A Parietal Memory Strength Signal Linked to Evidence Accumulation in Recognition Decisions

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Abstract

Recognising objects from memory requires an integration of sensory and mnemonic information. This process has been theorised to occur via a stochastic evidence accumulation process implemented within the parietal cortex. Here, we provide evidence for this type of mnemonic accumulator using a combination of electroencephalographic (EEG) recordings and generative computational modelling. We recorded EEG from participants making recognition judgements based on either studied or novel words. We first demonstrate that the widely studied parietal Late Positive event-related potential Component (LPC) reflects a dynamic decision-making variable occurring prior to the recognition judgments. By fitting Diffusion Decision Models to neural and behavioural data using specialised neural network tools, we then show that the LPC amplitudes are selectively associated with the rate of evidence accumulation, signifying memory strength. This association was stronger for previously studied words compared to novel words. Our findings therefore recast the LPC as a neural signature of mnemonic strength in evidence accumulation for recognition memory judgments, especially for memorised objects.

A central question in memory research concerns the neural signatures underlying memory retrieval[1-3]. Decades of research using electroencephalography (EEG) recorded during recognition memory tasks has consistently reported differences in event-related potential (ERP) components across recognized and novel items, such as the Late Positive Component (LPC) measured at parietal electrodes[4-6]. The LPC has been widely assumed to reflect a recollection process supporting the retrieval of detailed episodic information[7-9]. LPC amplitudes are more positive-going for previously-seen compared to novel items[7] (termed the old/new effect), decisions made with high confidence[10,11], self-reported recollection[12,13], and accurate source memory judgements[14,15]. The LPC has also been linked to blood oxygenation level dependent (BOLD) signals and lesions in the lateral parietal cortex[16,17], a region of brain that was shown to be critical to episodic memory and retrieval of event details[18-21]. Similar to LPC amplitudes, BOLD signals in left parietal regions are greater for 'old' than 'new' judgments and for retrieval of memory details[3,22-24]. However, despite research endorsing LPC as a correlate of recollection, it has not been formally tested within a mechanistic framework or linked to latent variables in computational models of recognition memory.

An alternative, plausible view of the LPC is derived from the mnemonic accumulator hypothesis relating to the parietal lobe. According to this hypothesis, parietal lobe neural activity during memory retrieval reflects the integration of mnemonic information from distributed brain regions, with the mnemonic evidence accumulating continuously over time[20,25,26]. Consistent with this view, BOLD signals in parietal regions were found to track the likelihood of items recognised as 'old'[27,28], and alpha band EEG power in the intra-parietal sulcus was reported to demonstrate features of an evidence accumulation signal[29]. This framework is often analogously compared to the classic Diffusion Decision Model (DDM)[30], which describes mnemonic evidence accumulating via a stochastic diffusion process toward 'old' or 'new' decision boundaries at an average speed represented by a drift rate parameter (Fig. 1a), simultaneously accounting for choices and response times (RTs). However, findings derived from previous functional magnetic resonance imaging (fMRI) studies were seldomly incorporated in evidence accumulation models for theoretical testing—a

limitation often attributed to the slow time course of BOLD signal fluctuations impeding detection of rapid decision dynamics[22,26].

Recent work has highlighted the advantages of using high temporal resolution EEG to study neural correlates of evidence accumulation processes[31-33]. In perceptual decision-making tasks, a prominent centro-parietal positivity (CPP) exhibits a gradual, positive build-up that peaks around the time of response. The CPP has been proposed as a domain-general marker of evidence accumulation, independent of motor preparation[34,35]. The rate of CPP amplitude build-up has been formally linked to drift rates in the DDM[34,36]. Pre-response CPP amplitudes have instead been associated with the extent of evidence accumulation at the time of a decision[31,37].

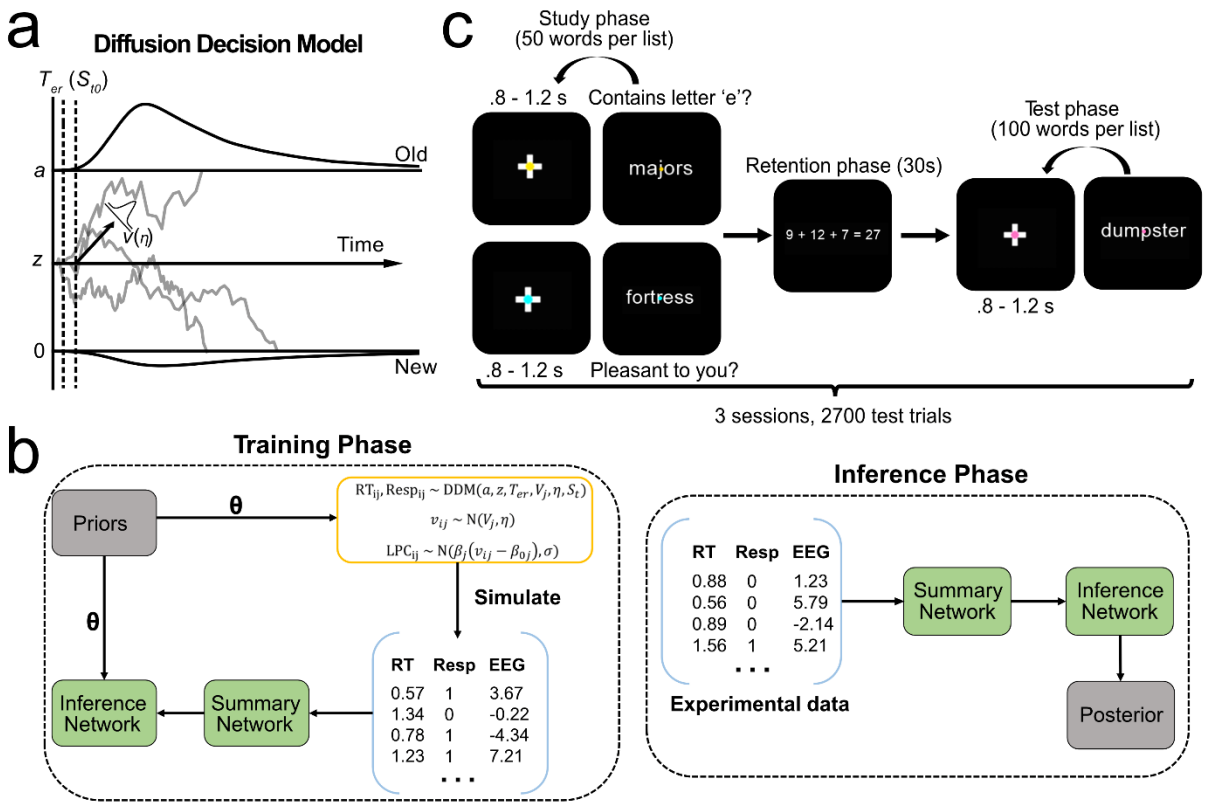
Importantly, the LPC shows similar characteristics to the CPP, suggesting that it may also reflect evidence accumulation processes. Specifically, LPC amplitudes scale positively with decision accuracy and confidence[7,11], and LPC amplitudes are also more positive-going for trials with faster RTs[38]. Within the DDM framework, greater memory strength (i.e., a higher drift rate) predicts an increased likelihood of 'old' responses, faster RTs, and higher confidence[39,40]. We recently demonstrated that the LPC, like the CPP, is time-locked to the response rather than occurring within the conventional post-stimulus measurement window (400–800 ms after probe onset)[41]. We observed that the LPC ramped up and peaked just prior to the behavioural response, indicating a process that is closely time-locked to the decision (such as mnemonic evidence accumulation) during memory retrieval tasks.

Accordingly, we investigated how features of the LPC relate to parameters in evidence accumulation models, and whether the function of the LPC can be reconceptualised within this modelling framework. Whilst earlier studies have typically examined these relationships by correlating neural signals with DDM parameters at the group, individual or trial level[38,42-44], we adopted a joint modelling framework that enables behaviour and neural data to be simultaneously predicted from the same underlying decision process[45]. Specifically, LPC amplitudes were predicted via a linking function with drift rate on each trial, with explicit modelling of additional noise commonly present in EEG signals—an aspect often overlooked in previous joint modelling implementations[46,47]. Importantly, this framework allowed us to disentangle

signal-driven contributions to the LPC from noise, offering a novel means of characterising the strength of the relationship between neural signals and evidence accumulation dynamics.

A critical technical limitation of fitting such models using conventional techniques is the need to develop closed-form likelihood functions for each unique implementation. Sometimes these models may be intractable. To address this limitation, we used a novel neural network method (BayesFlow) for amortised, likelihood-free inference[48]. This method approximates the likelihood function via neural likelihood estimation, whereby specialised neural networks are trained to map summary statistics from model predictions to parameter values sampled from prior distributions (Fig. 1b). Beyond providing a solution to the intractability problem, this approach enables efficient posterior parameter estimation and has been successfully applied across disciplines[49-52].

Figure 1. Diffusion Decision Model parameterisation, BayesFlow modelling pipeline and experimental structure.



Note. **a**. Depiction of the Diffusion Decision Model for recognition memory tasks and conventional model parameters. Following word presentation, evidence accumulates in a stochastic manner toward the 'old' or 'new' decision boundary. v = drift rate, a = boundary separation, z = starting point, η = across-trial drift rate variability, T_{er} = non-decision time, S_t = non-decision time variability. **b**. An illustration of the BayesFlow workflow with a modified DDM for jointly predicting behavioural and neural data. Training was done simultaneously for the inference and summary networks by simulating the model with sets of parameters drawn from prior distributions. During the inference phase, the summary statistics from experimental data were passed to the inference network to produce posterior model parameter estimates. **c**. Trial diagram for the recognition memory task. Participants were presented with a list of words during the study phase. They were required to judge whether each word contained the letter 'e' (perceptual task) or whether the word was pleasant (semantic task). The task was indicated by the colour of the fixation stimulus. Participants then answered mathematical questions during a retention phase and then viewed a list of test items. They reported whether the item was 'old' or 'new' via speeded keypress.

In the current study, we recorded EEG during a recognition memory task (Fig. 1c) and included two manipulations of memory performance – depth of processing (deep vs. shallow)[53] and word frequency[54]. Deeply/semantically encoded items are associated with better recognition performance and more positive-going LPC amplitudes than shallowly/perceptually encoded items[55-57]. Compared to high frequency words, low frequency words were observed to produce high hit (correct recognition) rates and low false alarm rates[58,59]. Regarding the LPC, more positive-going amplitudes and a stronger old/new effect have been observed for low compared to high frequency words[60-65].

We first replicated our previous finding that the LPC is time-locked to the response, and characterised the relationship between LPC amplitudes, decision accuracy, and RTs. Using our Bayesflow joint modelling pipeline, we assessed the strength of associations between ERPs and key model parameters across targets and lures, and identified a link between LPC amplitude and drift rate. We further examined how this association unfolds over time leading up to the keypress response. Our

extensive control analyses show that this association is specific to the LPC and drift rate rather than other ERP components or other model parameters. Taken together, our findings indicate that the LPC exhibits key characteristics that are consistent with a mnemonic strength variable in evidence accumulation.

Results

We recorded scalp EEG from 24 participants who each completed up to three sessions of a recognition memory task (Fig. 1c), with 900 test trials per session (maximum 2,700 trials per participant, 18 completed 3 sessions, 2 completed 2 sessions, 4 completed 1 session). Overall, participants performed well in the recognition task (mean hit rate = 64%, SD = 15%, mean correct rejection rate = 84%, SD = 13%). Compared to shallowly processed items, deeply processed items were associated with higher hit rates, mean difference = 16.5%, $t(23) = 4.83$, $p < 0.001$. Compared to high frequency words, low word frequency words had higher hit rates for targets, mean difference = 6.8%, $t(23) = 3.68$, $p = 0.001$, and higher correct rejection rates for lures, mean difference = 5.0%, $t(23) = 4.21$, $p < 0.001$. Our results were in line with existing work using these manipulations[53,54,66].

The LPC is time-locked to the recognition memory decision

By comparing hits and correct rejections, we first replicated the conventional stimulus-locked old/new LPC effect (Fig. 2a)[7]. The scalp distribution of the old/new effect was most prominent at left parietal electrodes. ERP amplitudes at channel P3 were more positive-going for hits between 533 and 895 ms after stimulus onset (Fig. 2a). These findings were consistent with the conventional definition of the LPC[7].

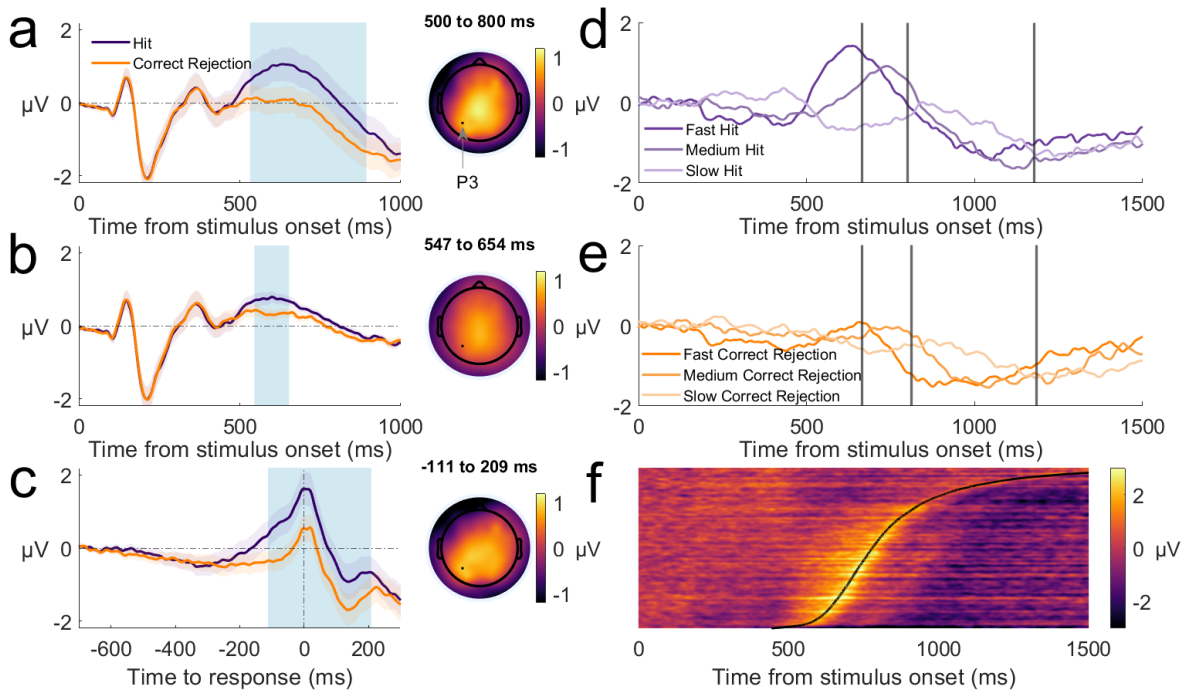
A critical limitation of these stimulus-locked ERP analyses is that they also capture contributions from response-locked ERP components. Condition differences in the amplitude and/or timing of response-locked components can lead to spurious interpretations of stimulus-locked effects[67,68]. Conversely, response-locked ERPs can also be biased by stimulus-locked ERPs[69,70]. To address this signal convolution problem, we separately estimated stimulus-locked and time-varying ERPs using the Residual Iteration Decomposition (RIDE) toolbox[68], as done in previous studies[41,71].

In the stimulus-locked ERP subcomponent, we observed a similar old/new effect between 543 and 659 ms post stimulus onset (Fig. 2b). Consistent with our previous observations[41], this effect was small and demonstrated a central parietal origin, rather than the left-lateralised topography of the conventional LPC. The stimulus-locked subcomponent in each condition was subsequently subtracted from the single-trial ERPs[71]. Response-locked segments of EEG data were then derived to better isolate the decision-locked signals[41,71].

In the response-locked data, the LPC gradually increased in amplitude and peaked at the time of the response (Fig. 2c). We observed more positive-going amplitudes for hits compared to correct rejections spanning -111 to 209 ms relative to the response. This ramp-to-peak feature of the LPC was clearer for hits than correct rejections. We further demonstrate this feature in the stimulus-locked data with the stimulus-locked subcomponents subtracted. For both targets and lures (Fig. 2d and 2e), the timing of the LPC shifted further away from stimulus onset for slower RT tertiles as observed for the CPP[34]. When sorting all trials in the experiment by RT (Fig 2f), the positive-going LPC signal is closely aligned with the time of the keypress response. Importantly, the topography of the response-locked LPC old/new effect showed a left-lateralised, parietal locus that is highly similar to the conventional stimulus-locked LPC effect (compare Fig 2a and 2c).

Taken together, this indicates that the LPC is time-locked to the response rather than the stimulus. It appears that the conventional stimulus-locked LPC effect arises from the contributions of overlapping, response-locked ERPs, where the RT distributions for hits and correct rejections closely align with the timing and duration of the conventional stimulus-locked effects.

Figure 2. Stimulus- and response-locked ERPs in the recognition memory task.



Note. **a**. The conventional LPC amplitude difference across hits and correct rejections (old/new effect), and the scalp distribution for hits minus correct rejections within the time window of statistically significant differences. Blue shading denotes time windows of statistically significant effects. **b**. The stimulus-locked ERP subcomponent estimated using RIDE and differences across hits and correct rejections. **c**. Response-locked LPC effects after subtracting stimulus-locked subcomponents from the data. **d**. Stimulus-locked ERPs for hits after subtraction of the stimulus-locked subcomponents, plotted by RT tertile bin. Vertical lines indicate average RTs for each bin. **e**. Stimulus-locked ERPs sorted by RT bin for correct rejections. **f**. ERPs for hits for all trials (pooled across participant) and sorted by RT, with the stimulus-locked subcomponents subtracted. Each row represents a single trial. Data were smoothed over 500-trial bins with a sliding Gaussian-weighted moving average filter.

Response-locked LPC amplitudes covary with accuracy and RTs

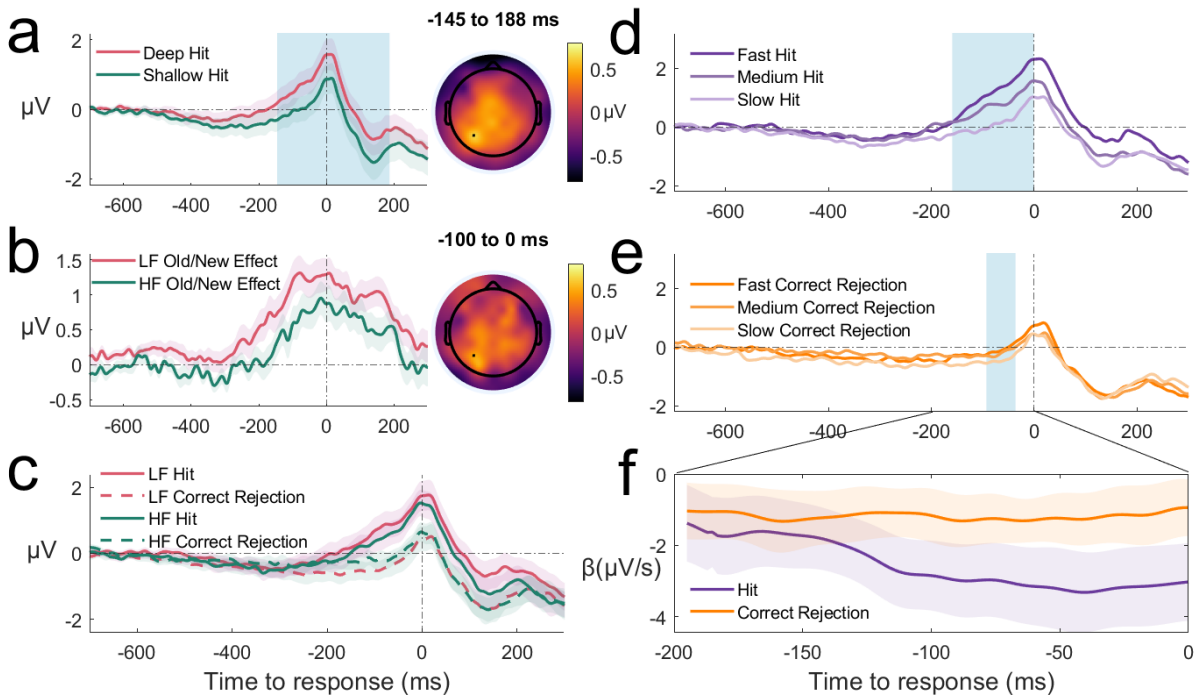
Building on existing findings related to the conventional stimulus-locked LPC, we tested for associations between the response-locked LPC and behavioural performance (accuracy and RT). First, we found that response-locked LPC amplitudes were sensitive to our manipulation of memory strength. LPC amplitudes were larger for deeply compared to shallowly processed targets (Fig 3a), consistent with the large

behavioural effect of deep/shallow processing on hit rates. However, we did not observe a significant difference in the old/new effects between high and low frequency words, or a word frequency effect on the LPC amplitudes for hits and correct rejections (Fig. 3b and 3c). We expected that LPC amplitudes would be higher for less frequent targets and lower for less frequent lures, based on the observation that lower frequency words are more likely to be correctly recognised and less likely to be falsely recognised[54]. The non-significant effect could be due to the small behavioural effect observed for our word frequency manipulation (Fig. 4a).

We also found that response-locked LPC amplitudes increased with faster RTs for both targets and lures (Fig. 3d and 3e). This partially explains the shifts and decreases in the LPC amplitude from fast to slow RTs in the stimulus-locked data (Fig. 2d and 2e). The β parameters from mixed effects models across time points were significantly lower than zero across time points prior to the response, indicating lower LPC amplitudes for faster RTs. Associations were stronger for hits than correct rejections, as evidenced by visual inspection and β values (Fig. 3f). Notably, for hits, the association was stronger at time points closer to the response.

Overall, LPC amplitudes were larger in conditions associated with a higher memory strength and for faster responses, and this effect was larger for targets. These characteristics are consistent with changes in the drift rate across conditions and trials within the DDM, where a higher drift rate will more likely result in faster RTs and a higher likelihood of 'old' responses.

Figure 3. Response-locked ERPs by depth of processing, word frequency and RT



Note. a. Response-locked ERPs for hits in deep/shallow conditions, and the scalp distribution of deep minus shallow differences during the statistically significant time window marked by the blue shaded area. b. Response-locked ERPs for hits in high and low word frequency conditions, and the scalp distribution of the low minus high difference during 100-ms time window prior to the response. c. Response-locked ERPs for correct rejections in high and low word frequency conditions. d. Response-locked ERPs for hits binned by RT tertile [0-33%, 33-66%, 66-100%]. Blue-shaded areas denote statistically significant associations between RTs and LPC amplitudes. e. Response-locked ERPs for correct rejections by RT tertile. f. β parameters for the effect of RT on ERP amplitude estimated from linear mixed effect models. Shaded error regions depict within-participant standard errors for each plot. LF = Low Frequency. HF = High Frequency

DDM predictions account for patterns of behavioural and neural data

To better understand the relationships between the LPC and behaviour, we jointly modelled accuracy, RT and response-locked LPC amplitudes using a modified DDM. Specifically, LPC amplitude on each trial was sampled from a normal distribution, where the standard deviation of the distribution reflects the noise in the neural measure and the mean is linked to the drift rate from the DDM via a linear

function (see Fig 1B). As a more positive-going LPC amplitude has been associated with more accurate recognition[7,8,72], we assume that LPC amplitudes positively scaled with drift rate. To construct a single measure of LPC amplitude for each trial, we calculated the averaged amplitude in a 200-ms time window prior to the response. We used BayesFlow (Version 1.0)[48] for amortised inference and parameter estimation.

We then fitted the model to the experimental data and generated 10,000 samples for each posterior distribution of parameters. The DDM was able to capture patterns of accuracy scores and RT distributions (summarized by the 10th, 50th, and 90th percentiles) across conditions in the experiment (Fig 4a-4c). The unique advantage of our joint modelling framework is its ability to capture relationships between accuracy/RT and LPC amplitude – in addition to capturing mean LPC amplitude across conditions that differ in accuracy (Fig 4b and 4c). Specifically, the model predictions closely match the mean LPC amplitudes across RT tertiles (Fig 4d), which showed a stronger relationship for targets than lures. We also found that the median parameters for posterior distributions across participants could be well recovered (Supplementary Fig. 1a). Notably, we also replicated the typical finding of a greater across-trial drift rate variability for targets than lures[73,74]. This demonstrates the advanced ability of the neural network to approximate likelihood functions for complex models.

Visual inspections of the model predictions against data (Fig 4b and 4c) indicated two main places of misfits for the neural data: LPC amplitudes between correct rejections and false alarms, and hits and misses for shallowly processed high word frequency targets. Here, we note that these apparent numerical differences did not correspond to statistically significant differences in our ERP measures. Specifically, there were no significant differences in LPC amplitudes between correct rejections and false alarms for either high or low frequency lures (p = 0.837 and p = 0.373), or between hits and misses for shallowly processed high word frequency targets (p = 0.379). Therefore, our model was able to capture changes in the neural data that corresponded to statistically significant ERP effects.

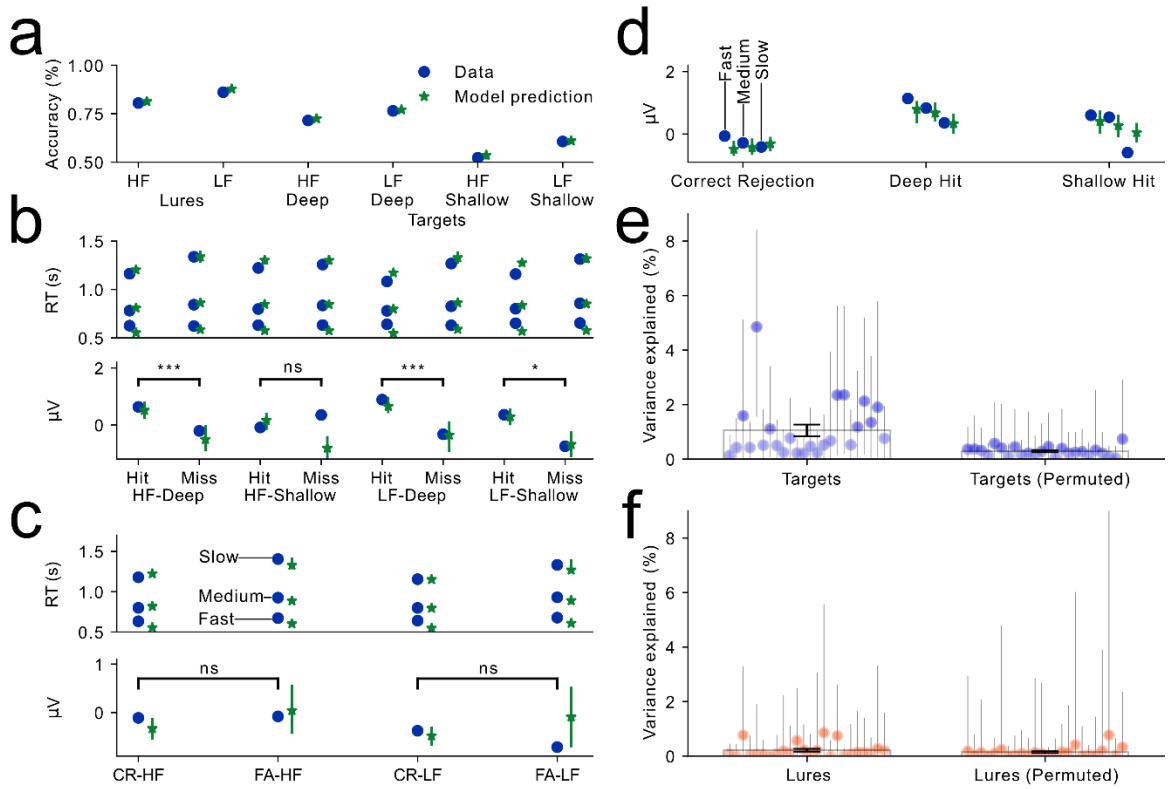
LPC amplitudes covary with single-trial drift rates

We next assessed whether LPC amplitudes covaried with drift rate estimates in each trial. An advantage of our modelling approach is the consideration of noise in EEG data, which could be understood as variance in EEG that was not explained by the specified model parameters. This parametrisation additionally allowed the estimation of the relative contributions to the LPC variance estimates from across-trial drift rate variability and noise. We used the median variance-explained values from the posterior distribution for statistical analysis. Across participants, we found this median variance-explained measures were significantly higher for targets than lures within participants (Fig 4d and 4e), $t(23) = 4.30$, $p < 0.001$. To validate this link between LPC and drift rate across trials, we permuted the trial-level match between LPC amplitudes and behavioural data across participants. Variance-explained measures were significantly lower than those from unpermuted data for targets, $t(23)= 3.58$, $p = 0.002$, but not for lures, $t(23)= 1.16$, $p = 0.260$, with the latter estimates close to zero. Therefore, the LPC amplitudes covaried with drift rate estimates in our models, and this association was much stronger for targets than lures.

While the variance-explained measures might be considered low (1.07 % on average for targets), within the context of the amount of noise in the data, they are substantial. This was demonstrated by estimating the maximum explainable variance in the LPC measures[75-77]. We first randomly split the dataset in half for each participant and calculated the rank-order correlation of averaged LPC measures across either target or lure words. We then applied the Spearman-Brown correction to the coefficients and squared them to calculate the estimates of maximum explainable variance. With 10,000 iterations, we found that averaged estimates were 0.84% (95% CI [0, 2.51%]) for targets and 0.67% (95% CI [0, 2.13%]) for lures. As this was only an estimation, the current mean of the variance-explained estimates exceeded the mean estimate of the maximum explainable variance. This suggested that while only a small amount of variance in the LPC was explainable, drift rate likely accounted for a large amount of the explainable variance for targets. The relatively high level of noise in single-trial EEG signals is also demonstrated by the small size of the LPC old/new effect (mean = 1.01 μV) relative to standard deviation of LPC measures across trials (9.08 μV). Nevertheless, the estimates observed here were of similar magnitude to the same estimates observed in a previous

recognition memory experiment[45]. These measures could also be well recovered by our model (Supplementary Fig 1b).

Figure 4. Model predictions and estimated variance-explained measures from the model using drift rates to predict LPC amplitudes.



Note. a. Model predictions of accuracy across conditions. The blue dots represent data averaged across participants. The green stars represent model predictions averaged across participants over 200 posterior samples (every 50th sample in the posterior distribution). Error bars denote 95% highest density intervals. b. Model predictions of RT percentiles and mean LPC amplitudes for targets across word frequency, processing depth and hit/correct rejection responses. c. Model predictions of RT percentiles [10th, 50th and 90th percentiles] and mean LPC amplitudes for lures across high/low word frequency conditions and correct rejection/false alarm responses. d. Model predictions for the LPC amplitudes averaged within equally binned RT tertiles [0-33%, 33-66%, 66-100%]. e. LPC amplitude variance-explained measures for targets and permuted data. Dots represent the median estimates for participants. Error bars depict 95% highest density intervals estimated from 10,000 posterior samples. f. Variance-explained

measures for lures. ns = non-significant, * = $p < 0.05$, *** = $p < 0.001$. LF = Low Frequency. HF = High Frequency. CR = Correct Rejection. FA = False Alarm

The relationship between the LPC and drift rate is strongest around the time of the response

To further understand how associations between LPC amplitudes and drift rates changes over time, we modified the DDM to simultaneously account for LPC amplitudes from three 100-ms time windows of LPC leading up to the time of the response. The LPC amplitude in each time window was independently predicted by the drift rates across trials. To account for the autocorrelation in EEG data, the three LPC time windows shared trial-level noise but also contained independent noise across time windows. Using a linear mixed-effect model we found that the later time windows (-200 to -100 ms, -100 to 0 ms) showed significantly greater variance-explained estimates than the -300 to -200 ms time window, $\beta = 0.67$, $p = 0.02$, $\beta = 2.20$, $p < 0.001$. A significant main effect of stimulus type was also observed, with the estimates being greater for targets than lures, $\beta = 0.62$, $p = 0.007$. This indicated a stronger association between LPC amplitudes and drift rates over time towards the time of the response across targets and lures. Notably, trial level noise was estimated to be more dominant than across-window noise (Supplementary Fig 2a), consistent with the autocorrelation assumption.

ERPs unrelated to mnemonic strength do not account for drift rate variance

We tested the selectivity of the relationship between the drift rate and the LPC by modifying our joint model to also include measures of the visual P1 component, measured at around from 50 to 150 ms after stimulus onset at electrode Oz. This component is generally assumed to reflect early visual processing across different tasks[78,79]. We therefore did not expect P1 to be strongly associated with memory strength. For the model specification, LPC and P1 amplitudes were predicted by drift rate in one model independently, and the noise was split into shared and independent components Overall, we found that the LPC explained a decent amount of the variance in drift rate, but no the P1.

Specifically, using a linear mixed effect model we found a significant main effect of stimulus type, with targets associated with significantly higher variance-explained

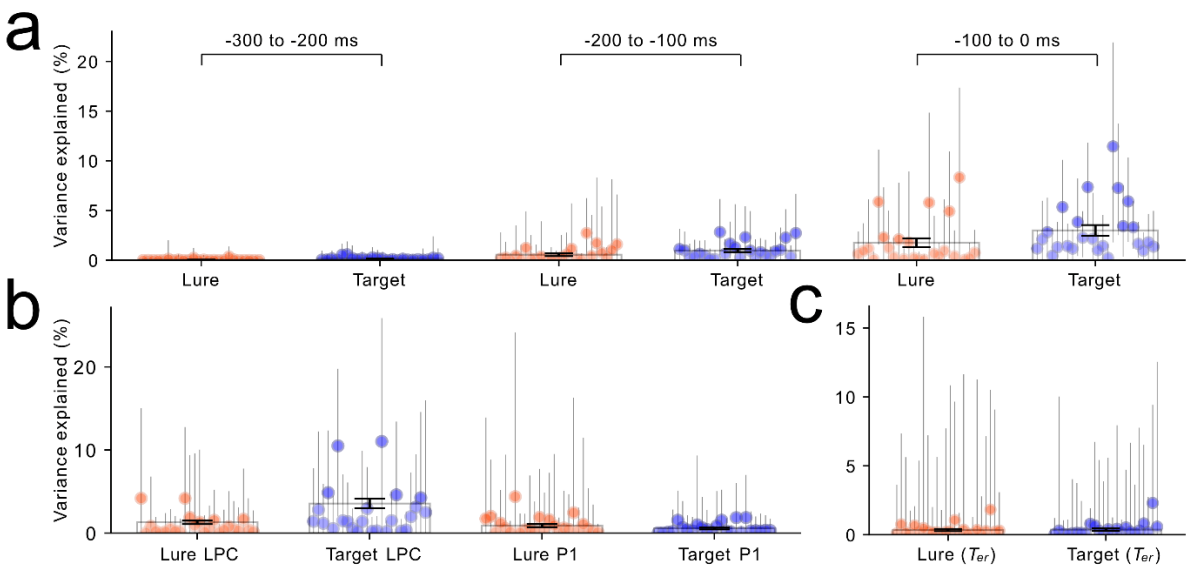
measures than lures, $\beta = 1.60$, $p = 0.001$. We also found a significant interaction between ERP component and stimulus type, with the difference between P1 and LPC being larger for targets than lures, $\beta = -1.95$, $p = 0.004$. For lures, the variance-explained measures did not differ significantly between LPC and P1, $\beta = 0.03$, $p = 0.945$.

As a validation to this model, we observed that the independent components of the EEG noise were more dominant than the shared noise (Supplementary Fig 3a). This was consistent with the fact that the the time windows between the LPC and P1 components were further apart than the LPC measures in the three-time-window model. Further, across participants, we found the estimated proportion of shared noise over overall noise showed a near-perfect correlation ($r = 0.96$, $p < 0.001$) with the correlation coefficients estimated between the LPC and P1 across trials (Supplementary Fig 3c). This suggests that the current assumption of Gaussian noises was able to track across-trial correlation of the outcome variables, which provides strong evidence that inferences from the model were based on information across trials.

The LPC is not related to variability in non-decision time

Finally, we validated our findings by testing whether changes in LPC amplitudes could be explained by other parameters in the DDM. We chose the non-decision time parameter[80,81], which similarly varies across trials (see Fig. 1). This model was otherwise the same as the first model except that LPC amplitude on each trial was predicted by the non-decision parameter sampled from a uniform distribution. Results from fitting such a model showed that non-decision time across trials could not account for model predictions of LPC amplitudes (Supplementary Fig 7) and the median variance-explained measures were close to zero (Fig. 5c). All three models mentioned so far could well account for the neural data (except for the non-decision time model) and behavioural data, and the parameters could be well recovered (Supplementary Figs 2 to 7).

Figure 5. Variance explained measures from the validation models.



Note. a. LPC amplitude variance explained measures estimated for lures and targets across three time windows leading up to the response. b. Variance explained measures estimated for lures and targets for LPC and visual P1 component amplitudes. c. Variance explained measures estimated for lures and targets for the LPC based on non-decision time variability.

Discussion

Inspired by the mnemonic accumulator hypothesis, we used a joint modelling framework to investigate whether the parietal LPC is a neural correlate of evidence accumulation variables in the DDM. We first build on our recent findings[41] to demonstrate that the LPC is more accurately characterised as a response- rather than a stimulus-locked ERP component. We also demonstrate properties of the LPC that are consistent with a mnemonic accumulation signal, akin to the CPP in perceptual decision making[31,35,82]. Using generative modelling to jointly account for behaviour and EEG signals, we found the pre-response LPC amplitudes were explained by across-trial variations in drift rate indexing mnemonic strength.

Our findings offer a fundamental reinterpretation of the LPC as a neural correlate of item memory strength driving evidence accumulation during recognition memory decisions. This contrasts with the conventional assumption that the LPC represents a high-threshold recollection process during memory retrieval[6,7,72], with LPC amplitudes indexing either successful or failed recollection. The diminished LPC

amplitudes were often interpreted as reflecting impairment in retrieving memory details[83-85]. Here, our results suggest LPC reflects a continuously varying strength signal associated with faster decisions, contrary to the assumption of recollection as a slow process[7]. While other theories proposed that recollection could also represent a continuous variable[86,87], it is important to note that models in previous studies of the LPC largely overlooked the role of decision-making processes, and analyses were primarily based on decision outcomes (e.g., accuracy and confidence). By directly integrating LPC measures into a computational model that specifies an evidence accumulation process, our findings reframe the LPC as a dynamic decision variable informed by memory evidence.

This alternative interpretation of the LPC is consistent with the mnemonic accumulator hypothesis[20], which proposes that the parietal cortex integrates mnemonic evidence rather than representing memory details, and particularly asymmetrically accumulating evidence for 'old' but not 'new' decisions[22,26]. The asymmetry hypothesis was based on observations that parietal BOLD signals and single-neuron recordings selectively track accuracy and confidence for old responses[3,24,27,28,88] and lesion studies showing selective impairments for old but not new decisions[89]. Similarly, for targets compared to lures, the LPC showed a clearer ramp-to-peak waveform and stronger pre-response associations with RT and drift rate across models. Therefore, a more positive-going LPC amplitude could be associated with a greater memory strength leading to a faster accumulation. It is important to note, however, we do not suggest that the LPC itself constitutes the mnemonic accumulator, which would require detailed tests of the accumulation trajectories along other decision-making dynamics (e.g., the level of caution).

Studies also reported evidence for a symmetrical accumulator in the parietal lobe, showing increased parietal activity for both high-confidence old and new decisions[22,88]. Likewise, a more positive-going central parietal EEG amplitude was sometimes observed for high confidence old and new responses[9,14,90,91], with recent findings demonstrating this component was also time-locked to the response and scaling with decision confidence[41]. This may account for the weak but present association between LPC and RT for lures. However, disentangling symmetrical and

asymmetrical accumulation signals with scalp EEG alone remains challenging, as these signal were suggested to be mixed within the parietal lobe[22] or even at the single-cell level[88]. Integrating models with a unifying account across modalities of neuroimaging data[92,93], such as simultaneous fMRI and EEG, offers a promising path to understanding the functional link to cortical regions. Our likelihood-free joint modelling framework provides a powerful basis for such extensions by enabling direct testing of complex neural hypotheses across data types.

Despite studies having demonstrated the ability of the DDM to fit behavioural data across recognition memory[94] and perceptual decision making tasks[95], our study constitutes an initial attempt to reconcile EEG signals in a memory task with formal evidence accumulation models. We demonstrated that LPC was a similar EEG signal to the CPP regarding the timing and relationships with different levels of accuracy and RT. Otherwise, our results indicated that the LPC could reflect mnemonic evidence gathered for old decisions but not the absence of mnemonic evidence for new decisions. This contrasts with the conventional assumption that CPP represents an evidence accumulation signal that is modality-invariant, accumulating evidence regardless of the type of response[34,35]. However, it is important to acknowledge that our observations for a lack of association in lures could be partially due to a small variability in the memory strength for lures. Therefore, we suggest further research is needed to understand the neural signals underlying evidence contributing to correct rejections of novel items with stronger manipulations of lures memory strength, such as the Deese-Roediger-McDermott paradigm[96]. More research could also be done to jointly model other components of decision-making, such as using Mu/Beta power tracing the motor action preparation[31,34]

Our study also demonstrates advanced methods for integrating neuroimaging and computational modelling. We applied EEG deconvolution (RIDE) to isolate response-locked ERP waveforms[41,71], mitigating concerns about temporal overlap with stimulus-locked signals[69]. We also leveraged a neural network-based, likelihood-free inference method[45] to jointly model behaviour and EEG signals, enabling robust hypothesis testing within a unified framework. As these tools continue to develop[97-100] and evolve—enabling functions such as model comparison[101] and misspecification

detection[102]—they hold promise for more precise mechanistic insights into cognitive processes and their neural correlates.

Finally, a challenging but meaningful extension of this work would be item-based inference linking LPC amplitude to item-level memory strength. While we demonstrated LPC fluctuations to reflect trial-level mnemonic strength, our framework does not estimate single-trial drift rates. Recent models derive item memory strength from stimulus similarity[103,104], which could be correlated with LPC amplitudes to inform model parameters. Although such approaches show promise—particularly for false recognition[105]—current models have yet to reliably capture strength variability for correctly recognised targets.

Conclusion

Our study provides compelling evidence that the LPC, traditionally seen as a marker of recollection, reflects a dynamic decision variable linked to mnemonic evidence accumulation during recognition judgments. We showed that LPC amplitude selectively tracks across-trial variations in drift rate within the DDM, consistent with the asymmetrical mnemonic accumulator hypothesis. Methodologically, we demonstrate the advantages of using deconvolution methods to isolate stimulus and response-locked signals, and a joint modelling framework to simultaneously fit behavioural and neural data. Further work in this field could further benefit from refining item-level inference, and leveraging multimodal data to further validate the computational property of LPC as well as identifying functionally equivalent cortical origins.

Method

Participants

We collected EEG data from 24 participants (19 female, 5 male, aged 18 – 33 years, M = 24.2, SD = 4). Participants were fluent in English and had normal or corrected-to-normal vision. One participant was left-handed. Two participants completed two out of the three sessions of data collection while four participants completed one session. No participants were excluded. All participants were paid 45

AUD per EEG session. This study was approved by the Human Research Ethics Committee of the Melbourne School of Psychological Sciences (ID 26845).

Stimuli

Stimuli were presented on a 24.5-inch ASUS ROG Swift PG258Q monitor (1920 x 1080 pixels, 60 Hz refresh rate). The stimuli are 900 words selected from UK-Subtlex database, which was an even split between low (2 - 5 per million) and high (80 - 500 per million) word frequency words. The word length was limited to 3 to 8 letters to reduce the need for making saccades. The word pool was re-used for each session but with random assignments to targets and lures. No words were repeated as stimuli within each session or list. There were 9 lists of words for the recognition task per session containing 50 targets and 50 lures, with balanced numbers of high and low word frequency words. The stimuli were presented in lowercase and the same words were not repeated within each session as either targets or lures. Stimuli were presented using the Psychopy Toolbox[106].

Paradigm

Participants were seated in a room with dimmed light and positioned 80 cm away from the monitor. For each session, participants completed a recognition memory task with EEG recordings. During the study phase, participants were instructed to view and memorise a list of 50 words presented serially at the centre of the screen. Each word was preceded by a fixation cross presented for 0.8 – 1.2 seconds (sampled from a uniform distribution). Each word was presented for 1.5 seconds along with a central fixation dot. Participants were instructed to fix their gaze on the fixation dot throughout the study phase.

Participants were additionally instructed to perform one of two tasks while memorising the word. They rated the pleasantness of the presented word (deep processing) for half of the study lists and indicated whether the presented word contained letter 'e' in it (shallow processing) for the other lists. The type of task to perform for each studied word was cued by the colour of the central fixation dot throughout the fixation and stimulus presentation periods (either blue or yellow, assignments counter-balanced across participants). Participants were instructed to

complete the task by pressing '1' or '2' on a numpad using their index and middle fingers from one hand. The allocation of keys and responding hand were counter-balanced across participants.

Following the study phase, participants were instructed to complete a set of arithmetic problems for 30 seconds. An addition equation (e.g., '2 + 3 + 5 = 12') was presented on the centre of the screen and participants indicated whether the equation was correct with a key response with the numpad (either '1' or '2'). Feedback (i.e., 'correct' and 'error') were provided for each response for 1 s followed by a 0.5 to 1 second interval.

For the test phase, a total of 100 words were presented serially in the centre of the screen, containing 50 targets from the study list and 50 lures. Each word was preceded by a fixation cross for 0.8 – 1.2 seconds (sampled from a uniform distribution), Words were presented for a maximum of 5 seconds. Participants were instructed to indicate whether the presented word was old (from the last study list) or new using key responses with the numpad (either '1' or '2'). If a response was made before the deadline, the word remained on screen for .5 seconds before the next fixation interval. Responses faster than .2 seconds or failures to respond within 5 seconds were immediately followed a screen displaying 'too fast' or 'too slow', respectively for 1 second. A pink fixation dot was presented in the centre of the screen throughout the test phase. As we used the same pair of keys ('1' and '2') across the three tasks, for each participant, the key for indicating pleasant words or words containing 'e' during the study phase, 'correct' responses for the arithmetic task and 'old' responses for the recognition memory task were kept the same to avoid confusion.

A practice block was first completed using a list of 12 words for the study phase, and 24 words for the test phase. Feedback was provided (correct or wrong) for 1 s for each type of task in the practice block to help participants understand the instructions. Each practice list took about 4 minutes to complete, and participants had the choice to repeat the practice block if needed.

EEG data acquisition and processing

EEG data was recorded at a sampling rate of 512Hz using a 64-channel Biosemi Active Two system. We added six electrodes around the eyes, two placed 1 cm from the outer canthi of each eye, and four placed above and below each eye. We processed the data using EEGLAB[107] (v 2021.1) running in MATLAB (The Mathworks). Code used for data processing and analysis will be available at https://osf.io/gqawp/ at the time of publication. First, we visually inspected the continuous EEG recording and excluded periods where EEG signals were excessively noisy. We also identified noisy channels (median number of noisy channels = 1, range = [0, 7]), which were excluded from the following data processing steps until they were interpolated. Second, we referenced the data to the average of all channels and low-pass filtered the data at 30 Hz (EEGLAB Basic Finite Impulse Response Filter New, zero-phase, -6 dB cutoff frequency 33.75 Hz, transition band width 7.5 Hz). We also removed channel AFz to account for the rank deficiency that can occur after the application of an average reference.

We applied Independent Component Analysis (ICA, RunICA extended algorithm) on a copy of the continuous EEG data[108] that was high-pass filtered at .1 Hz to improve stationarity of the signal. The resulting independent components were manually inspected, and components that resembled eye blinks and saccades were identified. These components were removed from the original continuous EEG data. Following this, we interpolated the noisy channels and channel AFz based on nearby channels (spherical spline interpolation). We then high-pass filtered the continuous EEG data at .1 Hz.

For the current study, we were only interested in EEG signals during memory retrieval. Therefore, the cleaned EEG data were segmented from -1,000 ms to 4,000 ms relative to the onset of each test probe. We then baseline-corrected the segmented data using a -200 to 0 ms time window prior to the onset of the test probe. As previous work[41] suggested that LPC should be best measured around the time of the response, we additionally constructed response-locked segments spanning -1,000 ms to 1,000 ms relative to the time of the response to each test probe, while using the same pre-stimulus baseline period. Please note that the response-locked data presented in the results section is after subtraction of the stimulus-locked subcomponent of the ERP, as described below.

ERP deconvolution and analyses

Following recommendations from previous work for measuring the LPC[41], we used the RIDE toolbox[68,109] to separate subcomponents of ERPs that are either time-locked to the onset of test probe or the response. Using the same parameter settings in previous work, we estimated two subcomponents: a stimulus-locked and a response-locked subcomponent, with time windows for estimation from 0 to 800 ms around stimulus onset, and -600 ms to 0 ms around response, respectively. For the input of the RIDE functions, we used the stimulus-locked epochs (-1,000 to 4,000 ms around stimulus onset) and estimated the subcomponent separately for each participant using pooled data across testing sessions. To reduce component overlap the stimulus-locked subcomponent was subtracted from all channels for analyses for response-locked data. This was done by subtracting the RIDE estimated stimulus-locked waveforms at each channel from each trial of EEG data before creating response-locked segments[71].

To identify the time windows that showed significant ERP amplitude differences, we performed mass univariate analyses with cluster-based permutation tests[110,111] implemented in the Decision Decoding Toolbox[112] to correct for multiple comparisons. This method groups adjacent time points with statistically significant amplitude differences between two conditions to form clusters of effects (cluster forming $p < .01$). The summed t values (the masses of the clusters) were then compared against the largest cluster masses obtained from each of 10,000 samples with permuted condition labels for a random subset of participants. The statistical significance of each cluster was judged by whether the cluster mass exceeded the 97.5th percentile of the permutation distribution (corresponding to an alpha level of 0.05, two tailed). For plotting purposes, we computed the within-participant standard errors across time points for comparing ERP waveforms[113].

To model changes in LPC amplitudes using the DDM, we calculated the averaged LPC amplitudes over a 200 ms time window prior to the time of the response. As the LPC effect has been consistently defined as a difference in EEG amplitude, we calculated the averaged amplitude over such a time window.

For the analyses regarding LPC amplitudes and RTs, we equally divided the EEG data within each participant into three equal tertiles based on the 33th and 66th percentiles, and then plotted the averaged waveforms from all participants based on both the stimulus-locked and response-locked data after subtracting the RIDE estimates. To investigate the association between pre-response LPC amplitudes and RT, we fitted a within-subject linear mixed effect model using RTs to predict LPC amplitudes in each trial, with random slopes and intercepts across participants. This was done for each sampling point within the 200 ms time window prior to the response for LPC. We then applied mass-univariate permutation tests to determine whether the β parameters obtained from linear mixed-effect models were significantly different to zero across time points.

Model parameterisation using BayesFlow

We employed BayesFlow to approximate likelihood functions for four versions of the DDM: a model linking LPC amplitudes with drift rate, a model predicting LPC amplitudes across three-time windows, a model predicting LPC and visual P1 amplitudes, and a model predicting LPC amplitudes based on non-decision time. Parameters shared by all three models are drift rate (v) varying according to stimulus type (old and new), deep/shallow processing and word frequency group, starting point (z), boundary separation (a), non-decision time (T_{er}), the across-trial variability parameters for non-decision time (S_t), and the drift rate variability parameter (η) varying across targets and lures. The four models differed in the how the neural component was predicted. The general modelling pipeline was an extension of previous work proposing this method[45] and details of each model are outlined below.

Model 1: predicting LPC amplitudes based on drift rate

For the neural component of this model, LPC amplitudes were linearly predicted by model parameter with a scale (β) and an intercept (β_0) separately for targets and lures, with a single measurement noise parameter σ. Parameter estimation from BayesFlow relies on the specialised neural network to learn the relationship between model parameters and data based on model simulations. Therefore, the model was simulated following the form below:

$$\mathrm{RT_{ij}}, \mathrm{Resp_{ij}} \sim \mathrm{DDM}(a, z, T_{er}, V_{\mathrm{j}},\ \eta_{\mathrm{j}}, S_t)$$

$$v_{\mathrm{ij}} \sim \mathrm{N}(V_{\mathrm{j}},\ \eta_{\mathrm{j}})$$

$$\mathrm{LPC_{ij}} \sim \mathrm{N}(\beta_{\mathrm{j}}(v_{\mathrm{ij}} - \beta_{0\mathrm{j}}), \sigma)$$

Where j represents each experimental condition and i represents each trial. Therefore, this model assumed that choices and RTs were generated by the DDM with different drift rate parameters for each experimental condition. On each trial, the drift rate was sampled from a normal distribution based on the mean drift rate estimated for each condition with a standard deviation of η. This drift rate was then linearly transformed as the mean of a normal distribution to generate the LPC amplitude on that trial, with a standard deviation of the noise parameter σ.

Model 2: predicting LPC amplitudes in different time windows based on drift rate

For this model, we used drift rate sampled on each trial to simulate LPC amplitude across time windows via three separate functions with different scales (β_{T1}, β_{T2}, β_{T3}) and intercepts parameters (β_{0T1}, β_{0T2}, β_{0T3}). Further, this model included shared and independent noise across neural measures and data were simulated in a general form below:

$$\mathrm{RT_{ij}}, \mathrm{Resp_{ij}} \sim \mathrm{DDM}(a, z, T_{er}, V_{\mathrm{j}},\ \eta_{\mathrm{j}}, S_t)$$

$$v_{\mathrm{ij}} \sim \mathrm{N}(V_{\mathrm{j}},\ \eta_{\mathrm{j}})$$

$$\mathrm{SharedNoise}_i \sim \mathrm{N}(0, \sigma)$$

$$\mathrm{LPCT1_{ij}} \sim \mathrm{N}(\beta_{T1_{\mathrm{j}}}\left(v_{\mathrm{ij}} - \beta_{0T1_{\mathrm{j}}}\right), c)\ + \mathrm{SharedNoise}_i$$

$$\mathrm{LPCT2_{ij}} \sim \mathrm{N}(\beta_{T2_{\mathrm{j}}}\left(v_{\mathrm{ij}} - \beta_{0T2_{\mathrm{j}}}\right), c)\ + \mathrm{SharedNoise}_i$$

$$\mathrm{LPCT3_{ij}} \sim \mathrm{N}(\beta_{T3_{\mathrm{j}}}\left(v_{\mathrm{ij}} - \beta_{0T3_{\mathrm{j}}}\right), c) + \mathrm{SharedNoise}_i$$

Whereas the shared noise component was the same for LPC int each time window signalling trial-level noise, it was assumed that LPC amplitudes in each 100-ms time window included unique noise that was not autocorrelated. This unique noise was simulated with an additional parameter c. Therefore, if the LPC amplitudes across three

time windows were highly correlated, then we should expect a relatively smaller estimate of unique noise as compared to shared noise.

Model 3: predicting LPC and P1 amplitudes based on drift rate

We measured the P1 as the mean amplitude from 50 to 150 ms relative to the stimulus onset at the electrode Oz. This model similarly assumed that LPC and P1 contains both shared and unique components of noise. Here, as LPC and P1 amplitudes were known to be associated with unique processes (memory vs. early visual processing), we included separate parameters for the unique noise component. EEG amplitudes for each measure were predicted with separate functions:

$$\mathrm{RT_{ij}}, \mathrm{Resp_{ij}} \sim \mathrm{DDM}(a, z, T_{er}, V_{\mathrm{j}},\ \eta_{\mathrm{j}}, S_t)$$

$$v_{\mathrm{ij}} \sim \mathrm{N}(V_{\mathrm{j}},\ \eta_{\mathrm{j}})$$

$$\mathrm{SharedNoise}_i \sim \mathrm{N}(0, \sigma)$$

$$\mathrm{LPC_{ij}} \sim \mathrm{N}(\beta_{LPC_{\mathrm{j}}}\left(v_{\mathrm{ij}} - \beta_{0LPC_{\mathrm{j}}}\right), c_{LPC})\ + \mathrm{SharedNoise}_i$$

$$\mathrm{P1_{ij}} \sim \mathrm{N}(\beta_{P1_{\mathrm{j}}}\left(v_{\mathrm{ij}} - \beta_{0P1_{\mathrm{j}}}\right), c_{P1})\ + \mathrm{SharedNoise}_i$$

To investigate whether the specification of the sources of noise was sensible, we computed the amount of shared noise relative to the overall noise estimated in the data:

$$\text{Shared variance } = \frac{\sigma^2}{{c_{LPC}}^2 + {c_{P1}}^2 + \sigma^2}$$

We then calculated the correlation between the amount of shared noise between the two EEG measures estimated by the model and the actual correlation across participants. It is expected that this measure should approximately track the trial-by-trial correlation between LPC and P1 amplitudes. This is because EEG is noisy, and it is expected that the neural/measurement noise should primarily contribute to the correlation between the two EEG component measures.

Model 4: predicting LPC amplitudes based on non-decision time

This model was otherwise the same as the Model 1 except that LPC amplitude on each trial was predicted by the non-decision time parameter sampled from a uniform distribution.

$$\mathrm{RT_{ij}}, \mathrm{Resp_{ij}} \sim \mathrm{DDM}(a, z, T_{er}, V_{\mathrm{j}},\ \eta_{\mathrm{j}}, S_t)$$

$$t_{\mathrm{ij}} \sim \mathrm{U}(T_{er_{\mathrm{j}}}, S_t)$$

$$\mathrm{LPC_{ij}} \sim \mathrm{N}(\beta_{\mathrm{j}}\left(t_{\mathrm{ij}} - \beta_{0_{\mathrm{j}}}\right), \sigma)$$

It is important to note that our simulation approach differed from studies that proposed the current Bayesflow method[45], as we simulated data across conditions at the same time. In previous studies, the posterior parameter distribution for different conditions was separately estimated based on a 'generic' DDM, which means condition-invariant parameters such as boundary separation were estimated multiple times to explain the full experimental dataset. This approach differs from traditional model fitting practices and provides less constraints on the parameter estimation. Further, we argue it is crucial in this case to have different parameters across conditions because numerous studies have shown that parameters such as drift rate and drift rate variability are different across targets and lures[74,114,115].

For all models we trained the neural networks with experience-replay learning implemented in BayesFlow[45]. Experience-replay is technique widely applied in deep reinforcement learning that allows samples to be stored in a memory buffer and re-sampled for training[116], which has been suggested to improve data efficiency. The ranges of priors from which the parameters values were drawn are listed in the supplementary material. We trained each neural network with 1,000 epochs, 1,000 iterations per epoch, a batch size of 32 with a buffer size of 100. This means the network was trained on 32,000,000 partially repeating samples. For each simulated dataset, the number of trials varied from 300 to 500. While this number was smaller than the actual data size, we have demonstrated that such network can generate robust parameter estimation and model predictions.

For parameter estimation, we generated 10,000 samples for each posterior distribution. For model predictions, we applied thinning and simulated data based on

every 50th sample of parameters in the posterior distribution (i.e., 200 data simulations in total). Importantly, we acknowledge that an inherent limitation of the neural network method is that the posterior estimates are informed by prior range but can sometimes fall outside of this range. To address this, we constrained the ranges for drift rate variability parameters and scale parameters with a logit-normal distribution. This involved first sampling unbounded parameter values from a normal distribution:

$$p \sim \mathrm{N}(0, 1.7)$$

These parameter values were used by the network for learning. The choice of standard deviation was to make the logit-normal distribution to approximate a uniform distribution, consistent with the prior distribution assumption for other parameters. Within the simulation functions, these parameters were transformed with a sigmoid function:

$$p = \frac{UpperLimit}{1 + e^{-p}}$$

Therefore, for both the simulation and posterior estimation, the values of the parameters were constrained between zero and the pre-defined upper limit of the prior range, and sampled from a logit-normal distribution approximating a uniform distribution.

Calculating the Variance in EEG Measures Explained by Models

Following previous work[45], we used parameters estimated from models to derive a variance-explained measure corresponding to the amount of variance in the EEG data explained by the model parameters. For example, for the model that linked LPC with single-trial drift rate, this ratio *r* was calculated as:

$$r = \frac{(\beta\eta)^2}{(\beta\eta)^2 + \sigma^2}$$

Where η is the across-trial drift rate variability parameter, β is the scale parameter of drift rate to produce the mean of LPC amplitude as a normal distribution with the noise parameter σ. For the model that linked LPC with non-decision time variability, the η parameter was replaced with the S_t for non-decision time variability.

For the model that included additional independent source of neural noise, this measure was calculated based on the joint noise:

$$r = \frac{(\beta\eta)^2}{(\beta\eta)^2 + \sigma^2 + c^2}$$

These estimates were produced based on parameter sets from the entire posterior distribution, the 95% highest density intervals of the measures were reported. We conducted statistical analyses (e.g., within-participant t tests and repeated measures linear mixed effect models) based on the mean participant-level estimates to determine how they differ across conditions.

To clarify, we did not intend to interpret the variance-explained measure as the true signal-to-noise ratio of the LPC, given that EEG data was generated based on simplified distributional assumptions rather than models with plausible neural mechanisms. However, as demonstrated in the results section, this measure was highly useful to understand and compare associations between neural data and model parameters under the same modelling framework. Crucially, as demonstrated in the supplementary material, these parameters as well as the measures could be recovered by the model. Further, we found that the variance-explained estimates in Model 3 could strongly track the across-trial correlation between different neural signals (Supplementary Fig.3c).

Model Parameter Recovery

To check whether the trained neural network can reliably generate posterior parameter estimates, we performed parameter recovery analyses. For each participant, we generated 200 simulations based on the median parameter estimates from the posterior distributions and re-fitted the model to each of the simulated datasets, generating 10,000 posterior samples. We used median estimates to avoid biases in the estimates by skewed posterior distributions, which was due to estimates of parameters being close to the prior boundary (e.g., low scale parameter estimates). We then plotted the median parameters against the 95% highest density interval for the recovered parameters. Specifically, given that we relied on the variance-explained measure for

inferring associations between model parameters and neural measures, we also focused on examining the recovery of this measure.

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