resulting from the oculomotor delay.

In other words, the size of the interval would remain the same, and the relative differences between curves would be preserved under a horizontal shift.

Appendix D

Idk, maybe oculomotor delay? That is kind of a disjoint section of this paper, and i don't think would get more than a mention, really, outside of a published paper. but its relevant for future direcitons, it's relevant for calling things what they are, it's not relevant for considering trace vs empirical results. Here is copy pasted out of the discussion of old paper what I had originally recordered for this:

With regards to the curve described, there are a number of avenues seemingly worthy of investigation. The most pressing of these appears to be methods to minimize the amount of bias present in scenario three, which presents the largest obstacle in the functional recovery of the data generating mechanism. Of special note here is the fact that the particular intervals in which this bias occurs can have a large effect on the overall bias, over and above that introduced by the occulomotor delay.

For example, consider the plot above in the situation in which there is an unknown random delay (blue curve). We may observe at 500ms the value of the data generating mechanism at 300ms (that is, we observed $g_{\theta}(500) = f_{\theta}(300)$), while $g_{\theta}(500) \approx f_{\theta}(500)$. In other words, the bias over this area is small if we make no correction.

In contrast, an observation at $t = 1000$ results in a highly biased estimate, as $g_{\theta}(1000) \ll f_{\theta}(1000)$. Accordingly, we note that the amount of bias at an observed point is a function of the derivative of the data generating function in a neighborhood of that point. Whether or not this observation proves profitable remains to be seen.

There also seems to be value in finding a way to incorporate the length of fixations into the modeling process. For example, we might consider the impact of weighting each saccade by the duration of its subsequent fixation, as it seems intuitive that saccades resulting in longer fixation periods are more likely initiated by activation rather than a searching pattern.

Appendix E

Why no double gauss treatment? simply for the reason that it is unable to find optimal fits in bdots curvefitters. in other words, with double gauss as it relates to the saccade method, there is a shortcoming in implementation rather than anything unique to it theoretically. as this is a theoretical paper, we limit presentation to those methods that may be implemented successfully on a computer as well.