Predicting continuous outcomes: Some new tests of associative approaches to contingency learning.

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Abstract

Associative learning models have traditionally simplified contingency learning by relying on binary classification of cues and outcomes, such as administering a medical treatment (or not) and observing whether the patient recovered (or not). While successful in capturing fundamental learning phenomena across human and animal studies, these models are not capable of representing variability in human experiences that are common in many real-world contexts. Indeed, where variation in outcome magnitude exists (e.g., severity of illness in a medical scenario), this class of models are, at best, approximate the outcome mean with no ability to represent the underlying *distribution* of values. In this paper, we introduce one approach to incorporating a distributed architecture into a prediction error learning model that tracks the contingency between cues and dimensional outcomes. Our Distributed Model allows associative links to form between the cue and outcome nodes that provide distributed representation depending on the magnitude of the outcome, thus enabling learning that extends beyond approximating the mean. Comparing the Distributed Model against a Simple Delta Model across four contingency learning experiments, we found that the Distributed Model provides significantly better fit to empirical data in virtually all participants. These findings suggest human learners rely on a means of encoding outcomes that preserves the continuous nature of experienced events, advancing our understanding of causal inference in complex environments.

Keywords: contingency learning; distributed representation; causal judgment; connectionist models; associative learning

Introduction

Contemporary models of associative learning are founded on their ability to reproduce phenomena observed across the animal kingdom and, critically, this includes effects that translate well to human contingency learning. Over the last 50 years, a wealth of contingency learning data has accumulated, tackling issues such as the sensitivity of causal judgments to contingency (Jenkins & Ward, 1965), biases in perceiving contingent relationships when none exist (e.g., Hamilton & Gifford, 1976; Matute et al., 2015), and cue competition (e.g., Kamin, 1969). The vast majority of these data come from experiments with simple and discrete stimuli that do not vary substantially in magnitude. Outcomes are usually visually represented as being present or absent, enabling the degree of association between a cue and an outcome to be determined by the covariation of binary events. This is, of course, a dramatic simplification of the events that a learner encounters in the real world, which often vary by degree rather than merely being present or absent. To illustrate, contingency learning tasks using a medical scenario typically involve presenting a medical treatment and evaluating whether the patient has recovered or not recovered (i.e., two possible outcome states; e.g., Vinas et al., 2025). Most health experiences however, involve fluctuating and noisy information and it is often unclear how one might characterise these experiences in a dichotomous way. The use of binary outcomes is convenient and justifiable in many circumstances (e.g., it helps to ensure we have theoretically tractable research questions), but it potentially misses a rich source of variation in the way we learn and avoids addressing challenges that models of learning must face in order to provide an adequate account of contingency learning in the real world.

More recently, much has been made about learning about complex properties, including distributional learning (people find it difficult to reproduce experienced distributions, e.g., Tran et al., 2017), and uncertainty related to making predictions (e.g.,

Courville et al., 2006; Dayan et al., 2000). A Bayesian approach to causal inference explicitly represents uncertainty in its model, and this approach has been extended from its original remit, predicting binary variables (Griffiths & Tenenbaum, 2005), to continuous ones (Pacer & Griffiths, 2011). However, for the most part, the Bayesian approach is concerned with defining and formalising the problem of causal inference, that is, *what* should be calculated for valid causal induction. In contrast, mechanistic models, including the prediction error learning networks we use in this study, are concerned with providing some account of the psychological mechanisms responsible for mental representation of events and how we learn about them.

Connectionist models assume that people track the covariation between events through mental links connecting representations of these events. The strength of these connections change with increasing experience. Many use a simple error-correction learning algorithm like that proposed by Rescorla and Wagner (1972; the RW model) in which learning is proportional to the discrepancy between what is expected and what actually occurs (i.e., *prediction error*). With increased experience of a putative cause (commonly referred to as a cue) and outcome co-occurring, associative weights linking the two events strengthen, such that the outcome is increasingly expected when the cue is present. Predictions about the outcome can be directly estimated by taking the summed associative strength of all cues present (i.e., how much the outcome is expected given cues present). Violation of expectancy—for example, when the outcome is expected but does not occur—produces a prediction error signal that leads to updating of associative strengths, i.e., learning. Thus, on each trial the model provides an estimate of the associative strength, and this estimate is updated based on the whether the outcome is present or absent.

The appeal of simple connectionist models like the RW model is that the process of learning is greatly simplified by providing a single point estimate for the outcome. In a contingency learning task where the outcome has some numeric value that changes trial-to-trial (e.g., magnitude of symptom severity), the associative strength will approximate the outcome mean. The implication of this is that prediction error models of this class are models of the outcome mean; they are not typically designed to provide an accurate fit of the *distribution* of outcomes, where variation in outcome magnitude exists, thereby missing a rich source of information about how we learn about variable events. However, there is scope for these models to represent outcomes in a distributed network. The present paper demonstrates one way in which a distributed representation of outcome values can be incorporated into a prediction error model of contingency learning.

Distributional representation in learning

Distributed stimulus representation is not new in associative learning. However, it is most commonly used to represent predictive *cues* that vary along a continuum (e.g., McLaren & Mackintosh, 2002; Livesey & McLaren, 2019; Thorwart, Harris & Livesey, 2012; Ghirlanda & Equist, 1998). Relatively little research within the human associative learning tradition has examined how well people learn about continuous *outcomes*. Previous work from our lab has shown that some phenomena typically found in contingency learning research using discrete outcomes were also present when participants were presented with continuous outcomes (e.g., outcome density effect; Chow et al., 2019; Double et al., 2020), suggesting that a common cognitive process may be involved in learning about discrete and dimensional outcomes. In some instances, researchers have used continuous outcomes to leverage a certain type of cognitive process. For instance, some of the debates about whether people show blocking for reasons to do with deductive reasoning or other decision processes

make use of outcomes that are demonstrably additive and/or submaximal on some magnitude scale (Beckers et al., 2005; Lovibond et al., 2003; Livesey & Boakes, 2004; Livesey et al., 2019). In these instances, however, variations in magnitude are a means to an end; these experiments are not concerned with whether people learn about continuous outcomes *per se*.

Distributed representations have previously been explored in other learning domains. For example, in function learning, the learner is asked to predict the magnitude of the outcome based on the level of input. The functional relationship between cue and outcome can be determined by some formula (e.g., a linear relationship) plus some noise (DeLosh et al., 1997; McDaniel et al., 2014): as the input at the cue level increases, the magnitude of the output also increases to a similar degree. Function learning has a place in everyday life, for example a doctor who is trying to estimate the change in the patient's body temperature given the severity of infection. This type of learning allows people to understand how the magnitude of an effect changes according to the strength of the cause, and thus make reliable predictions based on that relationship. There is evidence that people readily learn these functional relationships (Byun, 1995), and that knowledge can be captured in an associative framework (Krushcke, 1992; DeLosh et al., 1997). Associative learning models in function learning provide a starting point to consider how a distributed network for representing complex properties of outcomes can be implemented in contingency learning models.

The aim of the current project is to develop a connectionist model of contingency learning capable of representing continuous outcomes in a distributed fashion, and formalise a way in which distributions of responses might be derived from such a model. In particular, we are interested in what can be gained from a distributed approach to representing continuous outcomes in a prediction-error learning model, compared to a similar model which produces only a single point estimation that approximates the outcome mean. We will

first discuss one approach that could be used to represent distribution of outcome values, and then report the outcomes of a formal model comparison comparing the distributed model against a simple connectionist model that does not implement distributed outcome representation.

Modelling continuous outcomes with a prediction error algorithm

Simple Delta Model

We present here a description of a simple prediction error model (or delta rule model) commonly used to model trial-by-trial learning. The basic feature of a delta rule model is that the strength of connection between a single cue and outcome is updated on each trial according to a learning algorithm like the one shown in Equation 1:

$$\Delta V_{ij} = \alpha_i \times \beta_j \times (\lambda - \Sigma V)$$

Equation 1.

In this equation, the change in the strength of the connection between cue i and the outcome j, changes according to prediction error, that is, the discrepancy between the observed outcome, λ, and the extent to which it is predicted by the cues present, ΣV. Note that among the cue representations, it is assumed that the context is also represented by one or several nodes in the connectionist network, entering into associations with the outcome like other cue nodes. When cues are followed by the outcome, $(\lambda - \Sigma V)$ is typically positive, though the amount that will be learned about their co-occurrence depends on whether the outcome is surprising (larger prediction error = more learning) or expected (smaller prediction error = less learning). When cues are followed by the *omission* of the outcome, $\lambda - \Sigma V$ can be negative if the outcome was expected. That is, if ΣV is greater than λ, then the connection linking the cue and outcome is weakened, or becomes increasingly inhibitory if $V < 0$. This simple

interaction between expectation and error allows the algorithm to track contingencies between cues and outcomes accurately. Additional parameters α and β denote the learning rate of the cue and outcome respectively. The simplest instantiations of this model, those used to model a single outcome with two states (present vs absent) for instance, assume that the absence of the outcome does not have its own unique representation – it is simply represented as a situation in which $\lambda = 0$ in Equation 1.

In the present paper, as a starting point we assume that the representation of the outcome itself must have some relationship with the outcomes that are observed on each trial. Thus, on each trial λ takes on the observed outcome value, which in our case is a value between 0 and 100. We make the additional assumption here that participants are biased towards the center of the outcome scale at the start of the study, thus the associative strength V for the context starts at a value of 50. In all other respects, the model operates like a conventional delta rule model, and V for the cue approximates the mean outcome value at that point in time. Both associative strengths of the cue and the context update independently on trials where the cue is present, but only the associative strength of the context is updated when the cue is absent.

We additionally introduce a noise parameter, $sd_{expectancy}$, to capture unsystematic variability in predictions based on the learner's estimate of expected value; this is achieved by creating a gaussian function with a probability distribution centered around the expected outcome mean, and variance = $sd_{expectancy}$. Unlike the Distributed Model which we will present below, $sd_{expectancy}$ parameter is not implemented at the learning stage but only used to generate simulated predictions. The theoretical assumption here is that participants are solely tracking the outcome mean during learning, but may still produce predictions that vary around that mean. In contrast, a distributed model would encode that variability *during*

learning, resulting in a distribution of activation across outcome nodes. A corollary of this is that when generating simulated predictions with the Simple Delta Model, response probabilities are always normally distributed around ΣV, whereas in the Distributed Model response probabilities are, in principle, capable of demonstrating sensitivity to the shape and skew of the underlying outcome distribution.

In generating simulated predictions, we first assume that, all things being equal, the outcome value the person observes will be reflected in the predictions they subsequently make. Thus, like the participants in our experiments, the model can predict any integer value from 0 to 100 (101 distinguishable responses). In modelling human choices, the problem that is commonly faced is how discrete predictions are made on each trial. In many contexts, especially those involving discrete choices among several options, researchers assume that predictions are probabilistically determined. The *softmax* rule is often used for this purpose (Bridle, 1990; Rumelhart et al., 1995):

$$p(r) = \frac{ke_x}{\sum_{x=1}^{j} ke_x}$$

Equation 2.

Equation 2 calculates the probability of choosing each response from a set of j choices (the probabilities of all choices summing to 1). The probability of each choice is determined by the response probability, e_x, and by an inverse temperature parameter k. In the Simple Delta Model, e_x is generated using a gaussian function with a probability distribution centered around the expected outcome mean, and variance = $sd_{expectancy}$. Values of k are restricted to a range from 1 to 5 as is typical in the choice literature (e.g., Don et al., 2019), where higher values of k lead to more consistent choice of the outcome node with the highest activation, whereas when k is a small value, choice probabilities become increasing similar and a large

difference in relative activation is required to produce a choice response with greater uncertainty. In short, the greater the relative magnitude of outcome node activation, the greater the probability of choosing the response. Using this algorithm, we can get the model to produce a discrete prediction on each trial, and compare those predictions to participants' responses during the experiment.

Finally, we include α for the cue and the context to be separate parameters determined by model fit, where α is the learning rate of the cue, and α_x is the learning rate of the context. In some instantiations of such a model, learning rate of the context, α_x is assumed to always be less than the learning rate of the cue, α; this constraint was not applied to model fit in the current study.

Distributed Model

In this section, we will introduce one way in which distributed outcome representation can be incorporated into a delta rule model. One important difference between a simple delta rule approach and a distributed network approach is that in the former, there is a single associative value representing the strength of the connection between the cue and the outcome. In a distributed approach on the other hand, the cue forms an associative link with *multiple* outcome nodes along the dimension. Although it is not strictly necessary, for convenience, we used the same number of nodes as there are possible outcome values in our experiment. Thus, in the Distributed Model, there are as many associative links for each cue as there are outcome values. Learning then changes the strength of the connections between the cue and the outcome nodes. The strength of each connection depends on both the mean outcome value aggregated across trials, and the underlying distribution. This method of representing the outcome in a distributed network effectively tracks the occurrence of outcomes in a way that can differentiate between different outcome distributions that possess the same mean.

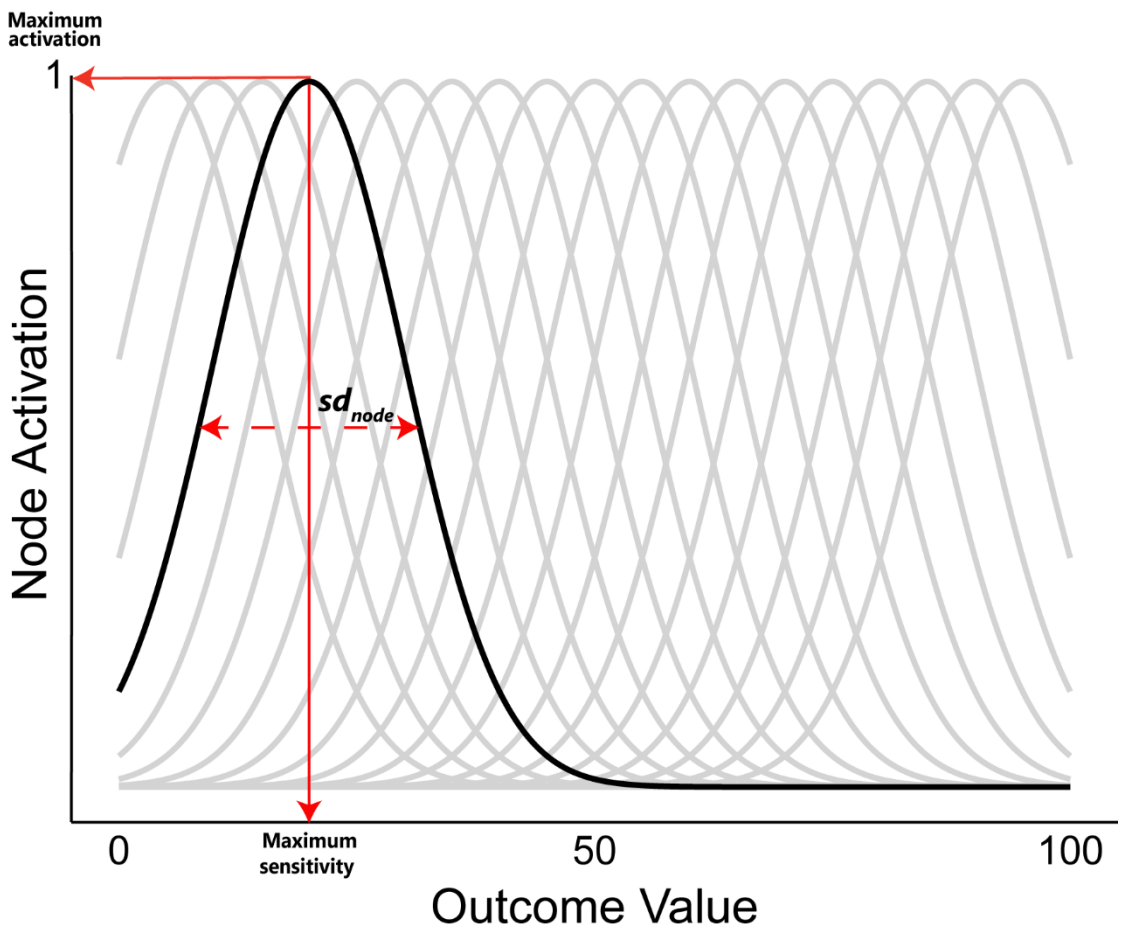
We incorporate a distributed architecture into the delta model by assuming that any given outcome magnitude is represented in a distributed fashion, resulting in activation of a series of outcome nodes that have overlapping sensitivities (see Figure 1). Activation of each node is a function of the distance between the observed outcome on that trial, and the peak sensitivity of the node. Each node is maximally sensitive to a particular outcome value, and activated incrementally less for outcomes that are larger or smaller than this value. For example, when the observed outcome has a value of 50, the outcome node corresponding to 50 is maximally activated. Values around 50 may also receive some level of activation, however node activation decreases as we move further away from the outcome node corresponding to the observed value. On every trial when an outcome value is presented, activity in each outcome node is determined according to a gaussian function, with a mean equal to the outcome value on that trial, and variance around the mean, sd_{node}. The activation of these units is then normalised so that they sum to 1. A corollary of this distributed architecture is that, unlike the Simple Delta Model (where we added unsystematic variance determined by $sd_{expectancy}$), response probabilities for making predictions can be directly determined based on ΣV (i.e., $e_x = \Sigma V$).

In contrast to the Simple Delta Model where the starting V for the context was set to outcome value of 50, starting values for V across the outcome dimension in the Distributed Model was randomly determined from values ranging between -.001 and +.001[1]. The strength of each associative link is updated on each trial following a prediction error algorithm in a similar fashion to the Simple Delta Model, that is by taking the difference in the outcome node activation and the expected value.

[1] Note that this difference in starting point should produce a greater advantage for the Simple Delta Model since participants tend to provide ratings at the mid-point of the scale. As we will see in the following section, the added advantage did not produce significantly better model fit for the Simple Delta Model.

Figure 1

Illustration of distributed architecture in the Distributed Model, where the presentation of an outcome value will result in maximal activation at the outcome node corresponding to that value with decreasing activation away from that node following a gaussian function with variance = sd_{node}. *Fixed parameters are presented as a solid line, and estimated parameters as a dashed line.*



We fit each model to individual participants' predictions made across the experiment using Maximum Log Likelihood Estimation (MLE). MLE selects parameter values that maximise the likelihood that the model produced the data observed in the empirical study. Both the Simple Delta and Distributed Model consisted of four estimated parameters required for model fitting and/or generating model predictions: context alpha (α_x), cue alpha (α), variance in a gaussian function for making predictions in the Simple Delta Model ($sd_{expectancy}$)

or activation spread during learning (sd_{node} in the Distributed Model), and temperature parameter for softmax, k required for model predictions. Best fitting parameters for both models for each participant in all four experiments can be found on the Open Science Framework (Chow et al., 2025).

In summary, both the Simple Delta and Distributed Model consist of the same number of estimated parameters for model fitting and making simulated predictions. Both models similarly rely on a prediction-error algorithm to update learning on each trial. The primary distinction between the two models is the ability for the Distributed Model to produce an associative link between the cue and each outcome node in the dimension (e.g., every value from 0 to 100 on a continuous scale). The relative activation of each outcome node across the outcome dimension allows the model to capture the underlying distribution of outcome values. In contrast, the Simple Delta Model updates a single associative value on every trial, producing a single terminal value (after many samples) that approximates the outcome mean; this approach has no ability to distinguish between cues with the same outcome mean but different underlying distributions (e.g., normal distribution vs negatively-skewed distribution).

In the following section, we will briefly describe a series of experiments and the empirical data used to determine model fit. We fitted both models using the same trial sequence and outcomes presented to participants in Experiments 1-4. We will provide a comparison of the two models across all four experiments using the Bayesian Information Criterion (BIC; Schwarz, 1978), followed by a discussion on model performance and simulated predictions.

Empirical Data

We report here four empirical studies designed to determine participants' sensitivity to outcome distributions. We designed these studies to include various factors that might influence learning, such as the presence of variability (Experiment 1), overall base-rate of outcome values (Experiment 2), and shape of the distribution in which outcome values are sampled from (Experiments 3 & 4). The purpose of these experiments was to obtain participant data that will later be used for model fit: we are interested in comparing the fit of both models (using best fitting parameters for each individual participant) to determine whether participants' prediction ratings during learning were better captured by the Simple Delta Model or the Distributed Model that is capable of representing the underlying distribution of outcome values. For brevity, we report only an overview of the design of each experiment and findings here; detailed description of the study design and empirical data can be found in Supplementary Materials.

Design & Procedure

Participants in all four experiments underwent a simple contingency learning experiment, where they were told to imagine that they were a medical researcher investigating a new illness and the efficacy of several fictitious drugs at treating this disease. Participants were shown a series of patients given one of the fictitious drug treatments and their subsequent health outcome on a scale from 0 (no recovery) to 100 (full recovery). The number of fictitious drug treatments differed between experiments (minimum = 8 [Experiment 3], maximum = 13 [Experiment 2; including filler cues]). Target cues were organised by distribution and mean outcome value. The allocation of stimulus name and image to cue type was random across participants. See Table 1 for a summary of the design of each experiment.

General procedure of all experiments

Participants were told that each trial represented a different patient who was given one of the drug treatments. Participants were first shown the drug treatment given to the patient and asked to predict the level of improvement in the patient's health they expect given the treatment provided. A prediction scale was presented below the drug cue ranging from 0 (no improvement) to 100 (full recovery) and participants were asked to make a prediction by clicking on a point on the scale. Clicking on the scale would result in the appearance of a horizontal bar extending from 0 to the final click location. Once a prediction has been made, participants could press the space bar to continue. The patient's actual observed health improvement was then presented on a near-identical scale below the prediction scale (the only difference being the colour of the horizontal bar inside the scale). Outcomes were animated as a growing horizontal bar along the scale starting at 0 to the final outcome value. Large improvements in health were indicative of the drug's efficacy in treating the disease. Prediction ratings made across the experiment were our primary measure of learning, and were thus used to determine model fit.

In addition to predictions across training, we also measured participants' average judgements at the end of the experiment; these include measures of outcome mean, outcome mode and causal ratings judging the efficacy of each treatment on patient recovery.

Table 1

Summary of the design of each experiment. Number in brackets represent the number of levels in the corresponding within-subjects factor.

Experiment	Design	
	Target cues	**Non-target cues**
1	Mean (3): 25, 50, 75 x Distribution (3): Fixed values, Uniform, Normal	Fixed outcome 0 and Fixed outcome 100
2	Mean (5): 15, 30, 50, 75, 80 All cues had outcomes sampled from a normal distribution	Six filler cues for between-subjects base-rate manipulation: 3 x Base-rate condition 30, 50, 70 Fixed outcome 0 and Fixed outcome 100
3	Mean (2): 35, 65 x Distribution (3): Normal, Positive-skew, Negative-skew	Fixed outcome 0 and Fixed outcome 100
4	Mean (2): 35, 65 x Distribution (3): Bimodal, Normal-Narrow (Low Variance), Normal-Wide (High Variance)	Fixed outcome 0 and Fixed outcome 100

Variations across experiments

Experiment 1 was designed to determine whether participants were sensitive to the presence of variability in outcome values. To achieve this, we presented cues with identical outcome means (mean = 25, 50, 75) but had outcomes that were either constant values (e.g., always 25 on a scale from 0 to 100), or variable values sampled from a uniform or normal distribution. The primary distinction between cues with outcomes from a uniform vs normal distribution lies in the frequency of different outcome values occurring. If participants were sensitive to, and made predictions based on, the experience of variability in outcome values, we might expect differences in their pattern of predictions for cues with constant vs variable outcomes

of the same mean. In addition, distribution of predictions is expected to differ for cues with normally distributed vs uniformly distributed outcomes; we expect the Distributed Model to be sensitive to variability in the probability of different outcome values, whereas the Simple Delta approach may fail to discriminate between the two.

In Experiment 2, we manipulated the base-rate of the outcome in a between-subjects design by presenting a number of filler cues, such that the overall outcome base-rate could be low, medium or high (30, 50 or 70) while the mean outcomes of target cues varied across the range of possible outcomes (mean = 15, 30, 50, 75, 80). All target cues had outcomes sampled from a normal distribution. Differences in the base-rate of the outcome have previously been shown to impact judgements people make about predictive cues (e.g., the inverse base-rate effect; Medin & Edelson, 1988; Don et al., 2021) and non-predictive cues (e.g., the outcome density effect in illusory causation; Alloy & Abramson, 1979; Blanco & Matute, 2020; Blanco et al., 2020a). Varying the base rate thus allows us to determine whether contextual factors assert any influence on predictions made to *specific* target cues, and whether these differences, if present, are captured by our two models.

In Experiments 3 & 4, we were interested in whether participants' ratings were sensitive to the range of probable outcome values. In Experiment 3, we drew outcomes from either a normal, positively-skewed or negatively-skewed distribution at two levels of outcome mean (35 and 65). The logic here is that these distributions differ in the outcome values that are likely to be sampled. For example, one cue A might be associated with a normally distributed outcome with a mean of 65, while another cue B was associated with a positively skewed distribution with the same mean. In the latter case, most of the outcomes shown were above 65 but occasionally outcomes of a lower value were shown. If participants' judgements reflect the mean outcome to which they are exposed in the presence

of that cue, then their judgements for A and B will be equivalent. If participants make judgments that reflect the most likely range of outcomes, then they are likely to predict that the outcome for B will be higher than for A. Alternatively, if participants are more strongly influenced by the rarer but more discrepant low outcomes then they may predict lower outcomes for B than for A.

Experiment 4 provided an additional test of the impact of distribution shape and variance by comparing cues with outcomes sampled from a normal distribution with a small variance (normal-narrow), normal distribution with large variance (normal-wide) and a bimodal distribution. Like in Experiment 3, mean outcome was 35 for half the target cues and 65 for the other half.

Results & Discussion

The pattern of predictions made for each cue type was compared using Kolmogorov-Smirnov (KS) test of equality on the final 10 presentation of each cue in Experiments 1 & 2, and last 20 presentations in Experiments 3 & 4 (the increase in trials used for analysis in the latter experiments were due to more subtle differences in the distributions being compared). The KS test provides a *D* statistic, which quantifies the difference in the cumulative distribution function of the two comparison distributions; it is therefore a measure of the how similar the two distributions are: larger *D* statistic indicates a larger difference between the two distributions. Using the *Matching* package in R (Sekhon, 2011), a bootstrapping procedure (number of bootstraps = 1000) was applied to calculate a bootstrap p-value for the KS test, with the null hypothesis that probability densities for the two comparison distributions are the same. Overall, the pattern of predictions across all four experiments were significantly different for cues with identical means but with outcome values sampled from different

distributions. A summary of the results obtained from comparisons across all four experiments are shown in Table 2.

Analysis of test ratings provided at the end of the experiment showed that ratings were based almost completely on the outcome mean alone, with the exception of small differences when comparing cues associated with the most starkly different distributions (e.g. bimodal vs normal). These results are reported in detail in Supplementary Materials. In summary, when asked to provide a single summary rating, participants aggregate their knowledge of the outcome values presented with each cue, and use the mean outcome value to inform their judgements, however predictions for individual trials made across training were sensitive not only to the mean value, but additionally informed by the underlying outcome distribution.

In the following section, we will describe the results from fitting participants' prediction ratings to the Simple Delta Model and Distributed Model, and provide a formal comparison of the two models in capturing participant behaviour during contingency learning.

Table 2

Summary of results from Kolmogorov-Smirnov (KS) test of equality. D statistic approaching 1 indicate large differences between distributions. Column values indicate outcome mean and each row is a contrast comparison involving outcome distribution.

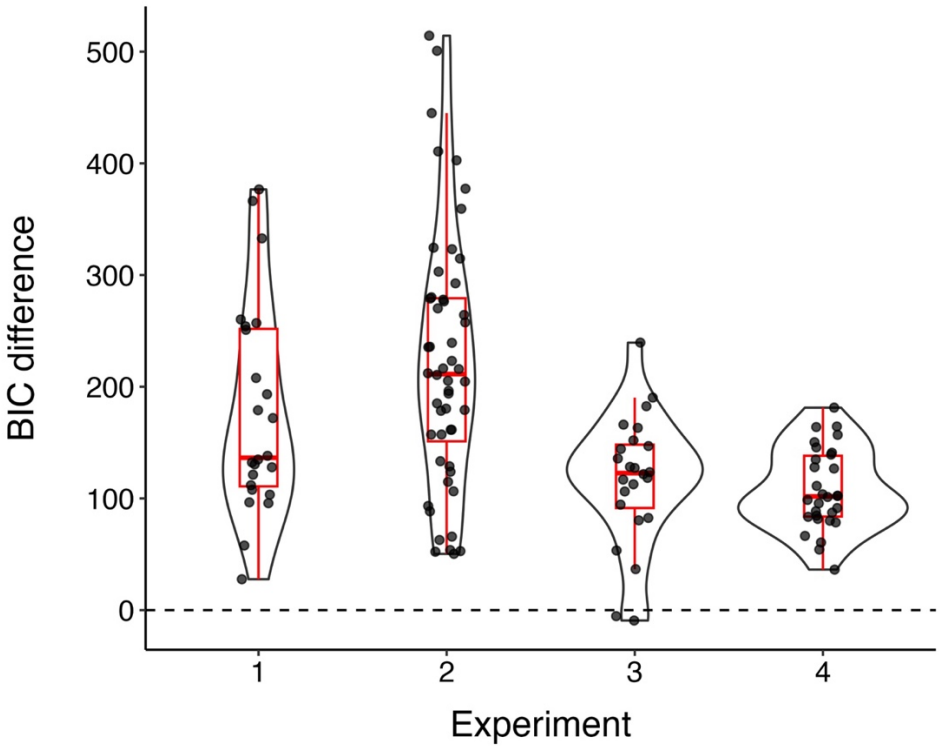
Experiment 1					
	25		50		75
Fixed vs Variable (average Uniform and Normal)	$D = .160$, $p = .001$		$D = .402$, $p < .001$		$D = .225$, $p < .001$
Uniform vs Normal	$D = .096$, $p = .220$		$D = .154$, $p = .007$		$D = .138$, $p = .021$
Experiment 2					
	15	30	50	70	85
Base-rate 30 vs Base-rate 50	$D = .083$, $p = .560$	$D = .072$, $p = .736$	$D = .133$, $p = .082$	$D = .161$, $p = .019$	$D = .150$, $p = .035$
Base-rate 30 vs Base-rate 70	$D = .126$, $p = .134$	$D = .095$, $p = .427$	$D = .100$, $p = .365$	$D = .156$, $p = .033$	$D = .167$, $p = .017$
Base-rate 50 vs Base-rate 70	$D = .074$, $p = .738$	$D = .128$, $p = .122$	$D = .128$, $p = .122$	$D = .223$, $p < .001$	$D = .218$, $p = .001$
Experiment 3					
	35			65	
Normal vs Positive Skew	$D = .085$, $p = .060$			$D = .046$, $p = .694$	
Normal vs Negative Skew	$D = .073$, $p = .156$			$D = .142$, $p < .001$	
Experiment 4					
	35			65	
Bimodal vs Normal (average Normal Narrow and Normal Wide)	$D = .083$, $p = .009$			$D = .042$, $p = .491$	
Normal Narrow vs Wide	$D = .085$, $p = .026$			$D = .073$ $p = .079$	

Model fit

For each participant, Bayesian Information Criterion (BIC) was calculated for the Simple Delta Model and the Distributed Model using best fitting parameters; a lower BIC value indicates better model fit. We examined the degree to which the Distributed Model fit the data better than the Simple Delta Model by computing a BIC difference score (ΔBIC) for each participant, $\Delta BIC = BIC_{delta} - BIC_{dist}$. A positive ΔBIC indicates better fit by the Distributed Model compared to the Simple Delta Model. Figure 2 is a plot of BIC difference scores for each participant in Experiments 1 - 4. It is noteworthy that almost all participants were better fit by the Distributed Model.

Figure 2

BIC difference score ($BIC_{delta} - BIC_{dist}$) calculated for each participant in Experiments 1 to 4. Positive BIC difference scores indicate better fit by the Distributed Model, whereas negative BIC difference scores suggest better fit by the Simple Delta Model. Boxplots illustrating the median, upper and lower quartile values are shown in red.



Finally, we transformed average ΔBIC for all participants in each experiment into a Bayes Factor, reporting the likelihood ratio of better model fit by the Distributed Model relative to the Simple Delta Model, using the formula described by Wagenmakers (2007): $BF_{10} = \exp(\Delta BIC / 2)$. Across all four experiments, there is strong evidence in favour of the Distributed Model; $BF_{10} > 10$ in all experiments (see Table 3). Overall, the Distributed Model provided a better fit of the data compared to the Simple Delta Model. These results suggest that a distributed architecture that enables representation of the underlying distribution of outcome values better captures how human participants are behaving as they make predictions about the outcome during learning. In the following section, we use best fitting parameters for each individual participant to illustrate the terminal associative strengths and discrete predictions generated by both models.

Table 3

Bayes Factor in favour of the Distributed model compared to the Simple Delta Model (BF_{10}) computed for each experiment. $BF_{10} > 10$ indicate strong evidence in favour of the Distributed model compared to Simple Delta Model.

Experiment	BF_{10}
1	2.13e+38
2	1.71e+48
3	2.57e+25
4	2.87e+23

Model output

Terminal associative strength

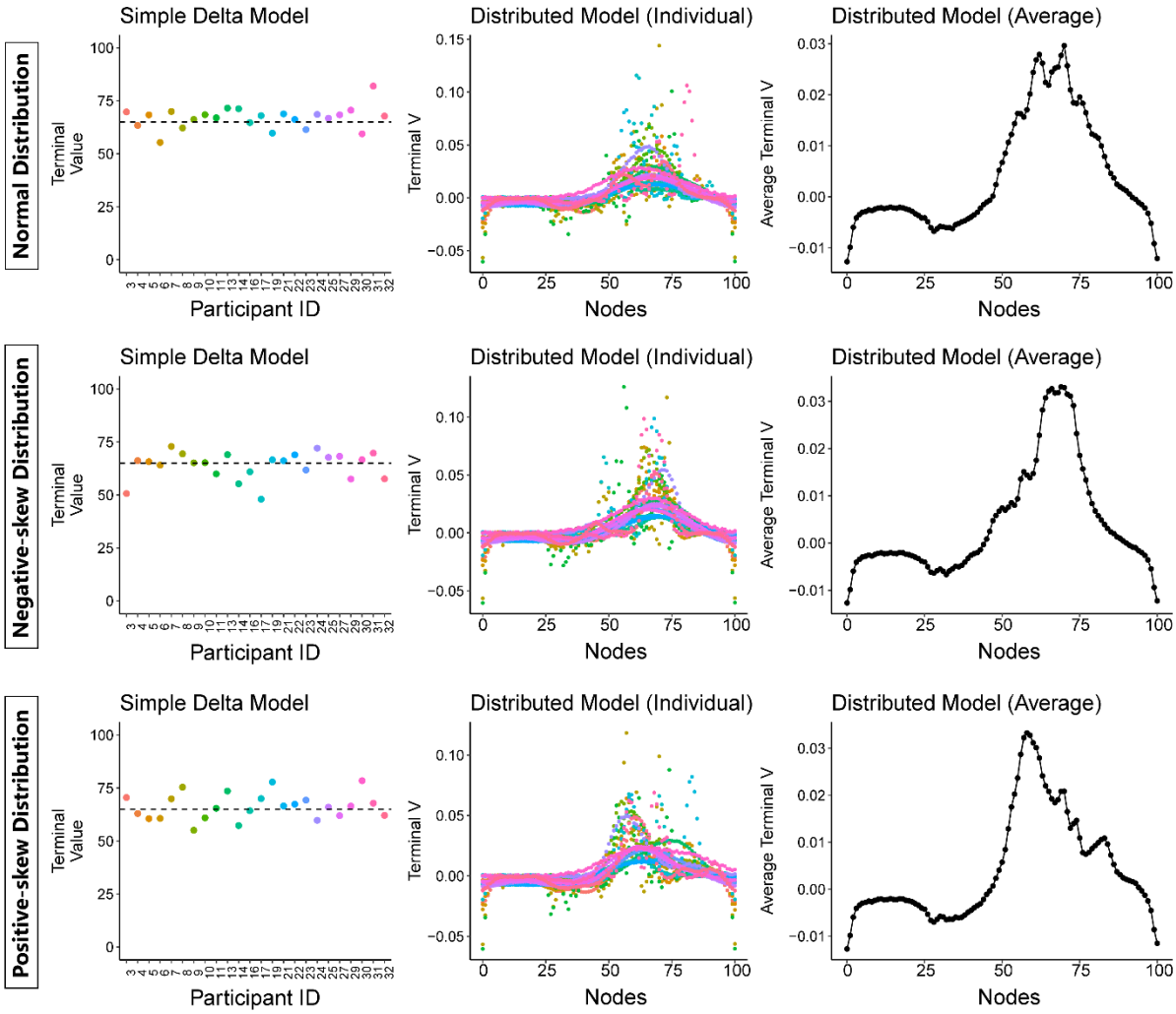
The ability for the Distributed Model to represent an associative connection between the cue and each level of the outcome is captured in the terminal associative strengths produced by the model. Unlike the Simple Delta Model which produces a single terminal

associative strength, the Distributed Model produces terminal associative strengths linking the cue to each outcome node. This produces a pattern of activation across the dimension that mirrors the underlying distribution (e.g., normal, positive and negative skew distributions). This is most obvious when associative values are averaged across all participants, as in the far right panel in Figure 3, but is also demonstrable at the individual participant level (middle panel in Figure 3).

Overall, terminal values generated by the model provide some indication that our models are performing as we expected. The Simple Delta Model accurately tracks overall outcome mean but is unable to distinguish between outcomes sampled from different-shaped distributions, whereas the Distributed Model shows sensitivity to the underlying distribution (Figure 3 middle and right panels).

Figure 3

Terminal associative values (ΣV; cue + context) generated by the Simple Delta and Distributed Model for three cues with outcome mean of 65, and outcomes sampled from a Normal Distribution (top row), Negatively-skewed Distribution (middle row), and Positively-skewed Distribution (bottom row). Terminal values were generated from models trained with sequences presented to participants in Experiment 3. Terminal values generated by the Simple Delta Model approximates the mean outcome value (dashed line), whereas the Distributed Model produces associative weights at each outcome node from 0 to 100. Different colour points represent model output from individual participant data generated by each model (far left and middle columns); average terminal values across all participants from the Distributed Model is depicted on the far right of each row.



Simulated model predictions

For both the Simple Delta and Distributed Model, we generated discrete predictions on each trial for every target cue based on Equation 2. This was completed following the trial sequence and outcomes presented to each individual participant, using best-fitting parameters for that participant generated from 100 simulated runs. Figure 4 shows density plots for the 6 target cues presented in Experiment 3, illustrating predictions made by participants (aggregated across all participants) overlayed with simulated predictions generated by the Simple Delta and Distributed model. There is greater overlap in the distribution of predictions generated by the Distributed model and participants' actual predictions compared to the Simple Delta model, particularly for cues whose outcomes were sampled from skewed

distributions. Predictions generated by the Simple Delta Model appear more diffused around the outcome mean. Figure 5 additionally shows an example of simulated predictions from a *single* participant in Experiment 3, plotted together with the participants' responses and observed outcome values. The purpose of both figures is to show that the distribution of predictions made by participants were sensitive to both mean and range of observed outcome values, and thus a model that was able to exploit the skewness of the outcome distribution, i.e., the Distributed model, was better able to generate predictions that more closely resembled participant responses.

Together, model outputs provide some assurance that the formulation of these models appropriately capture how a simple error-correction model will produce a terminal associative strength that approximates the outcome mean when the outcome has a numeric value, and selective activation of outcome nodes allows for learning about the shape of the underlying outcome distribution. Complete model output from these simulations can be found on the Open Science Framework (Chow et al., 2025).

Figure 4

Density plot of predictions made by participants and predictions generated by the Distributed and Simple Delta Model using best-fitting parameters for each participant, for each of the six target cues in Experiment 3.

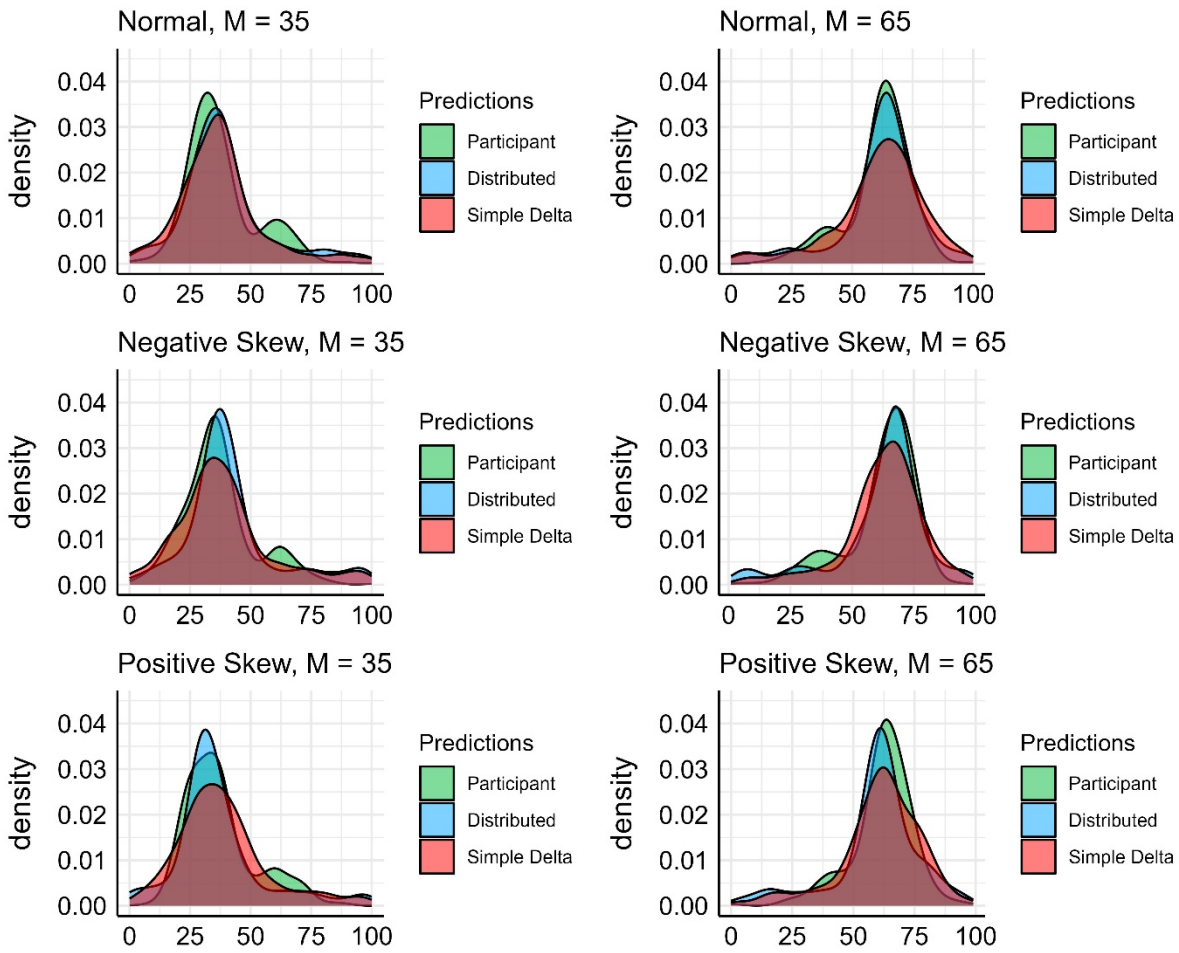
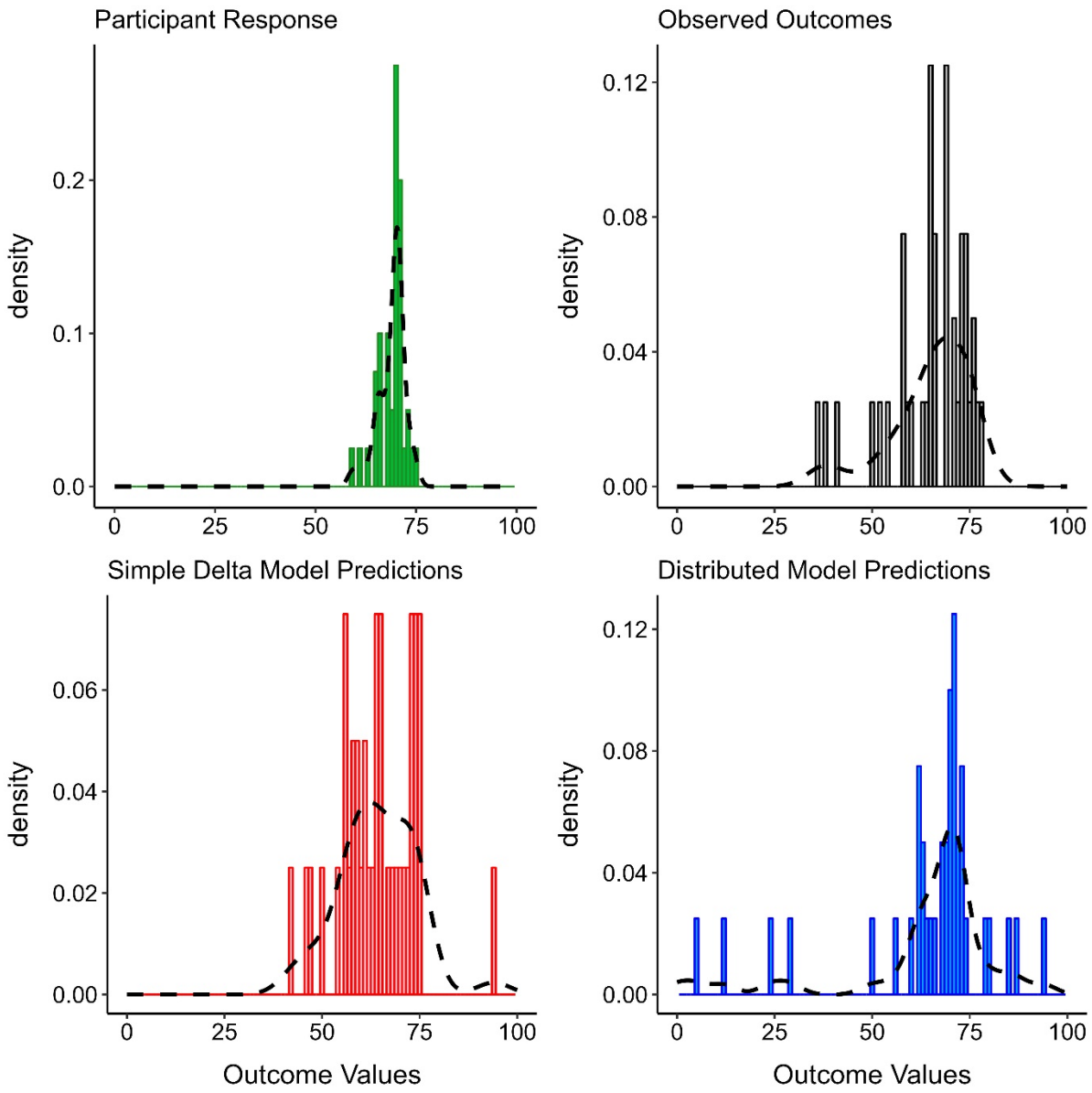


Figure 5

Density plot of (a) participant predictions, (b) observed outcomes, and simulated predictions from the (c) Simple Delta and (d) Distributed Model, for a cue with mean outcome = 65, and outcome values sampled from a negatively-skewed distribution. The illustrated data and model predictions are from a single participant in Experiment 3.



General Discussion

The goal of the present study was to determine whether the behaviour of human participants in a contingency learning task was better captured by a prediction error model

capable of representing a distributed network of outcome values, compared to a more conventional class of models that rely on a single point estimate. We first reported empirical data from human participants, and subsequently use participants' predictions from these experiments to fit the two models of interest. For almost all participants across four experiments, the Distributed Model provided a better fit to the data compared to the Simple Delta Model. Visual inspection of the simulated predictions indicate that the Distributed Model also produced predictions that more closely resembled the pattern of predictions produced by participants in the study. Thus, we show that a model of contingency learning which includes a distributed architecture is better at capturing how humans behave when making trial-by-trial predictions in a contingency learning task when the outcome has continuous values along a dimension.

The results of our experiments are compelling as they suggest that rather than just tracking an overall mean value, participants show some sensitivity to the continuous nature of the outcomes experienced, and are able to use this knowledge (to an extent) when making trial-by-trial predictions about outcomes associated with different cues. One explanation for these findings is that people can engage in distributional learning, and mentally represent outcomes in a probability distribution. This is consistent with a general assumption in cognitive science that people are able to spontaneously acquire knowledge about underlying probability distribution of events and use these learned frequencies to inform behaviour (Griffiths & Tenenbaum, 2006; Chater & Manning, 2006; Lindskog et al., 2021; Schlegelmilch & von Helversen, 2020). However, there is increasing debate about whether people are able to learn and reproduce the structure of underlying distributions: studies that explicitly interrogate distributional knowledge have failed to find evidence of accurate distributional learning when participants were asked to reproduce the distribution of spatial events (Tran et al., 2017), and require extensive guidance (Mason et al., 2024) or constraints

to task environment (Brehmer, 1980; Szollosi et al., 2023) in order to be accurate in their reconstruction. A less extreme and perhaps more plausible explanation of the present findings is that participants are not representing complete distributions, but they are sensitive to the frequency of different outcome values occurring for each cue type. This allows them to develop strategies or rules to distinguish between the cues, reflected in the differences in their pattern of predictions. This latter account is no less impressive, given the complexity of the present experiments where participants were sometimes presented with up to 13 different cues (intermixed) that differ in overall mean and outcome distribution. Under this account, participants are, at the very least, building sufficiently distinct representations or rules about each cue-outcome relationship that allow them to make predictions different from a similar cue whose outcomes are sampled from another distribution (see also Mason et al., 2024).

One alternate explanation of our findings is that participants are not learning about the outcomes associated with each cue *at all*, but are instead engaging in strategic matching behaviour by making predictions that match the outcome value experienced on the previous trial for that cue type. To account for this possibility, we compared the likelihood of such a "matching model" –using the outcome on trial n as a predictor of participants' predictions on trial $n+1$ for a given cue—to a "non-matching model", where responses are based on the average observed outcome up to trial n (i.e., learning). Overall, we found no convincing evidence that participants were systematically engaging in such a matching strategy (see Supplementary Materials: Additional Analysis). Indeed, when we correlated individual participant predictions made on trial $n+1$ to the observed outcome on trial n, we failed to find systematic evidence of a statistically significant correlation across participants and cue type, suggesting that participants' pattern of predictions cannot be fully explained by a simple matching strategy. A visual summary of this analysis is presented in Figure 6; we illustrate only the correlations from Experiment 3 where there may be increased incentive to use a

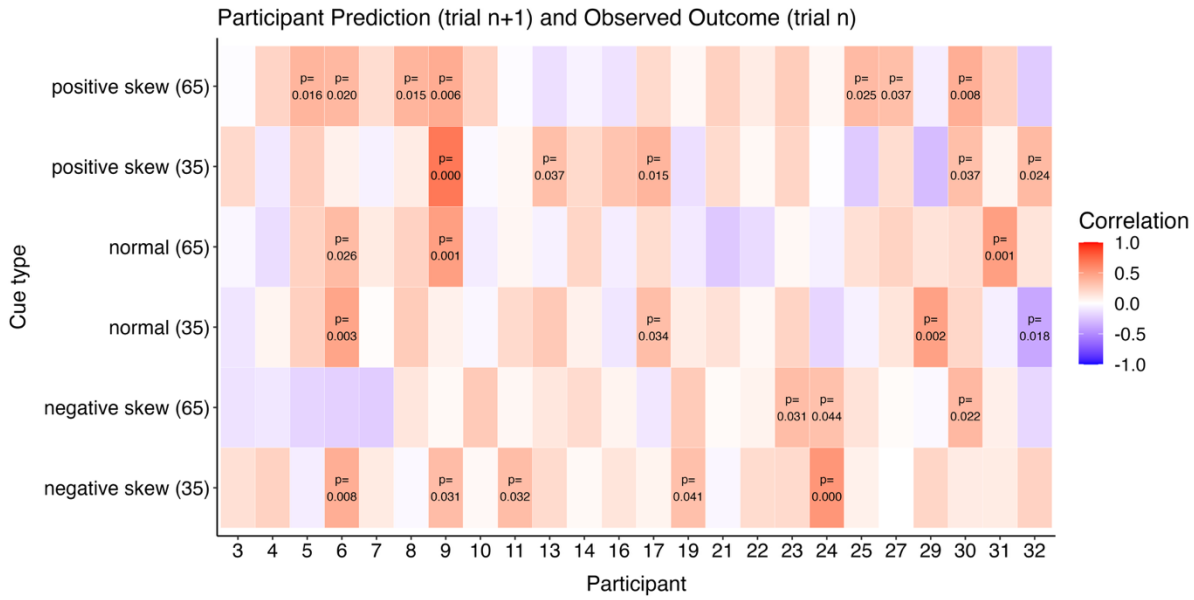
matching strategy due to greater similarity in the outcome distributions. We find that most participants do not systematically engage in matching behaviour when making predictions. A similar analysis comparing model predictions generated based on best-fitting parameters for the Simple Delta and Distributed Model for each individual participant showed that the Simple Delta Model substantially over-estimates the positive correlation, generating predictions that resemble matching behaviour significantly more so than participants' actual responses (see Figure S6 in Supplementary Materials); this pattern was not observed for predictions generated by the Distributed Model.

Taken together, we propose that when participants were presented with continuous outcomes in a contingency learning task, their pattern of predictions across learning goes beyond matching the outcome observed on the previous trial and a simple learning strategy that tracks the overall mean. Results from model fit and model comparison additionally provide good evidence in support of the idea that participants' behaviours are not simply a result of 'noisy' responding, but reflect a more sophisticated learning process that allows them to distinguish between similar cues by leveraging features of the underlying outcome distribution.

Figure 6

Correlation matrix illustrating the strength of the correlation between participants' predictions on trial n+1 *and the observed outcome on trial* n *for each cue type in Experiment 3. Each column represents data from an individual participant, and each row consists of one of the target cues presented in the study; row labels indicate the distribution type with the outcome mean in parentheses. Cells that appear more red in colour indicate increasingly positive correlation values, whereas bluer cells indicate increasingly negative correlation values, with colours close to white indicating little to no correlation. P-values for statistically*

significant correlations are included in the relevant cells, whereas correlations which did not reach statistical significance are not shown for visual clarity.



Measurements of learning

To our knowledge, the present experiments are the first to investigate learning about continuous events in a contingency learning experiment using predictions made *during* learning. In contrast, studies focused on eliciting distributional knowledge have focused on participants' ability to accurately reproduce the distribution of experienced events after the fact (e.g., Tran et al., 2017; Mason et al., 2024). Causal learning studies similarly rely on judgements made at the end of the experiment to determine what participants have learned about the cue-outcome relationship (e.g., Shanks & Dickinson, 1988; Wasserman et al., 1993; Vadillo et al., 2005). The appeal of measuring participants' learning at the end of the experiment is clear: it provides an opportunity for participants to integrate and report the acquired knowledge without interference from ongoing learning processes. This is pertinent in circumstances where the goal of the study is to determine *what* participants have learned (e.g., underlying task structure in distributional learning or the cue-outcome contingency in causal learning experiments), rather than *how* participants may be learning. Indeed, post-

experimental estimation tests are often limited as they do not require participants to reproduce the distribution of events, but rather encourage integration across the entire experiment (Collins & Shanks, 2002). In the present experiments, we similarly find an insensitivity of test ratings (average outcome, outcome mode, causal ratings) to the shape of the underlying distribution, despite differences in the pattern of predictions made during training.

In the present study, outcome representation is inferred through the predictions made by participants for different cues across the learning phase: we present participants between 8 and 13 different cues in an intermixed fashion, and they are required to make an outcome prediction on each trial. Participants were never told that they were being assessed on their distributional knowledge, and they were not constrained in how they could respond on any given trial—participants could, in theory, report the outcome mean on every trial, resembling predictions by the Simple Delta Model—thus eliminating any biases that might emerge from using a more explicit test of distributional knowledge, such as strong priors about normality and unimodality, as well as allowing for the influence of specific semantic knowledge like the proportion of people who benefit or do not benefit from treatment. In addition, the task is complex as it involves tracking multiple cues simultaneously. This complexity requires participants to use the outcomes to discriminate between different cues. This feature of the design may be pertinent for providing the necessary scaffolding for participants to learn the structure of the task environment: participants' attention may be drawn towards differences in outcome values associated with each cue in order to distinguish between them, thus encouraging representations that are sensitive to that distinction. This possibility is consistent with the idea that highlighting aspects of the task important for successful distributional learning increases performance accuracy (Szollosi & Newell, 2020), and evidence from the

multi-tasking literature showing greater separation of task representations when two tasks were performed in an intermixed fashion (Garner & Dux, 2015).

Although the use of prediction responses is well suited for our present purposes, we are cautious not to overclaim the benefit of this method for studying distributional learning *per se*, since participants' pattern of predictions were still significantly different from the actual observed outcome distribution (see Supplementary Materials), suggesting imperfect distributional knowledge despite the task design encouraging discrimination between cues. Nevertheless, the present method of measuring outcome representation is well suited to test the Distributed Model against a Simple Delta Model, and provides a good test bed for determining whether people are capable of more sophisticated means of encoding outcomes in a contingency learning task with continuous outcomes. To this end, the present findings suggest there is much to be gained from focusing on the pattern of predictions made across the course of learning. These predictions capture differences that are lost when asked to provide a single point estimate at test, at least when the research question is focused on understanding how participants are learning in a noisy and complex environment.

Applications to human contingency learning

We have shown that participants' predictions across learning are not merely a result of noisy approximations of observed outcomes, but instead reflect a degree of sophistication in encoding that preserves their continuous and distributed nature. Consequently, a model capable of representing continuous outcomes in a distributed fashion provides a more accurate representation of how participants engage with and learn about continuous outcomes during contingency learning. A notable advantage of the Distributed Model approach is its ability to capture variability in responding without explicit representation of uncertainty—a departure from most traditional models of decision-making. Most models require researchers

to specify uncertainty parameters, which may introduce arbitrary constraints or assumptions about how individuals perceive and respond to variability. In contrast, the Distributed Model allows for the emergence of variable responding without such a parameter, suggesting that the capacity to respond to uncertainty emerges naturally when there is sufficient complexity in how the model represents what is being learned, rather than a product of a distinct cognitive mechanism that needs to be explicitly represented in the model.

So far, we have presented two strengths of the Distributed Model for modelling contingency learning with continuous outcomes: first, it is a better model of what people are actually learning in these tasks compared to a Simple Delta Model, and second, it does not require additional assumptions about the role of uncertainty in learning. A third notable advantage of the distributional approach is that it presents a useful tool for understanding the learning processes underlying (biased) causal inference. This third advantage becomes particularly evident when considering how people reason from continuous rather than discrete events. The potential for ambiguity when interpreting continuous outcomes creates more opportunities for cognitive biases to shape both what is learned and the causal beliefs that follow.

The ability for people to learn about continuous outcomes is largely ignored in the contingency learning literature. Most prominent theories of causal learning are based on the assumption that people represent their experiences in a discrete way, where the cue and outcomes are either present or absent. The frequency of these discrete events (e.g., patient takes a medicine [cue present] and recovers from their illness [outcome present]) are subsequently used to form a causal judgement (e.g., delta p metric, Allan, 1980). Indeed, one interpretation of our findings is to assume that the different pattern of predictions generated for different cues are simply a product of participants applying *discrete* categorisation rules without appealing to the shape of the distribution—e.g., treatment A tends to have outcomes

around 50 but sometimes higher than 50. Nevertheless, the question of *how* we might classify noisy events in the real world into discrete categories is *also* largely ignored by existing covariation-based models of causal inference (Ahn et al., 2007; Marsh & Ahn, 2009). Take for example, the scenario used in the present studies. Participants were told that high-value outcomes indicate greater patient recovery, with the extreme ends of the scale indicating no recovery (outcome = 0) or full recovery (outcome = 100). When participants experience extremely high or low outcome values, categorisation of the outcome into patient recovered or not recovered may seem obvious, however what is less obvious is how to classify an outcome value around the middle of the scale (e.g., 50/100). These outcomes are ambiguous relative to the goals of the learner, and may present a challenge to contingency models that rely on discrete categorisation of events for causal inference.

We propose that the interpretation and classification of continuous and potentially ambiguous events in learning is a flexible process shaped by several factors, including the underlying probability distribution and the learner's prior beliefs. This flexibility in classification allows for a greater role of prior beliefs in shaping how individuals learn from variable and dimensional outcomes (see also Marsh & Ahn, 2009; Blanco et al., 2020b), and this in turn influences causal inference. Variability in learning may be especially pronounced under zero contingency scenarios, where there is no objective causal relationship between cue and outcome. It has previously been shown that when participants are uncertain about the causal status of a cue, like in zero contingency scenarios, there is a tendency to "explain away" conflicting information in ways that allow them to preserve their pre-existing beliefs (Chow et al., 2024; Spicer et al., 2020; Spicer et al., 2022). Thus, one possibility is that greater flexibility in processing continuous outcomes may allow factors known to increase causal illusions, such as prior beliefs, to exert even more influence, distorting individuals' learning of outcome distributions in ways that reinforce pre-existing beliefs. To illustrate, if

the learner has strong assumptions about the efficacy of treatment at causing recovery, they may be more likely to encode high outcome values on trials where the patient has been administered treatment for their illness than on trials where no treatment was provided, biasing their encoding and representation of the outcome distribution for treatment trials and no treatment trials in opposing ways. Thus, the Distributed Model may be a useful tool to examine how prior beliefs influence learning about distributions of continuous events in zero contingency conditions. This could provide insights into the formation and maintenance of false beliefs in real-world scenarios with variable and noisy outcomes.

Conclusions

In summary, we have shown that when presented with a continuous outcome, participants in a contingency learning task produced a pattern of responding that suggested sensitivity to the underlying outcome distribution. We modelled the results from four empirical experiments and found that model fits were significantly better for virtually all participants using a Distributed Model which has the capacity to represent a network of associative weights across the entire outcome dimension, compared to a Simple Delta Model which only tracks the overall mean. Contrary to most conventional models of learning that simplifies the content of what is learned, these findings suggest that variability in responding is not merely a product of noise, but that people are capable of more sophisticated encoding processes that preserve their continuous and variable nature. These findings help advance our understanding of how people learn from experienced events in a complex and noisy environment, and provides a framework for thinking about causal inference in the real world.

Data Availability Statement

All experimental data files are available from the Open Science Framework database (https://osf.io/f4wqd/). All code files are available from GitHub repository at DOI: https://doi.org/10.5281/zenodo.15636868.

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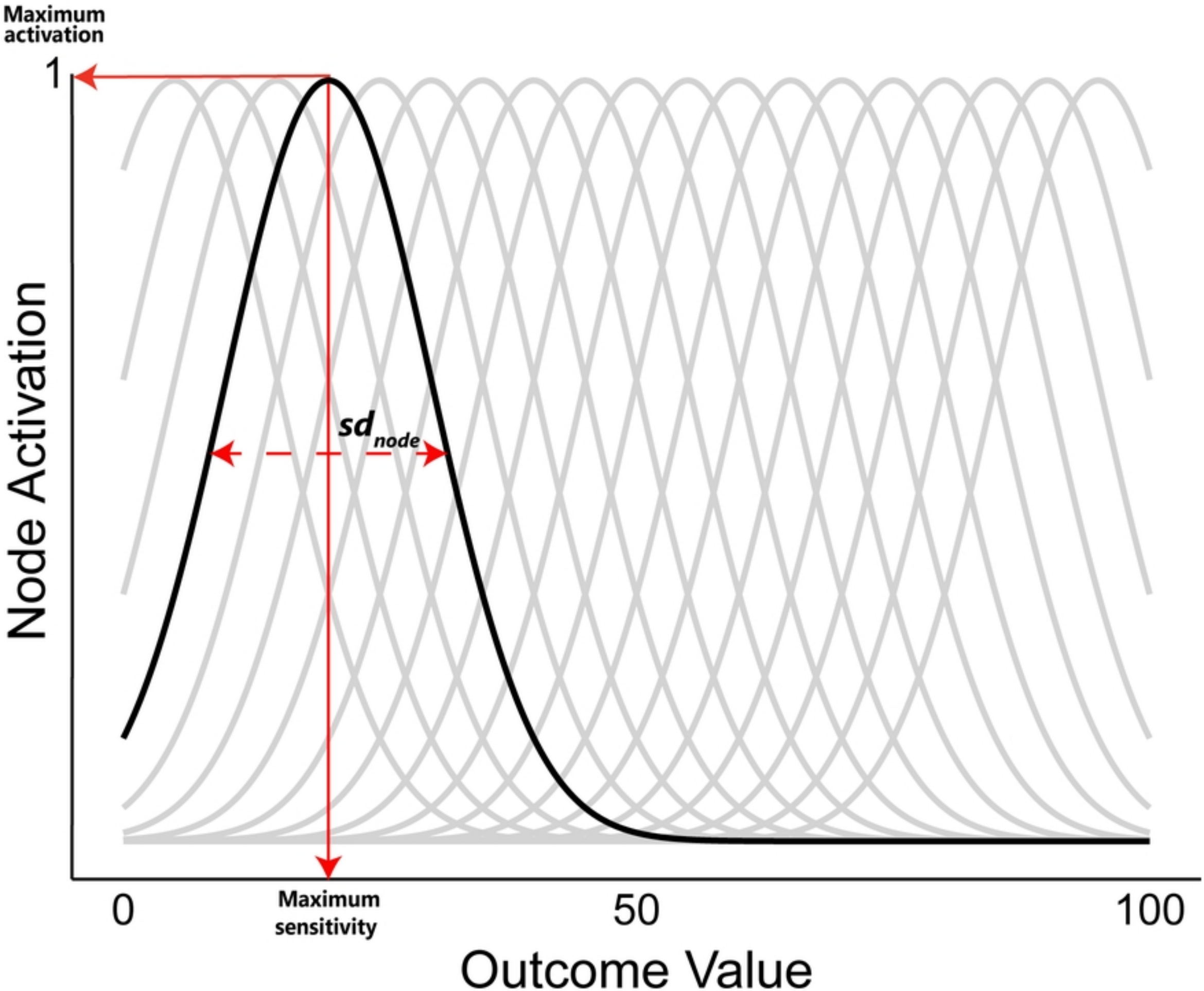
Maximum activation
1
Node Activation
sd_{node}
0
Maximum sensitivity
50
100
Outcome Value

Figure1

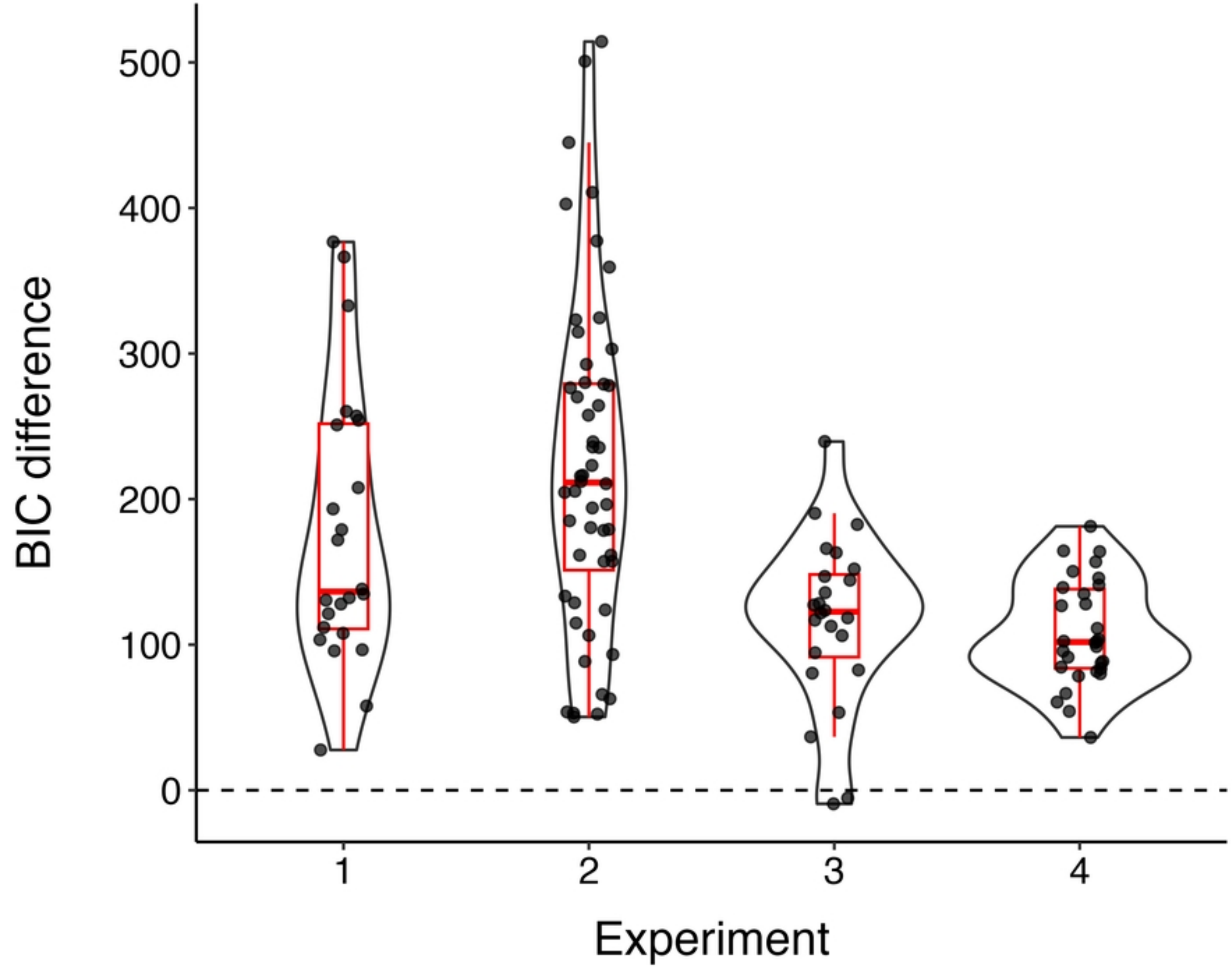

Figure2

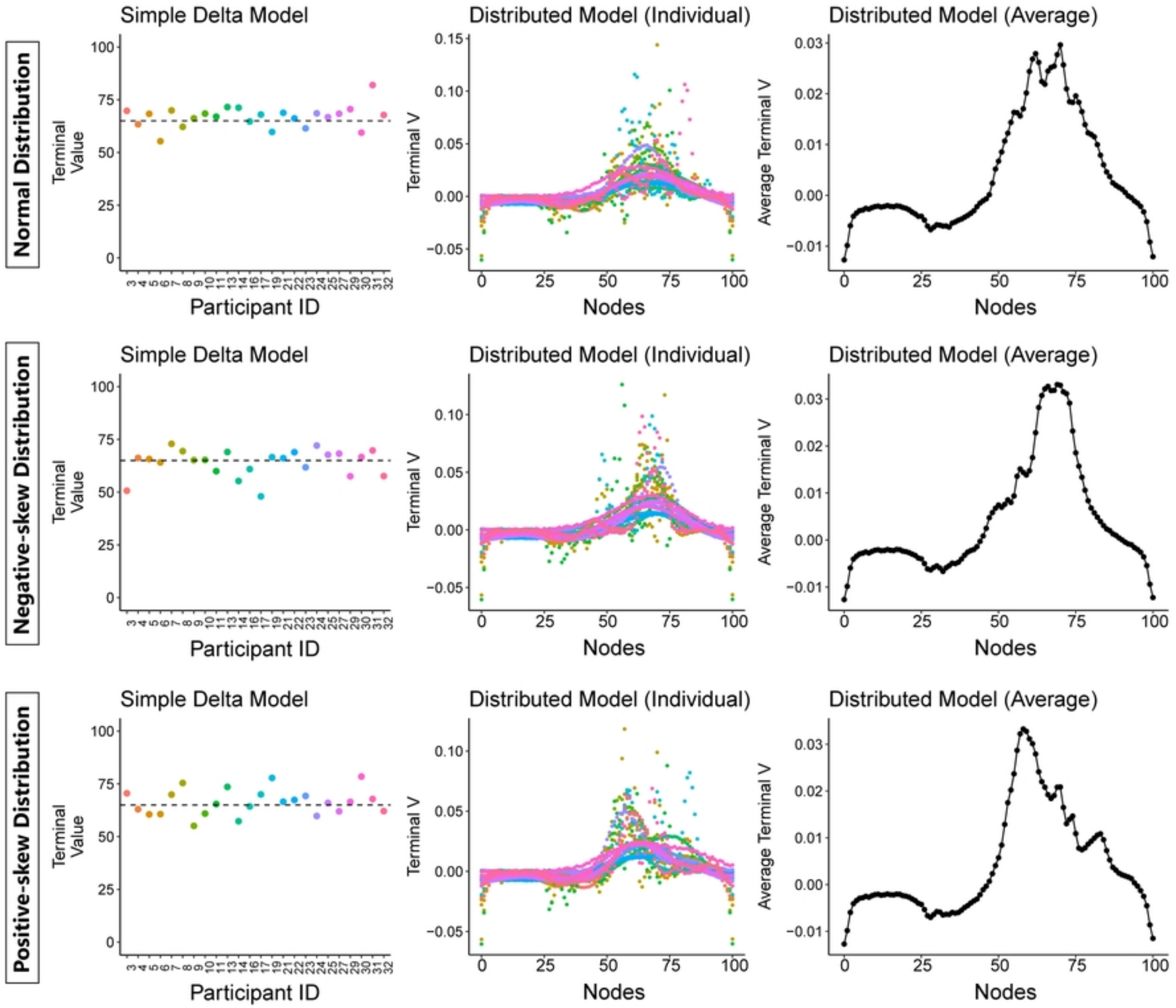
Normal Distribution
Negative-skew Distribution
Positive-skew Distribution
Simple Delta Model
Distributed Model (Individual)
Distributed Model (Average)
Terminal Value
Participant ID
Terminal V
Average Terminal V
Nodes

Figure3

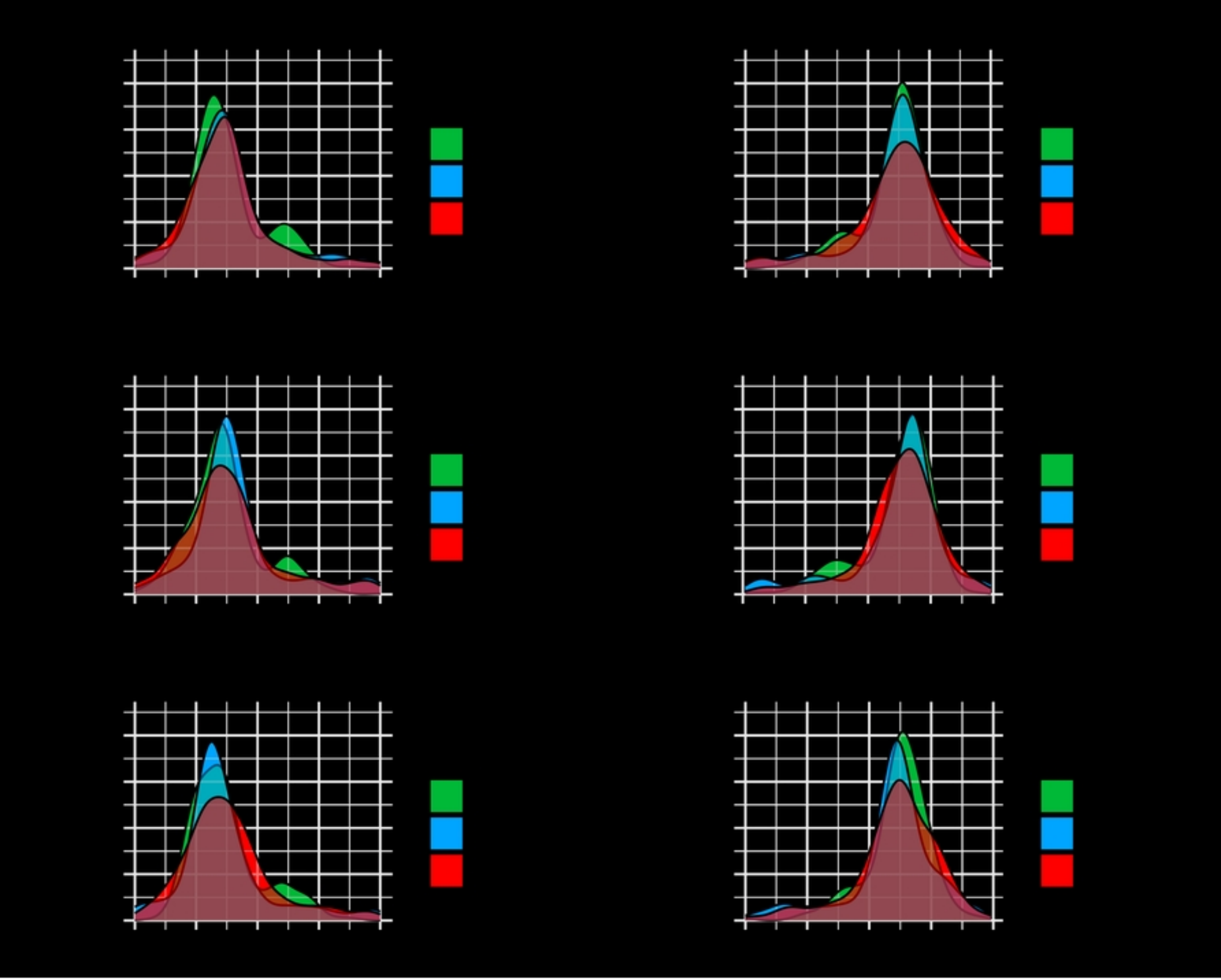

Figure4

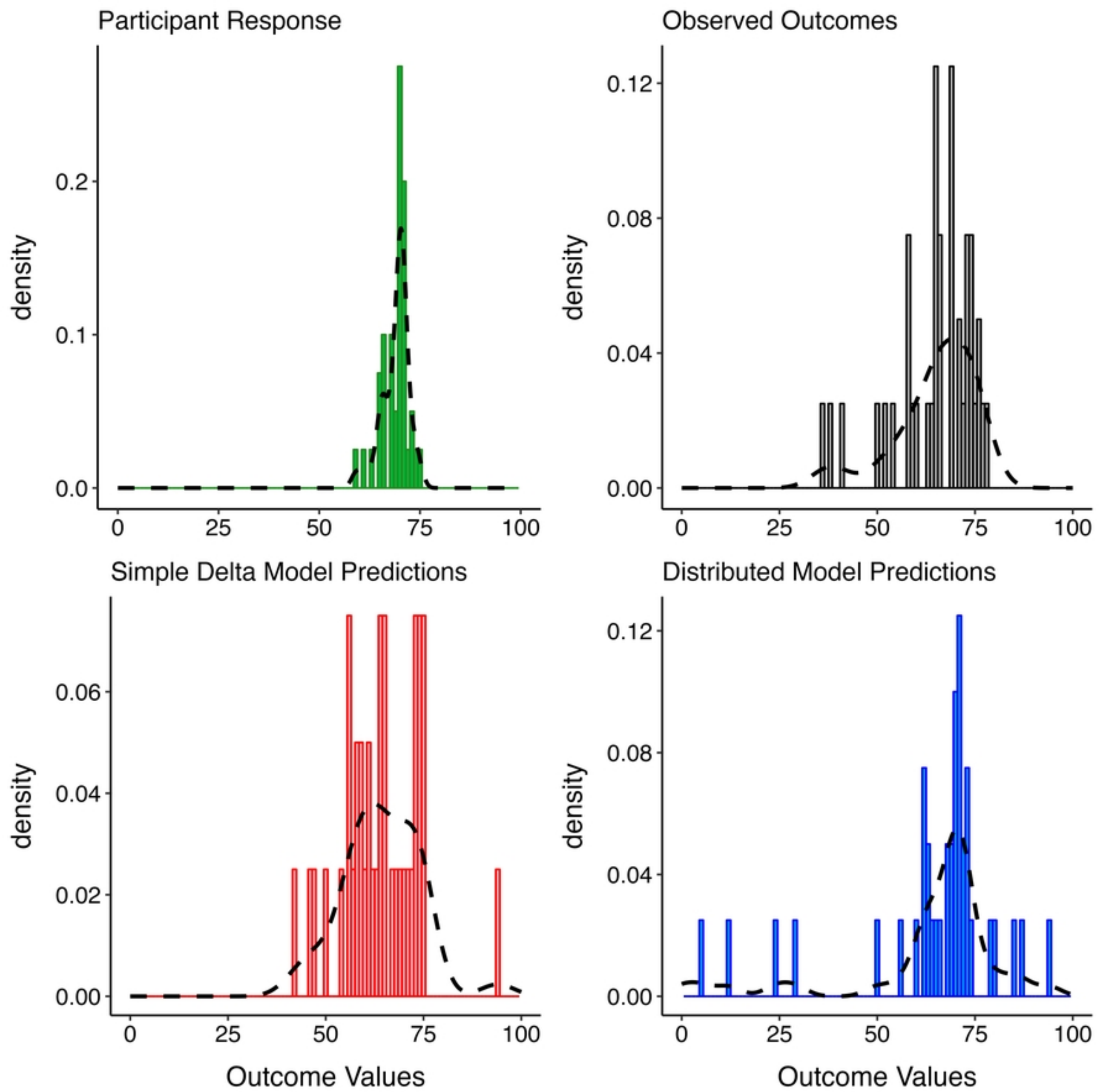
Participant Response
Observed Outcomes
density
0.0
0.1
0.2
0.00
0.04
0.08
0.12
0
25
50
75
100
Simple Delta Model Predictions
Distributed Model Predictions
0.00
0.02
0.04
0.06
Outcome Values

Figure5

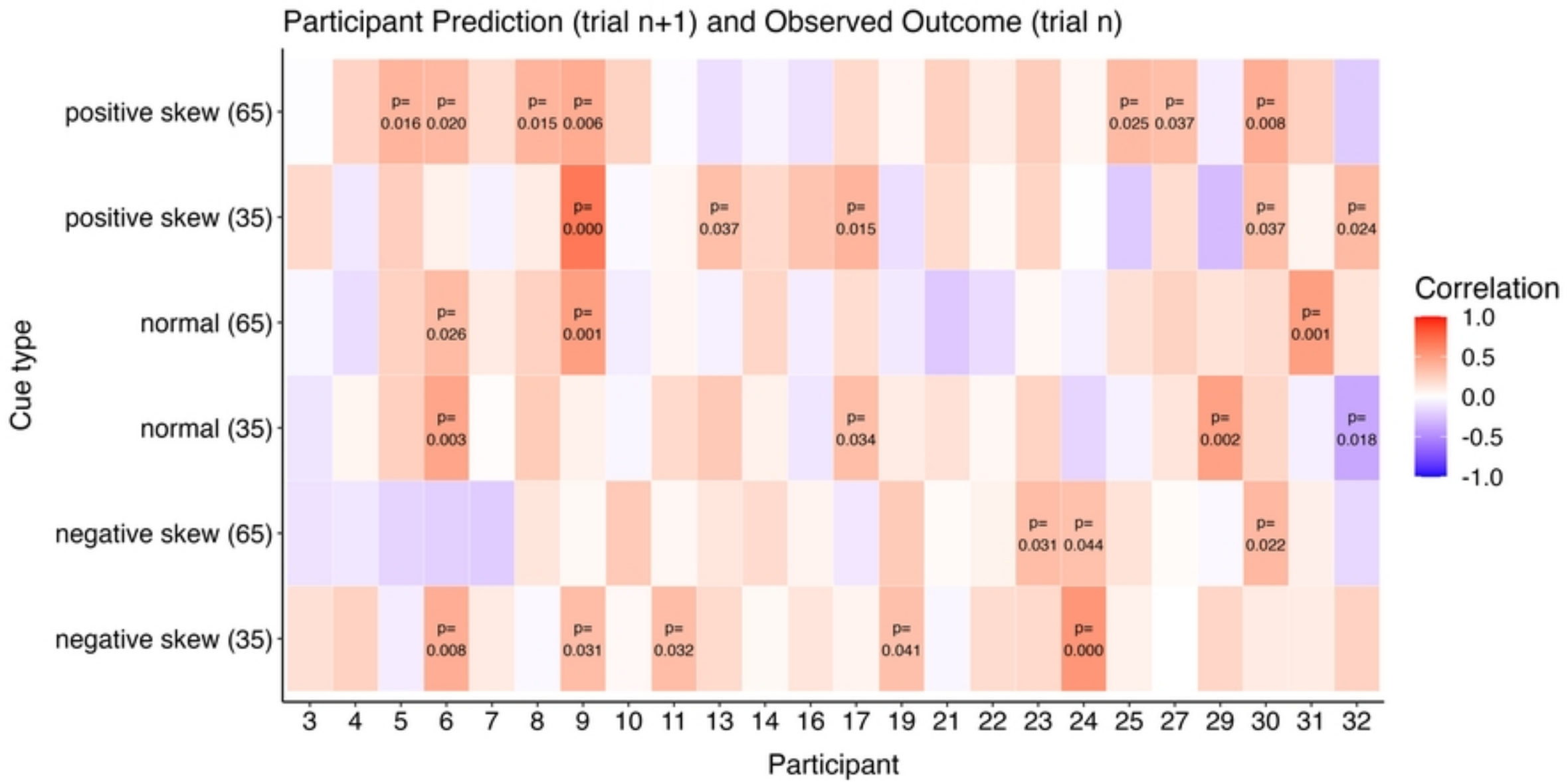
Participant Prediction (trial n+1) and Observed Outcome (trial n)
Cue type
positive skew (65)
positive skew (35)
normal (65)
normal (35)
negative skew (65)
negative skew (35)
p= 0.016
p= 0.020
p= 0.015
p= 0.006
p= 0.025
p= 0.037
p= 0.008
p= 0.000
p= 0.037
p= 0.015
p= 0.037
p= 0.024
p= 0.026
p= 0.001
p= 0.001
p= 0.003
p= 0.034
p= 0.002
p= 0.018
p= 0.031
p= 0.044
p= 0.022
p= 0.008
p= 0.031
p= 0.032
p= 0.041
p= 0.000
3 4 5 6 7 8 9 10 11 13 14 16 17 19 21 22 23 24 25 27 29 30 31 32
Participant
Correlation
1.0
0.5
0.0
-0.5
-1.0

Figure6