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Review

# The LATER model of reaction time and decision

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ABSTRACT

How do we choose one option rather than another when faced with uncertainty about the information we receive, and the consequences of what we decide? The LATER (Linear Approach to Threshold with Ergodic Rate) model has proved to be remarkably accurate in predicting how we respond in such situations. Given its conceptual simplicity, its grounding in fundamental Bayesian principles and its very few free parameters, it is being increasingly adopted for a wider range of choice tasks, helping us to understand the underlying neural mechanisms, and in applying this to clinical disorders. Here, we provide a thorough discussion of the history behind this model, and how it can be applied to more complex decisions, including anti-saccades, Go-NoGo, countermanding and other situations where newly-arriving information means that ongoing decisions must be modified. The neuroscience of decision-making is progressing rapidly, and we anticipate that wider understanding and application of this model will help simplify the interpretation of increasingly advanced decision behaviour both in the laboratory and clinic.


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## 1. Introduction

LATER is a model of response times, and of their underlying decision mechanisms. Conceived over 30 years ago (Carpenter, 1981), it has remained popular ever since because of its simplicity (the basic model has just two free parameters) and the ease with which it can be used to model behaviour in complex tasks, providing a detail of quantitative prediction that few if any other approaches can match. This simplicity has proved particularly attractive in the clinical field, where LATER can provide quantitative measures of pathological progression with a degree of precision that is rare in neurology (Antoniades et al., 2012; Burrell et al., 2013; Burrell et al., 2012; Dawson et al., 2011; Ghosh et al., 2010; Michell et al., 2006; Nouraei et al., 2004; Pearson et al., 2007; Perneczky et al., 2011; Smyrnis et al., 2009; Zoller et al., 2006); however, a discussion of these clinical applications (in Parkinson's and Huntington's disease, amyotrophic lateral sclerosis, dementia, progressive supranuclear palsy, endarterectomy, traumatic brain injury, schizophrenia, hepatic encephalopathy and migraine) lies outside the scope of this review. Necessarily, the majority of non-clinical studies using LATER have originated from our lab, as can be seen in the bibliography, but some that have not include (Avila and Lin, 2014; Beintema et al., 2005; Dolmenech and Dreher, 2010; Lauwereyns, 2010; Mackay et al., 2012; Montagnini and Chelazzi, 2005; Nakahara et al., 2006; Otto and Mamassian, 2012; Smyrnis et al., 2011).

Yet despite its widespread use, and application to an increasing range of types of behaviour, there is no single place where one can find a comprehensive description of LATER or of how it can be used, in particular for the increasingly complex tasks used to study decision-making. This article is an attempt to supply that need. It is essentially tutorial, and wholly LATER-centric, making no pretensions of comparing how good a job LATER does of explaining behavioural data with other models of reaction time and decision, such comparisons have been comprehensively undertaken elsewhere (for example by Gold and Shadlen (2007)); all it attempts is to set out the history of the LATER model in accounting for reaction times in terms of decision processes, and how its possible application to more complex tasks can improve our understanding of neural decision-making.

## 2. The relation between decision and response time

Experimentally, one can learn only a limited amount by studying people's actual decisions, for instance judgments of whether a stimulus is actually present. In the end, the answer is either yes or no, so that the rate of gathering information in real experiments is frustratingly low. It turns out that more can be learnt from studying something that at first sight may not seem very directly relevant, the time it takes to make a decision.

There are two basic facts about reaction time, both unexpected, that seem be true of the entire animal kingdom. The first is that reaction times are surprisingly long; the second is that they are unexpectedly variable.

One might well suppose that the length of reaction times would be explained by the extraordinarily long time it takes nerves to convey information. Even the fastest nerve fibres conduct at little more than 100 m/s, and to this must be added the millisecond or so that it takes for information to cross a synapses between one neuron and the next. In addition, it takes time for a stimulated muscle to begin to contract, and for a sensory receptor to generate a response to a stimulus. Yet actual reaction times are much greater than can be accounted for by the sum of all these factors.

Consider for instance the shortest anatomical route by which a visual stimulus could generate a saccade, through the superior colliculus. The colliculus is both a sensory and motor structure, in that neurons in the more superficial part respond to visual stimuli, forming a regular topographical map of the visual world; at deeper levels, electrical stimulation generates realistic saccades to particular points in space. Broadly speaking, these two maps correspond in the sense that the direction in visual space that best stimulates a certain point on the colliculus is also the destination of a saccade evoked by stimulating the same point (Cynader and Berman, 1972; Robinson, 1972). So one can think of the colliculus as providing a simple mapping function between visual stimuli and the eye movements needed to look at them. In the monkey, the visually-responsive cells begin to be active about 40 ms after we supply a visual stimulus; conversely, when we stimulate the deeper layers, the saccade begins after some 20 ms. So in principle this route ought to be able to generate a saccade in response to a stimulus with a latency of around 60 ms; yet average saccadic latencies are in practice at least three times as long. What is going on?

To understand why actual latencies are so much longer than one would expect, we need to understand something of the limitations of the colliculus. What it provides is a simple translation from a localised stimulus to a localized response. If all we ever had to look at were single targets in a darkened room, it would function beautifully. But it cannot cope even with two competing stimuli, let alone the huge number of potentially interesting targets typically present out in the real visual world at any moment. Furthermore, it lacks the kind of information needed to decide which of all these stimuli is actually worth looking at. Its visual cells localise visual objects, determining where they are: but they have no idea what they are, since they are incapable of discriminating either the shapes or the colours of what they are looking at. So they cannot be allowed to determine what we look at; this requires much more complex evaluative processes, of a kind the cerebral cortex in particular is better equipped to carry out. As this kind of processing is more time-consuming than the colliculus' simple mapping, these higher, cortical, levels must tonically suppress the colliculus, preventing it from making simple-minded responses, while these more sophisticated judgements are being made. Latency is, in other words, the result of procrastination: the brain deliberately withholds a possible early response in order to decide more carefully on a later one (Carpenter, 1981).

Corresponding to this, we see many inhibitory pathways descending from cortex, often through the basal ganglia, with neurons that fire continuously, often at a very high rate. Then, when the cortex has finished deciding about the next movement, this blanket of inhibition is lifted in a single localised area, permitting the colliculus then to carry out its basic function of converting visual location into an appropriately-directed eye movement (Hikosaka et al., 2000). A consequence of this procrastination is that latency is not telling us about conduction processes so much as about how long it takes the higher levels to work out what to look at. Reaction time is decision time.

Recognition that decision and reaction time are intimately intertwined, and that detailed quantitative analysis of one can shed light on the mechanism of the other, has been a powerful influence in advancing our understanding of this area (Gold and Shadlen, 2007; Schall, 2003a,b, 2005; Shadlen and Gold, 2004). This relationship underlies the way in which the LATER model was first conceived (as a purely empirical model of observed reaction time distributions), then interpreted (as a quasi-Bayesian model of how decisions ought ideally to be made), and finally used in more complex ways to explain behaviour in behavioural tasks requiring more than simple responses to single stimuli. The organization of the present review reflects this development.

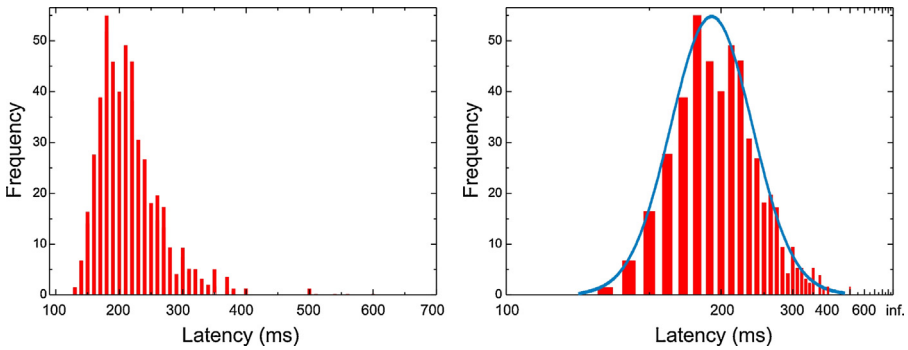



**Fig. 1.** A set of 486 saccadic latencies plotted as a conventional frequency histogram (left), showing the obvious skewness of the distribution. Right, the same data plotted using a reciprocal scale for latency: note that for convenience the latencies still increase to the right. The distribution is now relatively symmetrical, and indeed similar to a Gaussian (blue). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

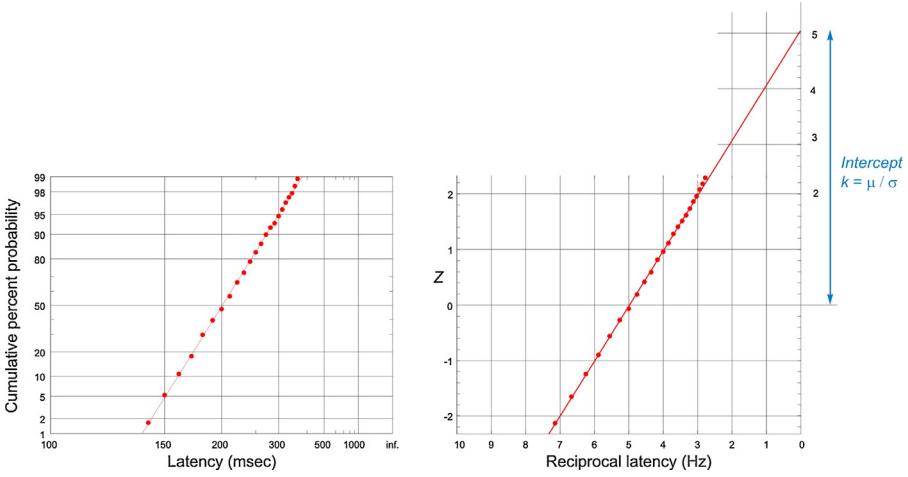



**Fig. 2.** Left, the same data as in the previous figure plotted as a cumulative histogram, using a probit scale (in effect an inverse error function transformation) that stretches the ends of the ordinate axis in such a way as to generate a straight line if the data is indeed Gaussian; since the latency uses a reciprocal scale, this is a reciprobit plot. Right, the same cumulative plot using alternative linearized axes: the latency scale is now explicitly of reciprocal latency ($1/T$), increasing leftwards to facilitate comparison; the probability scale uses units of $Z$. The data can be extrapolated to an intercept on the right-hand axis ($T$ = infinity or $1/T = 0$), whose value is $\mu/\sigma$ (in this case 5) in $Z$ units.

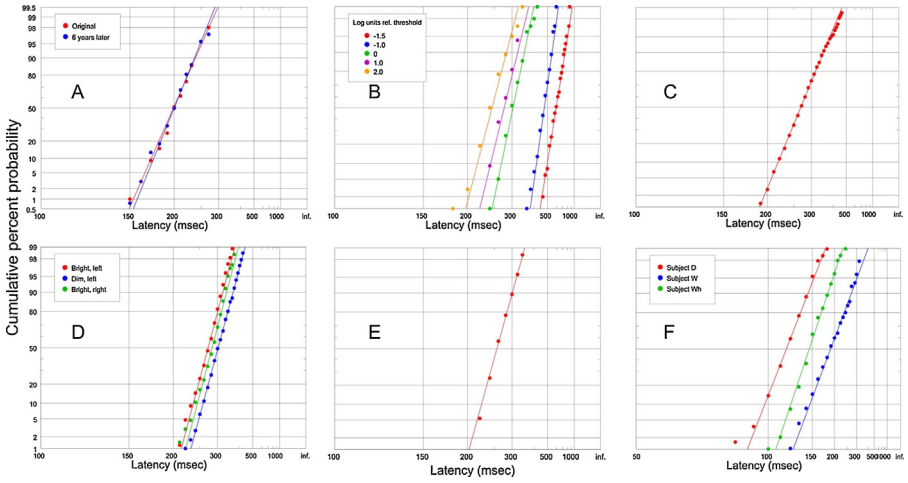



**Fig. 3.** Examples of reciprobit plots of reaction time distributions. (A) two different runs of around 100 saccadic trials at an interval of 6 years (R. H. S. Carpenter, unpublished data); (B) saccades to targets of different luminance, expressed in log units relative to foveal threshold, 100 trials per data set (Wheeless et al., 1967); (C) Human manual responses to a visual stimulus ($N = 825$) (Welford, 1959); (D) Human manual responses to visual stimuli (Johnson, 1918), comparing left and right hands, and bright and dim stimuli; (E) Feline paw responses (389 trials) to an auditory stimulus (Schmied et al., 1979); (F) Human manual responses to an auditory, stimulus in three subjects (194, 95 and 196 trials) (Wells, 1913).

## 3. LATER as an empirical description of how reaction times vary

Even in the very simplest tasks, like pressing a button in response to the illumination of a light, or moving the eyes to fixate a suddenly-appearing target, reaction times (RTs) vary substantially from one trial to another even when the target and circumstances are identical each time. This can be seen when a large number of

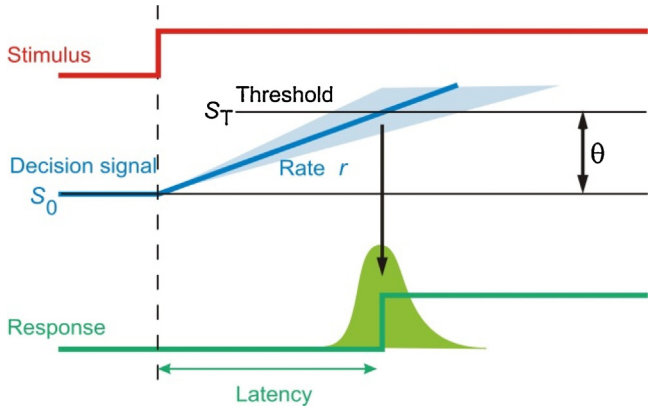



**Fig. 4.** LATER model. A decision signal whose initial value is $S_0$ begins to rise in response to the stimulus at a constant rate $r$ until it reaches a threshold at $S_T = S_0 + \theta$, when it triggers the response. On different trials, $r$ varies in a Gaussian manner with mean $\mu$ and variance $\sigma^2$; as a result the latency distribution (green) is skewed. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

them are plotted as a conventional frequency histogram (Fig. 1, left). A universal finding, true whatever the stimulus (visual, auditory or even tactile) or response (manual, oculomotor or nose-poke) or species is that such distributions are skewed, with a prolonged tail of longer latencies. Yet this universally recurring shape is not found to obey any of the standard mathematical functions (Gaussian, Poisson, binomial etc) that represent the basic stochastic processes commonly found in Nature. It was in 1981 that it occurred to the senior author that perhaps the reason for this frustrating state of affairs was that the problem was being viewed the wrong way round: we should be looking at the underlying mechanism for the variability, rather than its effect (Carpenter, 1981). Reaction time is after all the consequence of a process initiated by the stimulus that reaches completion in the response, as in a chemical reaction. So perhaps we should be asking ourselves how that rate varies from trial to trial, and analyse not the reaction time $T$, but its reciprocal, $1/T$. The effect of this change of viewpoint was almost magical: when plotted as a function of reciprocal RT, the distributions lost their skewness, and in fact turned out to obey the most fundamental of all stochastic laws, the normal or Gaussian distribution (Fig. 1, right).

The best way to illustrate this is to plot distributions as cumulative histograms, showing not the fraction of responses lying for instance between 200 and 210 ms, and then between 210 and 220, and so on, but rather the total percentage less than 200 ms, less than 210, etc. From a mathematical point of view such distributions are far more satisfactory than conventional frequency histograms, popular though these are: they look less noisy, allow multiple distributions to be displayed at once, do not depend in an arbitrary way on the size of the bin that has been chosen, and are normalized in the sense that the values must run from 0 to 1. Better still is to use not a linear scale for the vertical frequency axis, but a non-linear probit scale (embodying the inverse error function), which is stretched out from each side of 50% in such a way that a normal distribution will generate a straight line, as can be seen in Fig. 2 (left). One can then see at once whether a given distribution is normal (backed up by applying statistical tests such as the Kolmogorov–Smirnov one-sample for comparing an observed distribution with a theoretical one, or more specific tests for normality), and form a visual impression of the two parameters describing it, the median (which is also the mean, $\mu$), where the line intersects $p = 50\%$, and the standard deviation $\sigma$ which is determines the slope (a steep line has a small variance, a shallow one a greater one). One can also extend the line until it crosses the right-hand ($T$ = infinity) axis (Fig. 2, right): this intercept $k$, which is equal to $\mu/\sigma$, represents the probability of not making a response at all. It is convenient to refer to a plot of this kind as a reciprobit plot, and the underlying distribution itself as recinormal.

Fig. 3 shows some examples of reciprobit plots using a variety of types of data: with visual and auditory targets, manual and saccadic responses, and other species apart from humans. In general, larger data sets produce smoother and more regular curves, as is only to be expected, with the linear relation extending well into the tails of the distribution.

Purely empirically, the fact that reciprocals of reaction times follow a normal distribution means that entire data sets can be described with just two parameters, $\mu$ and $\sigma$; and from a purely empirical point of view this is highly desirable. But it is natural to wonder what kind of neural mechanism would give rise to such behaviour. The simplest model that behaves in this way is an embodiment of the idea presented earlier, that reaction time is the culmination of a process that proceeds at a certain rate towards completion. If we have a signal $S$, with an initial value of $S_0$, and imagine that in response to a stimulus it starts to rise linearly at a rate $r$ until it reaches a threshold $S_T = S_0 + \theta$ at which point the response is initiated, then the reaction time will be given by $T = \theta/r$. If, on different trials, $r$ varies randomly, following a normal distribution with mean $\mu$ and variance $\sigma^2$), then $(1/T)$ will also be distributed normally, generating the familiar skewed distribution of $T$ itself (Fig. 4). It is this extremely simple model that is designated by LATER, which stands for Linear Approach to Threshold with Ergodic Rate, as well as reminding us of the procrastination that underlies the whole process.

Changes in the underlying parameters have characteristic effects on reciprobit plots of the resultant latencies, shown in Fig. 5. Altering $\theta$ causes the distribution to swivel around the intercept $k$, changes to $\mu$ cause a horizontal, self-parallel, shift, and altering $\sigma$ rotates the plot around its median. It is important to realize that though at first sight this model looks as though it has four free parameters ($\mu$, $\sigma$, $S_0$ and $S_T$), this is not in fact the case. It is only the difference between $S_0$ and $S_T$, $\theta$, that is effective, and because the scale of the vertical axis is arbitrary, it is only the relative sizes of $\theta$, $\mu$ and $\sigma$ that matter: if all three are doubled, for instance, the behaviour is completely unchanged. In general, altering $\theta$ is exactly equivalent to altering $\mu$ and $\sigma$ together. Thus there are only two independent parameters, and in general it is usual to take them as $\mu$ and $\sigma$.

Note that for simplicity this model does not take into account the fixed delays that must occur in any complete sensorimotor system, due to such things as transduction by receptors, synaptic delay, conduction velocity, muscle activation etc (Noorani and Carpenter, 2015) It is difficult to make an accurate estimate of what this might amount to altogether, but in modelling work it is usually taken as some 50–60 ms for eye movements driven by visual stimuli. As can be seen in Fig. 6, a fixed delay of this magnitude has only a very small effect on the linearity of reciprobit plots, though for accurate modelling, particularly of more complex tasks such as countermanding (discussed below) it is normally explicitly included; but for many purposes, for example characterization of behaviour in a clinical setting, this fixed delay can be disregarded. In that case it is usual to describe observed populations in terms of the parameters $\mu$' and $\sigma$' that describe the distribution of reciprocal RTs, so that $\mu$' is it self the reciprocal of the median reaction time. $\mu$' is always smaller than the 'true' underlying $\mu$ of the rise-to-threshold mechanism, but for the purely empirical purpose of providing quantitative parameters to encapsulate the behaviour, this has no practical importance.

We noted earlier that a feature of the LATER model is that it is quite economical, in that it has only two free parameters, $\mu$ and $\sigma$, yet under most conditions is still capable of modelling actual observed data with considerable accuracy. While in general terms

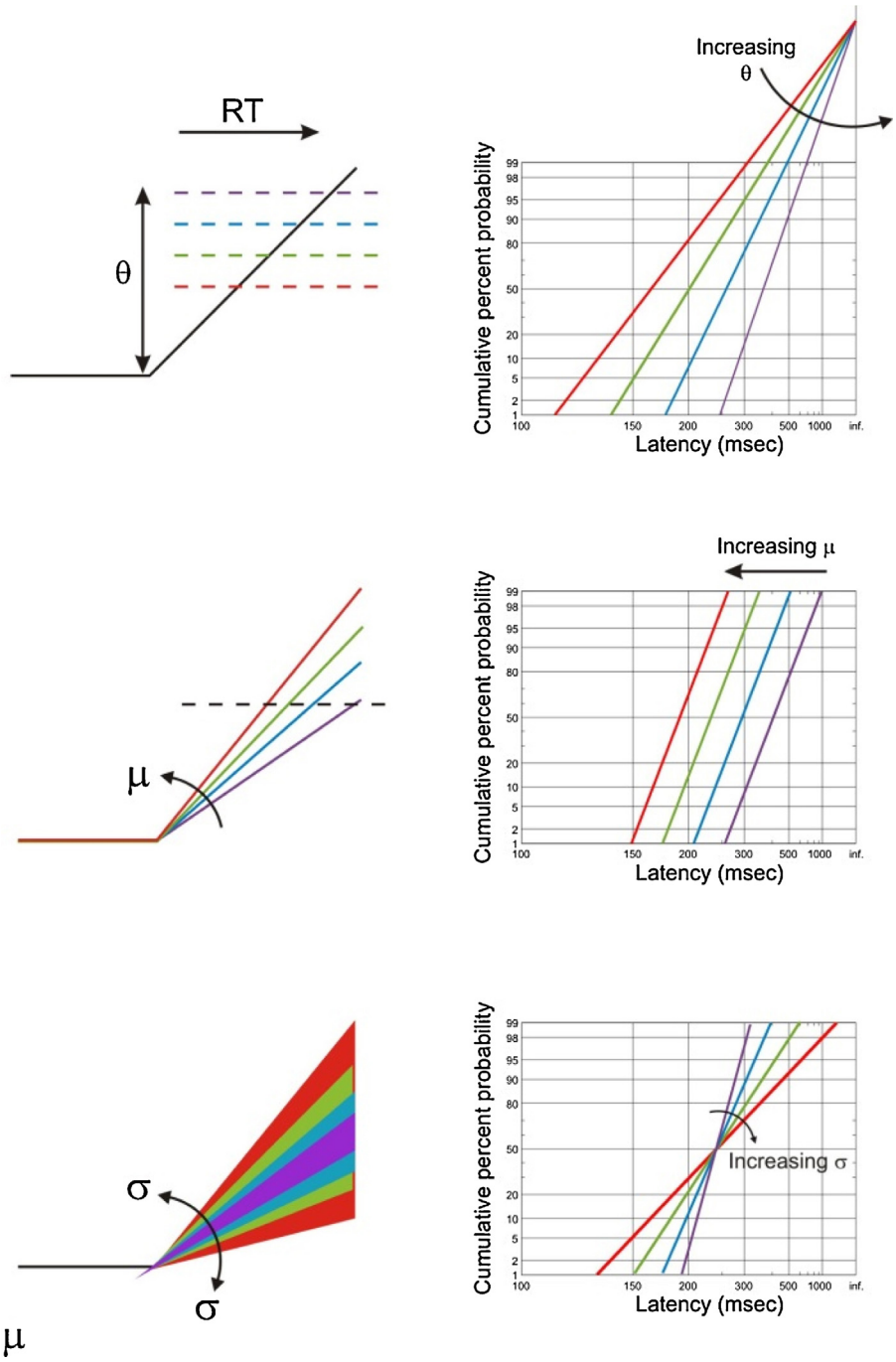



**Fig. 5.** Relation between parameters of the LATER model and parameters of the reciprobit plot. Top: variation in the threshold $\theta$ swivels the plot about the infinite intercept; middle: variation in the mean rate of rise $\mu$ leads to horizontal, self- parallel translation of the reciprobit plot; bottom: alterations in the variance $\sigma^2$ generate a change in the slope of the plot with no change in median latency.

economical models are scientifically attractive, it must always be true that even better fits will be obtained if we allow ourselves to add more parameters. Two particular examples of models that are based on LATER, but add an extra parameter, are the Linear Ballistic Accumulator or LBA model (Brown and Heathcote, 2008), and the Extended LATER model, or E-LATER model (Nakahara et al., 2006). In both cases the modification is in effect that the range $\theta$ is allowed to vary randomly from trial to trial, as well as the rate of rise. In tasks where either the urgency or prior probability or reward is not constant, but may vary over time, then in LATER itself this will be expected to alter either $S_0$ or $S_T$, and thus $\theta$; it is not entirely clear that in these circumstances it is helpful to consider this variation as simply random. An example is when the probability of the target appearing suddenly changes to a new value: the subsequent behaviour is well described – using the basic LATER model – in terms of a progressive systematic alteration in $S_0$\{Anderson, 2006 #6808\}.

### 3.1. Early responses

For some subjects, especially under conditions of high expectation or urgency, or when their attention is distracted by other demanding tasks, and much more often with saccades than other kinds of response, one tends to find more of the very earliest

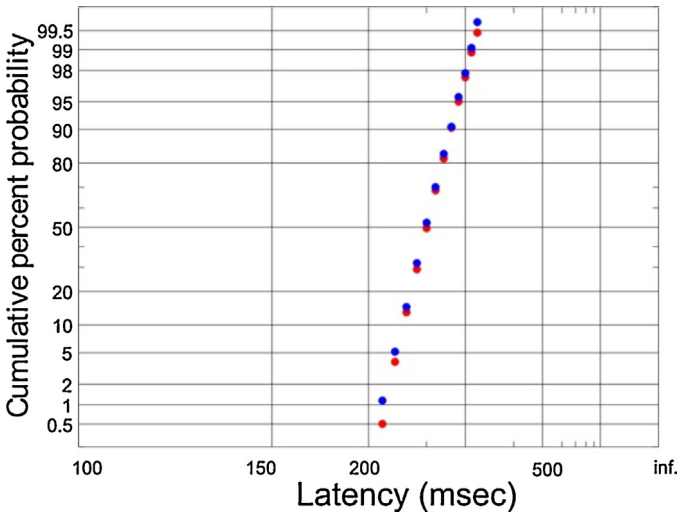



**Fig. 6.** Effect of a fixed delay. The blue data points show the distribution resulting from a 5000-trial simulation with $\mu = 4.0$, $\sigma = 0.33$; the red points show the same simulation with a fixed delay of 50 ms, $\mu = 5$ and $\sigma = 0.5$, having the same $\mu$' and $\sigma$'. It can be seen that the fixed delay has a barely discernible effect on the distribution.

responses than would be expected by LATER, so that instead of being straight, the distribution may be noticeably shallower at the left-hand end. Two examples are shown in Fig. 7; in data sets as large as these, it is usually found that this population of early responses lies along a different straight line, that extrapolates back to an intercept with $k = 0$.

This behaviour can be explained by imagining that in parallel to the ordinary LATER unit that is responsible for the main part of the distribution, there is another unit having $\mu = 0$, and a standard deviation, $\sigma_E$, that is larger than usual, thus generating a shallow slope on a reciprobit plot (Carpenter, 1994, 2012). If these two units race in parallel, with whichever is faster determining the time of the response, although most of the time the main unit will beat the early unit, just occasionally the parallel 'maverick' unit will happen to overtake it, generating the characteristic pattern of early responses (Fig. 8). There is some reason to think that the early unit represents primitive responses made by lower areas of the brain, perhaps in the colliculus, that are normally tonically inhibited by higher cortical regions, but escape this inhibition under conditions of distraction (Halliday and Carpenter, 2010), or increased expectation or urgency (Carpenter and Williams, 1995; Reddi and Carpenter, 2000): this is considered further below.

Although the early component is usually strikingly obvious when distributions are shown as reciprobit plots (because this stretches out the lower tail of the distribution), early responses normally form only a small proportion of the whole population (around 2% in the distributions shown in Fig. 7). A powerful reason for using reciprobits is that early responses are often virtually invisible on cumulative plots using a linear probability scale, even with a reciprocal time axis (Noorani and Carpenter, 2011a) (Fig. 9).

Another kind of short latency response that may be observed and also produces deviations from the straight line predicted by LATER is the express saccade, often confused with the early response. The essential difference is that although it too can be modelled by supposing a fast saccade generator in parallel with a main decision-making unit that is responsible for the majority of responses, in this case the fast unit has a larger value of $\mu$ than the main distribution, and a similar value for $\sigma$; it is therefore pre-emptive (when activated it is almost bound to reach threshold before the main unit), but is only activated probabilistically, on a small proportion of the trials. This generates a raw frequency histogram that is very different from that produced by early responses, in that it is bimodal, with a discrete fast peak corresponding to the larger value of $\mu$ (Fig. 10) (Fischer and Boch, 1983, 1984; Fischer and Ramsperger, 1984; Fischer et al., 1993). Neither for express nor early saccades are the supposed models very robust. Part of the problem is that both types of response are generally infrequent; with smaller datasets it is difficult to distinguish between different models on the basis of how well they fit the observations. In both cases, the neural mechanisms for their generation is equally unclear.: although there seems to be a consensus that the responses are due to lower-level, sub-cortical processes, perhaps in the superior colliculus, (Isa and Kobayashi, 2004) attempts to show that they are essentially collicular in origin have not been entirely compelling, whether behavioural in approach or neurophysiological (see for instance (Anderson and Carpenter, 2008; Edelman and Keller, 1996)).

## 4. LATER as an ideal Bayesian decision mechanism

So far, LATER has been presented as no more than an embodiment of the stochastic properties of the observations on which it is based. To be intellectually satisfying it needs to have some kind of functional basis: specifically, it ought to represent a plausible mechanism for making decisions. What would an ideal decision-maker look like?

It is generally accepted that the appropriate mathematical framework for making decisions on the basis of uncertain data is a Bayesian one. In general, it enables us to evaluate the probability $p(H)$ of some hypothetical mechanism H by observing the events E that it might have caused. This is sometimes called inverse probability because it is the opposite of the classic problems of elementary probability. Instead of having a 'set-up' H – for example tossing a coin – and estimating the probability of a consequent event E – like getting heads – we try to deduce something about H

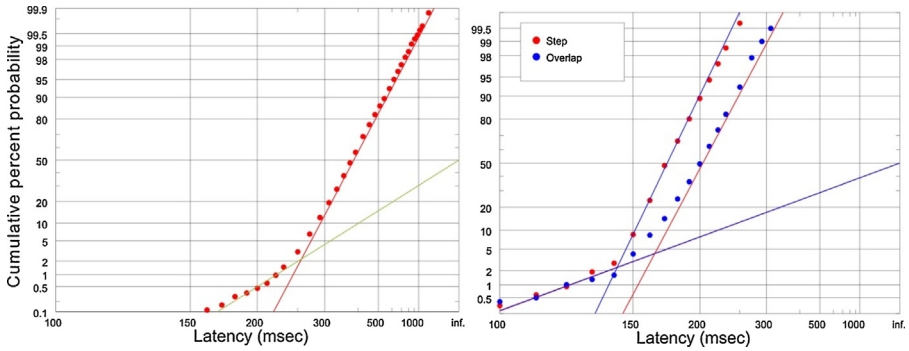



**Fig. 7.** Human reaction times with prominent early components. Left, manual auditory, 4436 trials (Green and Luce, 1971). Right, saccadic, in visual step (1875 trials) and overlap (998) tasks (Carpenter, 1994).

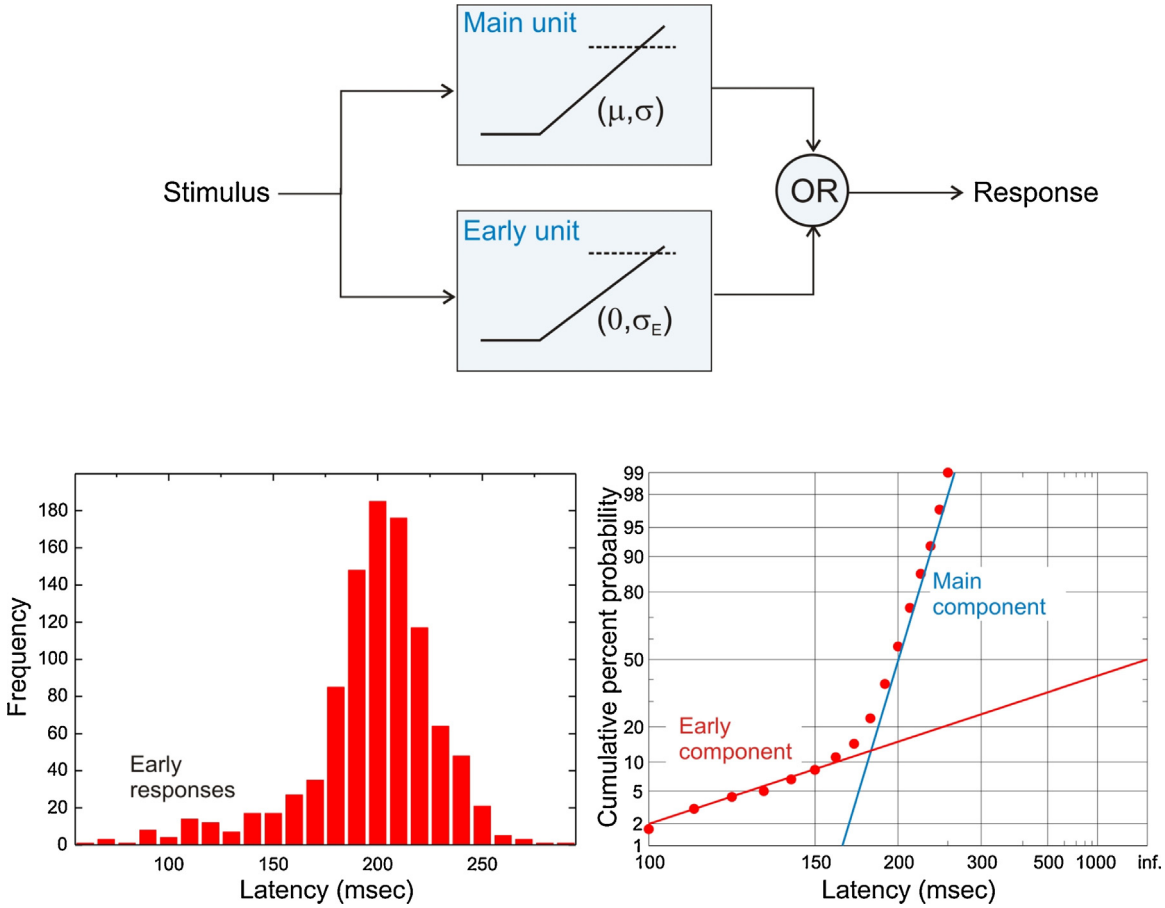



**Fig. 8.** Modelling the early responses. Above. a Main unit with parameters $\mu$ and $\sigma$ operates in parallel with a 'maverick' Early unit, whose parameters are $\mu = 0$ and $\sigma_E$. Whichever unit reaches threshold first triggers the response. Below, a distribution with a prominent early component, generated with this model. Left, frequency histogram; right, reciprobit plot: the lines correspond to the best-fit main (blue) and early (red) recinormal components. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

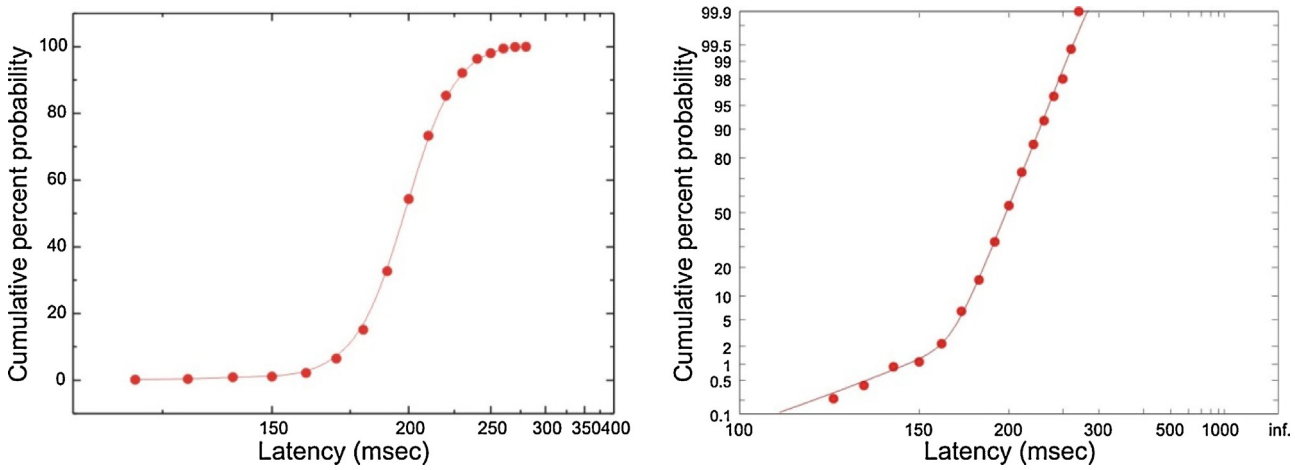



**Fig. 9.** A distribution with an early component which can barely be discerned when a linear cumulative scale is used (left), but whose characteristics are obvious on a reciprobit plot (right).

– for example, whether the coin is biased – by observations of E. In formal terms, we calculate not $p(E|H)$ (where '|' means 'given') but $p(H|E)$. This is the process of statistical inference that underpins the whole of empirical science. It also represents what is happening all the time in our sensory systems: for perception means judging the probabilities of hypotheses about the outside world on the basis of the sparse and noisy information – sensory events – that impinge on our sense organs. If it is a matter of using this information to make analogue adjustments, the effects can be immediate (Srimal et al., 2008). But for binary responses, only rarely, in a noisy and uncertain world, is observation of one event sufficient to make a robust decision concerning a particular hypothesis: more usually serial sampling must occur, with a gradual accumulation of evidence until it is sufficiently compelling.

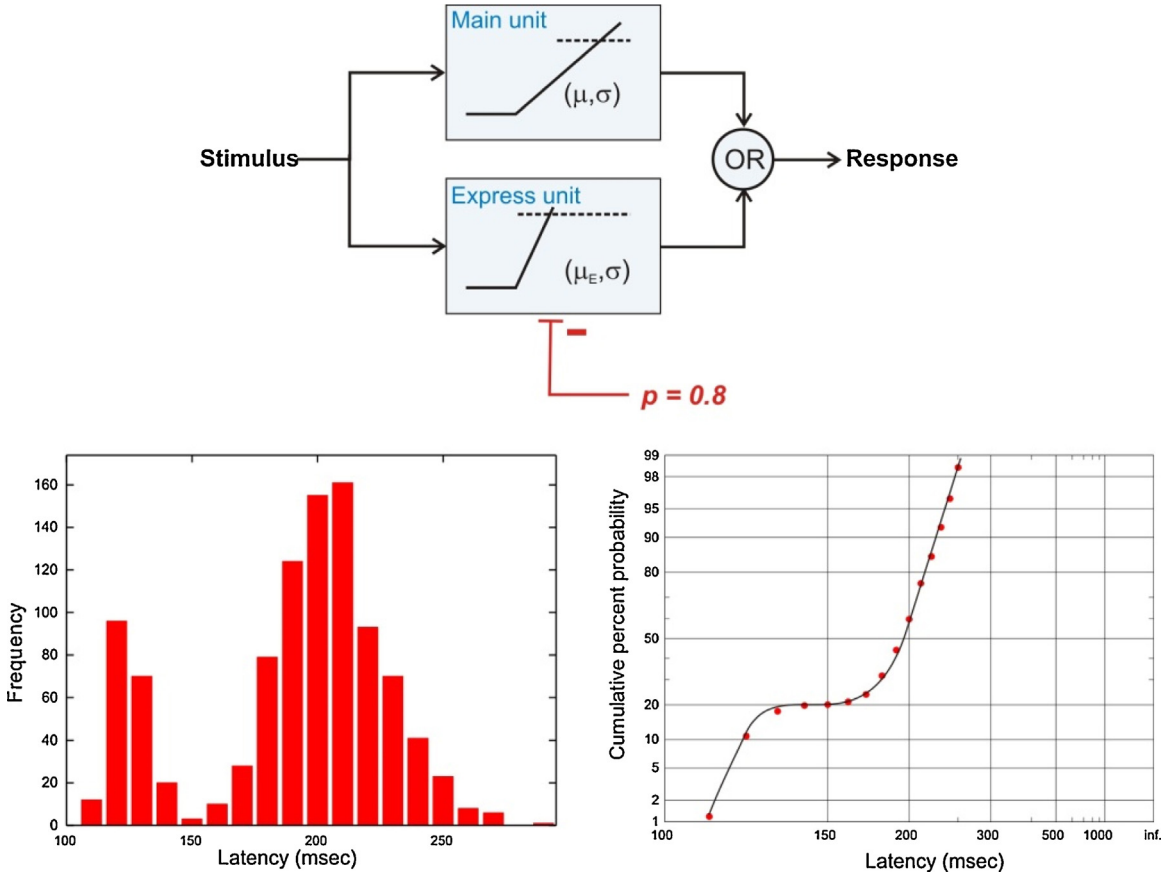



**Fig. 10.** The generation of express saccades. Above, an express unit with a large value of $\mu$ functions in parallel with a smaller $\mu$ that generates the main part of the distribution; but most of the time ($p = 0.8$) the express unit is inhibited and does not operate. Below, frequency (left) and reciprobit histograms of the resultant distribution, showing a clear peak of express responses around 120 ms.

There is a stylized situation that is often used as a paradigm of inverse probability. It takes the form of a game: you show me two identical urns, and tell me that one of them, A, contains 90 red balls and 10 black, whereas the other, B, has 10 red balls and 90 black. So we know that the chance of picking a red ball from A is 90%, but from B is only 10%. More precisely, if we represent the event of picking a red ball by E, then $p(E|A) = 0.9$ and $p(E|B) = 0.1$.

You now choose one of them at random by tossing a coin, and I have to guess whether it is A or B. They are equally likely, so in the absence of further information I would take $p(A)$ and $p(B)$ both as equal to 0.5; another way of expressing this is that the odds of the two hypotheses, $p(A)/p(B)$, is initially one. I'm now allowed to pick a ball at random from it and note its colour before replacing it: it turns out to be red. Intuitively it is natural to feel that the urn is more likely to be A, which has a higher proportion of red balls, but how can this be expressed more precisely? This is where Bayes' Law (Bayes, 1763) comes in.

If we have a hypothesis H and we have observed an event E, then Bayes' Law states that

$$p(E)p(H|E) = p(H)p(E|H) \quad (1)$$

Rearranging, this tells us how to use the fact that we have observed E to update our estimate of the probability from its old ('prior') value $p(H)$ to a new ('posterior') value, $p(H|E)$:

$$p(H|E) = p(H)\frac{p(E|H)}{p(E)} \quad (2)$$

$p(E|H)$ is the chance of observing E if H is true, and is called the likelihood, while $p(E)$ is the chance of observing E whether H is true or not. In practice we are nearly always comparing two or more hypotheses, so it is convenient to use Bayes' Law in odds form (this has the desirable consequence that $p(E)$ – in practice a difficult term to estimate – cancels out). So for two alternative hypotheses A and B we have:

$$\frac{p(A|E)}{p(B|E)} = \frac{p(A)}{p(B)} \times \frac{p(E|A)}{p(E|B)} \quad (3)$$

The term on the left is called the posterior odds, the next term is the prior odds, and the ratio of the conditional probabilities is called the likelihood ratio.

Returning to our game, the prior odds were 1, since $p(A)$ initially = $p(B)$; we already know $p(E|A)$ is 0.9 and $p(E|B)$ is 0.1, so the likelihood ratio is 9; therefore the posterior odds are $1 \times 9 = 9$: in other words it is now exactly nine times more likely that A was the urn chosen, rather than B.

If we sample again, and this second ball is also red, the likelihood ratio is again 9, so it is now 81 times more likely that the urn is A. Because the likelihood ratios multiply in this way as we make repeated observations, it is convenient to use Eq. (3) in logarithmic form, so that the multiplication becomes an addition (Edwards, 1972). Then:

$$\text{logposteriorodds} = \text{logpriorodds} + \text{loglikelihoodratio} \quad (4)$$

The log likelihood ratio or LLR represents the degree of support that the evidence has provided for A rather than B, and is additive. As we go on gathering more evidence, the total LLR increases each time, and the log odds in favour of the correct hypothesis will steadily get larger and larger, at a rate representing the amount of information gained each time.

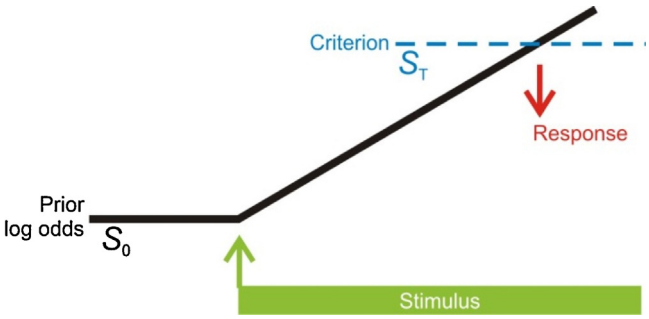



**Fig. 11.** LATER as a Bayesian decision-maker. A decision signal, representing log odds, starts at a level $S_0$ (the prior log odds); in response to the information provided by the stimulus, it starts to rise at a constant rate until it reaches the criterion level $S_T$, when it initiates a response.

It is not difficult to see how a system like this could be used to make decisions about the existence of stimuli in the outside world on the basis of the noisy sensory signals that they generate. What is needed is a decision signal $S$, representing the log odds: initially it has a value $S_0$, representing the log prior odds, for instance of there being a visual stimulus on the left as opposed to the right. When the stimulus actually appears, $S$ will start to rise steadily at a rate $r$ as the incoming sensory information provides evidence for one side rather than the other: eventually the log odds will reach a level ($S = S_T$) where the probability of the target being on the left is so overwhelming that it is justifiable to make a response (Fig. 11).

That figure ought to be familiar, because it is almost identical with what we saw to be the simplest model that would explain the observed variability of response latencies: in other words, LATER, which began life as an empirical way of describing reaction times, turns out also to be almost exactly what would also be expected of an ideal decision-maker. What we have not explained, of course, is the random variation of $r$ from trial to trial, but this explanation must wait until some other aspects of LATER, and decision-making in general, have been discussed.

Another point that needs to be addressed is how such a model would work if instead of merely two alternative hypotheses, as with the two urns, there are lots of them. Although in the laboratory we can set things up so there are just two visual targets, in the real world a typical visual scene consists of many rival targets we might want to look at. If there are $N$ of them, it would clearly be cumbersome to have to make comparisons of the likelihood ratios for each of the $N(N\text{-}1)$ possible pairs. An attractive solution is the idea of a race between LATER units representing the individual probabilities of each possible target, the unit that reaches threshold first determining the final response. Those that are more intrinsically likely, or are better supported by the evidence, will then be more likely to win. Each unit will still be Bayesian, but using the more basic form (Eq. (2)) rather than the odds version: taking logarithms, this equation becomes:

$$\log p(\mathrm{H}|\mathrm{E}) = \log p(\mathrm{H}) + \log p(\mathrm{E}|\mathrm{H}) - \log p(\mathrm{E}) \quad (5)$$

In other words, the new log probability is given by the prior log probability plus the log likelihood, minus a term embodying the probability of the event occurring at all. This final term is a nuisance, being difficult to calculate, but fortunately it is common to all the competing LATER units; so a decision made by racing between them will not be affected by it. Thus decisions to choose one rather than another of a number of possible options can be envisaged as a 'first past the post' race between competing rival LATER units, each associated with a particular response that is initiated when it wins.

### 4.1. Testing the Bayesian behaviour of LATER

To see whether this functional interpretation of the LATER model as a decision mechanism is justifiable we can do experiments in which we manipulate the three underlying Bayesian factors: prior probability $S_0$, the rate of information is supplied (which will affect $r$), and the criterion or threshold level $S_T$. There are two kinds of experimental variables that we can alter: circumstantial variables (such as expectation and the urgency with which a response needs to be made) that will affect $S_0$ and $S_T$ (and therefore $\theta$), and stimulus variables (such as luminance, contrast, noisiness) that will affect $r$, but not $S_0$ or $S_T$.

Expectation is relatively easy to manipulate. If for instance we arrange for the target to appear more often on the right than the left, we can set up any particular value for the prior probability we wish. LATER tells us exactly what to expect: if we increase $S_0$, reaction time will get shorter in proportion to the log probability, and the slopes of the reciprobit plots should become shallower, swivelling around a constant intercept $k$ (Fig. 12).

These predictions have been fully verified in human subjects (Carpenter and Williams, 1995), with the median reaction time being reduced by about 80 ms per log unit of prior probability (Fig. 13). It can be seen in the figure that the number of early responses is also increased as the expectation increases, and it is interesting that their distribution also appears to swivel around a fixed intercept.

We can manipulate the other main circumstantial variable, urgency, simply by telling subjects either to take their time and be careful not to make mistakes, or alternatively to respond as quickly as possible and not worry about errors. The intention is to change the criterion level $S_T$, which – as with $S_0$ – should result in changes of latency accompanied by swivelling of the reciprobit plot about a fixed intercept (Fig. 5). Quite a good task for seeing whether this prediction is fulfilled uses a stimulus called a random dot kinetogram (RDK) (Britten et al., 1993). It is a field of continually-moving random dots, a certain proportion moving steadily in one direction, and the remainder moving randomly. The subject's job is to make a saccade to a fixed target to indicate the perceived direction of the steady component of the movement. When told to respond as quickly as possible, the reciprobit plot is found to swivel as predicted around a fixed intercept, reducing the latency; but at the same time (as with increased expectation) the number of early responses also increases (Fig. 14; (Reddi and Carpenter, 2000)). Unfortunately we cannot set up in advance a known value of the parameter, as we can with expectation; but on the other hand determination of the slope provides, conversely, a probability scale by which we can assign numerical values to particular degrees of arousal or urgency.

We can also use the same RDK task to test what effect varying the rate of supply of information has on the distributions of reaction time, by changing the proportion of dots that are moving steadily in one direction rather than at random. In Bayesian terms, increasing the number of dots moving coherently should increase the information available to the subject, which should in turn increase $r$,

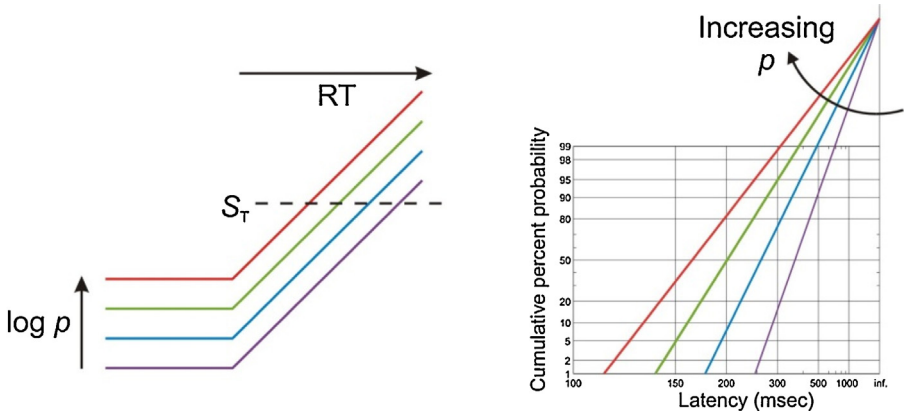



**Fig. 12.** Increasing prior probability in LATER reduces latency. Left, as $S_0$ (representing prior log odds) is increased, reaction time (RT) is reduced in proportion. Right, the result will be a reduction in the slope of the reciprobit plot, which will swivel around the intercept.

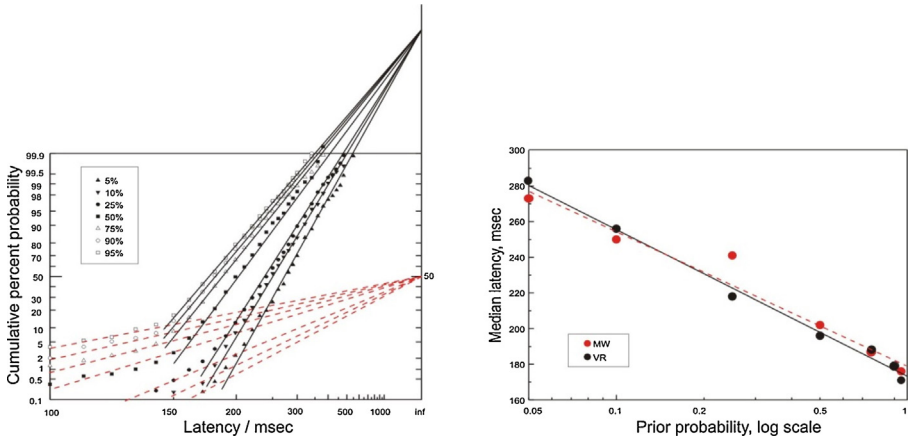



**Fig. 13.** Effect of expectation on human saccadic latency distributions. Left, reciprobit plot of saccadic latencies for one subject in trials with different prior probabilities, as shown: as expected, the distributions swivel around a constant intercept (as do the early responses: red asymptotes). Right, linear relation between median latency and log probability in this task, for two subjects. Redrawn from (Carpenter and Williams, 1995).

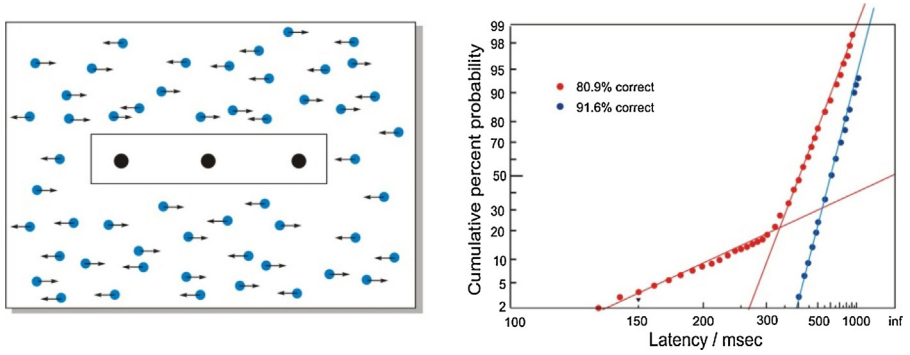



**Fig. 14.** The random dot kinetogram, and the effect of urgency instructions on reaction time in the task. Left, the subject is required to make a saccade from the central fixation target to one of the lateral targets to indicate the perceived overall direction of movement of the random dots (blue). Right: In this task, instructions to respond as quickly as possible (red data points) result in more errors, and (as predicted) swivelling of the reciprobit plot compared to when they are told to be more careful (blue data points); there is also an increase in early responses; these plots combine correct and error responses (Reddi and Carpenter, 2000). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the rate of rise of the decision signal, which will result not in swivel of the reciprobit plot, but a self-parallel displacement to the left (Fig. 5). As Fig. 15 shows, this is indeed what is actually observed: as the rate of information supply is increased the median reaction time steadily falls, with the reciprobit plots having constant slopes rather than swivelling (Reddi et al., 2003).

The conclusion of all these experiments is that the LATER model, originally conceived as the simplest possible empirical description of the stochastic behaviour of reaction times, also seems to represent something like the simplest possible system capable of making Bayesian-based decisions. But can LATER deal with the variety of responses from the more complex kinds of tasks that are found in the real world?

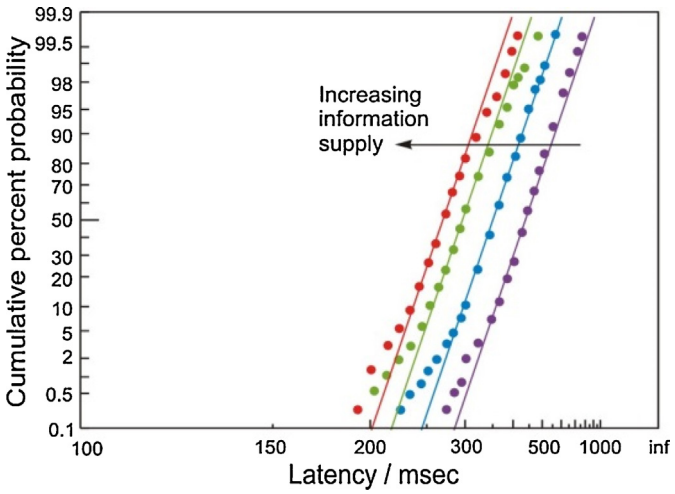



**Fig. 15.** Reciprobit plots of reaction times for a subject responding to the direction of overall motion in RDKs. Altering information supply results in sideways shifts of the reciprobit plots, but with constant slope (Reddi et al., 2003).

## 5. Errors

When required to make decisions based on probabilistic information, subjects inevitably make errors. There are two main kinds of error: errors of omission, when no response is made at all, and errors of commission, when a response is made that is incorrect.

Errors of omission are an inescapable feature of LATER: if the rate of rise $r$ follows a normal distribution, with mean $\mu$ and variance $\sigma^2$, then it must be true that there will be some occasions on which $r \leq 0$, so that $S$ never reaches $S_T$. This corresponds to the existence of the intercept at $k$ (Fig. 2), which represents the probability of this happening. When stimuli are high-contrast and not completely unexpected (as in the examples of reciprobit plots shown in Fig. 3) this probability is so small as to be negligible: for A in Fig. 3, for example, $k$ represents some 10 standard deviations from the mean, corresponding to a probability of about $10^{-24}$ ! But if $\mu$ is more comparable to $\sigma$, the resultant probability of not responding is large enough to be measured—and of course corresponds to what would be predicted from classic detection theory based on normal distributions (Green and Swets, 1966; MacMillan and Creelman, 1991) (Fig. 16).

Errors of commission can occur in two ways: as false positives, and as incorrect choices in discrimination tasks with more than one stimulus. The former are again expected from classic detection theory: specifically, even if there is no signal, so that $\mu = 0$, on half the trials the rising decision signal must eventually cross $S_T$ and initiate a response. This will generate a reciprobit plot for which the intercept $k$ is zero (Fig. 17), as in fact we have already seen in the case of the 'maverick' unit that is postulated to account for early responses (Fig. 8). Errors also arise whenever a subject is required to make a discrimination, choosing for instance one of two targets that differ in respect of some stimulus dimension such as size, colour or contrast; if they are sufficiently similar, mistakes are bound to be made. This can be modelled by two competing LATER units with different values of $\mu$; because of the random component of the rate of rise, sometimes the unit with the lower mean rate of rise $\mu$ will nevertheless reach threshold before the other, and initiate an incorrect response. In such cases it is helpful to use a modified version of the reciprobit plot (an incomplete plot) in which cumulative probability functions are plotted as a function of the total number of responses, including both correct and incorrect, rather than simply of those responses of the same category; an example for two competing units is shown in Fig. 17. It is important to note that in more complex tasks like this, plotting full reaction time distributions is essential for modelling the data without missing key features of the underlying decision processes: see (Noorani and Carpenter, 2011a) for further discussion.

At first the unit with the larger $\mu$ rises linearly, but as time passes the other unit begins to rise as well, causing the curves to flatten off until they reach the final asymptotes corresponding to the overall percentage responses. It can be shown (see the Appendix A) that the final proportion of 'wins' by the unit with greater $\mu$ is given by:

$$p = \frac{1}{2}(1 + \mathrm{erf}\frac{\Delta\mu}{2\sigma}). \quad (6)$$

Fig. 17 (right) shows an example of error responses of this kind in a task where the subject was required make a saccade to the larger of two targets, where the difference in size was in one case 6% and in the other, 2%. As would be expected, the easier discrimination results in a larger value of $\Delta\mu$ and thus a higher proportion of correct responses, and a larger separation of the pair of curves.

A related experiment, that happens to provide a great deal of information about competition of this kind, is the precedence task: here the rival targets, instead of being presented simultaneously when the central fixation target is extinguished, at time zero, are staggered, with one of them, chosen at random, appearing after a short delay $D$ (Leach and Carpenter, 2001). The instructions to the subject are simply to look at whichever of the two targets attracts their attention (if they are told to look at the one that comes on first, the experiment does not work, as they tend to agonize consciously about which it was). When $D$ is zero, subjects tend to choose both with equal probability; but as it is made to increase, it becomes more and more likely that they will select the one that comes on first (Fig. 18).

Two features of the behaviour in this task are informative. First, that even when $D$ is zero, so that the targets appear simultaneously, the latencies are longer than if one is presented alone (Fig. 19, left). This is surprising: for if what was happening was a simple race between two units, one would expect the average latency of the winner to be shorter, not longer. The simplest way of explaining this behaviour is that there is competitive lateral inhibition between the decision units: lateral inhibition is a common phenomenon in the nervous system, serving to enhance contrast and to sharpen up local differences in activity generally, and has often been demonstrated in the oculomotor system (Findlay, 1997; Findlay and Gilchrist, 2003; Hanes and Carpenter, 1999; Schall and Hanes, 1993; Schall et al., 1995; Wilson, 1999). In this case, the rightward shift of the reciprobit curves when two targets appear at once rather than one, with a free choice between them, can be modelled very well by

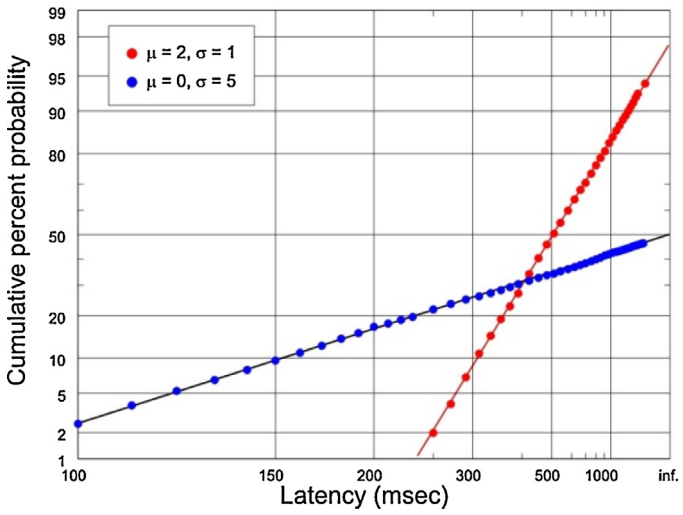



**Fig. 16.** False negatives and false positives. The red reciprobit plot is for $\mu = 2$, $\sigma = 1$; the intercept corresponds to a probability of 2.3% of not responding (simulation of 5000 trials); the blue one is for $\mu = 0$, $\sigma = 5$, with an intercept of $k = 0$ that corresponds to a 50% false positive rate.

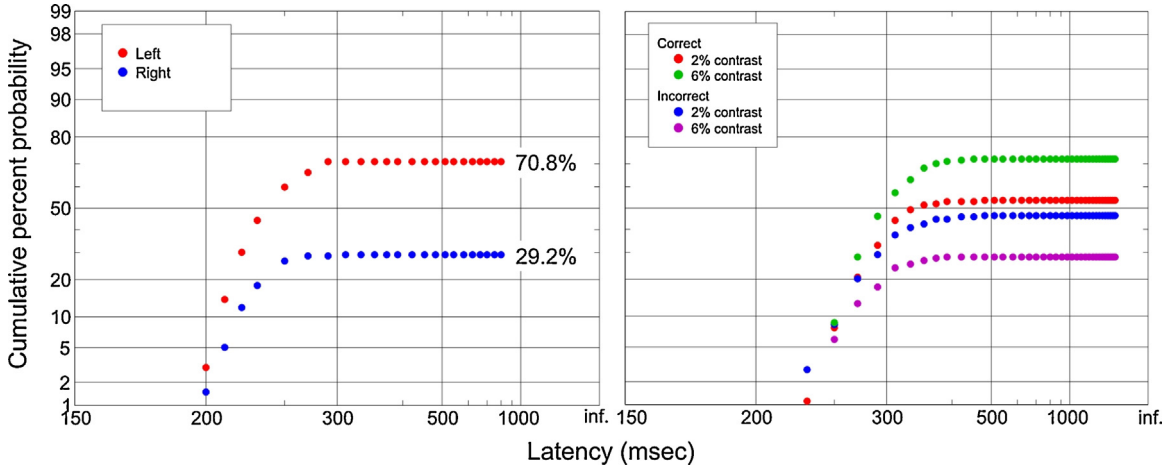



**Fig. 17.** Left, incomplete reciprobit plots of two simulated competing units, one (red) winning 70.8% of the time, and the other (blue) 29.2%. Right, distributions in a task in which the subject had to choose the larger of two targets, the difference in size being either 6% or 2%, showing incomplete distributions in each case for correct and incorrect responses, with the asymptotes dependent on the difficulty of the discrimination (RHSC unpublished data). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

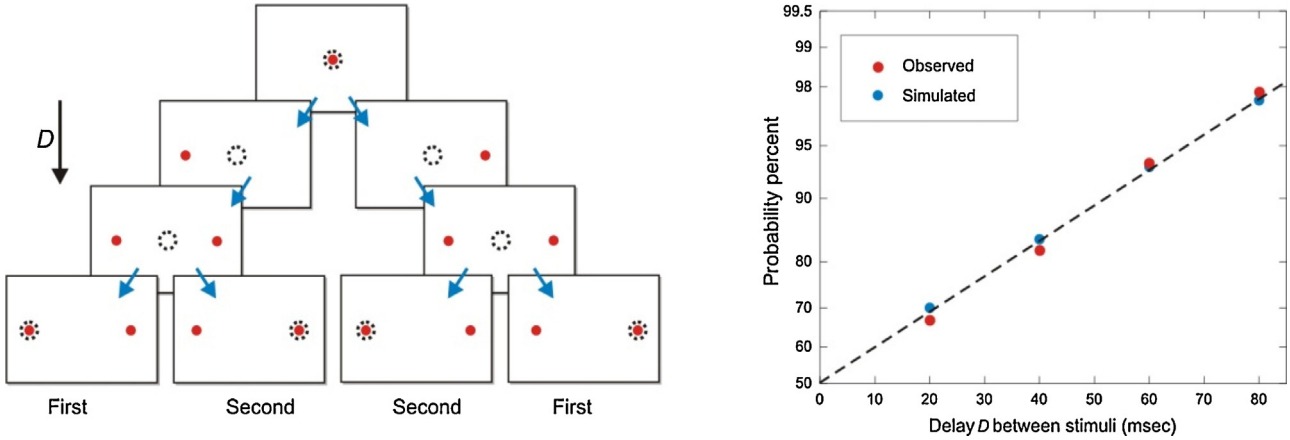



**Fig. 18.** The precedence task. Left, the protocol: after a fore-period, one target appears, randomly on right or left; then after a delay $D$ the mirror-image target appears as well (dotted circles indicate gaze position). Right, probability of making a saccade to the first target (probit scale) as a function of $D$; observations for one subject (red), and results from a simulation (blue), using a value of 38% for lateral inhibition. (Leach and Carpenter, 2001). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

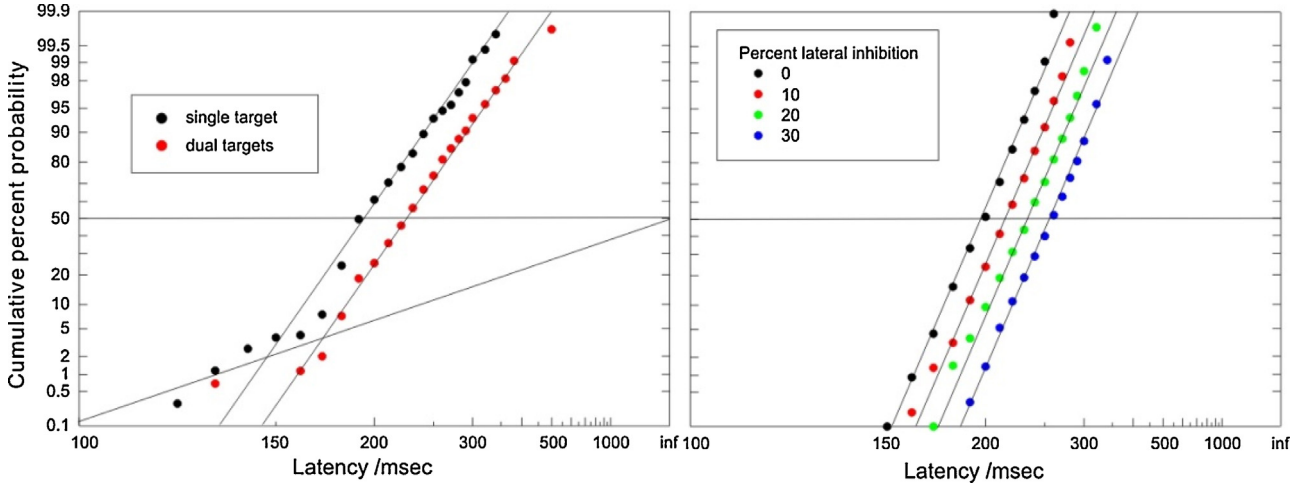



**Fig. 19.** Left, observed distribution for one subject in the precedence task (red), compared with control trials where just one target is presented, showing rightward shift. Right, simulated responses, showing how different degrees of lateral inhibition generate progressive rightwards shifts of the distribution. (Leach and Carpenter, 2001). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

adding a mechanism of lateral inhibition by which a fraction of the output of one decision signal is simply subtracted from the other; this has the effect of shifting the distributions to the right (Fig. 18). Second, the fact that when one target comes on before the other, nevertheless occasionally it is the second target that wins, implies something of great theoretical importance about the mechanism generating the underlying random variation in the rate of rise: it must be independently generated in the two units. If this were not so, and the variability was due, for instance, to something like relatively slow fluctuations of attention, then the occasions when the rate of rise happened to be low for the first unit would also be those where it was low for the second, so that the first unit would always win, which is not what is seen. This has obvious implications when considering the question of the origin of the randomness of reaction time.

Another way that lateral inhibition can arise is through interactions between the initial fixation target and the target that is eventually the destination for the saccade. So far we have been looking almost exclusively at step tasks, in which the initial fixation target is extinguished at the same moment that the peripheral target appears. In an appearance task, we do not turn the fixation target off at all: it remains throughout the trial. Alternatively, in an overlap task, it is on for a certain period after the appearance of the peripheral target, but is then extinguished. In both cases, the effect is to prolong the reaction times (Saslow, 1967). This can be seen very clearly in Fig. 7 (right), where the distribution is shifted to the right in a roughly parallel fashion by some 30 ms, as would be expected if the prolonged existence of the fixation target were causing lateral inhibition of the peripheral LATER unit: the similarity with Fig. 19 is obvious. However, even when in the step task the fixation target goes off at the same time as appearance of the peripheral target, it appears still to be exerting some kind of inhibitory effect, since if it is turned off before the final target appears (a gap task), latencies are reduced. The distributions are not simply shifted to the left, but show an element of swivel as well, suggesting that this reduction in latency is not simply due to reduced lateral inhibition but to something else as well, namely that extinction of the target shortly before the target appears will of course provide a predictive signal that is likely to lead to a higher value of the starting level $S_0$ of the decision signal. A model incorporating both these factors generates good predictions of the latency distributions that are observed over the whole range of gap, step and overlap tasks Fig. 20 (Story and Carpenter, 2009).

## 6. More complex tasks, involving stopping

Another type of complex behaviour that can be modelled successfully by small assemblies of LATER units is when unexpected stimuli cause previously-planned actions to be aborted and perhaps replaced with different actions. This simplest example is countermanding: here a subject performs a conventional step task in control trials, but in randomly interleaved experimental trials an extra stimulus (for example, reappearance of the fixation target) is presented a short time $D$ after the peripheral target. The subject has previously been instructed that when this happens they must cancel the saccade they would otherwise have made (Hanes and Carpenter, 1999; Hanes and Schall, 1995; Logan and Cowan, 1984; Logan et al., 1984). Successful cancellation is then found to be probabilistic: if $D$ is large, the chance of cancelling the saccade is small, and vice-versa. The behaviour in this task can be modelled by supposing that in addition to the LATER unit responsible for making a saccade to the peripheral target (the Go unit), there is another LATER unit, called a Stop unit, that is activated by the stop signal: when it reaches its threshold it disables the Go unit (Fig. 21). Successful cancellation is thus random because of the randomness in the rate of rise of the two LATER units. The overall behaviour is relatively easy to model in this way: the controls provide estimates of $\mu$ and $\sigma$ for the Go unit, and the interleaved countermanding trials enable one to estimate $\mu_{stop}$ and $\sigma_{stop}$, though with less precision. In general the rate of rise of Stop is found to be significantly higher than for Go.

A related task is Go/No-go, where a subject must make saccade to one class of target but not another (for instance, to blue targets but not to green). In this situation there is an analogy with the stop signal in countermanding, insofar as discriminative information (such as colour) takes longer to identify than mere existence of a target: as a result, it is found that the very earliest saccades have a virtually identical distribution whichever target it is. It is only later, when colour information arrives, that the two curves diverge: the No-go one levels off, whereas the Go distribution continues after a short hesitation (Fig. 22). This suggest quite strongly that the arrival of the colour information activates a stop unit that cancels any previously-initiated accumulation, at the same starting a LATER unit representing a response validly driven by the colour; a model of this kind is able to simulate the details of the (quite complex) latency distributions accurately (Noorani et al., 2011).

A drawback of both countermanding and Go/No-go is that a substantial proportion of the trials generate no response at all and

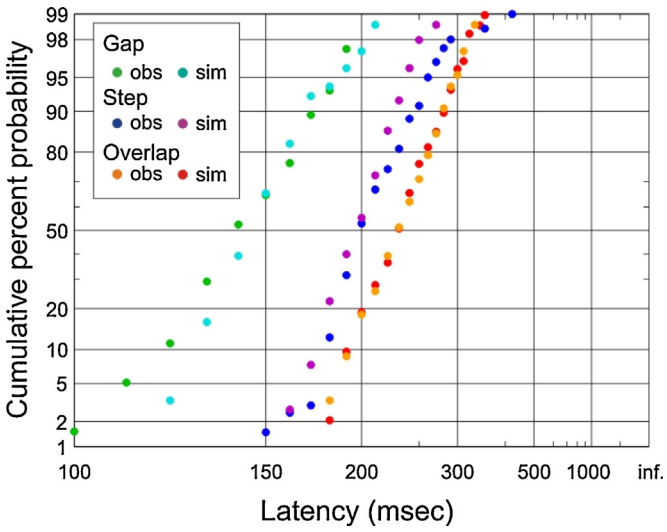



**Fig. 20.** Saccadic reaction times for a subject performing gap, step and overlap tasks, together with the results of simulation using a model incorporating lateral inhibition, and a predictive effect of offset of the fixation target. (Story and Carpenter, 2009).

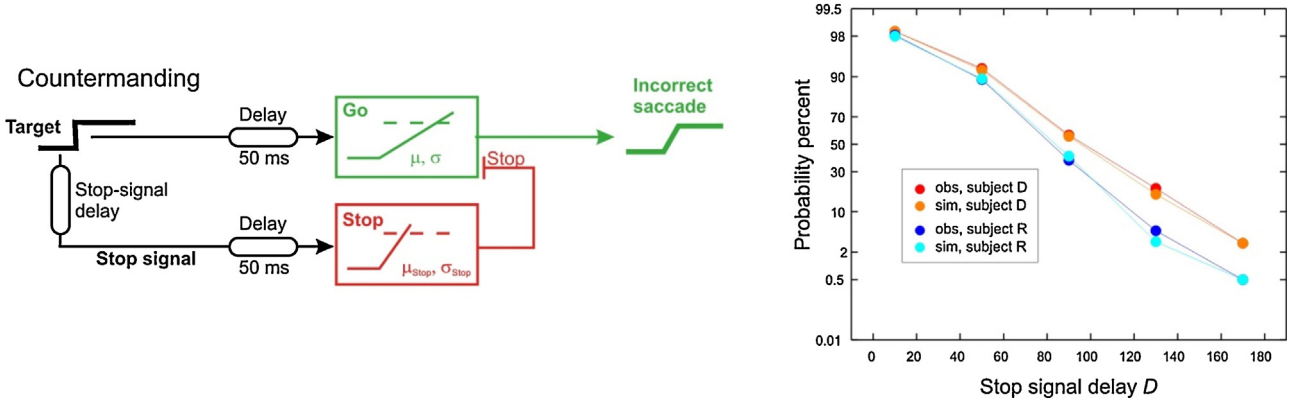



**Fig. 21.** Countermanding. Left, model of the countermanding process. A Stop unit, activated by the stop signal, disables the Go unit that would otherwise initiate the saccade, but may be too late. (Note that this model includes explicit constant delays to allow for such processes as sensory transduction, synaptic delay, conduction velocity, muscle activation etc). Right, probability of successful countermanding as a function of the stop-signal delay, *D*: observed and simulated functions for two subjects (Hanes and Carpenter, 1999).

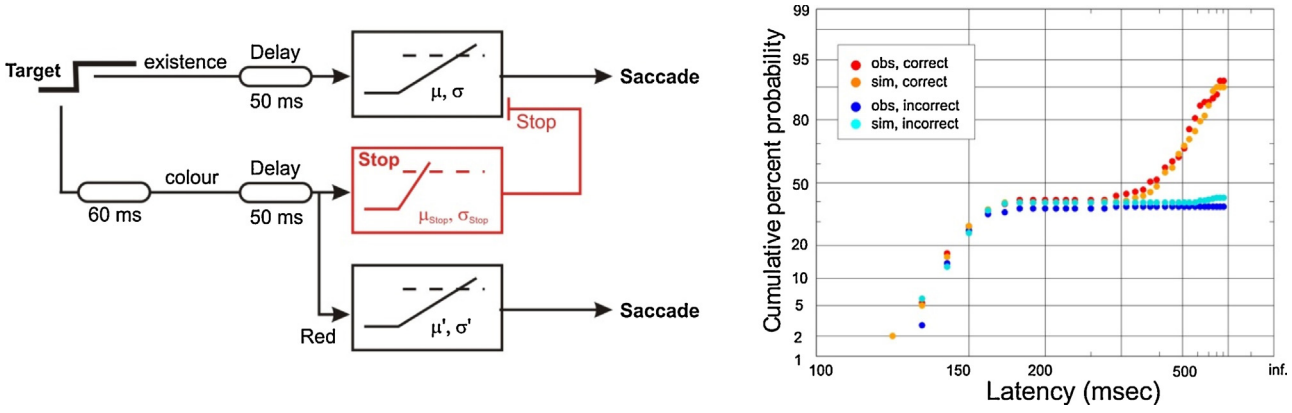



**Fig. 22.** Go/No-go task. Left, model of the process, in which colour information is postulated to arrive 60 ms after information about the existence of a target. Right, observed and simulated distributions for one subject in this task (red versus blue), showing early correspondence of correct and incorrect responses, followed by divergence. (Noorani et al., 2011). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

therefore do not contribute to the reaction-time distributions. The Wheeless task (Wheeless et al., 1966) is much better from this point of view. Here, in experimental trials, randomly interleaved with conventional step trials, after a short delay *D* the target jumps to its mirror-image position. Once again, the subject's behaviour is stochastic: sometimes a saccade is made to the first target (a Type A response), and sometimes the eye jumps straight to the final position (a type B response). If a type A response is made, then

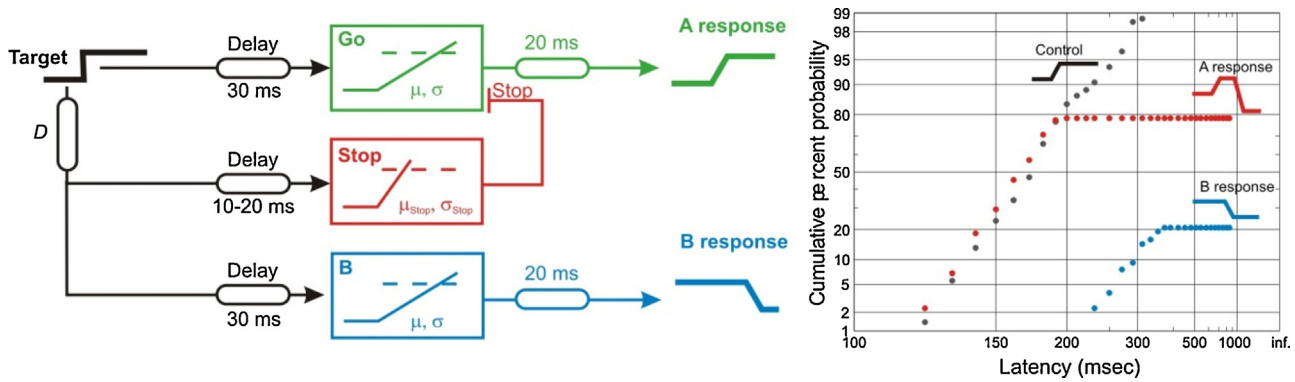



**Fig. 23.** The Wheeless task. Left, in the model, after a delay *D* the Stop unit is activated, which cancels the original A response if it has not already occurred; at the same time the B unit is activated. (For simplicity some other features of the model, including lateral inhibition and the mechanism that initiates the C responses, are not shown). Right, examples of A and B responses (for *D* = 100 ms), and of the controls distribution, in one subject, showing how the A responses are curtailed at about the same time that the B responses start to accumulate. Data from (Noorani and Carpenter, 2015).

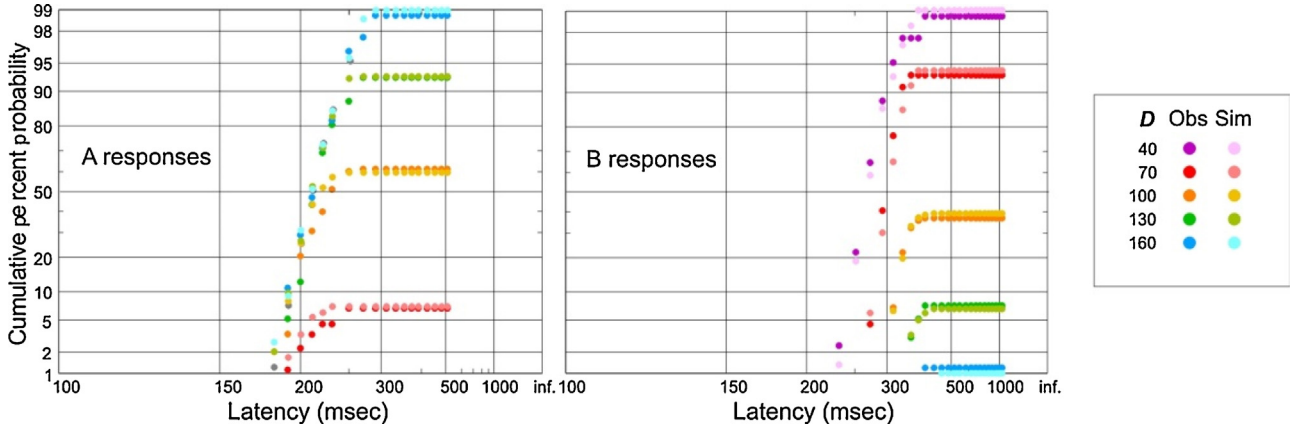



**Fig. 24.** Full distributions in the Wheeless task. Distributions of A and B responses for a full range of values of the delay *D*, in a single subject. As *D* increases, the proportion of A responses increases but of B responses decreases. Data from (Noorani and Carpenter, 2015).

it is eventually followed by a second saccade to the final target (a C response). As might be expected, the larger *D* is, the greater the probability of making a Type A rather than a Type B response. We can model the behaviour using an arrangement quite similar to what is needed for the Go/No-go task, resulting in quite accurate predictions of the distributions for all values of *D* (Noorani and Carpenter, 2015)(Figs. 23 and 24). Every trial generates some RT data, and a further advantage is that no explicit and arbitrary instructions are needed: as in a step task, the subject is simply told to follow the target. Earlier, Camalier et al. (2007) proposed a model of the Wheeless task and related two-step paradigms with a similar architecture to ours. However, whereas their model assumed that the decision units begin immediately on stimulus presentation, an essential feature of our model is that the Stop unit delay is 10–20 ms shorter than for the Go units, allowing it to predict full reaction time distributions for individual subjects in a variety of task conditions; it is also more consistent with recent neural recording data suggesting that onset time of decision activity critically influences latency (Pouget et al., 2011; Stanford et al., 2010).

Finally, a rather strange task that has achieved an extraordinary popularity over the last decade or two, especially amongst clinicians: the anti-saccade. Here the subject is presented with a jumping stimulus, exactly as in the conventional step task, but is instructed not to follow the target but rather to make a saccade in the opposite direction, of equal amplitude, either to a pre-existing static marker, or to a blank field (Antoniades et al., 2013; Evdokimidis et al., 2006; Hallett, 1978; Hallett and Adams, 1980). In practice, as with these other stopping tasks, it is found that the behaviour is stochastic: on a proportion of trials the subject cannot help making a saccade to the target rather than in the opposite direction, a pro-saccade. What this has in common with the tasks previously described is the need for a stop mechanism to prevent this happening. A suitable model thus consists, once again, of a pair of Go units, corresponding to the pro-saccade and anti-saccade, and a Stop unit. The anti-saccade unit is activated after a delay compared with the pro-saccade unit, corresponding to the extra time needed to effect the transformation from the actual target position to the mirror-image goal (Fig. 25). The model is able to predict the incidence and distributions of all the responses – correct anti-saccades and error pro-saccades – remarkably few free parameters for each individual, since $\mu$ and $\sigma$ for both Go units are taken from control trials using pro-saccades, $\sigma_{stop}$ for the Stop unit appears to be essentially constant across subjects, and only $\mu_{stop}$ needs to vary from one subject to another. It also predicts the distributions of the final corrective anti-saccades that normally follow error anti-saccades, by making the same assumption as for the C-responses in the Wheeless task, that the final corrective movement is triggered by the completion of the erroneous one (Noorani, 2014; Noorani and Carpenter, 2013).

In conclusion, it should be pointed out that in all these different varieties of tasks that involve stopping, estimates of the stop parameters are generally less certain than those for the Go units, which can be found from control step trials. This is because direct measures of cancellation times have been lacking, although recently air-puff triggered blinks and timing of antagonistic neck muscle recruitment that inhibit head movements in stop trials have proved

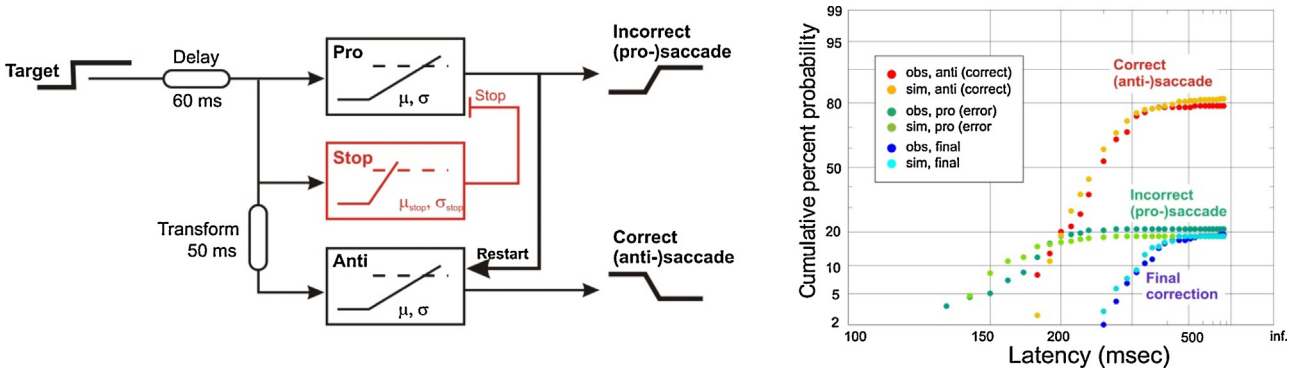



**Fig. 25.** Anti-saccades. Left, a model with two identical LATER units (Pro and Anti), the latter activated after a delay; a Stop unit cancels the erroneous pro-saccade: if it nevertheless occurs, on completion it restarts the Anti unit, generating the final corrective saccade. Right, observed and simulated distributions for one subject, showing pro-saccades, anti-saccades, and the final corrections when a pro-saccade has been made. (Noorani, 2014).

useful in this regard (Corneil et al., 2013; Goonetilleke et al., 2012; Walton and Gandhi, 2006). Moreover, although the default assumption has generally been that the fixed delay for the Stop unit is the same as for the other units, this may not necessarily be so: a shorter or longer delay can be offset by a smaller or larger value of $\mu_{stop}$, and for the Wheeless task in particular, there is some evidence that this delay may in fact be shorter, as in fact is shown in Fig. 23 (Carpenter, 2015).

## 7. Spontaneous behaviour

All the oculomotor tasks that have been discussed so far generate evoked saccades, made in response to one or more targets presented at specific times in a series of separate trials of short duration. A different kind of task, arguably more like the situation faced in real life, is when a subject is presented with a static visual scene, and makes a series of spontaneous saccades whose function is to obtain information by sequential scanning. Although these two kinds of task – evoked and spontaneous – are at first sight very different, it is clear that they have a number of features in common. At the end of each saccade in the sequence, a fresh retinal image is presented to the retina, so the next saccade could be thought of as a response to this suddenly-presented visual stimulus. So it would be plausible to conceive of the entire sequence of saccades as a series of isolated 'trials' comparable to what occurs in a typical evoked task in the laboratory. Of course, it could be that the spontaneous saccades are not in fact determined by each new visual stimulus at all, but the result of a larger-scale pattern of scanning driven by some kind of internal program that is essential independent of visual feedback. In reading, a highly specialized version of visual scanning, there is reason to think that the pattern of eye movements is made relatively independently of the visual stimulus of the words on the page, either in terms of their low-level visual features or of the their ultimate meaning, though both these factors can exert an influence on where the saccades are directed in particular circumstances (Carpenter and Kinsler, 1995; Just and Carpenter, 1980; McConkie et al., 1988; Rayner and Pollatsek, 2012).

Nevertheless, if we treat intersaccadic intervals as latencies (from the end of one saccade to initiation of the next), when plotted as reciprobits they reveal the same basic features found with evoked saccades; this is true for reading, for scanning static scenes, and also by the most basic type of spontaneous saccade of all, the 'quick phases' of nystagmus (Carpenter, 1994; Carpenter and McDonald, 2006; McDonald et al., 2005; Roos et al., 2008) (Fig. 26).

In all these cases, there is a main part of the distribution, usually with a longer latency than is typically seen with evoked saccades, and invariably a large early component. Both these features can be explained in terms of aspects of what is known of evoked saccades. The visual scene presented to the retina will typically have a very large number of possible targets competing to be the goal of the next saccade: as we have seen, this is likely to evoke lateral inhibition which will reduce the rates of rise of all the decision signals. It is also highly predictable; obviously so in time, since the moment of presentation is determined by the oculomotor system itself, but also in space since most of the image has already been seen, and its displacement is again known in advance. We saw earlier that one of the consequences of increasing expectation is to increase substantially the proportion of early responses, to an extent that is more than adequate to explain their prevalence in spontaneous viewing. Fig. 27 shows the result of an experiment in which a conventional step task (with random fore-period and unknown step direction) was followed by a return of the target to the original fixation point, after a constant delay, so that both the timing and target location were known in advance: as a result, there is a very large increase in the number of early saccades (Roos et al., 2008).

## 8. Detection versus decision, and the linear rise

An early, and very influential, idea about reaction times was that they were the result of something equivalent to a sequential probability ratio test, in which information is accumulated over time until a decision variable reaches a threshold criterion, in effect a statistical significance level: it is equivalent to accumulating log likelihood. It is most obviously applicable to situations in which a signal must be detected in the presence of accompanying noise. The attraction of this approach was that there is no more efficient way of using the information (Green and Swets, 1966; Wald, 1947; Wald and Wolfowitz, 1948), and also that it is in accord with what would be expected from information theory (and is observed), that the time to make such a discrimination should depend on the quantity of information required to perform the task (Gregory, 1956; Hick, 1952; Hyman, 1953; Welford, 1959), and thus on such factors as the signal-to-noise ratio. Stone (Stone, 1960) seems to have been the first to realize this, and the underlying idea has subsequently been adopted by many others (Grice, 1968; La Berge, 1962; Laming, 1968, 1973; Pacut, 1977, 1980; Ratcliff, 1978; Smith, 1980; Vickers, 1979; Watson, 1979), and is particularly attractive when modelling tasks such as the random dot kinetogram where there is clearly a great deal of noise in the stimulus.

There are two major respects in which this kind of model differs from LATER. First, that in order to function correctly, the successive samples in such a process must be statistically independent, which means that the rise of the decision signal to threshold will be in the form of a random walk, in which the velocity at each instant of time is a random, Gaussian, variable. This is very different from the linear rise envisaged in LATER. Second, that the effects of altering

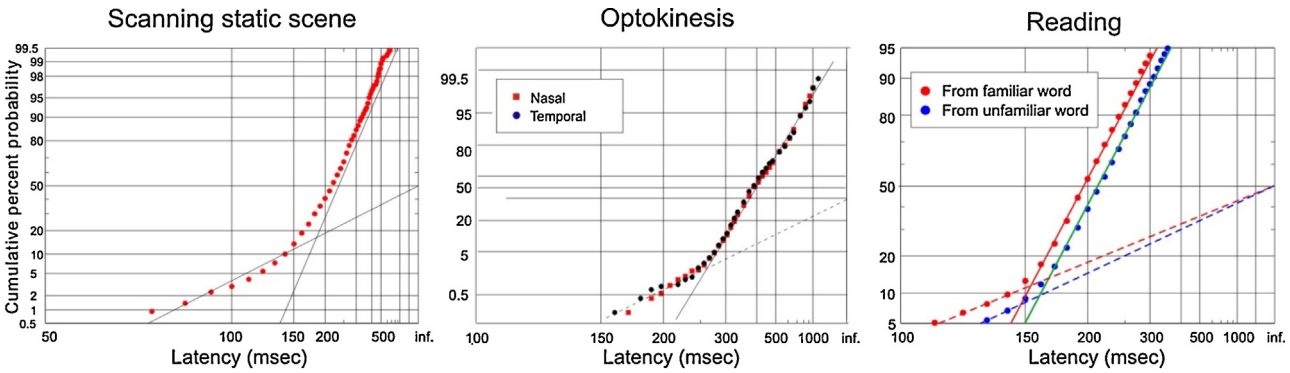



**Fig. 26.** Reciprobit plots of intersaccadic intervals for scanning a static visual scene, optokinetic nystagmus, and reading (Carpenter, 1994; Carpenter and McDonald, 2006; McDonald et al., 2005; Roos et al., 2008).

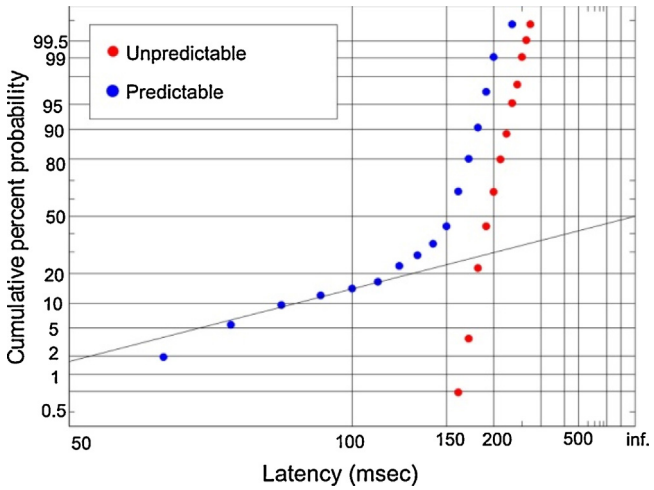



**Fig. 27.** Comparison of latency distributions for a subject making a saccade to a target moving at an unknown time to an unknown location (red), and performing a saccade of the same amplitude to the same target at a known time and to a known location (blue), showing the large increase in the prevalence of early saccades (Roos et al., 2008). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

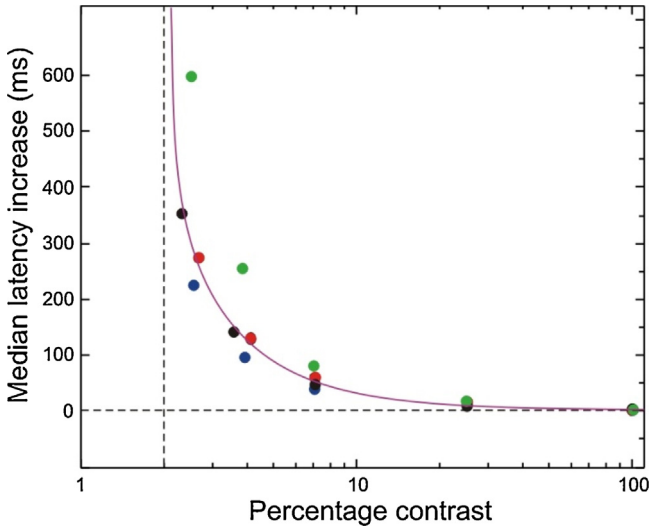



**Fig. 28.** Contrast and latency. The mean increase in saccadic latency $\Delta T$ relative to that for 100% contrast is plotted for four subjects as a function of target contrast $C$%; the purple curve shows the function $\Delta T = 200/(C-2)$. (Carpenter et al., 2009).

the rate of supply of afferent information are not always as would be expected of such a model. It is true, as we have seen, that altering the number of alternatives in a choice task, or the number of items of information in a task such as the random dot kinetogram do have the effects on latency predicted (and also predicted by LATER), this is not the case when the reduction in information supply comes about through lowering of the signal-to-noise level of the stimulus, for instance by reducing its contrast. When targets are easily

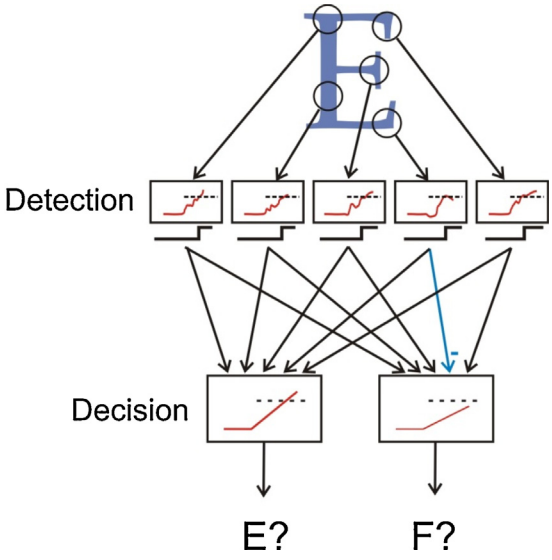



**Fig. 29.** The need for two stages. The process of detecting individual features of a letter that must be identified (top) is logically distinct from the decision processes needed to deduce from the pattern of these features what letter is actually present.

visible, altering their contrast has much less effect than would be expected if speed of response was limited by information supply; but nearer to threshold, latency is greatly increased when contrast is reduced further. In Fig. 28 it can be seen that for all the subjects shown, median latency increases about four times as much when contrast is reduced from 7% to 3% than when it falls from 100% to 7%. This suggests strongly that there are two stages in series that determine the overall latency, a first stage embodying a process of stimulus detection that is influenced by contrast, and a second stage of decision, about whether the target as a whole is present or not (Carpenter et al., 2009). At low contrasts, it is the first, detection, stage that dominates the behaviour, and at high contrasts it is decision that does so. Both stages act as integrators, but their behaviour appears different because of the kinds of signals they are integrating. The input to the first stage contains continually varying noise, so it generates a random walk; the second stage is integrating the relatively static signals coming from the detection units when they reach their threshold and raise their respective flags: it therefore generates a linear rise.

The clear logical distinction between these two processes, detection and decision, is obvious when one considers what is needed to perform simple pattern recognition. To recognise that a letter is E rather than F requires first of all that the various individual features of the letter be detected, and secondly that this information be gathered, and supplemented by such factors as prior probability and consideration of reward value that are obviously functions of the pattern as a whole and not of its individual elements (Fig. 29). A striking analogy (Reddi, 2001) is a criminal court of law, where an absolute distinction is made between the assessment of fragments of information provided individually by witnesses, and the separate question of whether these fragments, taken as a whole, are enough to amount to a conviction. Similarly, in the case of tasks driven by a random dot kinetogram, one can imagine first-stage units that detect the direction of motion within their own localized receptive fields, and that this information is then gathered together over the whole field to estimate the overall motion.

Arising out of this distinction between the two stages, and the linear rise of the second compared to the random walk of the first, is the question of the origin of the random variability. In the original single-stage models of reaction time, it was simply assumed that the random variability of latency was the natural result of that of the random walk itself, in turn ultimately due to the noise in the stimulus. But we have already seen that at ordinary contrast levels, the time taken to detect visual stimuli is insignificant in comparison with the overall latency, which is dominated by decision time. Yet this latency still shows a very large degree of random variability from trial to trial. If this variability is not coming from the stimulus, it can only be being generated internally.

This gratuitous randomness might seem at first sight to undermine the idea that LATER is in some degree an ideal decision-making process. Of course, one could argue that it doesn't matter very much if on different occasions our reaction times vary by a few tens of milliseconds. But as soon as one considers the situation in the real world, where we are presented not with single stimuli, but with large numbers of them, all competing to be looked at, then these small differences of timing matter a great deal: the individual LATER units are racing against each other, so that small random differences in latency get translated into randomness of choice. Often of course there will be an obvious winner, determined by the balance of prior probability and the strength of the evidence, and the choice will not be random at all. But if a pair of possible targets are evenly matched in this respect, they are likely to be subject to this randomness; and as we have seen, this kind of randomness of behaviour is indeed characteristic of what happens in the precedence task (Fig. 18) or very generally in tasks such as anti-saccades or countermanding in which there is a race between Stop and Go units.

Could this kind of deliberate randomness possibly be of any benefit? As early as 1934, R.A. Fisher – perhaps through his recognition of similar random processes in reproduction and genetics – realized that in some card games it is beneficial to introduce a degree of randomness into one's actions (Fisher, 1934). Such ideas became formalized as part of game theory, a field that flourished during the 1950s (von Neumann and Morgenstern, 1947; Williams, 1954). A familiar example of a game that lends itself to this kind of analysis is when I hide a coin in one of my hands, and invite you to win it by correctly guessing which. Clearly I would be foolish to hide it in the same hand every time, as you will soon predict correctly where it is. But equally, you should not always make the same guess, or I will ensure the coin is always in the other hand. Game theory proves that the optimum strategy here is for both players to be as randomly unpredictable as possible, and it is obvious that this is true of many less formal games, as with tennis, baseball, football penalties, or bowling in cricket. And in the biological world it is

equally true of the interactions between predator and prey: chased by a lion, a predictable wildebeest is more likely to end up as dinner than one who leaps around randomly. In foraging for food, it is equally true that an element of randomness in ones exploration is likely to result in a bigger pay-off than simply following the herd. There are thus powerful biological reasons for a certain amount of behavioural randomness (Carpenter, 1999); and at a more exalted level, what is human creativity and scientific discovery if not a systematic application of controlled randomness?

## 9. Neural correlates of LATER models

The theoretical and practical advantages of a LATERian description of neural decision-making and the success of the model in predicting decision times are certainly attractive features. However, one must also ask whether there is neurophysiological evidence that could corroborate its implementation at the neuronal or network level.

A key feature of LATER is the idea that variability of reaction time from trial to trial comes about not through random variation of the starting point of the cumulative process, or of the threshold for initiating action, but because of variation in the rate of rise. Recordings from movement neurons in the frontal eye field (FEF) of monkeys (Hanes and Schall, 1996) appear to show that this is the case. When a saccadic stimulus is presented, after a short delay their firing frequency rises during the latent period, and collapses again shortly after the saccade is actually executed. Although the reaction time varies randomly from trial to trial, neither the starting level nor the firing frequency at the moment of saccadic initiation alters concomitantly with the latency; rather, it is the rate of rise of activity that fluctuates randomly, and this correlates directly with the reaction time on each trial. In addition to the movement-related neurons, there is also a population of neurons in FEF whose activity is more closely related to the saccadic stimulus than to the response. Analysis of the relationship between their activity and that of the movement cells provides strong support for the idea that there are two stages in series, one representing a mechanism of sensory detection and the other of decision. In an 'oddball' task (where a monkey must make a saccade to one of a set of alternative targets that differs from the others in colour), at first the activity of the visually-driven cells rises with an identical time-course whether the colour means that the stimulus in the neuron's receptive field is or is not the required target; but after a delay (representing the time taken for colour information to reach the FEF), the time-course of the activity diverges sharply in the two cases, falling if the target is not the desired colour, but remaining high if it is to be the saccadic target (Thompson et al., 1997; Thompson et al., 1996). What is found is that the time at which this divergence occurs is essentially constant, and uncorrelated with the variability in the final reaction time. Furthermore, it is at this moment of divergence that the activity of the movement-related cells begins to rise. The conclusion is clear: the origin of the random variation in reaction time cannot lie in any kind of noisiness in the incoming sensory signal, nor in the process of detection: it must be injected into the system at the second stage, and reflected in the trial-by-trial variability of the rate of rise of the activity of the movement cells. Furthermore, neurons in MT demonstrate behaviour in RDK tasks during brief periods of motion that is very similar to the 'flag-raising' postulated in the two-stage model, with activity lasting 200 ms or more (Smith et al., 2001), implying the existence of a first stage of local integration.

Decisions often depend on the context, which may alter choice preferences even when the stimuli themselves are unchanged. Mante et al. (2013) explored how context affects decision processes by recording neurons in the frontal eye field of monkeys performing a context-dependent choice task. The monkeys were instructed by a contextual cue to identify either the overall colour or the direction of motion of a random-dot display, and to indicate their choice by making a saccade to the left or right. They showed that the decision process is distributed, rising towards threshold within a population of neurons, rather than in individual neurons, and that a recurrently connected prefrontal cortex would explain how context influences decision-making at the integration stage without prior filtering of inputs.

There is some evidence that neurons involved in decision processing may code the log likelihood ratio (LLR) of a choice, a major assumption of the LATER model. Yang and Shadlen, 2007 trained monkeys on a probabilistic reward task: monkeys had to choose between a pair of coloured targets after being shown a sequence of four shapes that determine which one of the targets is more likely to be rewarded. Recordings from neurons in the lateral intraparietal (LIP) area during this task demonstrated that these neurons develop responses much like an accumulation of evidence towards a threshold for a behavioural choice. Moreover, the firing rates of these LIP neurons correlate with the LLR denoted from the shape stimuli given in sequence. This study therefore is consistent with the notion that a neural decision signal represents the LLR of a given choice, a major feature of the LATER model.

What seems less clear, however, is precisely how neurons are accumulating evidence towards this threshold. In order to answer this question, investigators typically pool data from many different neurons that are being recorded and from many separate trials, and this averaging of the data may obscure fundamental underlying properties of the information being recorded. Very recently, Latimer and colleagues recorded from neurons in the LIP of macaques during choice tasks, whose activity appears to be accumulating gradually or 'ramping' towards threshold as models predict (Latimer et al., 2015). Importantly however, the group analysed single trial responses of these LIP neurons and demonstrated that although a 'ramping' response provides a satisfactory prediction of mean neural spike rates, a 'stepping' response is able to predict not only the mean spike rate but also the variance in the responses better than the ramping model. They found this to be true for the majority of neurons they recorded from. The stepping behaviour is where the spike rate of a neuron stochastically jumps from one rate to another during a single trial, essentially a 'discrete' rather than an 'analogue' form of neural implementation of decision-making.

In order to demonstrate whether decision signals vary randomly from trial-to-trial, as posited in the LATER model, one must be able to measure neural activity at the level of individual trials, rather than averaging across a large number of trials as is typically done in experiments. Smyrnis et al. (2011) achieved this by recording magnetoencephalography (MEG) signals from single-trials of humans performing a visuomotor decision task. They found that such signals from motor cortical areas predicted reaction times, and – very significantly – that these signals varied randomly from trial-to-trial and appeared to behave in a rise-to-a-fixed-threshold manner, as predicted by LATER. This corroborates in humans the earlier primate work of Hanes and Schall (1996), with decision network activity across cortical areas as opposed to responses from individual neurons.

As previously discussed in this review, altering the predictive information provided for decision-making ought to cause a swivel in the reaction time distribution if the distance to threshold of the decision process is the underlying mechanism accounting for the change in behaviour, according to the LATER model. Dolmenech and Dreher (2010) employed a Go/No-Go task and altered the level at which a subject would be able to predict an upcoming stimulus. Reciprobit analysis enabled differentiation between two possible mechanisms accounting for faster responses when subjects were given more predictive information: a change in rate of rise or

distance to threshold of the decision process. Indeed, they found the latter to explain the data very well. Moreover, by using fMRI, they were able to demonstrate the anterior cingulate cortex as the only brain region whose neural activity predicted the subjects' ability to use predictive information to modulate the distance to threshold, and that this region feeds information into the dorsolateral prefrontal cortex which encoded the decision variable itself. This study effectively combined behavioural modelling with imaging of human brains to demonstrate how LATERian decision-making may be implemented at the neural network level.

As discussed previously, under conditions of urgency in which a subject must respond as quickly as possible at the likely cost of accuracy of responses, the LATER model predicts that the response threshold is lower and this satisfactorily accounts for response times in these urgent conditions. The neurophysiological correlates of this are currently not clear. Heitz and Schall (2012) have recently attempted to explore this by recording from visually responsive neurons of monkeys' frontal eye fields in choice tasks requiring such a speed-accuracy tradeoff (SAT). In contrast to a simple lowering of response threshold, they found that these neurons modulated their baseline firing rate, sensory gain and the time taken for sensory processing. The group thus proposed an 'integrated accumulator model' to explain the data: this model allows more parameters to vary, in particular it explains the changes across SAT conditions by additional alterations in accumulation rate and baseline starting activity. However, this area is still uncertain, with other groups providing challenging opinions (Cassey and Brown, 2014; Hawkins et al., 2015).

The superior colliculus was traditionally thought to be a downstream output of motor decision areas such as the FEF, but now is increasingly recognised as a contributor to decision-making in its own right (Carello and Krauzlis, 2004; Krauzlis and Dill, 2002; Krauzlis et al., 2004; Phongphanphanee et al., 2014), or at least forming part of a distributed neural network whose activity reflects the ongoing decision. Basso and Wurtz (1998) investigated the effect of altering prior probability of build-up neurons (neurons with a delay period activity) in the superior colliculus of monkeys. Altering target probability, they found that the baseline activity of these neurons was consistently reduced as the probability of a saccade decreased and that it also predicted the saccadic latency, a necessary prerequisite if these cells are indeed contributing to the generation of these behavioural responses. Such neurophysiological experiments are in keeping with LATER, in which $S_0$, the starting level of activity, is modulated by prior probability. It should be noted that there are lateral inhibitory interactions via interneurons between fixation and saccade neurons in the intermediate layer of the ipsilateral and contralateral superior colliculus. Such lateral inhibition is thought to be important for maintaining stable visual fixation and initiating saccades appropriately to selected rather than unwanted targets (Cutsuridis et al., 2007; Kim and Basso, 2008; Munoz and Istvan, 1998; Munoz and Wurtz, 1993).

Over the last few years, the LATER model has been extended to increasingly complex tasks, with multiple choices including stopping. Stopping, in which one must cancel an impending response, is a special decision. Imagine the scenario where a pedestrian just crosses in front of a driver as he is about to step on the accelerator: the driver must quickly cancel his impending action in order to avoid disastrous consequences! The mechanism by which we stop actions is proposed to be a special Stop LATER unit: this follows the same principles as all LATER decision units, except that when it reaches threshold it cancels the activity of another LATER unit. It has been a crucial feature of LATER models of the Go/No-go task, antisaccades and the Wheeless task: modelling the details of the RT distributions of these tasks necessitates the use of a Stop unit (Noorani, 2014; Noorani and Carpenter, 2011b, 2013). A recent study has rather excitingly demonstrated that stopping actions in a Go/No-go task in rodents involves a race of neural activity between a pathway from the subthalamic nucleus to the substantia nigra (Stop pathway) and a pathway from the striatum to the substantia nigra (Go pathway). Significantly, the timing of activity in these two pathways predicted whether or not the rodents were able to cancel a response (Schmidt et al., 2013). Given the basal ganglia are the primary output of cortical decision areas, such as the FEF, and project to downstream areas such as the superior colliculus that can be thought of as implementing decisions, it is entirely plausible that this stop pathway may extend to stopping in other tasks, for example antisaccades (Noorani, 2014). For such complex tasks, one can conceive a race between multiple LATER units, each unit being composed of distributed neural activity in a specific region or pathway, which is well illustrated by this race in the basal ganglia.

## 10. Conclusions

Deciding what action is most likely to benefit us is in a sense the highest function of the brain, and its importance is reflected in our long and very variable response times. Various models have been proposed to explain this behaviour; most have been purely empirical but some, like LATER, based on principles of decision theory. Particularly in simpler decision tasks where there are often only one or two possible responses, the LATER model has gained increasing popularity. The main reason for this is its simplicity, reflected in its unusually small number of parameters, yet capable of modelling model increasingly complex decision behaviour in such tasks as Wheeless, Go/No-Go and antisaccades, often with several competing decisions occurring in parallel. Aside from the ease of modelling that the few parameters provide, this conceptual simplicity provides the possibility of interpreting clinical disturbances in terms of dysfunction of the underlying neural mechanisms. Although it is undoubtedly important to correlate model features with 'hard' neurophysiology, in the form of neural recordings, such vindication often has lagged behind novel predictions and features of decision models. Indeed, it was over a decade after the LATER model posited that the decision signal represents the log likelihood ratio of a potential decision (Carpenter and Williams, 1995) that neuronal responses in the parietal cortex were demonstrated to be exactly that by Yang and Shadlen (2007). Confirmation of model features by actual neurophysiology long after it was conceived is unusual in neuroscience but highlights the strength of this modelling approach. Bayesian-based decision-making is more and more recognised to be the simplest way of explaining how we face the tougher and more nuanced choices that our environment continually presents to us, reflected in correspondingly complex patterns of behaviour.

## Appendix A. Modelling reaction times

This Appendix brings together some of the more purely mathematical aspects of what has been discussed above.

### 8.1. Modelling reaction times

#### 8.1.1. The recinormal distribution

In a recinormal distribution the reciprocal of the variate is normally distributed. In the case of reaction times, this means that the probability $R(s)$ of a response in a particular trial having a latency $T$ whose reciprocal ($\lambda = 1/T$) lies between $\lambda$ and $\lambda + d\lambda$ is:

$$R(\lambda) = \frac{-1}{\sigma\sqrt{2\pi}} e^{-(\lambda-\mu')^2/2\sigma'^2} ds \quad (1)$$

where the mean (and also median) of the distribution is $\mu$' and $\sigma'^2$ is the variance.

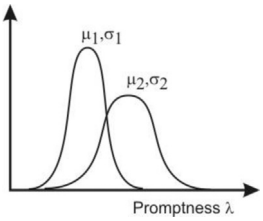
From this we can derive the probability density $L(t)$ of the set of original reaction times $T_i$:

$$L(t) = \frac{1}{t^2\sigma\sqrt{2\pi}} e^{-(1-\mu' t)^2/2t^2\sigma'^2} dt \quad (2)$$

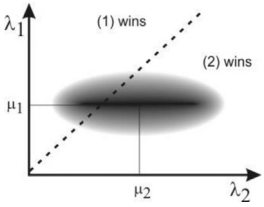
This is a positively skewed distribution whose median value $T_M$, is $1/\mu$', but whose mean and variance do not have simple analytical forms.

### 8.1.2. Theory of competing units

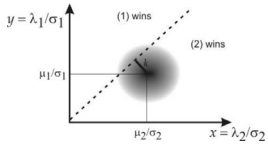
A situation that often arises is of two competing LATER units, $(\mu_1, \sigma_1)$ and $(\mu_2, \sigma_2)$. How can we calculate the proportion of wins by the faster one as a function of the parameters?




If the two races are independently random, a set of trials of $(\mu_1, \sigma_1)$ versus $(\mu_2, \sigma_2)$ will generate a bivariate distribution, with unit 1 winning when $\lambda_1 > \lambda_2$, i.e. when the corresponding point lies to the left of the line $(\lambda_1 = \lambda_2)$:

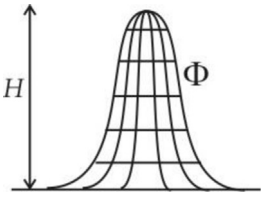



It is helpful to scale the axes $(y = \lambda_1/\sigma_1, x = \lambda_2/\sigma_2)$ to make the distribution circular in outline. Unit 1 wins when the point lies to the left of $(\sigma_1 y = \sigma_2 x)$. The distance $k$ of this line from the centre of the distribution is $\Delta\mu/\sqrt{\sigma_1^2 + \sigma_2^2}$, where $\Delta\mu = \mu_1 - \mu_2$




So we now need to know what volume is generated when we make a cut at a distance $k$ from the centre of a symmetrical Gaussian mound $\Phi = He^{-(x^2+y^2)/2}$ of unit volume.

A little bit of more advanced maths is needed.




The height of a thin slice $dt$ at $t$ from the centre is $He^{-t^2/2}$.

Therefore its volume is $\int_{-\infty}^{\infty} He^{-t^2/2} e^{-s^2/2} ds dt =$

$$He^{-t^2/2} dt \int_{-\infty}^{\infty} e^{-s^2/2} ds = H\sqrt{2\pi} e^{-t^2/2} dt$$

So the volume of the entire mound is $H\sqrt{2\pi} \int_{-\infty}^{\infty} e^{-t^2/2} dt = 2\pi.H$,

and this must be equal to 1 since it has unit volume. So $H = 1/2\pi$.

Therefore the volume of the offcut is $\frac{1}{\sqrt{2\pi}} \int_{-\infty}^{t=k} e^{-t^2/2} dt = P(k)$,

where $P$ is the normalised error integral, $P(x) = \frac{1}{2}\left(1 + \text{erf}x/\sqrt{2}\right)$.

Returning to the original problem, since $k = \Delta\mu/\sqrt{\sigma_1^2 + \sigma_2^2}$ the probability we were looking for is:

$$p = P\left(\frac{\Delta\mu}{\sqrt{\sigma_1^2 + \sigma_2^2}}\right) \quad (3)$$

For the special case of $\sigma_1 = \sigma_2$,

$$p = P\left(\frac{\Delta\mu}{\sigma\sqrt{2}}\right) = \frac{1}{2}\left(1 + \text{erf}\frac{\Delta\mu}{2\sigma}\right).$$

A Windows application, SPIC, is publicly available for download free of charge that enables complex simulations of interacting LATER units, with reciprobit analysis, as well as performing latency experiments and statistical analysis of data (Carpenter, 2015).

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## Nomenclature

*T:* Time
*i:* Trial index
*s*, $s_i$*:* Reciprocal time, set of reciprocal times
*N:* Number of trials
*T*, $T_i$*:* Latency, set of latencies
$\lambda$*:* Promptness (reciprocal latency, 1/*T*)
$T_M$*:* Median latency
*L(t):* Probability density function for latency
*R*($\lambda$)*:* Probability density function for promptness, $\lambda$
*C*($\lambda$)*:* Cumulative frequency of $\lambda$
*C(0):* Terminal frequency of *C*($\lambda$)
*P(x):* Cumulative normal function
*Q(x):* Complement of the cumulative normal function: *Q*=(1-*P*)
*S:* Decision signal
$S_0$*:* Initial value of *S*
$S_T$*:* Threshold value of S that initiates a response
$\theta$*:* Range of *S*: ($S_T$–$S_0$)
*r*, $r_i$*:* Rate of rise of *S*, set of rates of rise
$\mu$*:* Median of $r_i$
$\sigma^2$*:* Variance of $r_i$
$\mu'$*:* Mean of *R*(*s*)
$\sigma'^2$*:* Variance of *R*(*s*)
$\delta$*:* Delay
$\delta_0$*:* Transport delay
$\tau$*:* Time constant