A Burst-dependent Thalamocortical Substrate for Visual Rivalry

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Abstract

Investigations into the neural basis of conscious perception span multiple scales and levels of analysis. There is, however, a theoretical and methodological gap between advances made at the microscopic scale in animals and those made at the macroscopic scale in cognitive neuroscience. This divide constrains the neurobiological understanding of conscious perception to the scale of each specific measurement modality. Here, we use computational modelling to bridge this divide. Specifically, we show that the same (microscopic) mechanism that underlies conscious perception in a mouse model of threshold perception – namely, apical dendrite mediated burst firing in thick-tufted layer V pyramidal neurons – determines perceptual dominance in a thalamocortical spiking neural network model of visual rivalry – a staple paradigm in the (macroscopic) cognitive neuroscience of consciousness. The model conforms to the constraints imposed by decades of previous research into visual rivalry in human and non-human primates and explains the slowing of perceptual reversals caused by genetic perturbations to E/I balance in a mouse model of visual rivalry. In addition, the model's layer-specific structure and thalamocortical connectivity allowed us to simulate both cellular level (optogenetic and pharmacological) interventions and the downstream neuronal effects of psychological manipulations of attention and expectation. Our model, therefore, provides an empirically-tractable bridge between cellular-level mechanisms and conscious perception.

Introduction

Uncovering the neural mechanisms of conscious perception and conscious state remains one of the outstanding challenges in modern neuroscience. In spite of the rich backdrop of existing theoretical and experimental work (e.g., Brown et al., 2019; Lamme, 2006; Mashour et al., 2020; Tononi et al., 2016), there is limited interaction between theories proposed at different scales (Seth & Bayne, 2022). Perhaps most tellingly, it is rare for empirical predictions at one scale to be brought into contact with predictions at other scales: the large-scale correlates of consciousness are rarely couched in terms of their putative cellular underpinnings and *vice versa*.

This disparity is not without good reason. Cognitive neuroscience is largely based on human neuroimaging (e.g. Williams & Henson, 2018) where high-resolution (i.e., cellular specific) recordings and precise causal manipulations (e.g., optogenetic and pharmacological) are exceedingly difficult and often impossible (especially in humans). In turn, although it is possible to obtain high-resolution data with precise causal control in animal models, these experiments are enormously restricted in the range and complexity of tasks that they can perform. In the context of consciousness science, measures of perception often rely on indirect behavioural read-outs (e.g. eye movements and button presses), limiting contact with predictions associated with more nuanced aspects of perceptual awareness that are accessible in humans through self-report (e.g. perceptual awareness scale, post-decision wagering, and confidence ratings; for discussion see Sandberg et al, 2010). Indeed, a recent review summarising the consensus among consciousness researchers regarding open problems at the frontiers of the field emphasised the importance of integrating insights across scales through the use of experimental paradigms that can be run in both human and non-human animals (He, 2023).

Here, we use biophysical modelling to bridge the gap between the thalamocortical circuit identified in a recent rodent model of perception (Aru, Suzuki, et al., 2020; Takahashi et al., 2016, 2020) and the behavioural signatures of conscious perception studied in cognitive neuroscience. Specifically, we built a spiking neural network model of visual rivalry based on a circuit comprising thick-tufted LV pyramidal tract (LV_{PT}) neurons whose burst-firing state is gated by non-specific matrix thalamus, and is known to play a causal role in both perception and conscious level (Aru et al., 2019; Aru, Siclari, et al., 2020; Aru, Suzuki, et al., 2020;

Takahashi et al., 2016, 2020). We chose visual rivalry – which we use as a catch-all term for binocular rivalry, as well as related bistable perception paradigms, such as plaids (Hupé & Rubin, 2003) and continuous flash suppression (Tsuchiya & Koch, 2005) – as the primary explanatory target of the model as it is a staple behavioural paradigm in the cognitive neuroscience of consciousness (Overgaard, 2015). In addition, visual rivalry has been studied extensively in both empirical (Alais & Blake, 2005; Blake & Tong, 2008; Carmel et al., 2010; Tong et al., 2006) and computational (Grossberg et al., 2008; Laing et al., 2010; Laing & Chow, 2002; Safavi & Dayan, 2022; Shpiro et al., 2007, 2009; Wilson, 2003, 2007, 2017) settings, thereby imposing a wealth of constraints on the modelling process. Importantly, due to the highly robust nature of visual rivalry and recent advances in experimental paradigms, the subjective contents of perception can be reliably inferred from involuntary eye-movements without the need for subjective reports (Frassle et al., 2014; Kapoor et al., 2022; Naber et al., 2011) making it possible to study aspects of visual rivalry in mice (Bogatova et al., 2022; Palagina et al., 2017) as well as in human and non-human primates. This allows us to propose testable predictions about the neurobiological basis of perceptual awareness at both microscopic, mesoscopic, and behavioural scales.

Our neurobiological approach to modelling allowed us to: i) explore whether the cellular-level mechanism of visual awareness discovered in a rodent model could recover the lawlike psychophysical relationships (known as Levelt's laws) between stimulus properties and the perceptual phenomenology known to characterise visual rivalry (Brascamp et al., 2015); ii) validate the basic mechanism of perceptual competition and suppression in our model by comparing outputs of the model to genetic perturbations of E/I balance in a rodent model of visual rivalry. In agreement with empirical findings (Bogatova et al., 2022), a shift towards a high excitation regime led to a slowing of perceptual reversals. Crucially, this prediction is in contradiction to many large-scale (i.e. mean field) models of rivalry (e.g. Laing & Chow, 2002; Wilson, 2007); iii) interrogate the putative cellular basis of perceptual dominance through a series of *in silico* perturbation experiments, thus generating a suite of novel cellular-level predictions; and iv) leverage the novel thalamocortical connectivity backbone to simulate the downstream neuronal effects psychological manipulations of attention (Paffen & Alais, 2011), and expectation (Pinto et al., 2015), on visual rivalry, creating a tether between our cellular level model and the more sophisticated rivalry tasks studied in human psychophysics. In doing so, our model provides a neurobiological bridge between the cellular-level mechanisms

typically studied in systems and cellular neuroscience and the behavioural signatures of consciousness studied in human cognitive neuroscience.

Results

The dynamical elements of the model consisted of three classes of neurons – LV_{PT} cells, fast spiking interneurons (basket cells), and diffuse projecting (matrix) thalamocortical cells (**Fig 1 A-B)** – each of which were modelled using biophysically plausible spiking neurons (Izhikevich, 2003, 2004, 2006) coupled through conductance based synapses. The parameters of the basket cells and thalamus cells were taken unmodified from the whole brain model of Izhikevich and Edelman (2008). To capture the unique context-dependent bursting behaviour of the LV_{PT} cell (Larkum, 2004, 2022; Larkum et al., 2009), we created a novel dual-compartment variation of the Izhikevich neuron with (coupled) apical and somatic compartments. This allowed us to reproduce the behaviour of these cells that has been linked to conscious perception – namely, the change in dynamical regime in LV_{PT} cells that occurs when these cells receive coincident into their apical and basal dendrites. Specifically, we leveraged the insight (Izhikevich, 2003; Munn, Müller, Aru, et al., 2023; Munn, Müller, Medel, et al., 2023) that the difference between regular spiking and bursting behaviour (operationally defined by shorter inter spike intervals < 10 ms) can be modelled by changing the reset conditions of the somatic compartment – raising the reset voltage taking the neuron closer to threshold and reducing the magnitude of spike adaptation. We arranged the coupling between compartments such that the apical compartment controlled the reset conditions on the somatic compartment, with an apical spike causing a switch in the reset conditions of the somatic compartment from regular spiking to bursting parameters for 25 ms (in line with empirical measurements; Larkum, 2004). The membrane potential of the apical compartment was coupled to the somatic compartment through a conductance term allowing for somatic spikes to back propagate and contribute to the generation of apical spikes. In line with empirical findings and previous modelling (Larkum, 2004; Shai et al., 2015), the spiking activity in the apical compartment effectively controlled the gain of the somatic compartment's FR curve through increasing the amount of time each LV_{PT} cell spent in the bursting regime rather than the regular spiking regime (**Fig 1C**).

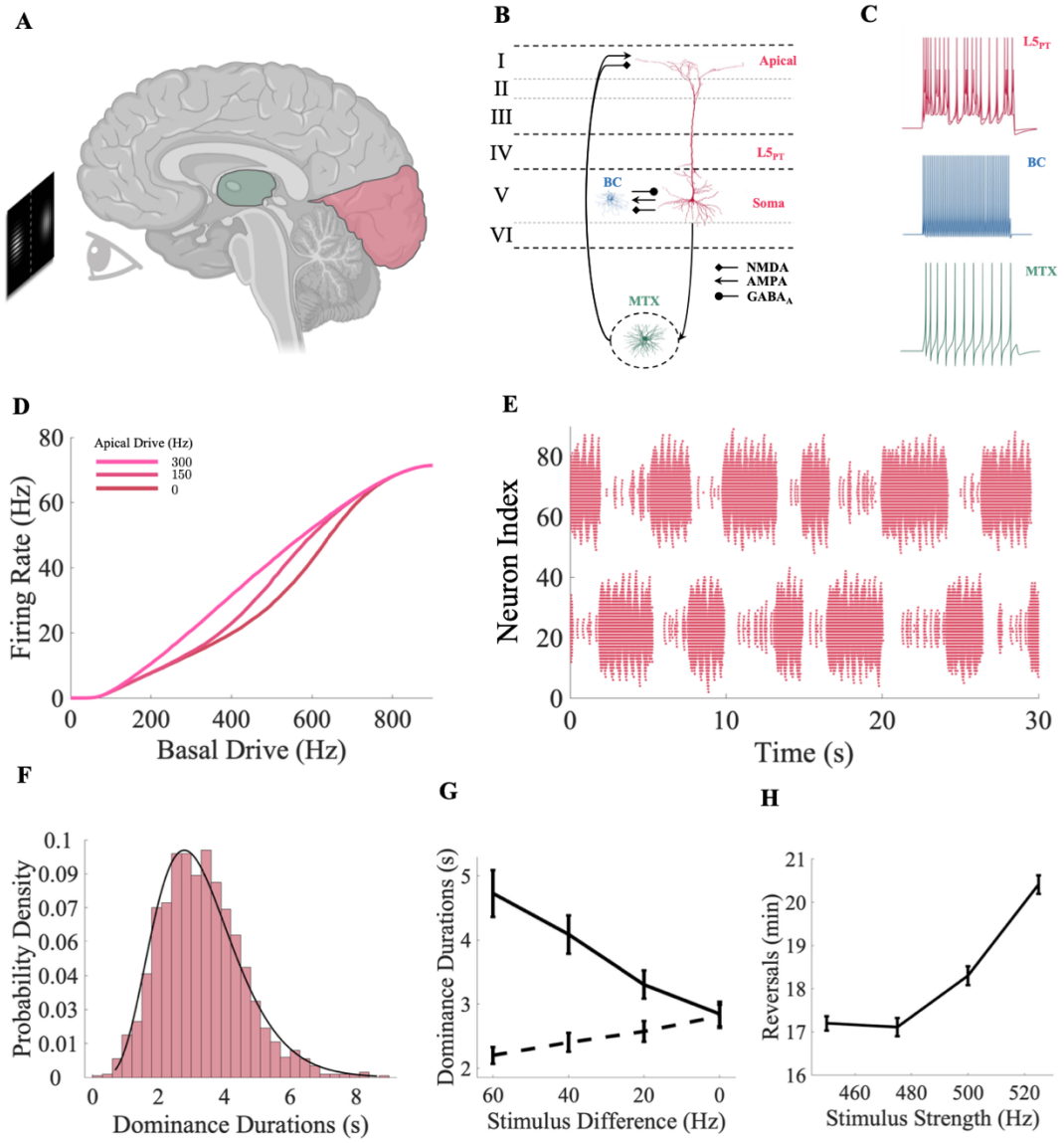

Figure 1. A-B) Anatomy of the model thalamocortical loop connecting non-specific matrix thalamus and LV_{PT} neurons. **C)** Single neuron dynamics of each class of cell used in the model. **D)** Firing rate of the novel dual compartment LV_{PT} neuron as a function of basal drive and apical drive. **E)** Example raster plot of the stochastic oscillations in somatic firing rate in the LV_{PT} neuron as the model is shown orthogonal grating stimuli. **F)** Example histogram of dominance durations for a simulation run with 475 Hz of basal drive. Black line shows the fit of a Gamma distribution with parameters estimated via MLE ($\alpha = 6.3546$, and $\beta = 2.1209$; the Gamma distribution is given by $f(x) = \beta^{\alpha}\Gamma(\alpha)^{-1}x^{\alpha-1}e^{-\beta x}$, where x is a random variable). **G)** Simulation of Levelt's second proposition. Solid line shows dominance duration of the population receiving the fixed external drive (520 Hz). Dash line shows the dominance duration of the population receiving the decreasing external drive. **H)** Simulation of Levelt's fourth proposition. Perceptual reversals (per minute of simulation time) increase as a function of external drive.

Based on previous spiking neural network models of rivalry (Laing et al., 2010; Laing & Chow, 2002; Wang et al., 2020), we used a one-dimensional cortical ring architecture (90 pairs of

LV_{PT} excitatory and fast-spiking inhibitory interneurons). Each point on the ring represents an orientation preference, with one full rotation around the ring corresponding to a 180° visual rotation. These modelling choices provided each neuron with a 2° difference in orientation preference, relative its neighbours. The (dimensionless) synaptic weights connecting neurons on the ring followed a Gaussian connectivity kernel with synaptic weight decaying as a function of the Euclidean distance between neurons. The conductance based synapses themselves had a bi-exponential form, with rise and decay time parameters chosen to recapitulate the dynamics of inhibitory ($GABA_A$) and excitatory (AMPA and NMDA) conductances (Dayan & Abbott, 2005; Gerstner et al., 2014). For a full description see **materials methods**.

Throughout this paper we draw on data from a wide array of experiments. Despite it being possible to fit the parameters of our model to each experimental finding individually, this approach permits too many degrees of freedom and risks overinterpretation of idiosyncratic aspects of individual experiments. Instead, in the spirit of hypothesis-driven science, we elected to challenge a single model to qualitatively reproduce a wide array of experimental findings with a minimal set of parameter changes carefully chosen to reflect experimental manipulations and perturbations. In line with common practice in models of rivalry (e.g. Moreno-Bote et al., 2007), we initialised the connectivity strengths such that the model was in a winner-take-all regime and then increased the slow-hyperpolarising current until the average dominance duration was in an empirically plausible range (Fig. 1E). To ensure the generality and robustness of our findings, we ran a large parameter sweep around the values supplied in **materials and methods table 2** and found consistent results (see **supplementary material S1)**.

We began by simulating a standard rivalry experiment by driving the model with input representing orthogonal gratings presented to each eye. External drive into the model simulated as two independent Poisson processes (representing indirect retinal input) exclusively targeted the basal dendrites of the somatic compartment of LV_{PT} cells and was weighted by a spatial Gaussian centred on neurons 90° apart on the ring (i.e., 45 neurons apart) mimicking the orthogonal grating stimuli commonly employed in binocular rivalry experiments (e.g., Logothetis & Leopold, 1996; Tong et al., 2006). In line with previous modelling work (Laing & Chow, 2002), we interpreted persistent 'bumps' of activity on the ring, which in our model was dominated by bursting, as representing the content of perception. In agreement with

previous models and psychophysical data (Wilson, 2003; Wolfe, 1983), at stimulus onset there was a brief period lasting ~150 ms where neuronal populations on both sides of the ring were equally dominant before the competition between populations for dominance was resolved and the model settled into an oscillatory regime where one of the two populations was dominant whilst the other was suppressed (**Fig 1D**). Also in agreement with psychophysical data, switches between persistent activity and suppression (perceptual reversals) appeared stochastic, with a right-skewed distribution of dominance durations (Brascamp et al., 2015; Levelt, 1967; Logothetis & Leopold, 1996; **figure 1E**). Across basal drive conditions, comparison of negative log likelihoods showed that the distribution of dominance durations was best fit by a Gamma distribution ($\ell = 9.7562 \times 10^{-4}$), compared to lognormal ($\ell = 9.9370 \times 10^{-4}$), or normal distributions ($\ell = 1.0053 \times 10^{-5}$). We found that the dynamical regime underlying these stochastic oscillations consisted of noisy excursions around a stable limit cycle (**supplementary material S2)**.

Levelt's Propositions

To validate the model psychophysically we tested Levelt's propositions (Brascamp et al., 2015) – a set of four statements that compactly summarise the relationship between stimulus strength (e.g., luminance contrast, colour contrast, stimulus motion) and the average dominance duration of each stimulus percept. Here, we focus on the second and fourth propositions, as they constitute the "core laws" of rivalry (propositions one and three are consequences of proposition two; Brascamp et al., 2015). Levelt's second proposition states that increasing the difference in stimulus strength between the two eyes will principally increase the average dominance duration of the percept associated with stronger stimulus (Brascamp et al., 2015), a finding that has been shown to hold across studies in both human and non-human primates (e.g., Leopold & Logothetis, 1996; Logothetis & Leopold, 1996). According to Levelt's fourth proposition, increasing the strength of the stimulus delivered to both eyes will increase the average perceptual reversal rate (i.e., decrease dominance durations; Brascamp et al., 2015), a finding that, like Levelt's second proposition, has been replicated across a wide array of experimental settings (Bonneh et al., 2014; Brascamp et al., 2006; Buckthought et al., 2008; Meng & Tong, 2004). Originally proposed in 1965 (Levelt, 1965), these psychophysical "laws" have proven to be remarkably robust needing only minor modification and contextualisation (Brascamp et al., 2015). Levelt's propositions have historically, therefore, served as a

benchmark for computational models of rivalry (e.g., (Grossberg et al., 2008; Laing & Chow, 2002; Shpiro et al., 2007; Wilson, 2007).

To simulate the experimental conditions described by Levelt's second proposition, we presented the model with external drive centred on neurons with orthogonal orientation preferences and then decreased the spike rate entering one side of the ring in steps of from 520 Hz to 460 Hz in steps of 20 Hz across simulations. In line with Levelt's second proposition (**Fig 1G**), the average dominance duration of the percept corresponding to the stronger stimulus showed a steep increase from ~2.75 s with matched input, to ~4.5 s with maximally different input whilst the average dominance duration on the side of the weakened stimulus decreased comparatively gradually to ~2.1 s with maximally different inputs.

To simulate Levelt's fourth proposition, we ran a series of simulations in which we increased the spike rate of the external drive in steps from 450 Hz to 525 Hz in steps of 25 Hz across simulations. In line with Levelt's propositions (**Fig 1H**), the perceptual reversal rate increased with input strength, starting at ~17 reversals per minute at the weakest stimulus and increasing to ~20.5 reversals per minute for the strongest.

We thus confirmed that our neurobiologically detailed model of the LV_{PT} non-specific matrix thalamus loop is capable of reproducing Levelt's propositions, which together with the right-skewed distribution of dominance durations, show the consistency of our model with the key lawlike psychophysical relationships known to characterise binocular rivalry.

E/I Balance and Perceptual Reversal Rate

Having confirmed that our model conforms to key psychophysical constraints, we next sought to test the model's mechanism of perceptual competition and suppression by comparing simulated E/I balance perturbations to genetic perturbations of E/I balance in a rodent model of visual rivalry (Bogatova et al., 2022). Specifically, Bogatova et al (2022) found that compared to non-transgenic litter mates, mice with a *MECP2*-duplication, which has been shown to reduce inhibitory IPSP currents (Chao et al., 2010), have a reduced perceptual reversal rate (**Fig 2A**). Crucially, this result is in direct contradiction to the predictions of a number of prominent mean field models of rivalry which all predict the exact opposite effect (e.g., Curtu et al., 2008; Laing & Chow, 2002; Wilson, 2007) – namely, that decreasing

inhibition should increase perceptual reversal rate through an increase in capacity of the suppressed population to escape from inhibition.

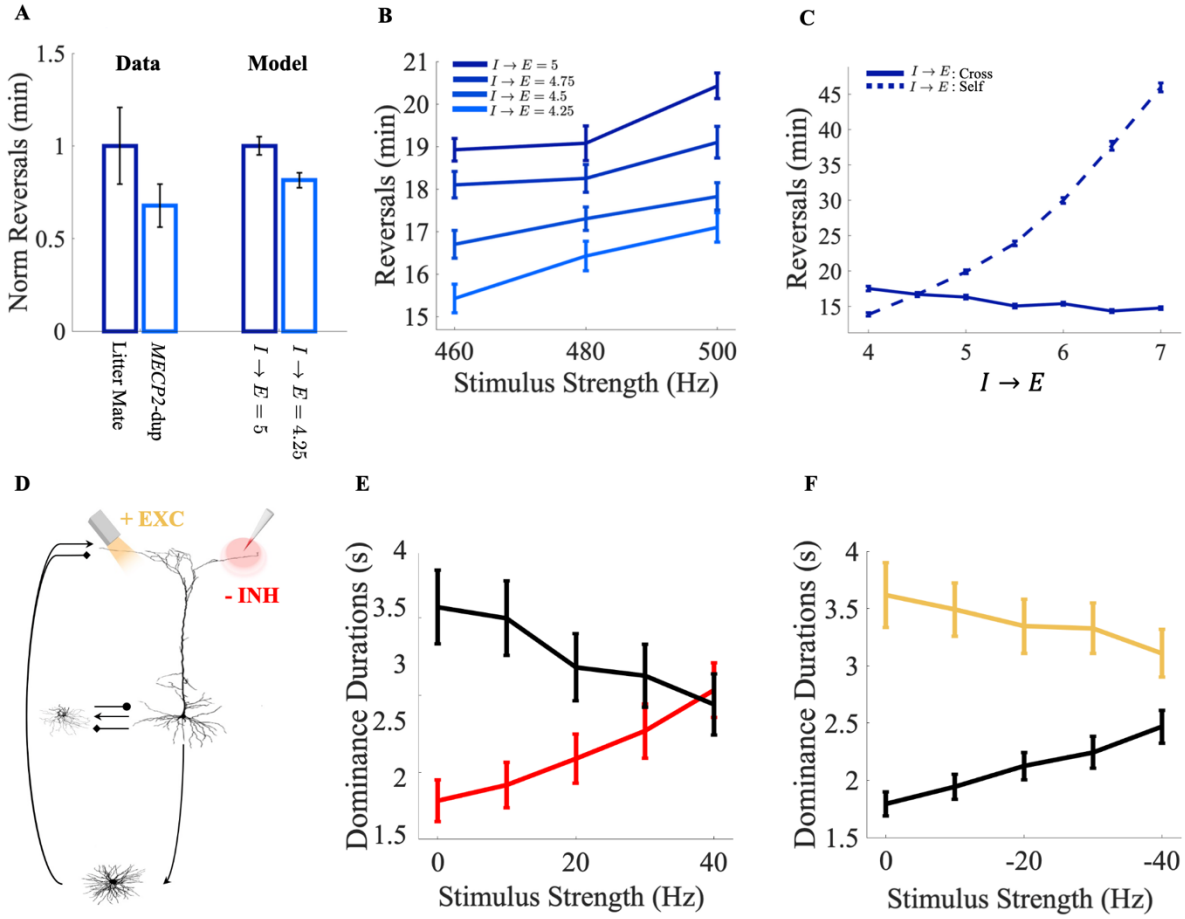

Figure 2. A) Comparison of empirical data acquired from figure 1 of Bogatova et al, (2022) showing reversal frequency between mice with and without a *MECP2*-duplication (which predominantly effects GABAergic signalling; Chao et al., 2010) with model output for $I \rightarrow E = 4.25$ and $I \rightarrow E = 5$. **B)** Effect of EI balance on perceptual reversal frequency across external drive frequencies. Decreasing the amplitude of $I \rightarrow E$ coupling slows rivalry. Reversal frequency was normalised by the max value of the mean. **C)** Effect of manipulating $I \rightarrow E$ coupling amplitude on perceptual reversal frequency for cross-inhibition (solid line) and self-inhibition (dashed line). **D)** Model thalamocortical loop highlighting the perturbation to apical dendrites designed to mimic optogenetic excitation (yellow), and pharmacological inhibition through the puff application of baclofen (red). **E)** Dominance duration as a function of the difference in external drive frequency entering the inhibited population. Red line shows the dominance duration of the population with inhibited apical dendrites and increasing basal drive. Black line again shows the dominance duration of the population with unperturbed apical dendrites and fixed basal drive. **F)** Dominance duration as a function of the difference in external drive entering the excited population. Yellow line shows the dominance duration of the population with excited apical dendrites and decreasing basal drive. Black line shows the dominance duration of the population with unperturbed apical dendrites and fixed basal drive. In all plots error bars show the standard error of the mean.

To test this in our model, we ran a series of simulations across a range of $I \rightarrow E$ coupling amplitudes from 4.25 to 5 in steps of $.25$ and, to ensure robustness, across a range of external drive frequencies (460 Hz, 480 Hz, 500 Hz). Consistent with the findings from the rodent model across the full range of external drive reducing the amplitude of inhibitory signalling reduced the rate of perceptual reversals (**Fig 2B**).

To understand the mechanism behind the decrease in reversal rates, we decomposed $I \rightarrow E$ coupling into local self-inhibitory connections and long-range cross-inhibitory connections. We then ran a series of simulations sweeping over a large range of $I \rightarrow E$ amplitudes from 4 to 7 in steps of $.5$ for both self-inhibitory and cross-inhibitory connections with a fixed external drive of 475 Hz. We settled on a lower bound of 4 for the coupling amplitude, as moving below this value resulted in unrealistically large excitatory conductances. We found that decreasing the coupling amplitude of cross-inhibition from 7 to 4 in isolation increased the rate of perceptual reversals, in line with the behaviour of existing mean field models of rivalry but in contradiction to empirical data. In contrast, when we decreased the coupling amplitude of self-inhibition in isolation, the rate of perceptual reversals decreased in line with empirical results (**Fig 2C**). Importantly, changes in self-inhibition had a vastly larger effect on the frequency of perceptual reversals compared to equivalent changes in cross-inhibition explaining the net slowing of perceptual reversals with decreasing $I \rightarrow E$ values. This suggests that the effect of *MECP2*-duplication on reversal rates is primarily mediated by the decrease in local self-inhibitory connectivity. This explains why the result was not predicted by mean field models of rivalry which do not include self-inhibition in the reduced description of neural activity. Finally, we note that the decrease in perceptual reversal rate as a function of decreased inhibition is redundant across $I \rightarrow E$ and $E \rightarrow I$ coupling parameters (see parameter sweeps in **supplementary material S1**). In fact, there is evidence that *MECP2*-duplications may (also) reduce inhibition through a reduction in basket cell excitability (Mierau et al., 2016) a mechanism which is consistent with the redundancy shown by our model.

In Silico **Cellular Perturbation Experiments**

We next ran a series of *in silico* perturbation experiments with the aim of interrogating the contribution of the apical compartment to perceptual dominance and to generate a set of novel cellular-level predictions. Specifically, we set out to mimic the optogenetic and pharmacological experiments carried out by Takahashi et al (2016, 2020) in the context of

threshold detection that originally motivated the hypothesis that bursting in LV_{PT} cells is the mechanism of conscious perception (Aru et al., 2019; Aru, Suzuki, et al., 2020). Based on the gain modulating effect of apical spikes (**Fig 1C**) at the single cell level, we hypothesised that exciting/inhibiting the apical dendrites on one side of the ring should be approximately equivalent to increasing/decreasing basal drive entering the population of neurons. Specifically, we predicted that artificially inhibiting the apical dendrites of a population should be comparable to decreasing their basal drive leading to a decrease in the dominance duration of the inhibited population and an increase in the uninhibited population. This difference should also be able to be compensated for by increasing the basal drive entering the inhibited population and should also hold in reverse for excitation.

To test this hypothesis in our model, we added a constant source of external drive ($\pm$ 100 pS) to the apical dendrites on one side of the ring that was designed to mimic optogenetic excitation or pharmacological inhibition (e.g., via puff application of baclofen) of the apical dendrites within an ocular dominance column (**Fig 2E**). Across a series of simulations we gradually increased/reduced the basal drive entering the inhibited/excited population from 480 Hz to 520 Hz for the inhibition simulation, and 480 Hz to 440 Hz for the excitation simulation (both in steps of 10 Hz). In agreement with our hypothesis, inhibition of the apical dendrites led to a dramatic increase in the dominance duration of the non-inhibited population relative to the inhibited population. This difference was gradually compensated for as basal drive into the inhibited side of the ring was increased (**Fig 2F**). Similarly, excitation of the apical dendrites led to a substantial jump in the average dominance duration of the excited population relative to the non-excited population, which was gradually compensated for by decreasing the basal drive into excited side of the ring (**Fig 2G**).

Attention and Expectation as Apical Gain Control

Both non-specific matrix thalamic and cortical feedback to superficial layers of cortex (i.e., apical dendrites of $L5_{PT}$ cells) have been linked to the cognitive capacities of attention (Halassa & Kastner, 2017; Kastner et al., 2020) and expectation (Bastos et al., 2020; Raizada & Grossberg, 2003), respectively. We reasoned that by manipulating these structures in our model (**Fig 3A**) – to mimic the downstream neural effects of attention and expectation – we should be able to reproduce the effects of attention and expectation on rivalry and in so doing provide a link between our cellular level model and the more sophisticated rivalry tasks studied in humans which manipulate these cognitive capacities (Denison et al., 2011; Paffen & Alais,

2011; Pinto et al., 2015). Importantly, we would like to emphasise that we only aimed to model the down-stream neural effects of attention and expectation, ignoring the more cognitive aspects of each capacity such as the voluntary direction of attention following a cue, or the formation of expectation through exposure to statistical regularities.

In the context of binocular rivalry, attention has been shown to modulates the relationship between dominance durations and stimulus strength. Specifically, reminiscent of Levelt's fourth proposition, Paffen et al (2006) showed that the removal of attention from the rivalry stimuli, through the addition of a motion discrimination distractor task (**Fig 3B**), caused the rate of perceptual reversals to slow as a function of the difficulty of the distractor task. They repeated the task across a range of stimulus contrasts and found that removing attention from the stimulus was equivalent to an decrease in stimulus contrast positing a gain control mechanism of attention as an explanation (also see John et al., 2018; Paffen & Alais, 2011). Based on the finding that non-specific matrix thalamus increases cortical excitability, enhancing the response to task-relevant stimuli (Schmitt et al., 2017; Zhou et al., 2016), and the existence of recurrent (closed loop) connections between non-specific matrix thalamus and LV_{PT} cells (Guo et al., 2020; Harris & Shepherd, 2015; Mease & Gonzalez, 2021; Wimmer et al., 2010), we hypothesised that the role of attention in rivalry could be reproduced by selectively modulating the amplitude of thalamic feedback onto apical dendrites in our model, which, consistent with the gain control mechanism posited by Paffen et al (2006), should speed perceptual reversals equivalent to an increase in external drive. To test this hypothesis, we conducted a series of simulations in which we increased the spike rate of the external drive from 475 Hz to 520 Hz in steps of 15 Hz, and increased the amplitude of thalamic feedback from 1 to 8 in steps of 3. In line with our hypothesis, at a fixed external drive, stepwise increases in the amplitude of thalamic feedback resulted in a stepwise increase the rate of perceptual reversals recapitulating the parametric effect of distractor task difficulty (**Fig 3C**). Similarly, across the full range of external drive rates, we found that increasing the amplitude of thalamic feedback from 5 to 8 was approximately equivalent to our 15 Hz step increase in external drive (**Fig 3 D-E**).

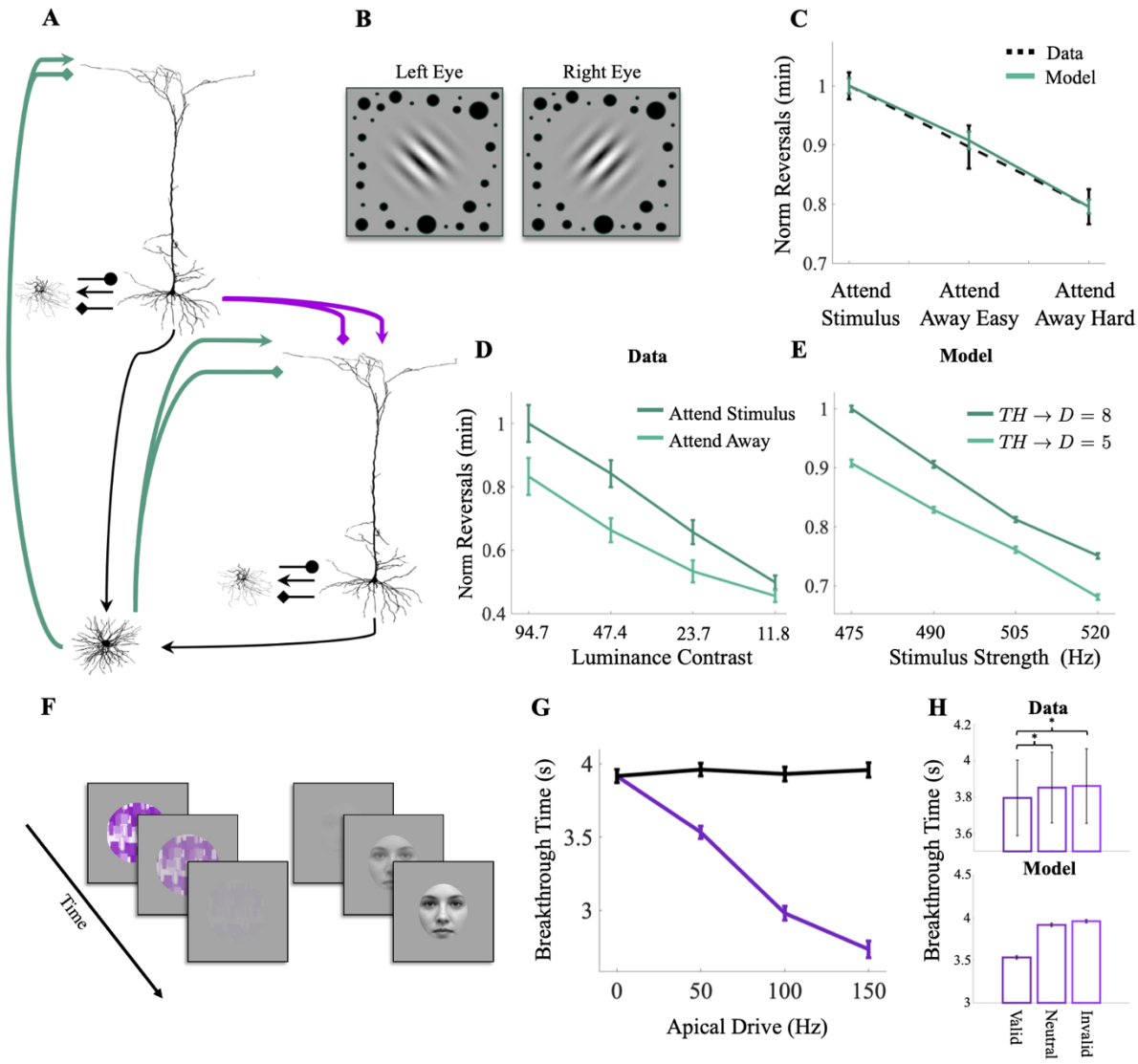

Figure 3. A) Model thalamocortical loop highlighting the closed loop projections from non-specific matrix thalamus to the apical dendrites of LV_{PT} neurons (green) and the projections from deep layers of "higher" regions of the cortical hierarchy to superficial layers of "lower" cortical regions (purple) simulated in our model by an (additional) external source of apical drive. **B)** Modified binocular rivalry task of Paffen et al, (2006). Motion discrimination distractor task in periphery required participants to track the overall direction of the moving dots allowing them to remove attention in a parametric manner. **C)** Comparison of empirical data taken from figure 1A of Paffen et al, (2006) and model output. The allocation of attention to the rivalry stimuli was modelled through a stepwise increase in the amplitude of thalamic feedback to the apical dendrites $\lambda^{TH \rightarrow D} = 1, 5, 8$. Reversal frequency was normalised by the max value of the mean. **D)** Empirical data of Paffen et al, (2006) adapted from figure 1C showing the equivalence between the allocation of attention and a stepwise increase in stimulus contrast. **E)** Model output of simulation designed to mimic the experiment of Paffen et al, (2006). **F)** Continuous flash suppression task of Pinto et al, (2015), mask stimulus entering one eye starts at full contrast and linearly decreases whilst the house stimulus entering the other eye starts at zero contrast and gradually increases. All trials start with a cue (not shown) which probabilistically indicates the category (e.g. face or house) of the low contrast stimulus. Pinto et al, (2015) used a range of response conditions including detection, identification, recognition, and breakthrough. As our model does not have the granularity to distinguish between these types of decision criteria

we focus on experiment 4 which examined the most basic - breakthrough time. **G)** Model breakthrough times as a function of apical drive (Hz) for valid (purple) and invalid (black) expectation conditions. **H)** Comparison of empirical data of Pinto et al, (2015) experiment 4 (adapted from figure 5) with model breakthrough times. In all plots error bars show the standard error of the mean.

Expectation, like attention, has been linked to feedback projections terminating in superficial layers of cortex, however, in the case of expectation, the source of the feedback projections is hypothesized to originate from deep layers of anatomically "higher" cortical populations (Bastos et al., 2012, 2015, 2020; Friston, 2005, 2018; Grossberg, 1995; Raizada & Grossberg, 2003). In the context of rivalry, predictive cues shown before the onset of rivalry stimuli bias which stimulus wins the initial competition for dominance towards the expected stimulus (Denison et al., 2011; Valuch & Kulke, 2020). Similarly, in continuous flash suppression (Tsuchiya and Koch, (2005); **Fig 3F**) – a variation of visual rivalry where participants are presented with a high contrast image to one eye that gradually decreases in contrast and a low contrast image to the other eye that gradually increases in contrast – expectation has been shown to decrease the time it takes for the low contrast image to "break through" and dominate perception relative to invalid or no expectation (neutral) conditions, a finding that has been conceptually replicated across a range of experimental conditions (Pinto et al., 2015).

We hypothesised that the empirically observed effect of expectation should be reproduced by introducing an external source of apical drive to one side of the ring representing content specific predictive feedback elicited by a predictive cue. Specifically, breakthrough time should decrease as a function of apical drive by increasing the amount of time each neuron spends in burst mode and be unaffected by invalid expectations which target the apical dendrites of neurons receiving insufficient basal drive to emit a spike. To test this hypothesis, we introduced an additional source of external drive that exclusively targeted the apical compartment of the model mimicking top-down feedback from an anatomically higher cortical region. To highlight the ability of our model to generalise beyond the classic binocular rivalry paradigm, we focused our simulations on the continuous flash suppression finding (for details see **materials methods**). To model the effect of valid expectations we centred the apical drive (which decayed according to a spatial Gaussian) on the same orientation as either the left or right eye stimulus. Similarly, we modelled invalid expectations by centring the apical drive on an orientation half-way between the orientations of left and right eye stimuli targeting a portion of the ring that received almost no basal drive. For both valid and invalid expectation conditions

we ran a series of simulations across a range of apical drive frequencies from 0 Hz to 150 Hz in steps of 50 Hz. In line with our hypothesis, breakthrough time decreased as a function of the apical drive frequency for valid but not invalid expectations which had no effect on breakthrough time (**Fig 3G**). At low values of apical drive (50 Hz), breakthrough times for valid, neutral (i.e., no apical drive), and invalid expectations, were comparable to the empirical data of Pinto et al, (2015) (**Fig 3 H**).

Discussion

The study of conscious perception in cognitive and systems neuroscience has so far proceeded largely in parallel – the former exploring the largescale neural dynamics and behavioural signatures of conscious perception across a rich array of experimental settings, and the latter characterising the cellular circuitry of perception in exquisite detail, and with precise causal control, but in highly constrained experimental settings. Leveraging a neurobiologically detailed model of the thalamocortical circuit, we have shown that a potential circuit-level mechanism of perceptual consciousness discovered in rodents (apical-mediation of bursting in LV_{PT} neurons; Aru et al., 2019, 2019; Takahashi et al., 2016, 2020) generalises to binocular rivalry – a core task used to study conscious perception in human cognitive neuroscience (Crick & Koch, 2003; Giles et al., 2016; Overgaard, 2015; Tong et al., 2006). In this way, we provide a roadmap for linking neuroscientific explanations of experimental paradigms relevant to the science of consciousness across spatial scales.

A crucial aspect this process is striking the right balance between neurobiological detail and interpretability. In line with our goal of exploring the generality of apically-mediated bursting in LV_{PT} we only included the elements of the thalamocortical circuit in our model that have been directly implicated in empirical work linking LV_{PT} bursting to conscious perception. Due to the fact that the same (or similar) aspects of visual rivalry can be studied in humans, non-human primates, and rodent models, it is possible to integrate insights across species into a single model, and to generate predictions at each scale. Indeed, in the initial simulations we validated our model psychophysically by showing that it was consistent with Levelt's laws and produced a right-skewed distribution of dominance durations findings that were initially discovered in humans (Levelt, 1965, 1967) and have been shown to generalise to non-human primates (e.g. Leopold & Logothetis, 1996)

The combination of neurobiological detail and transparency afforded by our model also allowed us to simulate experimental interventions that would not be possible in human or non-human primates with a degree of precision that is typically not found in largescale models of rivalry (Curtu et al., 2008; Laing & Chow, 2002; Wilson, 2007). Indeed, we were able to reproduce the finding that mice with a *MECP2*-duplication have a reduced rate of perceptual reversals compared to their healthy litter mates. Based on the finding that *MECP2*-duplication reduces inhibitory IPSP currents (Chao et al., 2010) we simulated the resulting changes to E/I balance by increasing the amplitude of $I \rightarrow E$ coupling. By decomposing inhibitory coupling in the model into self-inhibition and cross-inhibition and sweeping the amplitude of each individually we found that they had opposing effects (**Fig. 2C**). Decreasing self-inhibition reduced perceptual reversals, and consistent with mean field models of rivalry, decreasing cross-inhibition increased perceptual reversals (Curtu et al., 2008; Laing & Chow, 2002; Wilson, 2007). Changes in self-inhibition, however, had a much larger effect than changes in cross-inhibition so when we changed them in concert the net effect was to slow rivalry. Importantly, this result has a degree of redundancy in our model with reductions in the amplitude of both $I \rightarrow E$, and $E \rightarrow I$ coupling leading to a slowed rate of perceptual reversals (see parameter sweeps in **supplementary material S1**). We suspect that both $I \rightarrow E$ and $E \rightarrow I$ coupling are affected *in vivo* as *MECP2*-duplications have also been shown to reduce basket cell excitability (Mierau et al., 2016).

The model's layer-specific structure allowed us to mimic the optogenetic excitation and pharmacological inhibition of apical dendrites originally carried out by Takahashi et al (2016, 2020) in the context of threshold detection in the setting of visual rivalry leading to a novel prediction. Specifically, because the apical drive to LV_{PT} cells modulates the gain of the somatic compartment, inhibiting the apical dendrites of an ocular dominance column should be equivalent to a step decrease in stimulus strength (i.e., luminance contrast) leading to a decrease in the dominance duration of the stimulus associated with the inhibited population and a corresponding increase in the unperturbed stimulus percept. This finding also holds in the opposite direction – i.e., for optogenetic excitation of apical dendrites (**Fig. 2E-F**).

We then leveraged the thalamocortical connectivity backbone to simulate the downstream neuronal effects of attention (Paffen & Alais, 2011) on visual rivalry. There is a growing body of work linking non-specific matrix thalamus to attention through the boosting of task-relevant

feed-forward input (Halassa & Kastner, 2017; Shine et al., 2023). Briefly, in non-human primates, inhibition of the pulvinar (a region containing a high proportion of matrix cells) diminishes cortical responses to task relevant stimuli (Zhou et al., 2016), lesions to pulvinar result in an inability to ignore distractors (Kastner et al., 2020), and in rodents increasing the excitability of medial dorsal nuclei (again a region containing matrix-like cells) increases cortical excitability (Schmitt et al., 2017). We modelled this effect by increasing the amplitude of the coupling ($TH \rightarrow D$) between our matrix thalamus population and the apical compartment of our LV_{PT} cells, which arguably form a closed loop (Guo et al., 2020; Mease & Gonzalez, 2021; Wimmer et al., 2010) increasing the gain of the somatic compartment and thereby speeding perceptual reversals analogous to Levelt's fourth proposition. This reproduced the findings of Paffen et al (2006) and provided a neurobiological mechanism for their attention as gain modulation hypothesis (Paffen & Alais, 2011).

We showed that the biasing effect of expectation on the initial competition for perceptual dominance in rivalry was well described by the introduction of an additional source of apical drive, reproducing the empirically observed effect of both valid and invalid expectation on breakthrough time in continuous flash suppression (Pinto et al., 2015). A notable difference between our spiking model and canonical algorithmic models of top-down prediction which incorporate an explicit role for expectation such as predictive coding is that in our model, in line with empirical recordings from LV_{PT} cells, feedback has an amplifying effect on neural activity similar to adaptive resonance theory (Grossberg, 1995; Raizada & Grossberg, 2003), whereas, in most predictive coding models (Bastos et al., 2012; Friston, 2018; Rao & Ballard, 1999) feedback suppresses activity. We note, however, that this is not true of all predictive coding models, some implementations cast feedback as excitatory (Spratling, 2017). In addition, the implementation of predictive coding in spiking neurons is an open question and the effect of feedback is likely to be layer-specific.

To strike the right balance between neurobiological detail and interpretability, we made a number of simplifying assumptions that place notable limitations on our model. Most notably, we did not include a LII/III pyramidal neuron population – which are the primary source of long distance horizontal connections in the cortex (Douglas & Martin, 2004) – nor a specific core thalamic population which forms a targeted recurrent loop with LIV and LVI of cortex (Harris & Shepherd, 2015) preventing us from performing systematic lesion experiments on

our model highlighting the precise function of LV_{PT} cells and non-specific matrix thalamus in a more realistic cortical microcircuit. In addition, we did not include time delays between our corticocortical or thalamocortical connections preventing our model from providing a realistic model (e.g., Tahvili & Destexhe, 2023) of time-frequency components of common electrophysiological measures such as local field potentials which have played a large role in the empirical literature on visual rivalry in non-human primates.

By building a model based on circuitry identified in a rodent model of conscious perception that is able to reproduce the behaviour of human participants, we hope to help bridge the gap between the cognitive and cellular level study of consciousness. With this aim in mind, we conclude by highlighting three additional predictions that bridge microscopic and macroscopic scales. One, perceptual dominance should be characterised by bursting in LV_{PT} cells, a prediction that with recent advances in recording technology (i.e., primate neuropixel probes; Trautmann et al., 2023) and experimental techniques (Bogatova et al., 2022; Palagina et al., 2017) can be tested in both mouse models and non-human primates. Two, changes in perceptual dominance in visual rivalry should be evident in matrix nuclei of the thalamus (which forms a closed loop with LV_{PT} cells; Mease & Gonzalez, 2021). This prediction has, in fact, already received partial support from a recent fMRI study investigating the neural correlates of perception in a bistable colour illusion paradigm which found that matrix nuclei but not core nuclei distinguished between fused and non-fused perception (Seo et al., 2022). Three, a direct prediction of the model is that expectation, like attention, modulates the gain of LV_{PT} cells. Therefore the biasing effect of predictive cues on the initial competition for dominance in binocular rivalry should be equivalent to a stepwise increase in stimulus strength to the same eye, and should be compensated for by a stepwise decrease in stimulus strength to the same eye analogous to experiments on the role of attention in rivalry (Paffen & Alais, 2011; Paffen et al., 2006)

Materials Methods

The neuronal backbone of the model consists of Izhikevich quadratic adaptive integrate and fire neurons, a variety of hybrid dynamical system that, with biophysically fit parameters, is capable of reproducing a wide variety of the spiking behaviour whilst still being highly efficient to integrate numerically (Izhikevich, 2003, 2004, 2006).

$$C\dot{v} = k(v(t) - v_r)(v(t) - v_t) - u(t) + I_{ext} \tag{1}$$

$$\dot{u} = a\{b(v(t) - v_r) - u(t)\} \tag{2}$$

With the following reset conditions: if $v \geq v_{peak}$ then $v \rightarrow c, u \rightarrow u + d$. The equations are in dimensional form giving the membrane potential (including the resting potential v_r, spike threshold v_t, and spike peak v_{peak}), input I_{ext}, time t, and capacitance C, biophysically interpretable units (i.e., milivolts, picoamperes, milliseconds, and picofarads, respectively).The remaining three parameters k, a, and b, are dimensionless and control the timescale of the membrane potential, the timescale of spike adaptation, and the sensitivity of spike adaptation to sub-threshold oscillations. Crucially, Izhikevich (Izhikevich, 2006; Izhikevich & Edelman, 2008) fit parameters for a large class of cortical and sub-cortical neurons, thus affording our model a high degree of neurobiological plausibility while greatly reducing the number of free parameters.

The model included three classes of neurons: LV_{PT} cells, fast spiking interneurons (basket cells), and matrix thalamic cells (**Fig 1 A-C)**. The parameters for the fast-spiking interneurons and thalamic cells were taken unmodified from Izhikevich and Edelman (2008). For the LV_{PT} cells we employed a novel dual compartment neuron with (coupled) apical and basal compartments, each described by equations (1) to (2), allowing us to reproduce the change in dynamical regime in LV_{PT} cells that occurs when they receive coincident apical and basal drive. We leveraged the insight (Izhikevich, 2003; Munn, Müller, Aru, et al., 2023; Munn, Müller, Medel, et al., 2023) that the difference between regular spiking and bursting behaviour can be modelled by changing the reset conditions of equations (1) and (2) - raising the reset voltage (increasing c) taking the neuron closer to threshold, and reducing the magnitude of spike adaptation (decreasing d). We set up the coupling between compartments such that the apical compartment controlled the reset conditions on the somatic compartment with an apical spike switching the reset conditions of the somatic compartment from regular spiking to bursting

parameters for 25 ms. In turn, the membrane potential of the apical compartment was coupled to the somatic compartment by adding a conductance term ($20(v_{soma} - v_{dendrite})$) to equation (1) allowing for somatic spikes to back propagate and contribute to the generation of spikes in the apical compartment. Parameters of equations (1) – (2) for each neuron/compartment are given in **table 1**.

Neuron	C	k	v_r	v_t	a	b	c	d	v_{peak}
LV_{PT} dendrite	100 pF	1	− 50 mV	− 50 mV	0.8	15	−10	500	20 mV
LV_{PT} soma	150 pF	1.2	− 75 mV	− 45 mV	0.01	5	Reg: − 66 Burst: − 56	Reg: 260 Burst: 130	50 mV
Basket cell	20 pF	1	− 55 mV	− 40 mV	0.15	8	− 55	200	25 mV
Matrix thalamus	200 pF	1.6	− 60 mV	− 50 mV	0.01	15	− 60	10	35 mV

Table 1. Parameters for each class of Izhikevich neuron. LV_{PT} dendrite and soma parameters were modified from the active dendrite multi-compartment model (p.293) and intrinsic bursting model (p.290) described in Izhikevich (2006). Basket cell (fast spiking interneuron), and matrix thalamus parameters were taken, unmodified, from Izhikevich and Edelman (2008).

Based on previous spiking neural network models of rivalry (Laing et al., 2010; Laing & Chow, 2002; Wang et al., 2020) the cortical component of the network had a one-dimensional ring architecture. Each point on the ring represents an orientation preference with one full rotation around the ring corresponding to a 180° visual rotation. This mirrors the fact that a 180° rotation of a grating results in a visually identical stimulus and also ensures periodic boundary conditions. The cortical ring contained 90 LV_{PT} neurons and 90 fast spiking interneurons. Each pair of excitatory and inhibitory neurons was assigned to an equidistant location on the ring (unit circle) giving each neuron a 2° difference in orientation preference relative to each of its neighbours. The (dimensionless) synaptic weights $w_{i,j}^{\kappa\omega}$ connecting neurons ($E \to E$, $I \to E$, and $E \to I$), were all to all with magnitudes decaying as a function of the Euclidean distance $\delta_{i,j}$ between neurons (equations (3)) according to a spatial Gaussian footprint (equation (4)).

$$\delta_{i,j} = \sqrt{(\cos\theta_i - \cos\theta_j)^2 + (\sin\theta_i - \sin\theta_j)^2} \tag{3}$$

$$w_{i,j}^{\kappa\omega} = \lambda^{\kappa\omega} e^{-\frac{1}{2}\left(\frac{\delta_{i,j}}{\sigma^{\kappa\omega}}\right)^2} \tag{4}$$

Where θ is the location of the neuron on the unit circle, κ and ω denote the pre- and post-synaptic neuron, λ controls the magnitude of the synaptic weights, and $\sigma^{\kappa\omega}$ the spatial spread. In line with empirical constraints inhibitory coupling had a larger spatial spread and magnitude than excitatory to excitatory coupling (Naka & Adesnik, 2016). Each 9 cortical neurons were coupled to 1 thalamic neuron which then projected back up to the apical dendrites of the same 9 cortical neurons in line with the diffuse projections of higher order thalamus onto the apical dendrites of LV_{PT} neurons in layer I (Mease & Gonzalez, 2021; Shepherd & Yamawaki, 2021). For simplicity we set projections to from the thalamus to a constant value (e.g., $w_{i,j}^{TH\rightarrow D} = \lambda^{TH\rightarrow D}$). Synapses had a bi-exponential form (equation (5)) with rise (τ_{rise}) and decay (τ_{decay}) constants chosen to recapitulate the dynamics of inhibitory ($GABA_A$), and excitatory (AMPA and NMDA) synapses (Dayan & Abbott, 2005; Gerstner et al., 2014).

$$g_i(t)_{syn} = \sum_j w_{i,j}^{\kappa\omega} g_\infty \left(1 - e^{-\frac{(t-t_j^f)}{\tau_{rise}}}\right) e^{-\frac{(t-t_j^f)}{\tau_{decay}}} \Theta(t - t_j^f) \tag{5}$$

We set g_∞ to 1 for $GABA_A$ and AMPA synapses, and $g_\infty = \frac{1}{1+[Mg^{2+}]\beta e^{-\alpha v_i(t)}}$ for NMDA synapses making the conductance voltage sensitive, where Θ denotes the Heaviside function, and t^f is the time that the neuron passes the threshold $v \geq v_{peak}$. The conductance term entered into the input I_{ext} through the relation $I_{i,syn} = g_i(t)_{syn}(E_{syn} - v_i(t))$ where E_{syn} is the reverse potential of the synapse.

External drive into the model exclusively targeted the somatic compartment of LV_{PT} cells representing input into the basal dendrites. We decomposed the external drive into four terms, $I_{ext} = I_C + I_{AMPA} + I_{NMDA} + I_{adapt}$, consisting of a (subthreshold) constant drive (I_C), two glutamatergic conductance based terms ($I_{AMPA} + I_{NMDA}$), and a slow hyperpolarising adaptation current I_{adapt}. We obtained I_{AMPA} and I_{NMDA} by convolving a Poisson spike train with equation (5). Separate monocular inputs into the network were modelled with independent Poisson processes (representing input from the left and right eyes with rates varying between 450 Hz and 525 Hz depending on the simulation). The constant drive took the neuron closer to threshold reducing the firing rate of the conductance based input necessary to drive the model circumventing a number of known numerical issues that arise when conductance values get too

large (Izhikevich, 2010). The adaptation current was described by $I_{adapt} = g_i(t)_{adapt}(E_{adapt} - v_i(t))$ with $g_i(t)_{adapt}$ obtained from numerical integration of equation (6).

$$\dot{g}_{adapt} = -\frac{g(t)_{adapt}}{\tau_{adapt}} + \Delta g \delta(t - t^{tf}) \tag{6}$$

Where Δg is the contribution of each spike to the hyperpolarising current and τ_{adapt} the time constant. External drive (with the exception of I_{adapt}) was weighted by the spatial Gaussian shown in equation (7) centred on neurons 90° apart on the ring (i.e., 45 neurons apart) corresponding to the orthogonal grating stimuli commonly employed in binocular rivalry experiments.

$$h_i = e^{-\left(\frac{(i-N_L)}{\sigma^{\varepsilon}}\right)^2} + e^{-\left(\frac{(i-N_R)}{\sigma^{\varepsilon}}\right)^2} \tag{7}$$

Here i denotes the index of the ith neuron, N_L and N_R control the orientation of the stimulus delivered to the left and right eyes, and σ^{ε} controls the spatial spread with the superscript indicating the source of the external drive (i.e., feedforward stimulus drive vs feedback projections). To model continuous flash suppression we increased the external drive centred on N_L from 300 Hz to 500 Hz and decreased the external drive entering centred on N_R from 300 Hz to 500 Hz in steps of $\sim$ 3 Hz every 100 ms over a 6 second simulation period.

The equations were integrated numerically in MATLAB using the hybrid scheme introduced by Izhikevich (2010) with a step size of 1 ms, along with a linear interpolation method, that adjusts the integration step size of spike adaptation in line with the timing of the spike (i.e. inferring the value t when $v(t) = v_{peak}$ rather than just taking the value of t when $v(t) \geq v_{peak}$).

Simulations of Levelt's propositions were run for 120 s, all subsequent simulations, which swept over a larger number of parameter, were run for 60 s. To ensure robustness all results shown in the main text were averaged over 40 random seeds. We defined perceptual dominance in terms of the average firing rate of neurons on opposite sides of the ring which we treated as separate populations. Due to the fluctuations in the average firing rate of each population we required that a population have a 5 Hz greater firing rate than the other to be counted as

dominant. Parameters for the model components described by equations (3) – (7) are supplied in **table 2**.

Parameter	Description	Value
$\lambda^{E\to E}$	Amplitude of excitatory to excitatory coupling	$\frac{3.25}{\sigma^{E\to E}\sqrt{2\pi}}$ a.u.
$\lambda^{E\to I}$	Amplitude of excitatory to inhibitory coupling	$\frac{1}{\sigma^{E\to I}\sqrt{2\pi}}$ a.u.
$\lambda^{I\to E}$	Amplitude of inhibitory to excitatory coupling	$\frac{4.5}{\sigma^{I\to E}\sqrt{2\pi}}$ a.u.
$\lambda^{E\to TH}$	Constant excitatory to thalamic coupling constant	4 a.u.
$\lambda^{TH\to D}$	Constant thalamic to apical dendrite coupling	1 a.u.
$\sigma^{E\to E}$	Spread of excitatory to excitatory coupling	0.5 a.u.
$\sigma^{E\to I}$	Spread of excitatory to inhibitory coupling	2 a.u.
$\sigma^{I\to E}$	Spread of inhibitory to excitatory coupling	2 a.u.
τ_{rise}: AMPA	Rise time of AMPA conductance	1 ms
τ_{decay}: AMPA	Decay time of AMPA conductance	8 ms
τ_{rise}: $GABA_A$	Rise time of $GABA_A$ conductance	1 ms
τ_{decay}: $GABA_A$	Decay time of $GABA_A$ conductance	6 ms
τ_{rise}: NMDA	Rise time of NMDA conductance	10 ms
τ_{decay}: NMDA	Decay time of NMDA conductance	75 ms
Mg^{2+}	Extracellular magnesium concentration	1.2 mM
β	Backward rate constant of voltage dependent NMDA conductance	3.57^{-1} mM
α	Forward rate constant of voltage dependent NMDA conductance	0.062 mV
τ_{adapt}	Decay time of adaptation current	2000 ms
Δg	Contribution of each spike to adaptation current	0.045 nS
σ^{ext}	Spatial spread of external drive	10 a.u.
σ^{dend}	Spatial spread of apical drive	5 a.u.
I_C	Constant component of external drive	200 pS

Table 2. Parameter description and values for the components of the model described by equations (3) – (7)

Supplementary Material

S1. Parameter Sweeps

To understand the contribution of each parameter to the model dynamics we ran a sweep across coupling amplitudes anchored around the values supplied in **Table 2**. All sweeps were run with 475 Hz of basal drive for 60 s and averaged across 40 random seeds. Variation in each parameter value behaved as expected. For $E \to E$ coupling the frequency of perceptual reversals decreased as coupling amplitude increased. For low values there was insufficient recurrent excitation in the network to sustain prolonged periods of bursting leading to rapid alternations between populations in a regime dominated by regular spiking (**Fig S1A**). For $E \to I$ coupling, perceptual reversals increased with increased coupling amplitude (**Fig S1B**).

Increasing $I \rightarrow E$ coupling also speeded perceptual reversals but to a lesser degree than $E \rightarrow I$ (**Fig S1C**). In accordance with the gain modulating effect of apical drive increasing the $TH \rightarrow D$ coupling amplitude had a speeding effect on perceptual reversals with a small initial slowing effect for lower parameter values (**Fig S1D**) qualitatively similar to the effect of basal drive shown in simulations of Levelt's fourth proposition (see main text **Fig 1H**). Varying $E \rightarrow TH$ had a very minor speeding effect on the model dynamics (**Fig S1C**). This is an artefact of the small number of thalamic neurons which each received input from 10 LV_{PT} neurons and so were close to the saturation point of their FI curves. In a larger network we expect that varying $E \rightarrow TH$ would have an identical effect to varying $TH \rightarrow D$.

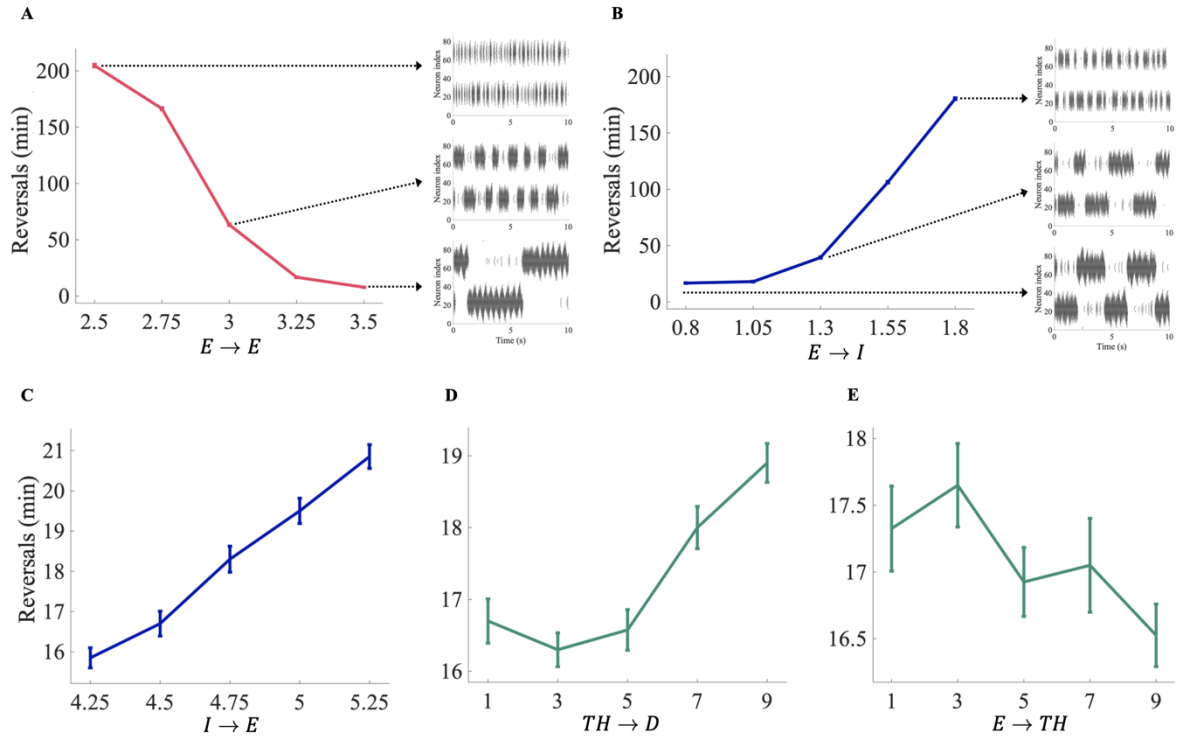

Figure S2. A-E) Alternation frequency (per minute of simulation time) as the amplitude of each corticocortical and thalamocortical parameter is varied. Representative raster plots are shown for $E \rightarrow E = [2.5, 3, 3.5]$ and $E \rightarrow I = [0.8, 1.3, 1.8]$.

S2. Dynamical Regime

To interrogate the structure of the dynamical system underlying the stochastic oscillations we ran the model with constant drive and asymmetric initial conditions that ensured that the system converged to a state where one of the populations was dominant whilst the other was suppressed. We reasoned that if the oscillations were driven by stochastic jumps between stable fixed points with basins of attraction only weakly affected by adaptation then in the absence of noise oscillations should disappear. In contrast, if adaptation exerts a large effect, the stochastic

oscillations should consist of noisy excursions around a stable limit cycle and the model should continue to oscillate in the absence of noise (c.f. Moreno-Bote et al., 2007; Shpiro et al., 2009). In agreement with the stable limit cycle hypothesis in the absence of noise the model continued to oscillate (**Fig S1A**). To test the stability of the limit cycle we: i) confirmed the existence of an unstable structure inside the limit cycle; and ii) confirmed that perturbations to the limit cycle decayed back to a stable orbit. Indeed, with symmetric initial conditions the model converged to a state where excitatory activity on each side of the ring was perfectly matched. Perturbations to this state consisting of a 1 ms pulse of constant drive (50 pS) delivered to the basal dendrites of the somatic compartment of a single LV_{PT} neuron caused the orbit to converge to the surrounding limit cycle (**Fig S1B**). Such perturbations had little effect on already oscillating orbits confirming the stability of the limit cycle (**Fig S1B**).

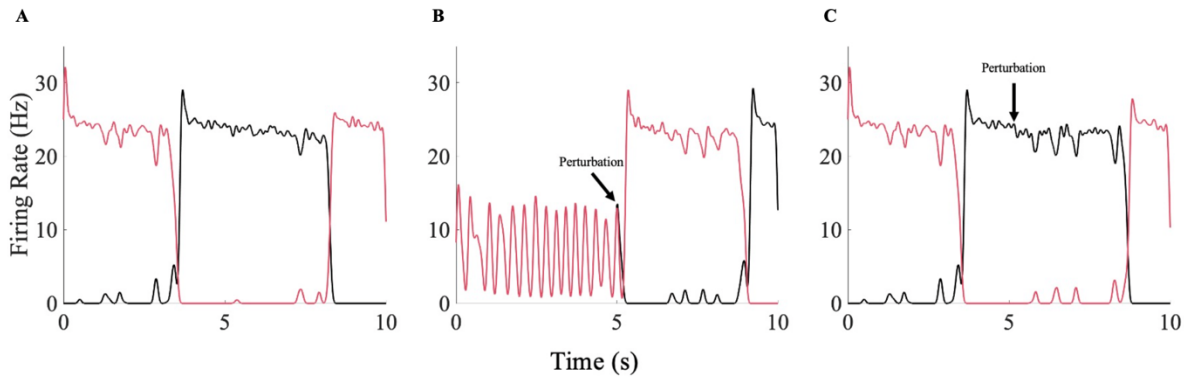

Figure S2. A) Average firing rate of neuronal populations on opposite ends of the ring driven by constant drive with asymmetric initial conditions that ensured the population represented by the pink line would win the initial competition for dominance. **B)** Average firing rate of neuronal populations simulated with constant drive and symmetric initial conditions. A perturbation was delivered at $t = 5000$ sending the population orbit to the surrounding stable limit cycle. **C)** Average firing rate of neuronal populations driven by constant drive with asymmetric initial conditions. Perturbation delivered at $t = 5000$ had no substantial effect on the already oscillating orbit indicative of a stable limit cycle.

Code and Data Availability

MATLAB code illustrating the basic operation of the model can be found at https://github.com/cjwhyte/LV_VisualRivalry. Complete code, and simulated data, will be made available upon full publication of the paper.

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