Neuron, Volume *101*

Supplemental Information

Engagement of Pulvino-cortical Feedforward and Feedback Pathways in Cognitive Computations

Jorge Jaramillo, Jorge F. Mejias, and Xiao-Jing Wang

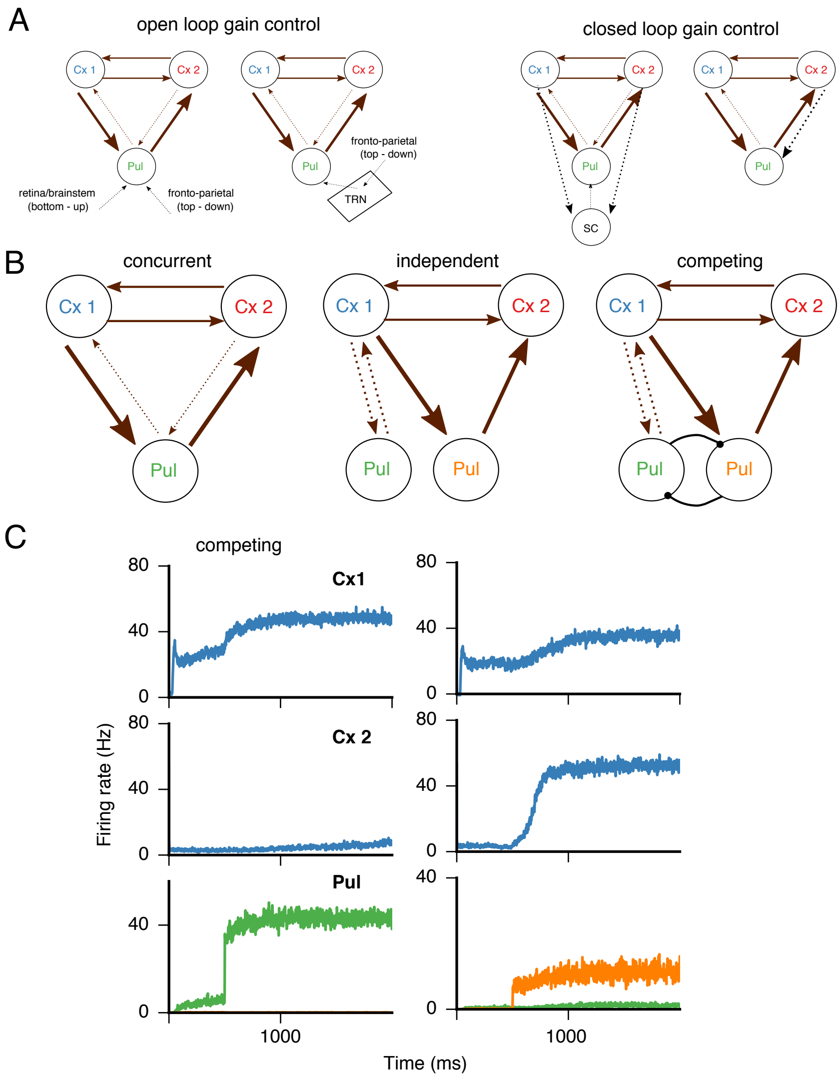

Figure S1: (Related to Fig. 1) Pulvinar gating and pulvino-cortical interaction motifs. ***A,*** Pulvinar excitability can be regulated via different mechanisms. If the origin of the gating signal is external to the pulvino-cortical circuit, we refer to it as open-loop, whereas if the gating signal originates from the circuit itself, we refer to it as closed loop. Note that the gating mechanism might depend on the pulvinar sector being biased, e.g., the ventro-lateral pulvinar might only receive top-down attentional modulation indirectly through the TRN. SC: superior colliculus.***B,*** Three possible architectures connecting pulvinar to cortex: concurrent (left),independent (center), competing (right). ***C,*** For the "competing" architecture in ***B,***, right, there is a tradeoff depending on which of the two pulvinar populations, green or orange, is activated: either a strengthening of a local cortical representation (left, green population active) or propagation of that representation to the next cortical area (right, orange population active). If the strength of recurrent connections is a proxy for noise correlation structure (Helias et al., 2014), this intra-pulvinar competition scenario is consistent with a increase (decrease) of noise correlations across (within) cortical areas as a result of attention (Ruff and Cohen, 2016), here modeled as a bias to a pulvinar population. Furthermore, we suggest that the cortical area whose representation was strengthened will have precedence over the control of subcortical motor centers via projections from layer V axons. This proposal is distinct from - although not necessarily incompatible with - another hypothesis of motor control whereby corticothalamic projections arising from layer V are efference copies relayed to higher cortical areas to monitor impending actions (Sherman and Guillery, 2013; Sherman, 2016)

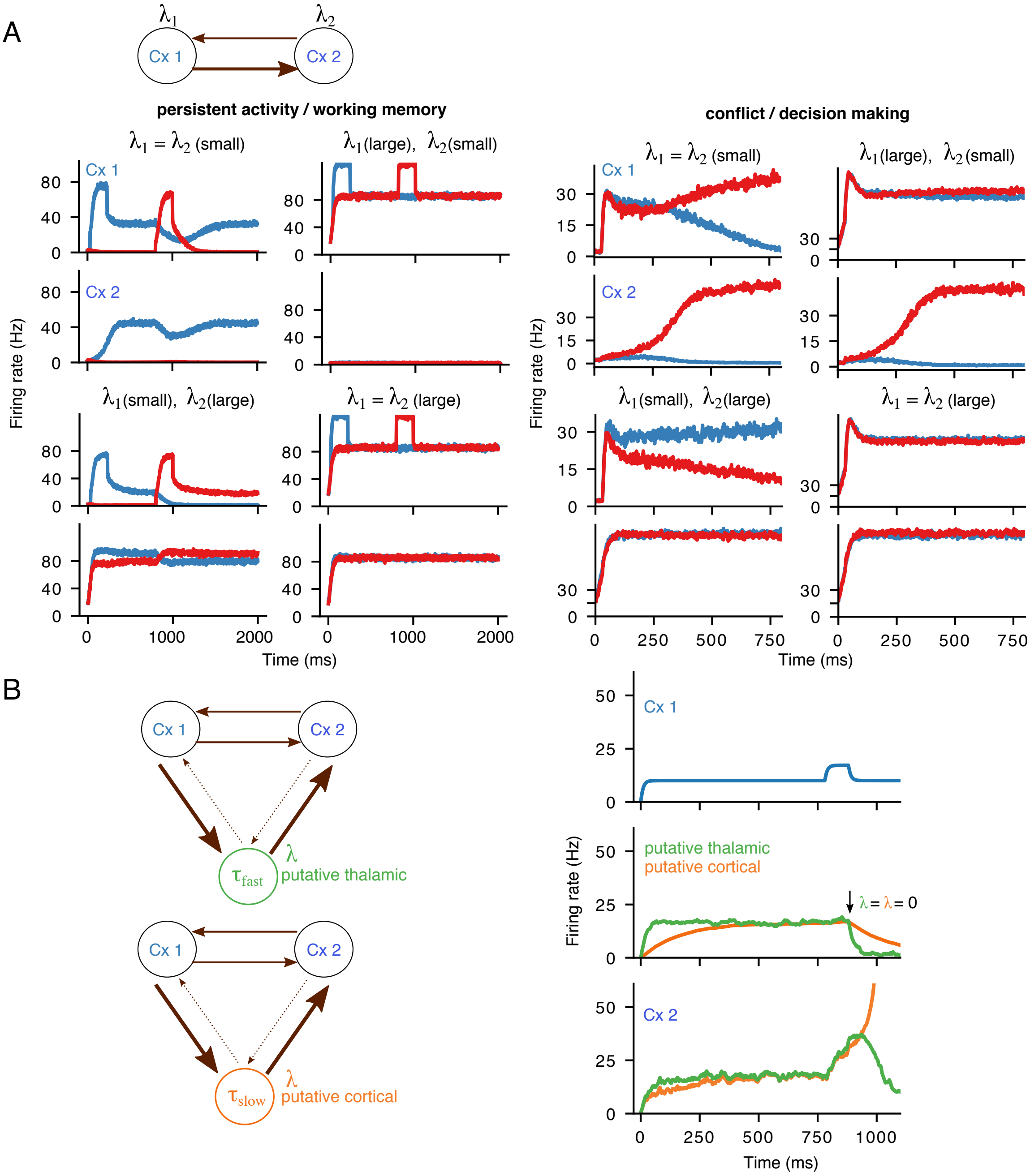

Figure S2: (Related to Figs.3 and 4) Comparison of gating mechanisms for cortico-cortical transmission: cortex-only vs cortex with thalamus ***A***, The schematic depicts a gating mechanism for cortico-cortical transmission (from Cx1 to Cx2) that depends on gain modulation of the cortical areas through the gain parameters λ_1 and/or λ_2. For both working memory (left) and decision-making (right) computations, the proposed cortical gain modulation mechanisms - increasing λ_1 and/or λ_2- is not able to reproduce the results from Figs. 3 and 4, namely, 1) a 'remember last' regime for working memory and 2) conflict resolution in favor of cortical area 1 for decision making. Indeed, direct cortical modulation modifies the dynamical regime of the cortical modules so that well-separated high and low states – for working memory – and winner-take-all competition – for decision making are not easily obtainable. ***B***, We consider a cortico-cortical gating mechanism that involves a third intermediate module which, for comparison, is either putative thalamic (as in the main text) with a time constant $\tau_{\text{fast}} = 20$ ms or putative cortical with a time constant $\tau_{\text{slow}} = 180$ ms (for time constants of spontaneous fluctuations in cortex in vivo see Murray et al. (2014)). We study the transmission of an input signal in Cx1 to Cx2. The input stimulus to the system is applied through cortical area Cx1 (top) and is constant until t = 800 ms, when a 100 ms pulse is subsequently applied. A faster time constant in the putative thalamic module (green, middle) compared to the putative cortical module (orange, middle), results in rapid tracking of the stimulus as also observed in cortical area Cx2 (bottom). Moreover, after the transient pulse in cortical area Cx1, setting the gain of the intermediate module to zero (arrow) results in a rapid cancellation of the input signal only via the putative thalamic area (compare green vs orange, middle). Interestingly, after the gain of the intermediate module is set to zero, the slow decay in the putative cortical module (orange, middle) leads to an undesired transition to the high state in Cx2 (orange, bottom), assuming that Cx2 is bistable.

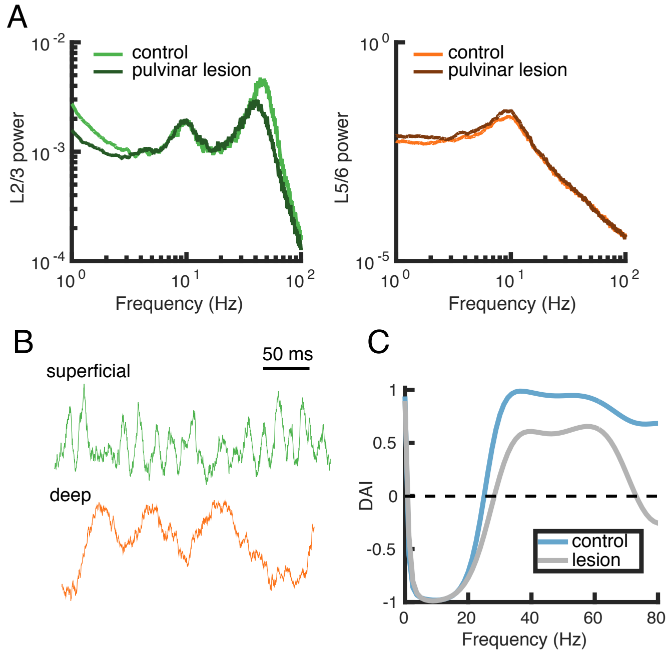

Figure S3: (Related to Fig. 7) Temporal and spectral profiles of the pulvino-cortical circuit before and after pulvinar lesions. ***A,*** Power spectrum for superficial (left) and deep (right) layers for cortical area 1 in control and pulvinar-lesion conditions. ***B,*** Example oscillatory firing-rate traces for superficial and deep layers in cortical area 1. ***C,*** The directed asymmetry index (DAI) for the functional connections between cortical area 1 and cortical area 2 is obtained by normalizing Granger-causality profiles in Fig. 7D.

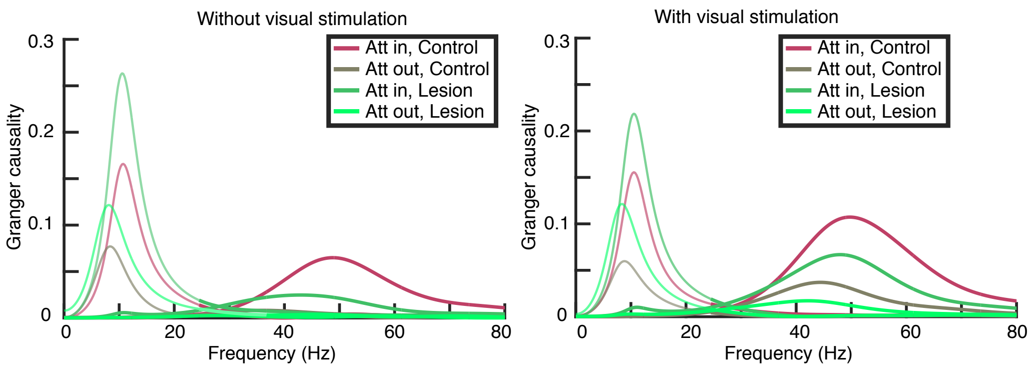

Figure S4: (Related to Fig. 7) Granger interactions for an attentional task without (left) and with (right) visual stimulation. In both scenarios, attention increases interactions between V4 and IT, predominantly in the gamma range (feedforward, V4 $\rightarrow$ IT, thick lines) and alpha range (feedback, IT $\rightarrow$ V4, thin lines). Pulvinar lesions increase Granger interactions in the alpha range but decrease interactions in the gamma range. Both attention and pulvinar lesions have the capability of incrementing alpha power in the circuit, but via different mechanisms (see details in main text). Finally, the absence of visual stimulation is reflected in reduced gamma- and increased alpha-range coupling (see also Saalmann et al. (2012)).

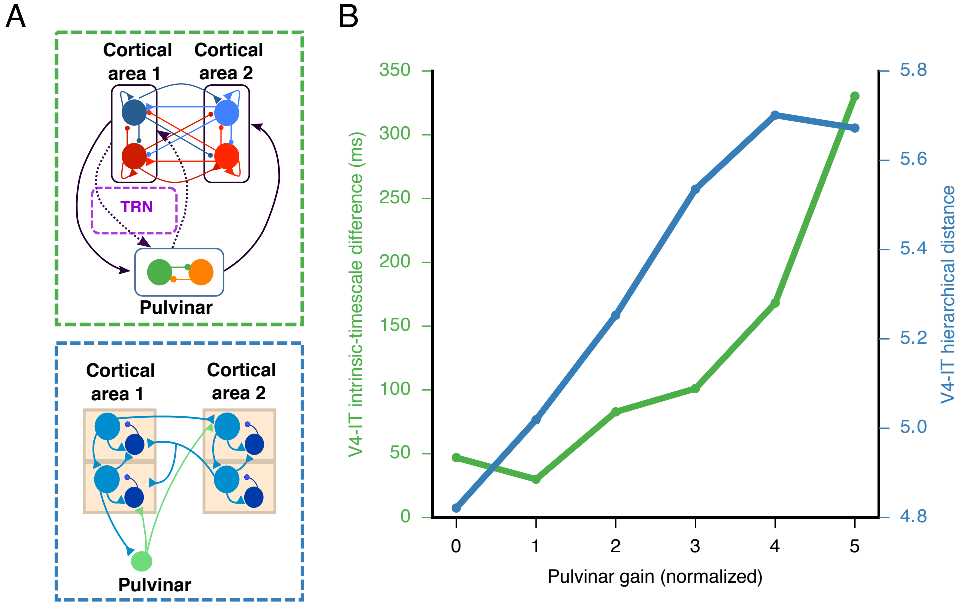

Figure S5: (Related to Fig.1, 3, 4, and 7) Pulvinar gain modulates the hierarchical distance between two cortical areas. ***A,*** Two instantiations of the thalamocortical model: non-linear model for 2AFC tasks (top, see also Fig. 1) and laminar model for oscillatory coupling between areas (bottom, see also Fig.7) ***B,*** Both intrinsic timescale difference (green) as well as oscillation-based hierarchical distance (blue) increase as a function of pulvinar gain.