

RESEARCH ARTICLE

Computational Neuroscience

Choice anticipation as gated accumulation of sensory predictions

 Brandon Caie,¹  Dominik Endres,²  Aarlenne Z. Khan,³ and  Gunnar Blohm¹

¹Queen's University, Kingston, Ontario, Canada; ²Université de Montréal, Montreal, Quebec, Canada; and ³Philipps-University Marburg, Marburg, Germany

Abstract

Predictions are combined with sensory information when making choices. Accumulator models have conceptualized predictions as trial-by-trial updates to a baseline evidence level. These models have been successful in explaining the influence of choice history across-trials, however, they do not account for how sensory information is transformed into choice evidence. Here, we derive a gated accumulator that models the onset of evidence accumulation as a combination of delayed sensory information and a prediction of sensory timing. To test how delays interact with predictions, we designed a free-choice saccade task where participants directed eye movements to either of two targets that appeared with variable delays and asynchronies. Despite instructions not to anticipate, participants responded before target onset on some trials. We reasoned that anticipatory responses reflected a trade-off between inhibiting and facilitating the onset of evidence accumulation via a gating mechanism as target appearance became more likely. We then found that anticipatory responses were more likely following repeated choices, suggesting that the balance between anticipatory and sensory responses was driven by a prediction of sensory timing. By fitting the gated accumulator model to the data, we found that variance in within-trial fluctuations in baseline evidence best explained the joint increase of anticipatory responses and faster sensory-guided responses with longer delays. Thus, we conclude that a prediction of sensory timing is involved in balancing the costs of anticipation with lowering the amount of accumulated evidence required to trigger saccadic choice.

NEW & NOTEWORTHY Evidence accumulation models are used to study how recent history impacts the processes underlying how we make choices. Biophysical evidence suggests that the accumulation of evidence is gated, however, classic accumulator models do not account for this. In this work, we show that predictions of the timing of sensory information are important in controlling how evidence accumulation is gated and that signatures of these predictions can be detected even in randomized task environments.

anticipatory saccades; free choice; gated accumulation; predictive processing; saccadic choice

INTRODUCTION

Pattern recognition is important for adaptive behavior. Sometimes, the patterns we observe are representative of causal relationships in our environment, so we can use this knowledge to adapt the way we process sensory information and inform predictions about future events (1–3). Indeed, both empirical evidence and theoretical accounts point toward the importance of predictive feedback on even the earliest stages of sensory processing (4–6). However, although predictions are often studied under conditions where they have an adaptive purpose, predictions may also be based on relationships that arise spuriously. In

laboratory environments, it is common to randomize the presentation of sensory data such that information from one trial does not have predictive value for the subsequent trial. Despite this, people often show evidence for history effects, even when this confers no obvious advantage. Although history effects in randomized tasks have been extensively studied in the psychological sciences (7–14), less is known about how history effects influence the sensorimotor transformations that occur as we make decisions in real time.

Reaction times between the presentation of a sensory stimulus and the execution of a saccadic eye movement are thought to be determined by the time it takes to integrate sensory information from a baseline to a threshold (15–23).



Correspondence: B. Caie (brandon.caie@queensu.ca).
Submitted 25 January 2024 / Revised 6 March 2024 / Accepted 12 February 2025



In decision-making tasks reported through eye movements, predicting the likelihood of one alternative favoring another has traditionally been accounted for through shifts in the baseline evidence (18, 21, 24–26), although a direct and unique link has been questioned (27–29). Reinforcement-learning approaches have since been applied to explain how predictions influence eye movements through trial-trial updating of baseline evidence (30, 31), thus casting baseline evidence as a readout of a prediction that is updated on a trial-trial basis according to the outcome of a choice. However, baseline evidence is a fixed model parameter, aligned to the experimentally controlled onset of a stimulus, that abstracts over the sensory processing that occurs before evidence accumulation (32)—it has no obvious relationship to a biophysical process. Thus, it is unclear how discrete trial-trial baseline updating in classic accumulator models is related to sensory processing that occurs before evidence accumulation, and how this may lead to the observation of discrete baseline evidence shifts in classic accumulator models.

Predicting the timing of future sensory information is informed by prior experience (33–35). One natural timing problem faced in the evidence accumulation process is transmission delay between the onset of sensory information in the environment and its relay to regions thought to be involved in evidence accumulation, such as the frontal eye fields (36, 37) and the lateral intraparietal area (38, 39). In the visual system, it is generally known that different brain regions must adapt preparatory activity to account for variations in latency (40–43). Although timing the onset of the evidence accumulation process is an important component of decision-making (44, 45), any sensory processing that occurs prior to evidence accumulation is typically not addressed in models within the accumulator framework. An exception to this is the gated accumulator model of the visual-motor cascade in the frontal eye fields (46, 47), wherein transient responses to visual activity trigger the onset of the evidence accumulation. Thus, instead of describing the onset of evidence accumulation with a fixed baseline plus some sensory delay, the onset of the decision process is explicitly modeled as a consequence of upstream visual processing. However, this activity has been considered as a purely feedforward process, so the role of history effects are not well understood.

Here, we show behavioral data from a free-choice saccade experiment that suggests a prediction of sensory timing is combined with delayed visual information to trigger the onset of evidence accumulation. To explain this, we developed a gated accumulator model, in which a continuously fluctuating baseline evidence signal is combined with delayed sensory information to trigger the onset of evidence accumulation. By explicitly modeling the delay interval with baseline fluctuations, the model captures expected reaction time distributions for multiple delay intervals concurrently, and can additionally account for anticipatory response distributions arising from baseline fluctuations in the absence of external stimuli. To test how predictions derived from previous trials may be reflected in the gating process, we designed a free choice saccade task where participants had to balance withholding anticipatory responses with responding as fast as possible to one of two choice targets that were presented with variable asynchrony. A choice

history analysis suggested that the outcome of the previous trial, despite randomization, influenced the balance between anticipatory and sensory-guided reaction times. By fitting the gated accumulator model to choice history data, we show that the gated accumulator could capture both anticipatory and sensory-guided reaction times, and that trial-trial updates to the variance in baseline dynamics best explained the influence of recent history on reaction times. This suggests that evidence accumulation during saccadic choice is gated by combining delayed sensory information with predictions from previous trials, even in randomized protocols, to adapt the timing of accumulation onset. Furthermore, this suggests that variability in the baseline dynamics underlying gated accumulation may be subject to trial history effects.

METHODS

Free Choice Experimental Procedures

Participants.

Six participants took part in the study. All had normal or corrected-to-normal vision. The participant gave written consent before undergoing the procedure. The study was conducted with an experimental protocol approved by Health Sciences Research Ethics Board, which adheres to the principles of the Canadian Tri-council Policy Statement on Ethical Conduct for Research Involving Humans and the principles of the Declaration of Helsinki (2013).

Task.

We adapted a free choice saccade task, first introduced by Schiller and Chou (48) and further studied by Noudoost and Moore (49) and Soltani et al. (50). The subject began by fixating on a central target projected onto a screen (20 in. Mitsubishi Diamond Pro [16 × 12 in.] 1,280 × 1,024 pixels, 60 Hz, contrast 75.2%, brightness 0%). Two targets, one in each visual field, were then presented with randomly assigned temporal asynchrony values (16 ms, 33 ms, 66 ms, and 99 ms) and onset times drawn from U (750 ms, 1,250 ms). The subject was instructed to look at either target, so long as it was done as fast as possible, without anticipating. The subjects were informed prior to the beginning of the experiment that the targets would come up at different times relative to each other. The subject was instructed to blink in between trials to minimize disruption of stimulus perception. The script for the task was written in MATLAB using Psychtoolbox.

Data acquisition and filtering.

We collected a total of 80,550 trials from 52 sessions across our six participants. During each trial, eye movements were tracked via EyeLink 1000 Tower Mount (SR Research, Mississauga, ON, Canada) with a 1,000 Hz infrared camera that tracked retinal position. Retinal position was calibrated before each session. The EyeLink apparatus was placed 60 cm from the screen containing the saccade targets. Saccades were calculated offline using a saccade detection algorithm with a velocity criterion of 50°/s, and were individually verified through a custom visual marking program. Trials where the tracker lost the eye (either through tracking error or participant lapse) were excluded. Trials were also excluded if the eye position deviated more

than 15° from the horizontal. The first saccade following target onset, or the first saccade within 3° of the future target position prior to target onset, were taken to calculate the saccadic reaction time and endpoint error. Saccades with a positive endpoint horizontal position were given a choice value of 1, and saccades with a negative endpoint horizontal position a choice value of 0.

Psychometric fitting.

All data analysis was performed using custom scripts in Python. Psychometric functions were fit using the Psignifit toolbox with a Bayesian Inference fitting procedure (51). We fit psychometric functions to a cumulative Gaussian distribution. Choices occurring before 70 ms (sensory delay cutoff) after target onset were discounted from psychometric fitting. To compare the slopes of the psychometric functions across choice sequences, we bootstrapped the marginal distribution that estimates the slope parameter and used the position in the choice sequence as an ordinal variable to fit a linear regression model. The model therefore tests the hypothesis that there is a linear relationship between the slope of the psychometric function and the position in the choice sequence, against the null model that they are independent.

Statistical testing of differences in reaction times.

Qualitatively, we observed an increase in specific portions of the distribution of reaction times as a function of choice history and delay intervals. From the perspective of our modeling framework, causal change in the underlying process may influence certain portions of the observed reaction time distribution without changing typical summary statistics such as the mean reaction time. Thus, analysis of variance tests were not appropriate to analyze the effects that may not be manifest as changes to mean reaction times. To avoid this issue, we chose to take the standard deviation as a metric that would be sensitive to changes to anticipatory and express saccades. To estimate the confidence interval around the standard deviation, we bootstrapped each reaction time distribution 1,000 times and computed the 95% confidence interval of the standard deviation. We then compared each successive choice sequence and determined if the intervals overlapped.

Fitting to free choice saccade data.

We fit a gated accumulator model (see, *Gated Accumulation of Sensory Timing Expectations Accounts for Choice History and Anticipation*) to reaction time data from the free choice saccade task. We used differential evolution, a stochastic optimization technique in the *Scipy* optimization toolbox, to fit a parameter set for each delay bin. This algorithm allowed us to avoid local minima in the parameter space that interfere with gradient-based methods. We used the “best1bin” search method, with a maximum of 1,000 iterations performed, population size of 15, recombination constant of 0.7, and convergence tolerance of 0.01. The five parameters of the gated accumulation model were given the following boundary conditions

$$\begin{aligned} \theta &: (0.4, .8), \kappa : (0.3, 1.3), \sigma_{OU} : (.05, .2), \\ \mu_r &: (3/1000, 6/1000), \sigma_r : (1/1000, 4/1000). \end{aligned} \quad (1)$$

Before fitting, five alignment points for delay times were chosen (evenly spaced between 750 and 1,250 ms), and

reaction time data were discretized into these five bins relative to the delay time (first target onset). Thus, each data set contained five overlapping reaction time probability distributions. A model was then generated with a target onset time at each of these five delay times. To obtain a model error, the Kullback–Leibler divergence between each delay bin (model vs. data) was summed across the five delay bins. It was this summed error that was used as a cost function for the differential evolution algorithm.

$$\sum_{i=1}^N D_{KL}(P_i||Q_i) \quad (2)$$

$$D_{KL}(P_i||Q_i) = \sum_{x \in \mathcal{X}} P(x) \log \left(\frac{P(x)}{Q(x)} \right) \times \lambda, \quad (3)$$

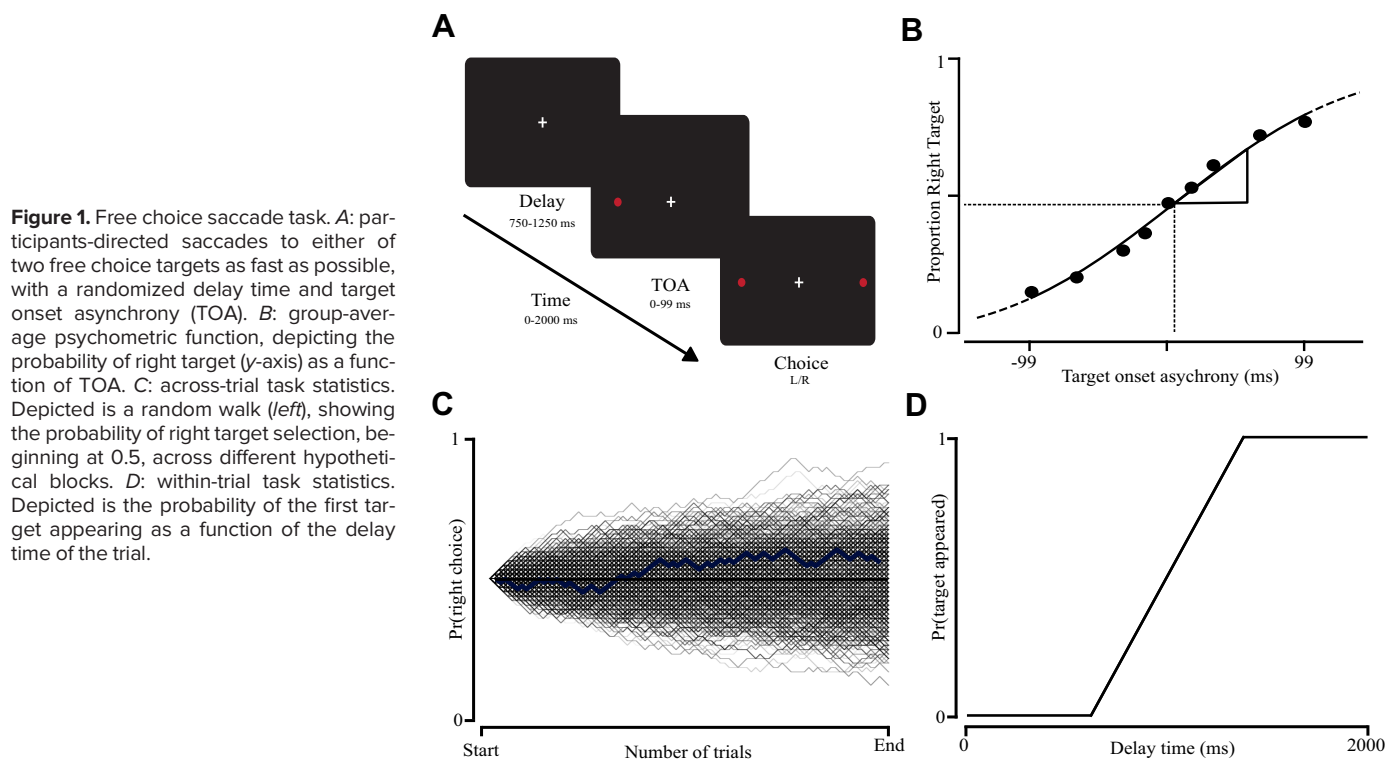
where i is the delay bin start, generated by N equal spacings between 750 and 1,250 ms, $P_i(x)$ is the reaction time model probability for delay bin i and reaction time bin x , $Q_i(x)$ is the same for the data probability distribution, and λ was a constant scaling term to prevent numerical underflow. The discrete model probability was calculated from the full probability density function by sampling 100,000 times from the continuous model over the same set of 50 discrete reaction time bins as the data histogram was computed from.

RESULTS

We designed a free choice saccade task (see, *Free Choice Experimental Procedures*) where participants had to rapidly respond to the onset of sensory information while withholding anticipatory responses. Each trial begins with a central fixation period drawn from a uniform distribution between 750 and 1,250 ms (Fig. 1A). This period was followed by the appearance of two visual targets, one in each visual hemifield. The targets appeared at different times relative to each other, with the target onset asynchrony (TOA) randomized across nine discrete values 0, +/− 16, 33, 66, 99 ms. Positive asynchrony values will denote the right target appearing prior to the left, and negative asynchrony values will denote the left target appearing before the right. Participants were instructed to direct a saccade to either target as fast as possible, without anticipating. Although both delay and TOA were randomized, any given sample of trials from a uniform distribution may have biased statistics in the spatial/across-trial (target location) and temporal/within-trial (target onset and asynchrony) domain (Fig. 1, C and D).

Choice Probability Is History-Dependent

Psychometric analysis revealed that choices are probabilistic with respect to the magnitude of asynchrony (Fig. 1B). Choice behavior binned by asynchrony indicated a progressive increase in rightward choices when the right target appeared earlier. The binned choice data was fit to a sigmoidal curve with a psychometric fitting procedure using Psignifit (52). Note in this analysis only choices occurring after the target onset plus a minimum delay of 70 ms were included, as is often standard when excluding anticipatory trials (40, 53). Synchronous targets resulted in choices closest to chance with respect to left or right direction, whereas a target



appearing well before another increased the likelihood of response toward it. This indicated that participants were aware of the task instructions and that the range of TOAs used in the task were sufficient to evoke a change in choice direction probability.

In randomized tasks, trials are uncorrelated, so analysis of behavior often rely on treating trials as statistically independent. However, it is common to infer patterns in short sequences of randomized information (7, 9). In our task, there is no inherent advantage to relying on information from past trials, but each choice trial is embedded in a unique sequence. A binary choice tree for five trials in the free choice task is schematized in Fig. 2A. For future analyses, we compared sequences of choice repetitions to the same target direction, and sequences of choices alternating targets, as measures of choice sequences with a stereotyped local structure that may induce bias away from the true generative statistics of the task (54). Figure 2, B and C shows a histogram of the number of repetitions and alternations in the present data set. Because the data become increasingly limited with higher sequence number, analyses were conducted up to a sequence length of 5.

Psychometric analysis (see, *Psychometric fitting*) revealed that choice probability was influenced by the number of previous repetitions and alternations. In Fig. 2E, psychometric fits to group-averaged repetition data are plotted as a function of repetition number (blue gradient), showing a progressive decrease in the slope of the sigmoid function. We interpret this as indicating that following choice repetitions, participants relied less on the TOA of the current trial, and more on information derived from past trials. To quantify this, we estimated parameters of the psychometric function using a Bayesian procedure (see, *Psychometric fitting*). We then bootstrapped estimates of the slope from the resulting probability

distribution over the slopes, and performed a Kruskal–Wallis to ask if the parameter estimates came from the same distribution ($F = 2,829.13$, $P = 0$). This suggests that the distribution of best-fitting psychometric slopes changed across repetitions. We performed this same analysis for sequential alternations, finding that choice probability was also not constant ($F = 476.23$, $P = 9.18 \times 10^{-102}$).

In addition to the TOA, we also assessed the role of the delay interval. We next assessed whether the length of the delay interval influenced choice probability. To do this, we performed the same binning procedure as previously outlined for choice sequence, but instead, we binned the delay period into five equally spaced intervals between 750 ms and 1,250 ms. The resulting psychometric functions are shown in Fig. 2D. Again, we performed a Kruskal–Wallis test, which supported the qualitative observation that the best-fitting psychometric functions were not constant when separating data by the length of the delay interval ($F = 384.77$, $P = 5.43 \times 10^{-82}$). In combination, these analyses support the conclusion that both choice sequence and delay time influenced choice behavior.

Choice History Jointly Influences Anticipation and the Accumulation of Choice Evidence

Analysis of reaction time distributions provides a window into the processes underlying saccade triggering (18, 32, 36). To better understand how the choice process evolved throughout the trial, we therefore performed a reaction time analysis of the free choice saccade task based on the delay interval and previous trials. Saccades are often grouped into three classes based on reaction time: anticipatory responses that occur before stimulus onset, sensory-guided responses, and an “express saccade” regime that lies between the two (55). In the psychometric analyses, only choices occurring

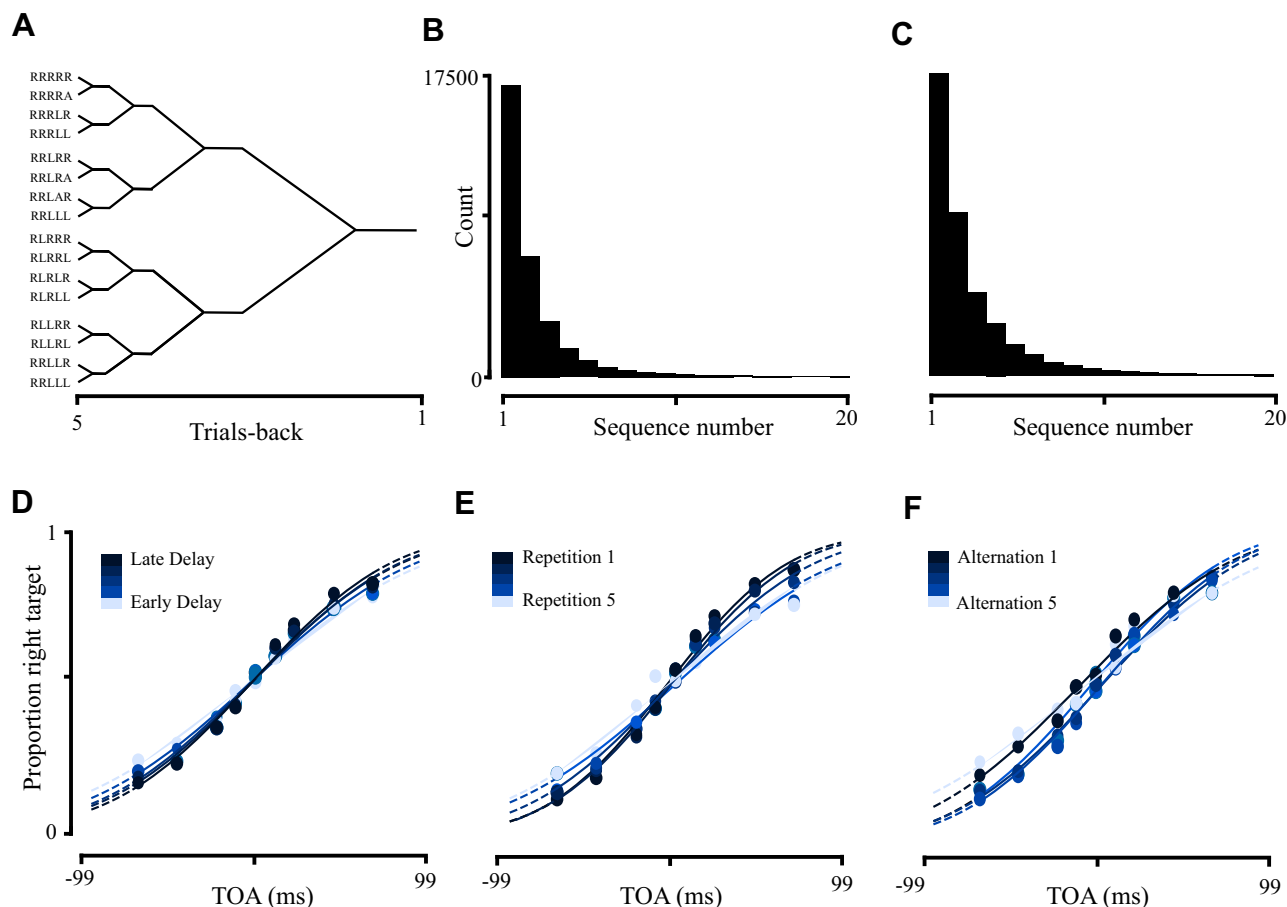


Figure 2. Choice history and delay time influence choice probability. *A*: choice history tree depicting the possible alternatives of a binary choice up to four trials back. *B*: histogram of the number of choice repetitions across all participants. For each sequence number, a single tally was recorded if the same choice direction had been chosen for that many trials previously. *C*: the same as *B*, but for the number of successive choice direction alternations. *D*: group-average psychometric functions with respect to target onset asynchrony (TOA), where the data has been split into four delay bins equally spaced between 750 and 1,250 ms. The color gradient denotes the delay period, low to high. *E*: psychometric functions split into the number of previous choice repetitions. Here, each repetition sequence was given an independent repetition score, such that no trials in one repetition set are contained in another. *F*: the same as *E*, for choice alternations.

after sensory onset plus a minimum delay of 70 ms were considered (40), so as to analyze the role of target onset asynchrony. However, this was only a subset of the total behavior; while participants were asked to try not to anticipate, they were also asked to go as fast as possible, so in a subset of trials participants responded before target appearance.

First, we assessed the influence of the length of the delay interval on anticipatory and sensory-guided responses. [Figure 3](#) plots the cumulative distributions of reaction times split into five equally sized delay bins between 750 and 1,250 ms. We aligned the distributions in two ways. First, we aligned relative to the onset of the target, and plotted anticipatory responses as negative time relative to this ([Fig. 3A](#)). Next, we aligned the data to the onset of the trial, such that each delay bin will have a mixture of onset times bounded within the delay bin ([Fig. 3B](#)). In each view, it can be seen that anticipatory responses increase, and sensory-guided responses get faster, when the delay interval was longer ([23, 56](#)). Qualitatively, this resulted in a broadening of the distribution as a function of the delay interval, since an increase in anticipatory responses corresponds with an increase in

the early tail of the distribution. To quantify this, we randomly sampled from each distribution and bootstrapped the empirical standard deviation, and computed the 95% confidence interval for each. This supported the conclusion that reaction time variance was dependent on the delay interval.

We next analyzed reaction times as a function of choice history (sequential repetitions and alternations). [Figure 4](#) plots the reaction times aligned to the target dependent on how many previous repetitions ([Fig. 4A](#)) and alternations ([Fig. 4B](#)) preceded the current trial. Here, it can be seen that sequential repetitions were associated with an increase in anticipatory responses, as seen in the cumulative proportion lying before the dashed line indicating target onset, and faster posttarget responses. However, this speed advantage was negated for responses in the median range, and even reversed for later responses, consistent with the idea that choice sequence influenced an association with baseline evidence in accumulator models ([57, 58](#)). This effect was detectable but noticeably diminished in magnitude for alternations, consistent with the idea that sequence effects may operate over different timescales when targets alternate ([54](#)). In summary, our results suggest that reaction times were jointly

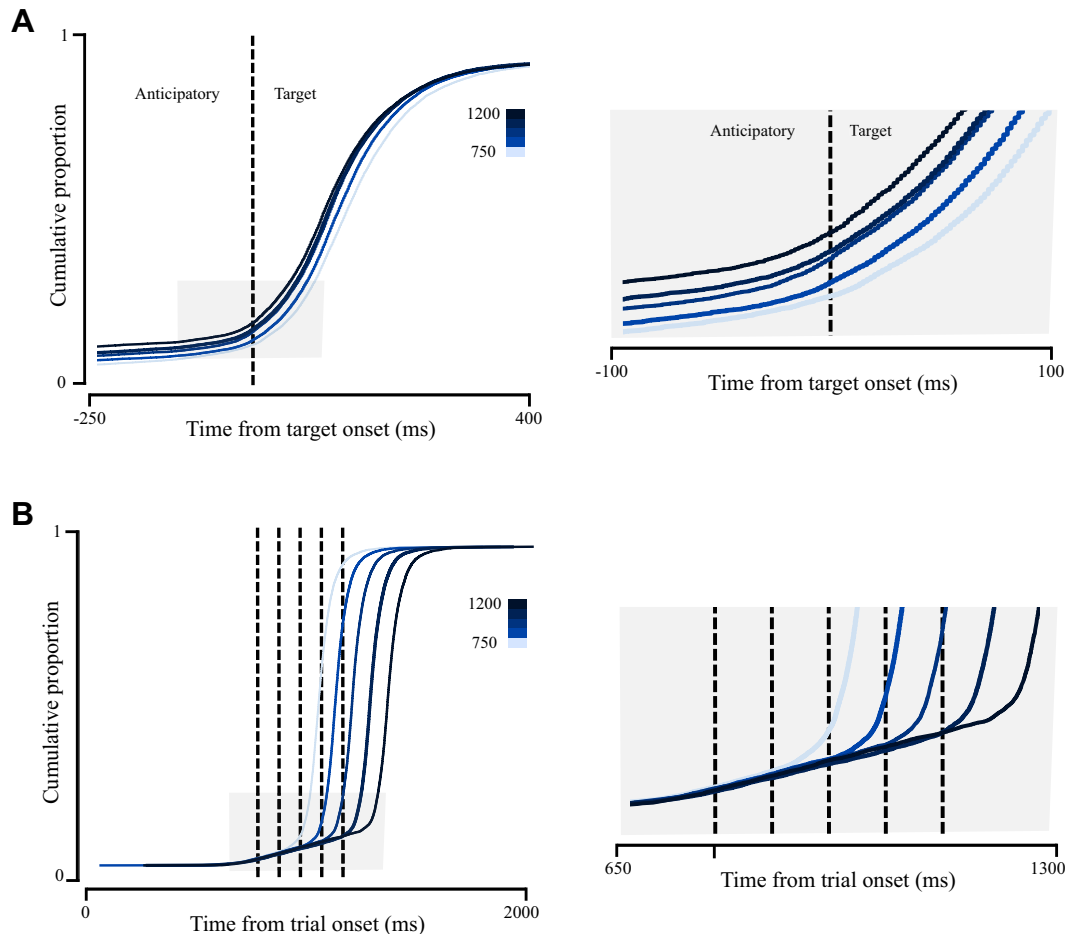


Figure 3. Anticipatory and sensory-guided reaction times are influenced by delay time. **A:** cumulative probability of reaction times as a function of delay time (5 delay bins equally spaced between 750 and 1,250 ms) aligned to the onset of the target. **B:** the same reaction time delay bins, aligned to the onset of the trial.

influenced by choice history and the delay time of the trial. By analyzing reaction times not by summary statistics such as the mean or median, and instead viewing entire distributions relative to different onset times, we were able to conclude a joint influence of delay time and the previous trial on early and express saccade responses.

Modeling Saccade Triggering as a Gated Accumulation

The gating of inputs to different neural structures is an established mechanism of information transfer in the brain (59). In the study by Purcell et al. (46), the accumulation of evidence favoring one of several choice alternatives is hypothesized to be gated below some threshold for saliency, allowing for the selective accumulation of information in the presence of a continuous stream of changing visual scenes. It is commonly thought that such evidence is integrated or amplified toward a threshold, upon which a corresponding motor command is produced (32, 60). Models of the accumulation process have served a dual role, bridging an algorithmic explanation of the heavy-tailed distributions characteristic of reaction times with a mechanistic explanation of the delayed ramping activity of single neurons in regions of the brain such as the frontal eye fields (36, 61). In

recognizing both the necessity and empirical evidence for the control of the accumulation process by a gating mechanism, Purcell et al. (46) extended the descriptive capacity of accumulator models to explicitly include the visual transformations occurring before the onset of evidence accumulation, thereby replacing the discrete baseline evidence of traditional accumulator models (such as the drift-diffusion model, LATER model, and linear ballistic accumulator) with a continuous visual transformation.

In this section, we derive a gated accumulator that receives a continuous input to an evidence accumulator during the delay interval of a trial. We first decompose the model into two sections: a section explaining the baseline dynamics, or how baseline evidence fluctuates throughout the trial before the onset of sensory information, and a section explaining the evidence accumulation. Then, we explain how these processes may be linked by a gating mechanism, and how we combine the two processes algorithmically to obtain a single probability density for reaction times.

Baseline dynamics.

We first describe the baseline dynamics (Fig. 5) that evolve throughout the delay interval of a trial before the onset of a visual stimulus. We chose an Ornstein-Uhlenbeck (OU)

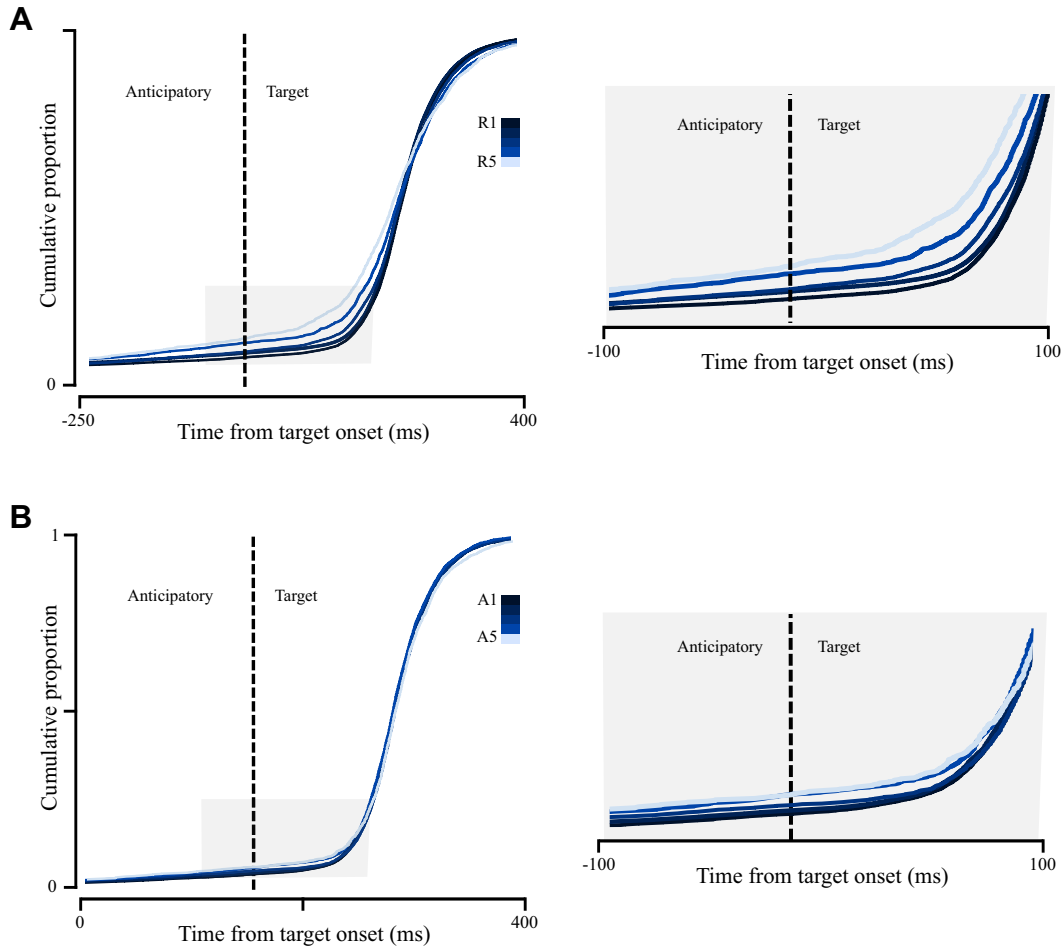


Figure 4. Anticipatory and sensory-guided reaction times are influenced by choice history. *A*: cumulative probability of reaction times as a function of previous choice repetitions, aligned to the onset of the target. *B*: cumulative probability of reaction times as a function of previous choice alternations.

process to model the baseline dynamics for the free-choice saccade data. This was chosen because it is a well-characterized dynamical system with tractable properties that can describe the evolution of a stochastic process from a baseline value to a steady state. However, it should be noted that this does not constitute an argument for the necessity of an OU-like process in the neural processes underlying gated accumulation, merely its sufficiency to describe the data. The OU process is a stochastic differential equation that describes the time evolution of a random variable changing proportionally to the distance from an expected mean (Fig. 2). Let $B_1, B_2, \dots, B_n$ be a set of processes that evolve as a time series during a trial of length $T_{0:Tmax} = 2,000$, where $b_i = b_1, b_2, \dots, b_n$ is a single process satisfying the stochastic differential equation

$$db_t = \lambda(\theta - b_t)dt + \sigma_B dW_t, \quad (4)$$

where λ is the drift rate, θ is the steady-state baseline the process reverts to as time approaches infinity, and W is a Wiener process scaled by σ_B .

We further assume that if this system reaches the expected baseline θ before the onset of a choice target, then an anticipatory responses is triggered. Thus, the rate λ dictates if and how close to θ the system reaches before target onset, and thus if an anticipatory response is triggered. The anticipatory response distribution is thus defined by the first-passage

problem where the threshold is equal to the long-run mean θ , which is described by Lipton and Kaushansky (62) as the solution to the problem

$$T_{A,C} = \inf \{t \geq 0 : B_t = A | X_0 = C\} \quad (5)$$

with change of variables

$$\begin{aligned} \tilde{t} &= \lambda t \\ \bar{C} &= \frac{\sqrt{2\theta}}{\sigma_B} (C - \theta) \\ \bar{A} &= \frac{\sqrt{2\theta}}{\sigma_B} (A - \theta) \end{aligned} \quad (6)$$

The anticipatory density function for the case where $\bar{A} = 0$ is known.

$$\begin{aligned} P_B(t) &= \lambda \sqrt{\frac{2}{\pi}} \cdot |\bar{C}| \cdot \frac{\exp(-\theta t)}{(1 - \exp(-2\theta t))^{3/2}} \\ &\cdot \exp\left(-\frac{\bar{C}^2 \cdot \exp(-2\theta t)}{2 \cdot (1 - \exp(-2\theta t))}\right). \end{aligned} \quad (7)$$

Because $\bar{A} = 0$ is equivalent to setting the threshold crossing A to equal θ , this equation becomes the first passage time solution for when the threshold $A = \theta$. Thus, this equation describes the probability density of how long it takes the OU

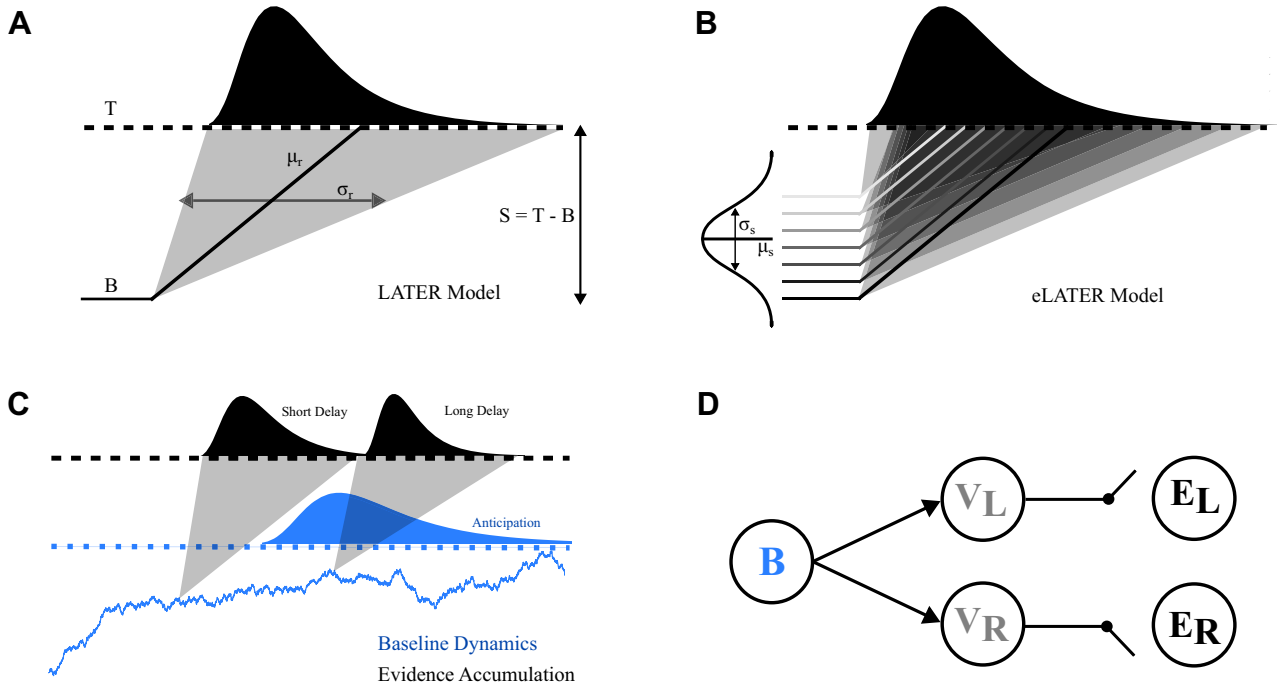


Figure 5. Gated accumulation of choice evidence. **A:** schematic of a linear approach to threshold with ergodic rate (LATER) model. Sensory evidence is integrated upon a baseline level of choice evidence toward a movement-triggering threshold. The strength of sensory evidence typically controls the average rate of rise. Variability in the rate of rise yields variability in reaction times (top curve). Time in the model begins relative to the onset of a repeated stimulus. **B:** extended LATER model (eLATER), with variance in the baseline giving rise to a distributed integration process. The resulting probability density function is integrated over the threshold-baseline distribution. **C:** gated accumulation. Baseline dynamics, governed by an Ornstein-Uhlenbeck process, are combined with the delay time of a given trial to yield a joint distribution of anticipatory and sensory-guided reaction times. **D:** circuit diagram of a two-alternative gated accumulation process, with the baseline dynamic B combined with visual transients V to trigger evidence accumulation E .

process to reach the long-run mean θ , scaled by the rate parameter λ .

A graphical depiction of the baseline dynamics of a sample OU process is depicted in Fig. 6. In Fig. 6A, different instances of the same OU parameter set are plotted in the blue traces, with each evolving according to the difference between its current state and a mean reversion parameter $\theta = 1.5$ at a rate according to λ with variance σ_b . We can solve analytically for the first passage time for a given parameter set, yielding our anticipatory response function, as the time it takes to reach the steady state (Eq. 4). In addition, we have a Gaussian probability density of baseline activity at each time point throughout the delay period (Fig. 6C), which we will use to model a distributed evidence integration process with variability in the baseline, as in the study by Nakahara et al. (57).

Evidence accumulation.

We next describe the integration of evidence to a threshold following the onset of sensory information. For simplicity, we chose to describe the evidence accumulation process that occurs after the onset of a choice target as minimally as possible, so as to focus on the effects of input during the delay period. A simple and interpretable evidence accumulation model is the linear approach to threshold with ergodic rate (LATER) model (18, 58, 63). The LATER model captures reaction time as a deterministic signal with variability across trials using three free parameters. Any single trial is determined by

$$s = rt \quad (8)$$

where t is the response time, s is the threshold-baseline difference, and r is the rate of rise. The distribution of reaction times can be derived using process parameters μ_r , the mean rate of rise, s the distance from baseline to threshold, and σ_r , the variance of the rate of rise

$$P_L(t) = \frac{1}{t^2} \frac{1}{\sqrt{2\pi}\mu_r/s} \exp \left[\left(-\frac{1}{2(\frac{\sigma_r}{s})^2} \right) \left(\frac{1}{t} - \frac{\mu_r}{s} \right)^2 \right]. \quad (9)$$

In the LATER model, the baseline is a single value, and the variance required to explain a distribution of reaction times comes from trial-trial variance in the integration rate. Although the LATER model fits the majority of a reaction time distribution, it does not account for anticipatory responses and the short latency reaction times typical of eye movements, often termed “express saccades” (58). To treat the entire reaction time distribution under this framework, and crucially the responses close to the time of target onset, a derivation of the LATER model with variance to the baseline value was proposed (57). This “extended LATER” model has an analytical solution for the reaction time probability density if the prior is Gaussian, $N(\mu_s, \sigma_s)$

$$P_E(t) = \frac{t + r_1 \alpha^2}{(t^2 + \alpha^2)^{3/2}} \frac{1}{\sqrt{2\pi}\Sigma} \exp \left(-\frac{1}{2\Sigma^2} \left(\frac{1}{t} - r_1 \right)^2 \frac{t^2}{t^2 + \alpha^2} \right), \quad (10)$$

where $\alpha = \frac{\sigma_s}{\sigma_r}$, $\Sigma = \frac{\sigma_r}{\mu_s}$, $r_1 = \frac{\mu_r}{\mu_s}$.

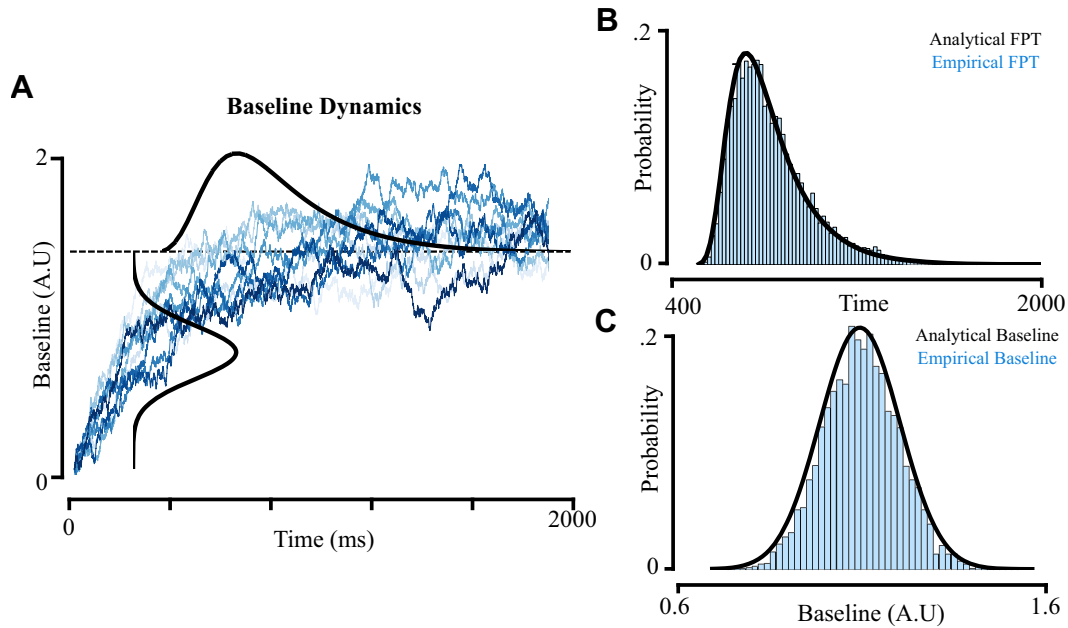


Figure 6. Baseline dynamics. **A:** sample paths of an Ornstein-Uhlenbeck process used to model the baseline dynamics. Each blue trace is one sample path drawn from the same parameter set: $\theta = 1$, $\mu = 1.5$, $\sigma B = 0.5$, $X_0 = 0$. **B:** the first passage time for this parameter (black: analytical solution, blue: empirical histogram for 1,000 processes) set is plotted as the probability that the OU process has reached the μ value at time t . This is also seen in the heavy-tailed distribution above the threshold in **A**. **C:** probability distribution of baseline values at $t = 250$ ms. The distribution at any time follows a Gaussian distribution (color scheme same as **B**).

The extended LATER model allows us to calculate reaction time distributions for different distributions of baseline activity, rather than using the single baseline parameter in the LATER model. Because of this, we can then model the expected reaction time distribution for different delay lengths by combining the extended LATER model with a time-varying baseline dynamic.

Combining evidence accumulation with baseline dynamics via gating.

We next describe how the two previously described probability density functions, the anticipatory response distribution P_B derived from the baseline dynamics, and the evidence accumulation distribution P_E , are combined into a common process with a single probability density function. We first treat the simplest case, where only a single target onset time is used. Thus, we need to compute the anticipatory first passage problem for the OU process, and the mean and variance of the process itself at the corresponding delay time, which we will use as the mean and variance to the baseline Gaussian distribution in the extended LATER model. We can thus compute the mean of the extended LATER baseline μ_r at time t as the expectation of the Ornstein-Uhlenbeck process, which is well-known

$$\mathbb{E}[x(t)|x_0] = x_0 e^{-\kappa t} + \theta(1 - e^{-\kappa t}) \quad (11)$$

and the covariance

$$\text{COV}[X(s), X(t)] = \frac{\sigma^2}{2\theta} (e^{-\theta|t-s|} - e^{-\theta(s+t)}), \quad (12)$$

where $s = 0$ and $t = y$. To convert the rising baseline of the OU process into a threshold-baseline distance for the extended LATER model (where a smaller value indicates a

shorter distance and thus a faster time to threshold and reaction time), we translate μ_r as

$$\mu_r(t) = 1 - x(t) \quad (13)$$

where μ_r is bounded between 0 and 1. Thus, while adding four free parameters from the OU process, we reduce two free parameters from the extended LATER process pT by allowing the OU process to control the baseline Gaussian at a given target onset time t . We can then define the gating process, G , as a combination of the anticipatory first passage distribution P_B , and the extended LATER solutions P_L for a distribution of delay intervals D over the interval $[a, b]$.

$$G = P_A + \int_a^b P_E(t_0 = D) dD$$

A key distinction between the way in which we have derived this model and other evidence accumulator models is that by explicitly accounting for the delay interval and anticipatory responses, the onset of sensory information is no longer a fixed, experimentally controlled parameter that references $t = 0$, or $t = 0$ plus some fixed sensory delay parameter. Instead, the model output is the combination of the parameters of the model and the chosen delay times.

Figure 7 depicts a cartoon scenario illustrating the impact of the delay time of a given trial on the outcome of different baseline dynamics. If two different baseline dynamics, depicted in dark and light blue respectfully, were aligned relative to the onset of a stimulus but had different delay times (Fig. 7A), they may produce identical baselines at the time of target onset, and as such not be dissociable. However, if the same baseline dynamics were shifted such that the common starting time was the trial, and not the target, the same baseline dynamics would lead to an observable difference in reaction times (Fig. 7B). This

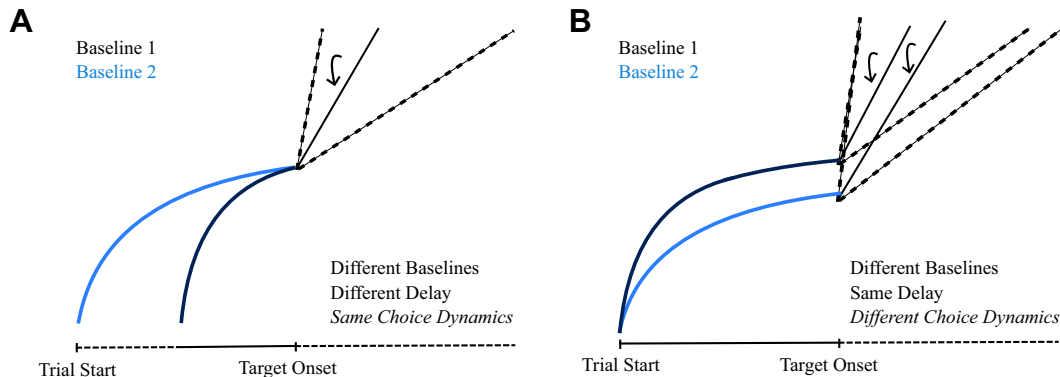


Figure 7. Inferring difference in baseline dynamics depends on starting point. *A*: two trials where baseline dynamics (high rate parameter: light blue, low rate parameter: dark blue) with the same delay time reach the different baseline levels, resulting in distinct accumulation processes. *B*: two baseline dynamics with different delay interval lengths, resulting in identical baseline evidence at target onset, and thus indistinguishable reaction time distributions.

highlights that distinctions between underlying processes can be masked by the way in which reaction time data is aligned, whereas identical processes can result in quantitatively different behavior if the temporal statistics of the task are appropriately varied.

Gated Accumulation of Sensory Timing Expectations Accounts for Choice History and Anticipation

Biophysically motivated models of evidence accumulation have suggested that the onset of accumulation is gated to prevent prepotent responses to the continuous stream of sensory inputs we are subject to in natural visual scenes (46, 47). We derived a model in which the gating of evidence accumulation is not driven solely by delayed sensory information, but also by within-trial fluctuations in baseline evidence dynamics. Here, we show that trial-trial changes in the baseline dynamics can account for the dependence of reaction times on choice history that was observed empirically.

To do this, we fit the group-averaged reaction time data to the gated accumulator model, split up by choice repetitions so as to isolate the strongest observed effect of choice history (see, *Fitting to free choice saccade data*). The model fitting procedure yielded a joint probability density function for reaction time data continuously throughout the trial period, which was post hoc binned into five delay quantiles for visualization. Figure 8 depicts the result of the model fitting. In Fig. 8A, the baseline dynamics for the best fitting model are shown (the mean baseline is shown in the black trace, with the shaded blue depicting the standard deviation of the baseline at each time point, and the dotted line depicting the threshold).

Here, a dependence of the model fitting results on the choice repetition sequence on the variance of the baseline dynamic σ_B was observed. There was a progressive increase in σ_B as repetitions increased ($R_{1\sigma_B}$: 0.112, $R_{2\sigma_B}$: 0.119, $R_{3\sigma_B}$: 0.127, $R_{4\sigma_B}$: 0.157, $R_{5\sigma_B}$: 0.183), whereas no consistent trend was observed in the other baseline dynamics parameters θ or κ , nor the sensory integration parameters μ_r or σ_r . This indicates that the trial-trial changes observed in the reaction time data may not be best explained by shifts in the baseline per se, but the variance in the process that gives rise to its average value. As a result, the model best explains

the data by a combination of increased anticipatory responses due to a higher variance in the baseline OU process, and a higher variance in resulting threshold-baseline distance in the eLATER sensory integration process.

To further illustrate the consequences of this result, we simulated the result of a sequential updating scheme, where single parameters of the baseline dynamic were updated according to the expected probability of repetition in a Markov process tracking the choice direction outcome. Figure 9A depicts this process (left), with the resulting repetition probability approximating a random walk (right), wherein the expected value remains constant, but the variance of the process increases—as such, any given process becomes more likely to have diverged from the expected value as the trial numbers increase, despite the expected value remaining constant. Figure 9, B–D plots the baseline dynamics (left), anticipatory distributions (middle), and cumulative reaction time distributions (right) for two delay times (750 ms and 1,250 ms) for sequential updating of θ (Fig. 9B), κ (Fig. 9C), and σ_B (Fig. 9D). Here, two observations should be noted. First, it can be seen that all three parameters can result in changes to the proportion of anticipatory responses, although this may only be apparent if the delay time is sufficiently long to realize this. However, each parameter update has a differential effect on the resulting baseline evidence distributions at different delay times, influencing the trajectory of the evidence accumulation process.

In light of this, the finding that the variance of the baseline, σ_B , best explained the choice repetition results should be appraised. Like the other parameters, σ_B can result in an increased anticipatory density. However, each parameter has a differential influence on the evidence accumulation process. Theta influences the average baseline, which may or may not be in turn influenced by the delay time, depending on the rate parameter κ . In contrast, the variance parameter σ_B influences the variance of the baseline and the anticipatory density function. Taken together, this suggests that the choice repetition effects were best explained by a combination of increased anticipatory responses and an increased variance of the threshold-baseline distance in the extended LATER model, as has been previously reported to underlie pre-target processes such as prediction (57).

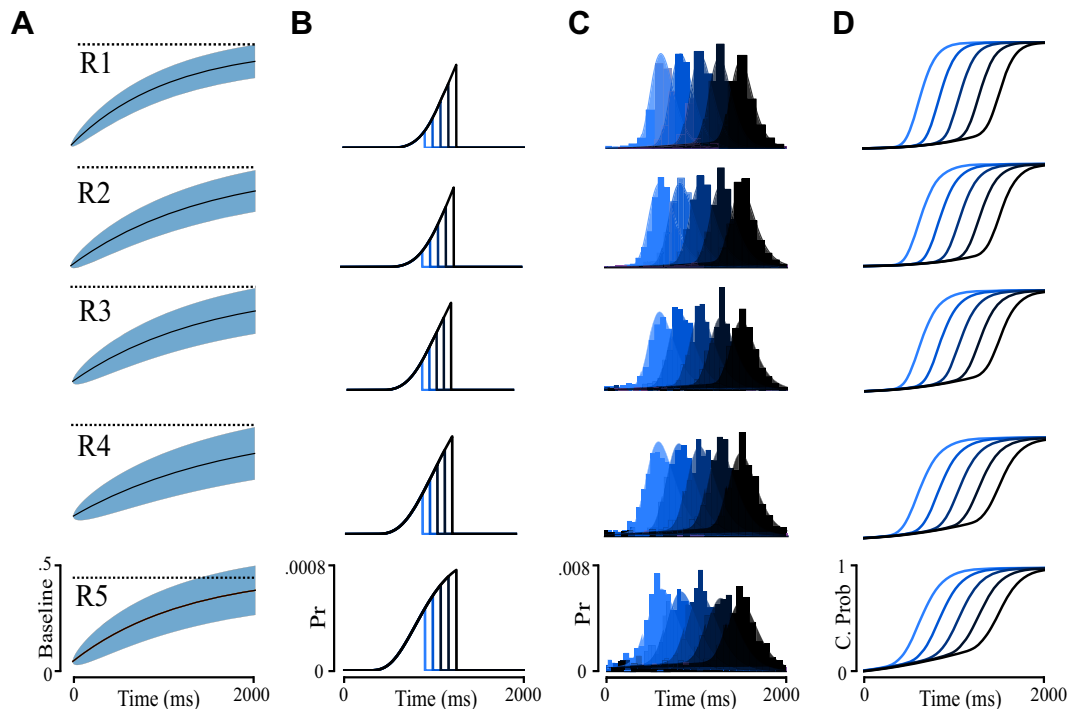


Figure 8. Gated accumulation explains dependence of reaction times on choice sequence and delay time. *A*: best fitting baseline dynamic for each data set, where each row denotes the fit to repetition 1 (top row) through 5 (bottom row). The mean of the baseline dynamic is plotted in black, and the variance is plotted in the shaded blue. The dotted line indicates the value of the mean-reversion parameter θ . *B*: anticipatory first passage of the model for 5 delay times, each cut off at target onset. *C*: model reaction time density functions (shaded curves) are plotted against the data (histograms). *D*: cumulative density functions from the best-fitting model.

DISCUSSION

In this paper, we combined behavioral experiments with computational modeling to show that predictions of sensory timing influence how evidence accumulation is gated during saccadic choice, even in a randomized task where history effects may not be expected. In a free choice saccade task to randomized targets presented with variable delays and variable asynchronies (48, 49, 64). We found evidence for an interaction between trial history effects based on the choice sequence, and the influence of the delay period (observed via anticipatory responses and delay-dependent changes to evidence accumulation). We developed an extended version of a gated accumulator model (46, 47) to account for this behavior. In doing so, this work provides a technical advancement on the evidence accumulation framework by explicitly modeling reaction times in reference to a delay interval, rather than as aligned to the onset of a stimulus. This allowed us to estimate latent dynamics in the baseline of an evidence accumulator that may go unobserved in classic accumulator models that align the decision process to the onset of a repeated stimulus. By fitting the model to data, we found that different phenomena in saccadic choice—anticipation, urgency, and history effects—could be accounted for in our data through variance in the baseline dynamics occurring prior to the onset of evidence accumulation.

Anticipating the timing of sensory information is useful when interacting with recurring patterns in the environment (65), particularly in the presence of delays. In this study, we found that anticipation reflected a balance between lowering

the amount of evidence required to trigger a saccade and inhibiting movement generation prior to the appearance of sensory information. The onset of evidence accumulation has been suggested to be under active control and subject to task-specific modulation (44, 45), but in the analysis of choice tasks, anticipatory responses are usually treated as outliers to be discard—as such, the relationship between anticipation and the evidence accumulation may have been underappreciated. When it has, anticipation has been explained as arising from an independent and parallel process (66), supporting claims for a mechanistic division between proactive and reactive processing (67, 68). In contrast, our model explicitly links anticipatory processing and evidence accumulation as a dependent process, in accordance with our experimental evidence. However, future work is required to assess whether the dependence of baseline evidence and anticipatory response generation is strictly coupled by a common physical process, or only coupled through a joint correlation with another unmeasured factor (69).

We also report urgency-like effects in our task, that is, a decrease in reaction time as a function of the length of the delay period. We reason this reflected the increasing probability of a target appearing in our task as time goes on. In accumulator frameworks, urgency has classically been associated with a change in the rate of evidence integration (56). An additional class of models eschews evidence accumulation altogether, instead arguing that urgency is represented as an independent signal that is combined with sequentially sampled sensory information (60). Differentiating these models empirically has been a matter of debate (70–72). As

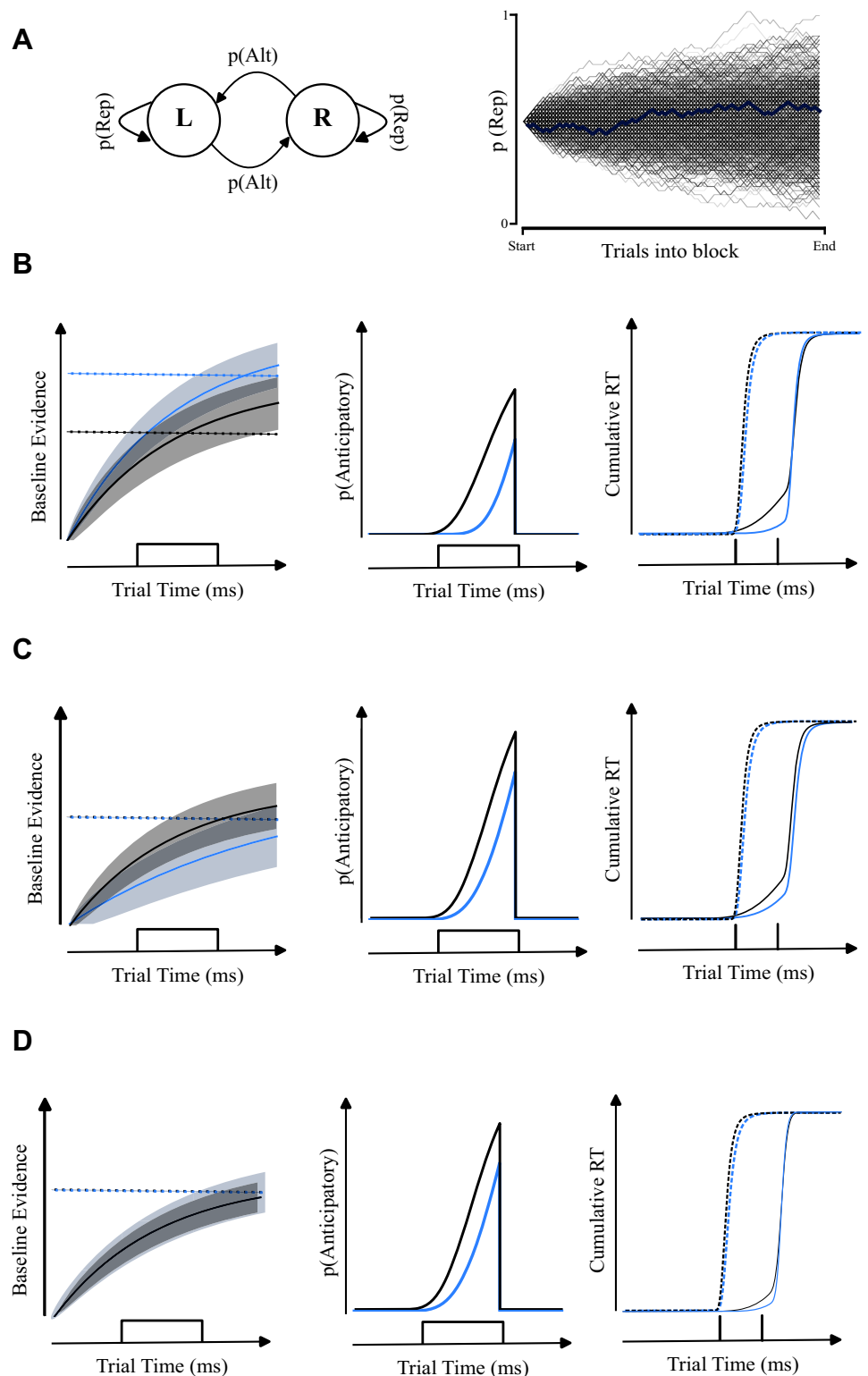


Figure 9. Sequential updating of baseline dynamics parameters. **A:** depicted is a Markov process tracking the probability of choice repetitions and alternations, with the probability of a repetition shown as a random walk over trials (single block in blue, many processes shown in gray). **B:** updating of the mean-reverting parameter θ . Black: high repetition probability results in a lower mean-reversion parameter (left depicts the baseline dynamics for each), and thus more anticipatory responses (middle). The right shows reaction time distributions for early target onsets, where the two processes are indistinguishable, and late target onsets, where they are distinguishable. **C:** updating of the rate parameter κ . Higher probability of repetition leads to the baseline dynamics reaching a steady-state faster, thus more anticipatory and faster sensory responses.

we have not performed a formal model comparison with other potential post-target dynamics, it is unclear to what degree the models can be dissociated empirically through behavioral data alone, particularly without manipulating the strength of sensory information over time (60). Thus, caution is warranted in interpreting the mathematical

form of the accumulation process we model as a necessary component of neural processing, rather than a sufficient explanation of the data we present.

The trial-to-trial use of prediction we show evidence for is suboptimal with respect to the instructions and statistics of the task. This sort of sequential dependency is often likened

to model-free learning, i.e., updating behavior purely based on the outcome of the previous choice. The alternative, model-based learning, requires forming an internal representation of the statistics of a task. Model-free strategies have been the de facto metaphor for incremental learning in neural systems (73–76), whereas “models” are invoked to explain the learning of more complex structures (77–83). Model-free strategies, defined this way, are particularly useful for novel or volatile environments, as they can adapt to structures that may not be known a priori. For example, a model-free learner in our task could track the recent history of the free choice task with a trial-trial update of the probability of the left target appearing before the right. The disadvantage of such an approach in our task is that while this may be a good approach to learn the average statistics of the task, the probability of being biased from the mean on any single trajectory would approximate a random walk, thus it would actually increase over time (84). Thus, a model-free learning algorithm may do a poor job of learning the statistics of the task if given few opportunities to do so, and sequential biases in randomized tasks would emerge as a by-product of the learning process.

Environmental changes occur over multiple timescales (85, 86), so the time horizon with which that change is considered is critical in the ongoing evaluation of behavior (87, 88). When making inferences about model-free or model-based learning, it can be convenient to categorize learning by the timescales over which they change: model-free behavior closely resembles the immediate past, whereas model-based learning may not. It is worth noting that while model-free learning is based on the recent past, learning based on the recent past is not all model-free. Although model-free learning refers to an update based solely on the outcome of a previous event, fast-timescale updating could reflect a nuanced model of the volatility of an environment, where rapid switching is favored in or outside the contexts of a laboratory task. Although we may be able to quantify the timescale of learning through correlating choices with recent and long-term history, this does not mean we can falsify the generative model (69), where what appears to be a suboptimal lingering effect of the previous trial may actually be the output of a learning rule of arbitrary complexity. Indeed, even in simple neural circuits such as those studied in the classic studies of the crab and lobster stomatogastric ganglia, very different physical processes (i.e., synaptic conductances) were shown to be capable of leading to similar emergent circuit properties (prinz_similar_200, 89). Such redundancy should similarly be considered in future work evaluating history-dependence in gated accumulation—can multiple different across-trial learning schemes describe trial-averaged data equally well? Thus, while we can conclude that our gated accumulation model can describe the trial-averaged statistics of history-dependence we observe, our results do not determine anything about specific learning rules or cost functions that gave rise to the history-dependence, nor why participants failed to behave in an algorithmically random fashion.

Our model is statistical in nature, but was inspired by a biophysical model that was originally proposed as a model of the transformation of visual information into accumulated evidence in the frontal eye fields (FEF) (46, 47). Recording

single-neuron activity following the onset of a visual stimuli evokes transient changes to the firing rate of some FEF neurons (40, 47) at latencies between 50 and 100 ms. These “visual” neurons (but see Ref. 90) are thought to be located in superficial cortical layers, which receive visual input from intraparietal sulcus (91, 92), extrastriate cortex (93, 94), and the mediodorsal nucleus of the thalamus (95), among others. Pyramidal cells in layer V FEF, on the other hand, innervate the superior colliculus (96) and basal ganglia (97); neurons of this category are thought to correspond with movement neurons (97–99), which begin a ramping discharge after the visual response, reaching a threshold firing rate that correlates with saccade onset (36, 100). Given the similar profiles of these motor neurons and accumulator models, it has been suggested that the delay in FEF motor neuron activity reflects a transformation of visual information into a motor command. Gated accumulation in FEF was proposed as a mechanism by which ongoing visual transients are filtered from the accumulation of evidence, allowing only salient information to be passed on. The model captures the timecourse of firing rates from visual and motor neurons in different speed-accuracy conditions. The neural implementation of such transformations are a matter of debate (47, 101).

Our work suggests that the temporal dynamics of this transformation involve a prediction of sensory timing sensitive to trial by trial changes. Classically, the striatum is associated with trial by trial learning (75, 102–107). In the striatum, the substantia nigra tonically inhibits collicular activity necessary for saccade generation; this inhibition is released following bursting activity from the caudate, which is reciprocally connected to cortical regions responsible for saccade planning, such as FEF (108). Thus, in addition to reinforcement learning, the striatum is also thought to be important for balancing the facilitation and inhibition of motor plans. Layer V of FEF is reciprocally connected with the striatum (109, 110). Pharmacological manipulation of dopamine D2 receptors, primarily found in deep layers, in FEF resulted in a trial-trial repetition bias in the same free choice saccade task used in this paper, whereas D1 receptors, distributed throughout cortex, resulted in an alternation bias that is typically correlated with sensory processing (50, 111), suggesting that the behavioral dissociation observed in the saccadic system between these biases has a physical correlate.

The striatum is also implicated in timing behavior. Electrophysiological evidence suggests that monkey striatum amplifies decision dynamics in cortex as a function of urgency (112). Timekeeping deficits are also correlated with damage to the striatum in humans, along with classical symptoms of impaired movement initiation (for review, see Ref. 113). So in addition to inhibition of movement and reward-based learning, there is evidence for striatal involvement in temporal processing. Schall et al. (47) suggested the striatum as a potential physiological source for providing an inhibitory signal to movement neurons based on the strength of ongoing visual information, along with axo-axonal shunting and intermediary interneurons within FEF. In our model fitting results, we found that the variance in the baseline dynamics of the gated accumulator best explained the choice repetition

data. Given the importance of spiking variability on preparatory eye movement signals in FEF, future work may assess if and how this variability is modulated by trial history.

Our modeling work adds to a growing list of varieties of accumulator models, many of which have very different episodic intents. Accumulator models in this respect are in a somewhat unique position, in the sense that they are used as both an abstraction of a physical process that makes predictions about mechanisms, and a statistical model that is used to differentiate underlying physical processes across groups or individuals. Perhaps as a result of this, most formulations of accumulator models contain a mixture of theoretical and technical arguments that combine to fit a reaction time distribution. Technical assumptions are usually justified by mathematical convenience rather than logic or theory, yet are necessary to constrain models such that the selective influence of parameters or different model classes (collapsing bounds, baseline variability, etc.) can be dissociated by data. Indeed when these models have technical assumptions removed, they are redundant in the sense that they can represent any reaction time distribution even when restricting some degrees of freedom (114).

Part of the difficulty in dissociating models and model parameters comes from an additional degree of freedom that must be allowed when one is forced to consider nonstationarity as an additional source of variability. Previously, it has been suggested that baseline evidence in an accumulator framework can be thought of as analogous to a prior belief over the likelihood of one of a limited number of choice alternatives (18, 61), motivating the adoption of reinforcement learning approaches to model trial-trial variability in saccadic reaction times (31, 115, 116). We showed in behavioral data that anticipatory and sensory-guided reaction times are jointly influenced by choice history. In our model, we can account for this dependency by allowing the parameters of the baseline dynamic to remain free across fits to choice sequence data. This is a claim for sufficiency of trial-trial variability to capture sequence effects for a specific data set, however, it does not rule out history-dependent variance in sensory integration, as has been reported elsewhere (27, 117). Without ground-truth knowledge of a system's dynamics, it is difficult to estimate the cause of trial-trial variance in model parameters, simply because the hypothesis space over potential models becomes intractable when forced to consider each individual trial as a singularly observable outcome of a class of models that is inherently stochastic and designed to explain ensembles of trials (118).

Because of this, we wish to elaborate on the distinction between our analysis of choice history, and any conclusions that may be drawn with respect to the functional form of underlying history-dependence. What we do report is a dependence on the outcome of previous trials on anticipatory and sensory-guided reaction times for a particular task. From the perspective of the instructions of the task, history effects may be considered to denote some form of suboptimality in the decision process—as the task statistics were randomized, information from the previous trial contained no evidentiary value beyond providing a single sample of the task statistics. Arguments for why history effects persist in different tasks are numerous. Some have argued that

history effects in randomized experiments (often binary series) reflect a capacity limitation of the system (119–121). Others reframe the question in the context of optimality by considering these history effects as carry-over from rational behavior in other contexts where statistical regularity is more prevalent, or question if bias is truly present (122–124). From our data, there is little to be concluded about why history effects are present here, simply because the number of sufficient explanations is such that redundancy is likely (89), and falsification is a challenge (69). However, we can conclude that saccadic choice was not independent of previous trials, and this history-dependence could be explained by a gated accumulation model driven by a prediction of the timing of future sensory information.

DATA AVAILABILITY

Python code for the gated accumulator model, data analysis, and model fitting can be found on <https://github.com/BJCaie/Gated-Accumulation>. Data are available at <https://doi.org/10.17605/OSF.IO/6ZJKD>.

ACKNOWLEDGMENTS

The first author would like to thank the members of the Computational Vision and Kinematics Group and the Theoretical Cognitive Science Group.

GRANTS

Financial support for this project was provided by the Natural Sciences and Engineering Research Council of Canada (NSERC), the Canada Foundation for Innovation (CFI), and the Deutsche Forschungsgemeinschaft.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

B.C. and G.B. conceived and designed research; B.C. performed experiments; B.C. and G.B. analyzed data; B.C., D.E., A.Z.K., and G.B. interpreted results of experiments; B.C. and G.B. prepared figures; B.C., A.Z.K., and G.B. drafted manuscript; B.C., D.E., A.Z.K., and G.B. edited and revised manuscript; B.C., D.E., A.Z.K., and G.B. approved final version of manuscript.

REFERENCES

1. Griffiths TL, Tenenbaum JB. Optimal predictions in everyday cognition. *Psychol Sci* 17: 767–773, 2006. doi:10.1111/j.1467-9280.2006.01780.x.
2. Gershman SJ, Horvitz EJ, Tenenbaum JB. Computational rationality: a converging paradigm for intelligence in brains, minds, and machines. *Science* 349: 273–278, 2015. doi:10.1126/science.aac6076.
3. Richards BA, Frankland PW. The persistence and transience of memory. *Neuron* 94: 1071–1084, 2017. doi:10.1016/j.neuron.2017.04.037.
4. Rao RPN, Ballard DH. Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. *Nat Neurosci* 2: 79–87, 1999. doi:10.1038/4580.
5. Clark A. *Surfing Uncertainty: Prediction, Action, and the Embodied Mind*. Oxford: Oxford University Press, 2016, p. 424.

6. **Friston K, Kilner J, Harrison L.** A free energy principle for the brain. *J Physiol Paris* 100: 70–87, 2006. doi:10.1016/j.jphysparis.2006.10.001.
7. **Jarvik ME.** Probability learning and a negative recency effect in the serial anticipation of alternative symbols. *J Exp Psychol* 41: 291–297, 1951. doi:10.1037/h0056878.
8. **LaPlace PS.** *A Philosophical Essay on Probabilities*. New York: Dover, 1951.
9. **Nickerson RS.** The production and perception of randomness. *Psychol Rev* 109: 330–357, 2002. doi:10.1037/0033-295X.109.2.330.
10. **Bakan P.** Response-tendencies in attempts to generate random binary series. *Am J Psychol* 73: 127–131, 1960. doi:10.2307/1419124.
11. **Gilovich T, Vallone R, Tversky A.** The hot hand in basketball: on the misperception of random sequences. *Cogn Psychol* 17: 295–314, 1985. doi:10.1016/0010-0285(85)90010-6.
12. **Bar-Hillel M, Wagenaar WA.** The perception of randomness. *Adv Appl Math* 12: 428–454, 1991. doi:10.1016/0196-8858(91)90029-1.
13. **Mookherjee D, Sopher B.** Learning behaviour in an experimental matching pennies game. *Games Econ Behav* 7: 62–91, 1994. doi:10.1006/game.1994.1037.
14. **Abrahamyan A, Silva LL, Dakin SC, Carandini M, Gardner JL.** Adaptable history biases in human perceptual decisions. *Proc Natl Acad Sci USA* 113: E3548–E3557, 2016. doi:10.1073/pnas.1518786113.
15. **Stone M.** Models for choice reaction time. *Psychometrika* 25: 251–260, 1960. doi:10.1007/BF02289729.
16. **Busmeyer JR, Townsend JT.** Decision field theory: a dynamic-cognitive approach to decision making in an uncertain environment. *Psychol Rev* 100: 432–459, 1993. doi:10.1037/0033-295X.100.3.432.
17. **Dzhafarov EN.** Grice-representability of response time distribution families. *Psychometrika* 58: 281–314, 1993. doi:10.1007/BF02294577.
18. **Carpenter RH, Williams ML.** Neural computation of log likelihood in control of saccadic eye movements. *Nature* 377: 59–62, 1995. doi:10.1038/377059a0.
19. **Usher M, McClelland JL.** The time course of perceptual choice: the leaky, competing accumulator model. *Psychol Rev* 108: 550–592, 2001. doi:10.1037/0033-295X.108.3.550.
20. **Ratcliff R, Smith PL.** A comparison of sequential sampling models for two-choice reaction time. *Psychol Rev* 111: 333–367, 2004. doi:10.1037/0033-295X.111.2.333.
21. **Bogacz R, Brown E, Moehlis J, Holmes P, Cohen JD.** The physics of optimal decision making: a formal analysis of models of performance in two-alternative forced-choice tasks. *Psychol Rev* 113: 700–765, 2006. doi:10.1037/0033-295X.113.4.700.
22. **Wong KF, Wang XJ.** A recurrent network mechanism of time integration in perceptual decisions. *J Neurosci* 26: 1314–1328, 2006. doi:10.1523/JNEUROSCI.3733-05.2006.
23. **Standage D, Wang DH, Blohm G.** Neural dynamics implement a flexible decision bound with a fixed firing rate for choice: a model-based hypothesis. *Front Neurosci* 8: 318, 2014. doi:10.3389/fnins.2014.00318.
24. **Ratcliff R, McKoon G.** The diffusion decision model: theory and data for two-choice decision tasks. *Neural Comput* 20: 873–922, 2008. doi:10.1162/neco.2008.12.06.420.
25. **Mulder MJ, Wagenmakers E-J, Ratcliff R, Boekel W, Forstmann BU.** Bias in the brain: a diffusion model analysis of prior probability and potential payoff. *J Neurosci* 32: 2335–2343, 2012. doi:10.1523/JNEUROSCI.4156-11.2012.
26. **Glaze CM, Kable JW, Gold JI.** Normative evidence accumulation in unpredictable environments. *eLife* 4: e08825, 2015. doi:10.7554/eLife.08825.
27. **Urai AE, de Gee JW, Tsetsos K, Donner TH.** Choice history biases subsequent evidence accumulation. *eLife* 8: e46331, 2019. doi:10.7554/eLife.46331.
28. **Simen P.** Evidence accumulator or decision threshold—which cortical mechanism are we observing? *Front Psychol* 3: 183, 2012. doi:10.3389/fpsyg.2012.00183.
29. **Fan Y, Gold JI, Ding L.** Frontal eye field and caudate neurons make different contributions to reward-biased perceptual decisions. *eLife* 9: e60535, 2020. doi:10.7554/eLife.60535.
30. **Bitzer S, Park H, Blankenburg F, Kiebel SJ.** Perceptual decision making: drift-diffusion model is equivalent to a Bayesian model. *Front Hum Neurosci* 8: 102, 2014. doi:10.3389/fnhum.2014.00102.
31. **Kim TD, Kabir M, Gold JI.** Coupled decision processes update and maintain saccadic priors in a dynamic environment. *J Neurosci* 37: 3632–3645, 2017. doi:10.1523/JNEUROSCI.3078-16.2017.
32. **Schall JD.** Accumulators, neurons, and response time. *Trends Neurosci* 42: 848–860, 2019. doi:10.1016/j.tins.2019.10.001.
33. **Jazayeri M, Shadlen MN.** Temporal context calibrates interval timing. *Nat Neurosci* 13: 1020–1026, 2010. doi:10.1038/nn.2590.
34. **Taatgen N, van Rijn H.** Traces of times past: representations of temporal intervals in memory. *Mem Cognit* 39: 1546–1560, 2011. doi:10.3758/s13421-011-0113-0.
35. **Acerbi L, Wolpert DM, Vijayakumar S.** Internal representations of temporal statistics and feedback calibrate motor-sensory interval timing. *PLoS Comput Biol* 8: e1002771, 2012. doi:10.1371/journal.pcbi.1002771.
36. **Hanes DP, Schall JD.** Neural control of voluntary movement initiation. *Science* 274: 427–430, 1996. doi:10.1126/science.274.5286.427.
37. **Gold JI, Shadlen MN.** Neural computations that underlie decisions about sensory stimuli. *Trends Cogn Sci* 5: 10–16, 2001. doi:10.1016/S1364-6613(00)01567-9.
38. **Huk AC, Katz LN, Yates JL.** The role of the lateral intraparietal area in (the study of) decision making. *Annu Rev Neurosci* 40: 349–372, 2017. doi:10.1146/annurev-neuro-072116.
39. **Shadlen MN, Kiani R.** Decision making as a window on cognition. *Neuron* 80: 791–806, 2013. doi:10.1016/j.neuron.2013.10.047.
40. **Schmoleksy MT, Wang Y, Hanes DP, Thompson KG, Leutgeb S, Schall JD, Leventhal AG.** Signal timing across the macaque visual system. *J Neurophysiol* 79: 3272–3278, 1998. doi:10.1152/jn.1998.79.6.3272.
41. **Shadmehr R.** Control of movements and temporal discounting of reward. *Curr Opin Neurobiol* 20: 726–730, 2010. doi:10.1016/j.conb.2010.08.017.
42. **Lee J, Joshua M, Medina JF, Lisberger SG.** Signal, noise, and variation in neural and sensory-motor latency. *Neuron* 90: 165–176, 2016. doi:10.1016/j.neuron.2016.02.012.
43. **Lee J, Darlington TR, Lisberger SG.** The neural basis for response latency in a sensory-motor behavior. *Cerebral Cortex* 30: 3055–3073, 2020. doi:10.1093/cercor/bhz294.
44. **Teichert T, Grinband J, Ferrera V.** The importance of decision onset. *J Neurophysiol* 115: 643–661, 2016. doi:10.1152/jn.00274.2015.
45. **van den Brink RL, Murphy PR, Desender K, de Ru N, Nieuwenhuis S.** Temporal expectation hastens decision onset but does not affect evidence quality. *J Neurosci* 41: 130–143, 2021. doi:10.1523/JNEUROSCI.1103-20.2020.
46. **Purcell BA, Schall JD, Logan GD, Palmeri TJ.** From salience to saccades: multiple-alternative gated stochastic accumulator model of visual search. *J Neurosci* 32: 3433–3446, 2012. doi:10.1523/JNEUROSCI.4622-11.2012.
47. **Schall JD, Purcell BA, Heitz RP, Logan GD, Palmeri TJ.** Neural mechanisms of saccade target selection: gated accumulator model of the visual-motor cascade. *Eur J Neurosci* 33: 1991–2002, 2011. doi:10.1111/j.1460-9568.2011.07715.x.
48. **Schiller PH, Chou IH.** The effects of frontal eye field and dorsomedial frontal cortex lesions on visually guided eye movements. *Nat Neurosci* 1: 248–253, 1998. doi:10.1038/693.
49. **Noudoost B, Moore T.** Control of visual cortical signals by prefrontal dopamine. *Nature* 474: 372–375, 2011. doi:10.1038/nature09995.
50. **Soitani A, Noudoost B, Moore T.** Dissociable dopaminergic control of saccadic target selection and its implications for reward modulation. *Proc Natl Acad Sci USA* 110: 3579–3584, 2013. doi:10.1073/pnas.1221236110.
51. **Kuss M, Jäkel F, Wichmann FA.** Bayesian inference for psychometric functions. *J Vis* 5: 478–492, 2005. doi:10.1167/5.5.8.
52. **Schütt HH, Harmeling S, Macke JH, Wichmann FA.** Painfree and accurate Bayesian estimation of psychometric functions for (potentially) overdispersed data. *Vision Res* 122: 105–123, 2016. doi:10.1016/j.visres.2016.02.002.
53. **Nijhawan R.** Visual prediction: psychophysics and neurophysiology of compensation for time delays. *Behav Brain Sci* 31: 179–198, 2008. doi:10.1017/S0140525X08003804.

54. **Cho RY, Nystrom LE, Brown ET, Jones AD, Braver TS, Holmes PJ, Cohen JD.** Mechanisms underlying dependencies of performance on stimulus history in a two-alternative forced-choice task. *Cogn Affect Behav Neurosci* 2: 283–299, 2002. doi:10.3758/CABN.2.4.283.
55. **Kingstone A, Klein RM.** What are human express saccades? *Percept Psychophys* 54: 260–273, 1993. doi:10.3758/BF03211762.
56. **Reddi BA, Carpenter RH.** The influence of urgency on decision time. *Nat Neurosci* 3: 827–830, 2000. doi:10.1038/77739.
57. **Nakahara H, Nakamura K, Hikosaka O.** Extended LATER model can account for trial-by-trial variability of both pre- and post-processes. *Neural Netw* 19: 1027–1046, 2006. doi:10.1016/j.neunet.2006.07.001.
58. **Noorani I.** LATER models of neural decision behavior in choice tasks. *Front Integr Neurosci* 8: 67, 2014. doi:10.3389/fnint.2014.00067.
59. **Gisiger T, Boukadoum M.** Mechanisms gating the flow of information in the cortex: what they might look like and what their uses may be. *Front Comput Neurosci* 5: 1, 2011. doi:10.3389/fncom.2011.00001.
60. **Cisek P, Puskas GA, El-Murr S.** Decisions in changing conditions: the urgency-gating model. *J Neurosci* 29: 11560–11571, 2009. doi:10.1523/JNEUROSCI.1844-09.2009.
61. **Gold JI, Shadlen MN.** The neural basis of decision making. *Annu Rev Neurosci* 30: 535–574, 2007. doi:10.1146/annurev.neuro.29.051605.113038.
62. **Lipton A, Kaushansky V.** On the first hitting time density of an Ornstein-Uhlenbeck process (Preprint). *arXiv*, 2018. doi:10.48550/arXiv.1810.02390.
63. **Carpenter RHS.** Oculomotor procrastination. In: *Eye Movements: Cognition and Visual Perception*, edited by Fisher DF, Monty RA, Senders JW. Hillsdale, NJ: Lawrence Erlbaum Associates, 1981, p. 237–246.
64. **Soltani A, Khorsand P, Guo C, Farashahi S, Liu J.** Neural substrates of cognitive biases during probabilistic inference. *Nat Commun* 7: 11393, 2016. doi:10.1038/ncomms11393.
65. **Nobre AC, van Ede F.** Anticipated moments: temporal structure in attention. *Nat Rev Neurosci* 19: 34–48, 2018. doi:10.1038/nrn.2017.141.
66. **Hernández-Navarro L, Hermoso-Mendizabal A, Duque D, de la Rocha J, Hyafil A.** Proactive and reactive accumulation-to-bound processes compete during perceptual decisions. *Nat Commun* 12: 7148, 2021. doi:10.1038/s41467-021-27302-8.
67. **Hikosaka O, Isoda M.** Switching from automatic to controlled behavior: corticobasal ganglia mechanisms. *Trends Cogn Sci* 14: 154–161, 2010. doi:10.1016/j.tics.2010.01.006.
68. **Evans JS.** Dual-processing accounts of reasoning, judgment, and social cognition. *Annu Rev Psychol* 59: 255–278, 2008. doi:10.1146/annurev.psych.59.103006.093629.
69. **Caie B, Blohm G.** History-dependence shapes causal inference of brain-behaviour relationships (Preprint). *arXiv*, 2024. doi:10.48550/arXiv.2403.00947.
70. **Carland MA, Thura D, Cisek P.** The urgency-gating model can explain the effects of early evidence. *Psychon Bull Rev* 22: 1830–1838, 2015. doi:10.3758/s13423-015-0851-2.
71. **Winkel J, Keuken MC, van Maanen L, Wagenmakers EJ, Forstmann BU.** Early evidence affects later decisions: why evidence accumulation is required to explain response time data. *Psychon Bull Rev* 21: 777–784, 2014. doi:10.3758/s13423-013-0551-8.
72. **Evans NJ, Hawkins GE, Boehm U, Wagenmakers EJ, Brown SD.** The computations that support simple decision-making: a comparison between the diffusion and urgency-gating models. *Sci Rep* 7: 16433, 2017. doi:10.1038/s41598-017-16694-7.
73. **Neftci EO, Averbach BB.** Reinforcement learning in artificial and biological systems. *Nat Mach Intell* 1: 133–143, 2019. doi:10.1038/s42256-019-0025-4.
74. **Montague PR, Dayan P, Sejnowski TJ.** A framework for mesencephalic dopamine systems based on predictive Hebbian learning. *J Neurosci* 16: 1936–1947, 1996. doi:10.1523/JNEUROSCI.16-05-01936.1996.
75. **Schultz W.** Predictive reward signal of dopamine neurons. *J Neurophysiol* 80: 1–27, 1998. doi:10.1152/jn.1998.80.1.1.
76. **Barto AG.** Adaptive critics and the basal ganglia. In: *Models of Information Processing in the Basal Ganglia*, edited by Houk JC, Davis J, Beiser D. Cambridge, MA: MIT Press, 1995, p. 215–232.
77. **Harlow HF.** The formation of learning sets. *Psychol Rev* 56: 51–65, 1949. doi:10.1037/h0062474.
78. **Tolman EC.** Cognitive maps in rats and men. *Psychol Rev* 55: 189–208, 1948. doi:10.1037/h0061626.
79. **Acuna DE, Schrater P.** Structure learning in human sequential decision-making. *PLoS Comput Biol* 6: e1001003, 2010. doi:10.1371/journal.pcbi.1001003.
80. **Doya K.** What are the computations of the cerebellum, the basal ganglia and the cerebral cortex? *Neural Netw* 12: 961–974, 1999. doi:10.1016/s0893-6080(99)00046-5.
81. **Daw ND, Kakade S, Dayan P.** Opponent interactions between serotonin and dopamine. *Neural Netw* 15: 603–616, 2002. doi:10.1016/s0893-6080(02)00052-7.
82. **Nakahara H, Hikosaka O.** Learning to represent reward structure: a key to adapting to complex environments. *Neurosci Res* 74: 177–183, 2012. doi:10.1016/j.neures.2012.09.007.
83. **Nakahara H.** Multiplexing signals in reinforcement learning with internal models and dopamine. *Curr Opin Neurobiol* 25: 123–129, 2014. doi:10.1016/j.conb.2014.01.001.
84. **Levy P.** Sur certains processus stochastiques homogènes. *Compos Math* 7: 283–339, 1940.
85. **Kording KP, Tenenbaum JB, Shadmehr R.** The dynamics of memory as a consequence of optimal adaptation to a changing body. *Nat Neurosci* 10: 779–786, 2007. doi:10.1038/nn1901.
86. **Murray JD, Bernacchia A, Freedman DJ, Romo R, Wallis JD, Cai X, Padoa-Schioppa C, Pasternak T, Seo H, Lee D, Wang XJ.** A hierarchy of intrinsic timescales across primate cortex. *Nat Neurosci* 17: 1661–1663, 2014. doi:10.1038/nn.3862.
87. **Levins R.** *Evolution in Changing Environments: Some Theoretical Explorations*. Princeton, NJ: Princeton University Press, 1968.
88. **Cheong KH, Tan ZX, Xie NG, Jones MC.** A paradoxical evolutionary mechanism in stochastically switching environments. *Sci Rep* 6: 34889, 2016. doi:10.1038/srep34889.
89. **Marder E.** Variability, compensation, and modulation in neurons and circuits. *Proc Natl Acad Sci USA* 108, Suppl 3: 15542–15548, 2011. doi:10.1073/pnas.1010674108.
90. **Lowe KA, Schall JD.** Functional categories of visuomotor neurons in macaque frontal eye field. *eNeuro* 5: ENEURO.0131-18.2018, 2018. doi:10.1523/ENEURO.0131-18.2018.
91. **Huerta MF, Krubitzer LA, Kaas JH.** Frontal eye field as defined by intracortical microstimulation in squirrel monkeys, owl monkeys, and macaque monkeys. II. Cortical connections. *J Comp Neurol* 265: 332–361, 1987. doi:10.1002/cne.902650304.
92. **Stanton GB, Bruce CJ, Goldberg ME.** Topography of projections to posterior cortical areas from the macaque frontal eye fields. *J Comp Neurol* 353: 291–305, 1995. doi:10.1002/cne.903530210.
93. **Schall JD, Morel A, King DJ, Bullier J.** Topography of visual cortex connections with frontal eye field in macaque: convergence and segregation of processing streams. *J Neurosci* 15: 4464–4487, 1995. doi:10.1523/JNEUROSCI.15-06-04464.1995.
94. **Jouve B, Rosenstiehl P, Imbert M.** A mathematical approach to the connectivity between the cortical visual areas of the macaque monkey. *Cereb Cortex* 8: 28–39, 1998. doi:10.1093/cercor/8.1.28.
95. **Huerta MF, Krubitzer LA, Kaas JH.** Frontal eye field as defined by intracortical microstimulation in squirrel monkeys, owl monkeys, and macaque monkeys. I. Subcortical connections. *J Comp Neurol* 253: 415–439, 1986. doi:10.1002/cne.902530402.
96. **Sommer MA, Wurtz RH.** Frontal eye field neurons orthodromically activated from the superior colliculus. *J Neurophysiol* 80: 3331–3335, 1998. doi:10.1152/jn.1998.80.6.3331.
97. **Segraves MA, Goldberg ME.** Functional properties of corticotectal neurons in the monkey's frontal eye field. *J Neurophysiol* 58: 1387–1419, 1987. doi:10.1152/jn.1987.58.6.1387.
98. **Sommer MA, Wurtz RH.** Composition and topographic organization of signals sent from the frontal eye field to the superior colliculus. *J Neurophysiol* 83: 1979–2001, 2000. doi:10.1152/jn.2000.83.4.1979.
99. **Ferraina S, Pare M, Wurtz RH.** Comparison of cortico-cortical and corticocollicular signals for the generation of saccadic eye movements. *J Neurophysiol* 87: 845–858, 2002. doi:10.1152/jn.00317.2001.
100. **Hanes DP, Patterson WF 2nd, Schall JD.** Role of frontal eye fields in countermanning saccades: visual, movement, and fixation activity. *J Neurophysiol* 79: 817–834, 1998. doi:10.1152/jn.1998.79.2.817.

101. **Sajad A, Sadeh M, Crawford JD.** Spatiotemporal transformations for gaze control. *Physiol Rep* 8: e14533, 2020. doi:[10.14814/phy2.14533](https://doi.org/10.14814/phy2.14533).
102. **Kawagoe R, Takikawa Y, Hikosaka O.** Expectation of reward modulates cognitive signals in the basal ganglia. *Nat Neurosci* 1: 411–416, 1998. doi:[10.1038/1625](https://doi.org/10.1038/1625).
103. **Kravitz AV, Tye LD, Kreitzer AC.** Distinct roles for direct and indirect pathway striatal neurons in reinforcement. *Nat Neurosci* 15: 816–818, 2012. doi:[10.1038/nn.3100](https://doi.org/10.1038/nn.3100).
104. **Skelin I, Hakstol R, VanOyen J, Mudiayi D, Molina LA, Holec V, Hong NS, Euston DR, McDonald RJ, Gruber AJ.** Lesions of dorsal striatum eliminate lose-switch responding but not mixed-response strategies in rats. *Eur J Neurosci* 39: 1655–1663, 2014. doi:[10.1111/ejn.12518](https://doi.org/10.1111/ejn.12518).
105. **Yttri EA, Dudman JT.** Opponent and bidirectional control of movement velocity in the basal ganglia. *Nature* 533: 402–406, 2016. doi:[10.1038/nature17639](https://doi.org/10.1038/nature17639).
106. **Yttri EA, Dudman JT.** A proposed circuit computation in basal ganglia: history dependent gain. *Mov Disord* 35: 704–716, 2018. doi:[10.1002/mds.27321](https://doi.org/10.1002/mds.27321).
107. **Hwang EJ, Link TD, Hu YY, Lu S, Wang EH, Lilascharoen V, Aronson S, O'Neil K, Lim BK, Komiyama T.** Corticostriatal flow of action selection bias. *Neuron* 104: 1126–1140.e6, 2019. doi:[10.1016/j.neuron.2019.09.028](https://doi.org/10.1016/j.neuron.2019.09.028).
108. **Hikosaka O, Wurtz RH.** Visual and oculomotor functions of monkey substantia nigra pars reticulata. IV. Relation of substantia nigra to superior colliculus. *J Neurophysiol* 49: 1285–1301, 1983. doi:[10.1152/jn.1983.49.5.1285](https://doi.org/10.1152/jn.1983.49.5.1285).
109. **Stanton GB, Goldberg ME, Bruce CJ.** Frontal eye field efferents in the macaque monkey: I. Subcortical pathways and topography of striatal and thalamic terminal fields. *J Comp Neurol* 271: 473–492, 1988. doi:[10.1002/cne.902710402](https://doi.org/10.1002/cne.902710402).
110. **Parthasarathy HB, Schall JD, Graybiel AM.** Distributed but convergent ordering of corticostriatal projections: analysis of the frontal eye field and the supplementary eye field in the macaque monkey. *J Neurosci* 12: 4468–4488, 1992. doi:[10.1523/JNEUROSCI.12-11-04468.1992](https://doi.org/10.1523/JNEUROSCI.12-11-04468.1992).
111. **Fecteau JH, Au C, Armstrong IT, Munoz DP.** Sensory biases produce alternation advantage found in sequential saccadic eye movement tasks. *Exp Brain Res* 159: 84–91, 2004. doi:[10.1007/s00221-004-1935-9](https://doi.org/10.1007/s00221-004-1935-9).
112. **Thura D, Cisek P.** The basal ganglia do not select reach targets but control the urgency of commitment. *Neuron* 95 95: 1160–1170.e5, 2017. doi:[10.1016/j.neuron.2017.07.039](https://doi.org/10.1016/j.neuron.2017.07.039).
113. **Buhusi CV, Meck WH.** What makes us tick? Functional and neural mechanisms of interval timing. *Nat Rev Neurosci* 6: 755–765, 2005. doi:[10.1038/nrn1764](https://doi.org/10.1038/nrn1764).
114. **Jones M, Dzhafarov EN.** Unfalsifiability and mutual translatability of major modeling schemes for choice reaction time. *Psychol Rev* 121: 1–32, 2014 [Erratum in *Psychol Rev* 121: 150, 2014]. doi:[10.1037/a0034190](https://doi.org/10.1037/a0034190).
115. **Pedersen ML, Frank MJ, Biele G.** The drift diffusion model as the choice rule in reinforcement learning. *Psychon Bull Rev* 24: 1234–1251, 2017. doi:[10.3758/s13423-016-1199-y](https://doi.org/10.3758/s13423-016-1199-y).
116. **McDougle SD, Collins AGE.** Modeling the influence of working memory, reinforcement, and action uncertainty on reaction time and choice during instrumental learning. *Psychon Bull Rev* 28: 20–39, 2021. doi:[10.3758/s13423-020-01774-z](https://doi.org/10.3758/s13423-020-01774-z).
117. **Steingrover H, Wabersich D, Wagenmakers EJ.** Modeling across-trial variability in the Wald drift rate parameter. *Behav Res Methods* 53: 1060–1076, 2021. doi:[10.3758/s13428-020-01448-7](https://doi.org/10.3758/s13428-020-01448-7).
118. **Carpenter RHS.** A neural mechanism that randomises behaviour. *J Consciousness Stud* 6: 13–22, 1999.
119. **Baddeley AD.** The capacity for generating information by randomization. *Q J Exp Psychol* 18: 119–129, 1966. doi:[10.1080/14640746608400019](https://doi.org/10.1080/14640746608400019).
120. **Tune GS.** A brief survey of variables that influence random-generation. *Percept Mot Skills* 18: 705–710, 1964. doi:[10.2466/pms.1964.18.3.705](https://doi.org/10.2466/pms.1964.18.3.705).
121. **Kahneman D, Tversky A.** Subjective probability: a judgment of representativeness. *Cogn Psychol* 3: 430–454, 1972. doi:[10.1016/0010-0285\(72\)90016-3](https://doi.org/10.1016/0010-0285(72)90016-3).
122. **Lopes LL.** Doing the impossible: a note on induction and the experience of randomness. *J Exp Psychol Learn Mem Cogn* 8: 626–636, 1982. doi:[10.1037/0278-7393.8.6.626](https://doi.org/10.1037/0278-7393.8.6.626).
123. **Ayton P, Hunt AJ, Wright G.** Psychological conceptions of randomness. *Behav Decision Making* 2: 221–238, 1989. doi:[10.1002/bdm.3960020403](https://doi.org/10.1002/bdm.3960020403).
124. **Kareev Y.** Not that bad after all: generation of random sequences. *J Exp Psychol Hum Percept Perform* 18: 1189–1194, 1992. doi:[10.1037/0096-1523.18.4.1189](https://doi.org/10.1037/0096-1523.18.4.1189).