A spiking neuron model for binocular rivalry

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Abstract. We present a biologically plausible model of binocular rivalry consisting of a network of Hodgkin–Huxley type neurons. Our model accounts for the experimentally and psychophysically observed phenomena: (i) it reproduces the distribution of dominance durations seen in both humans and primates, (ii) it exhibits a lack of correlation between lengths of successive dominance durations, (iii) variation of stimulus strength to one eye influences only the mean dominance duration of the contralateral eye, not the mean dominance duration of the ipsilateral eye, (iv) increasing both stimuli strengths in parallel decreases the mean dominance durations. We have also derived a reduced population rate model from our spiking model from which explicit expressions for the dependence of the dominance durations on input strengths are analytically calculated. We also use this reduced model to derive an expression for the distribution of dominance durations seen within an individual.

1. Introduction

Binocular rivalry occurs when the two eyes are presented with drastically different images. Only one of the images is perceived at a given time, and every few seconds there is alternation between the perceived images. The perceived durations of the images are stochastic and uncorrelated with previous perceived durations (Fox and Herrmann, 1967; Walker, 1975). Also, changing the contrast of the images will change the dominance durations of the perceptions in specific ways.

It is not yet clear exactly what is rivaling during binocular rivalry (Lee and Blake, 1999; Logothetis et al., 1996). It was traditionally thought that the rivalry was between the two eyes (Blake, 1989; Lehky, 1988). However, there is more recent evidence that the neurons at the site(s) of rivalry have access to information from both eyes (Carlson and He, 2000; Kovacs et al., 1996; Lumer et al., 1998; Ngo et al., 2000), and these experimental results cannot be explained in terms of "eye rivalry" (although see Lee and Blake (1999) for an indication of how changing stimulus characteristics can produce either "eye rivalry" or "stimulus rivalry").

Recordings in the cortex of monkeys undergoing binocular rivalry indicate that the neuronal activity of binocular rather than monocular neurons is correlated with the perception of one of the presented images (Leopold and Logothetis, 1996; Leopold and Logothetis,

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1999; Logothetis, 1998; Logothetis et al., 1996; Logothetis and Schall, 1989). The proportion of neurons which are active only when one of the images is perceived increases as one moves up the visual pathway (Leopold and Logothetis, 1999; Logothetis, 1998). It should be noted that while some neurons are more active when their preferred image is perceived, others are more active when their preferred image is suppressed, and yet others show little selectivity during nonrivalrous stimulation but become more selective during rivalrous stimulation (Leopold and Logothetis, 1996; Logothetis, 1998; Logothetis and Schall, 1989).

Several explanations of binocular rivalry have been proposed (Dayan, 1998; Gomez et al., 1995; Lehky, 1988; Lumer, 1998)

We include two slow processes. The first is spike frequency adaptation due to a calcium–dependent potassium current (Huguenard and McCormick, 1992; McCormick and Huguenard, 1992). This is sufficient to cause oscillations in the network's activity, although they occur on a similar time–scale to the time constant of the decay of this current, ~ 80 ms. We also include synaptic depression in the excitatory to excitatory connections that has a larger time–constant (Abbott et al., 1997). We find that synaptic depression alone is not sufficient to cause switching — we need a slow hyperpolarizing current as well. The switching phenomenon is quite robust with respect to the exact strengths and time–scales of these slow variables.

For simplicity, we explicitly model only those neurons whose activity increases when their preferred stimuli are perceived. Those neurons that respond preferentially when their preferred stimuli are suppressed may be part of a different circuit that is involved in suppression of a particular image or eye, and those whose selectivity changes when the stimulus is changed from rivalrous to nonrivalrous may be manifesting the effects of attention on perception (Leopold and Logothetis, 1996; Logothetis, 1998; Logothetis and Schall, 1989). Neurons in these last two classes are not explicitly modeled. Those neurons possibly involved in suppressing an image are similar to those that fire when their preferred stimulus is dominant (both groups fire when one image is suppressed) and our model could be augmented to include such neurons.

2.1. Simulation Results

Figure 2 shows a rastergram of the firing events of the excitatory neurons in the network given two current stimuli centered at neurons whose preferred orientations differ by 90 degrees. At every moment in time, the activity is localized into a bump which is centered at either of the two locations of maximum external current input. A bump in one of these locations is thought to represent a perception of bars of the corresponding orientation. The inhibitory neuron activity is very similar although it has a greater angular spread. Note the wide spread of activity, lasting less than 100 ms, when the activity initially moves to another location. The decrease in width after this period is probably due to the adaptation current saturating. This type of high activity at the onset of a percept is seen in some neurons in superior temporal sulcus and inferior temporal cortex during binocular rivalry (Leopold and Logothetis, 1999; Sheinberg and Logothetis, 1997). Experimentally, bursting behavior is also seen in some of these neurons. Replacing some of the fast ex-

The switching can be understood heuristically. In Sec. 3 we give a more quantitative explanation. Consider two input stimuli 1 and 2. Connections between excitatory neurons promote activity centered at stimulus 1 or 2 while inputs from the inhibitory population prevent this activity from spreading over the whole network. This inhibitory activity is also strong enough to suppress activity at the site corresponding to the stimulus that is not perceived. (For sufficiently strong inputs, two bumps may coexist). Suppose that population 1 is active and 2 is suppressed and consider the effects of the slow current responsible for spike frequency adaptation. This current increases at site 1 and decreases at site 2 until eventually the adaptation remaining from activity at site 2 has decreased sufficiently that the neurons at site 2 are able to fire again, immediately suppressing the neurons at site 1. The adaptation current at site 2 then builds up, the adaptation current at site 1 wears off sufficiently, and the cycle repeats. A similar argument can be made if synaptic depression is the cause of the switching: both the recurrent excitation at site 1 and the inhibition of the neurons at site 2 weaken, allowing neurons at site 2 to fire and suppress neurons at site 1.

One well–known aspect of binocular rivalry is that if the strength of the stimulus to one eye is changed, it is largely the mean dominance duration of the other eye that is affected, not the mean dominance duration of the eye whose stimulus strength is being changed. This effect is sometimes known as Levelt's second proposition (Bossink et al., 1993; Levelt, 1968) and has been observed many times (Leopold and Logothetis, 1996; Logothetis et al., 1996; Mueller and Blake, 1989). More specifically, if the strength of the stimulus to eye 1 is decreased, the mean dominance duration of eye 2 typically increases markedly in a nonlinear fashion, while the mean dominance duration of eye 1 decreases by a small amount. We performed this experiment with our model and the results are shown in Figure 6 (together with data from the reduced model that is presented in Sec. 3). They agree well with observations, and an explanation for this behavior is given in Sec. 3.

Another experiment that has been performed involves changing the angle between the two sets of bars presented to the two eyes. It has been observed that decreasing the angle from 90 degrees causes the mean dominance durations to increase (Andrews and Purves, 1997). We performed this experiment on our model and the results are shown in Figure 7. The variation is small (as it is in experiments, (Andrews and Purves, 1997)) but significant. (Smaller angular differences could not be tested, as this caused the two bumps to "merge" into one that spanned both input positions. This is due to the widths of the Gaussians used in coupling neurons — if these widths were reduced, smaller angular differences could have been tested, but the total number of neurons in the network would have then had to be correspondingly increased, resulting in prohibitively long simulation times.) An explanation for the dependence of dominance duration on angle between bars is given in Sec. 3.

where all constants are positive. Here u_i represents the spatially averaged net excitatory activity of each localiz

has worn off by a sufficient amount. For the parameters shown, population 1 switches on when $a_1 = I_1 - \beta = 0.03$ and population 2 switches on when $a_2 = I_2 - \beta = 0.1$.

We can calculate the dominance period by following

thetis, 1996), however, when interpreting these results one should keep in mind that the relationship between "stimulus strength" and "current input" is not at all clear.

The full system, (1)-(6), shows qualitatively similar oscillations and dependence of dominance durations on input strengths as the two special cases examined above, and we suggest that in practice it may well be a combination of adaptation and synaptic depression (and possibly more than one mechanism for each of these) that causes switching. As a specific example of the behavior when both adaptation and depression are present in (1)-(6), we show in Figure 6 linearly–

a Gaussian distribution, and a_0 is randomly chosen from another Gaussian distribution, each time a^d is reset. The distribution of dominance durations is then

$$p(T) = \Omega \left(\frac{e^{-T/\tau}\left[\gamma + \eta\kappa e^{-T/\tau}\right]}{[\gamma + \kappa e^{-2T/\tau}]^{3/2}} \right) \exp\left(\frac{-\left[e^{-T/\tau} - \eta\right]^2}{2(\gamma + \kappa e^{-2T/\tau})} \right) \tag{12}$$

where Ω, γ, κ and η are related to the parameters of the two Gaussian distributions. See Appendix C for the derivation. This function is plotted in Figure 4 together with data from the simulation of the full Hodgkin–Huxley network. It fits the data well and has the typical skewed shape seen in experimental data (Kovacs et al., 1996; Logothetis et al., 1996).

4. Discussion

Our cortical circuit of excitatory and inhibitory neurons is able to reproduce many of the observed dynamical characteristics of binocular rivalry. We are also able to compute analytically the dependence of the dominance period on the input strengths, and this shows how Levelt's second proposition can arise naturally in a network with mutual inhibition.

We find that the input strength to the network strongly influences the dominance duration. This allows large variations in the dominance durations even with fixed adaptation and synaptic depression time scales. The large distribution in mean times between subjects could be due to the differential input to the local circuit — this may be especially true of feedback from higher level cortical areas — and the strength of this contribution could vary widely between subjects and even change within a subject. The neuromodulators acetylcholine, histamine, norepinepherin and serotonin are all known to decrease the effects of spike frequency adaptation in human cortex (McCormick and Williamson, 1989) and if adaptation is the main mechanism for switching, changes in their concentration would significantly affect mean dominance durations. It is known that there is some training effect in binocular rivalry and multistable perception (Leopold and Logothetis, 1999), and systematic changes in switching frequency on the time scale of several minutes have been observed (Borsellino et al., 1972; Lehky, 1995). Also, knowledge that a stimulus is ambiguous and the possible perceptions of it plays a role in switching (Rock et al., 1994).

There are instances when rivalry does not take place. It is known that if the stimulus contrast is reduced the images from the two eyes can fuse into a single merged percept (Leopold and Logothetis, 1999). Presumably, this [illegible]

Our reduced model was anticipated by Lehky (1988) who proposed a neural network model of binocular rivalry which involved reciprocal inhibitory feedback between signals from the two eyes, prior to binocular convergence. He created an electronic circuit to represent the network, and for strong enough reciprocal inhibition the circuit oscillated. The oscillations stopped for weak inhibition which Lehky attributed to fusion. He could reproduce Levelt's second proposition by changing the adaptation rates on either neuron and postulated that changing stimulus strength changes adaptation rates.

Recently, Kalarickal and Marshall (2000) numerically studied a model similar to (1)-(6), with noise, but not including adaptation. Their model reproduced Levelt's second proposition, the lack of correlation between successive dominance durations, and the results of Mueller and Blake (1989) relating to synchronized changes in input strengths. They also realized that it is the total input to the inactive population that determines the time for which the active population remains active (thus explaining Levelt's second proposition and the results of Mueller and Blake (1989)), but the advantage of our reduced model (1)-(6) over

latter it is nonperiodic, as seen from Figure 4. Thus such a model provides few benefits over a spatially–averaged rate model such as (1)-(6).

Our model does not specify whether the rivalry is "stimulus rivalry" or "eye rivalry". Recent results of Lee and Blake (1999) may indicate that both are occurring. These authors presented orthogonal gratings to the two eyes and investigated the effects of both flickering the images at 18 Hz and swapping the images between the two eyes (as done by Logothetis et al. (1996)). Their results suggest that both the 18 Hz flicker and the swapping of the images continually produce transient effects that significantly change perception of the images, and that either "eye rivalry" or "stimulus rivalry" can result from very similar stimuli. Other recent results (O'Shea, 1998) suggest that binocular rivalry consists of two components: alternations between two images that are independent of eye of origin, and alternations between two images that depend on eye of origin. It is possible that networks with our proposed connectivity exist in various regions of the cortex and produce rivalrous dynamics.

The temporal dynamics of the perception of other ambiguous stimuli such as the Necker cube are similar to those investigated in this model (Borsellino et al., 1972; Gomez et al., 1995), which lends weight to the idea that binocular rivalry is another manifestation of competition between alternative representations of a stimulus, rather than being a phenomenon that is restricted to the ocular system (Leopold and Logothetis, 1999), and it may be possible to extend this type of modeling to include more complex visual stimuli, for example, blurred images (O'Shea et al., 1997).

Acknow

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Appendix

A. Methods

The eq

neurons (and the number of inhibitory neurons),

$$g_{ee}^{jk} = \alpha_{ee}\sqrt{\frac{50}{\pi}}\exp\left(-50[(j-k)/N]^2\right) \tag{15}$$

and

$$g_{ie}^{jk} = \alpha_{ie}\sqrt{\frac{20}{\pi}}\exp\left(-20[(j-k)/N]^2\right) \tag{16}$$

Similarly, the synaptic current entering the jth inhibitory neuron is

$$\frac{1}{N}\left[(V_{ei}-V_i^j)\sum_{k=1}^{N} g_{ei}^{jk}s_e^k + (V_{ii}-V_i^j)\sum_{k=1}^{N} g_{ii}^{jk}s_i^k\right] \tag{17}$$

where $V_{ei}=0$, $V_{ii}=-80$, V_i^j is the voltage of the jth inhibitory neuron,

$$g_{ei}^{jk} = \alpha_{ei}\sqrt{\frac{20}{\pi}}\exp\left(-20[(j-k)/N]^2\right) \tag{18}$$

and

$$g_{ii}^{jk} = \alpha_{ii}\sqrt{\frac{30}{\pi}}\exp\left(-30[(j-k)/N]^2\right) \tag{19}$$

A typical I_{ext} for the excitatory population is

$$I(i) = 0.4\left[\exp\left(-\left\{\frac{20(i-N/4)}{N}\right\}^2\right) + \exp\left(-\left\{\frac{20(i-3N/4)}{N}\right\}^2\right)\right] - 0.01 \tag{20}$$

where $i = 1\ldots N$, i.e. two Gaussians centered at $1/4$ and $3/4$ of the way around the domain together with a constant negative current. I_{ext} for the inhibitory population is 0. Typical values for the coupling strengths are $\alpha_{ee}=0.285$, $\alpha_{ie}=0.36$, $\alpha_{ei}=0.2$, $\alpha_{ii}=0.07$.

B. Derivation of Reduced Model

Here we derive the reduced model, equations (1)–(6). We first note that spike frequency adaptation and synaptic depression are both slow processes relative to the time over which a spike occurs. Both are driven by the post-synaptic activity. Focusing on adaptation we can write

$$\frac{da_i}{dt} = -a_i/\tau + A_i(t) \tag{21}$$

where a_i is a generalized adaptation variable (e.g. the calcium concentration in system (13)) and $A_i(t)$ is proportional to the cell activity (instantaneous firing rate) of neuron i. We then assume that the neuronal activity is driven by the synaptic inputs through a gain function f,

$$A_i(t) = f\left(\sum w_{ij}U_j(t) - a_i + I_i\right) \tag{22}$$

where w_{ij} represents the synaptic weight from neuron j to neuron i, and $U_j(t)$ is the post-synaptic response of neuron j. We assume the influence of the adaptation is linear and I_i

where

$$A = \frac{\mathfrak{v}^{-2T/\tau}}{2\sigma_\theta^2} + \frac{1}{2\sigma_a^2}, \qquad B = \frac{\mu_a}{\sigma_a^2} + \frac{\mu_\theta \mathfrak{v}^{-T/\tau}}{\sigma_\theta^2}, \qquad C = \frac{\mu_\theta^2}{2\sigma_\theta^2} + \frac{\mu_a^2}{2\sigma_a^2} \tag{28}$$

and Ω is a normalization constant defined through $\int_{-\infty}^{\infty} p(T)\, dT = 1$. So

$$p(T) = \Omega \mathfrak{v}^{-T/\tau} \mathfrak{v}^{B^2/(4A)-C} \int_{-\infty}^{\infty} a \exp\left(-A\left(a - \frac{B}{2A}\right)^2\right) da \tag{29}$$

$$= \Omega \mathfrak{v}^{-T/\tau} \mathfrak{v}^{B^2/(4A)-C} \int_{-\infty}^{\infty} \left(u + \frac{B}{2A}\right) \mathfrak{v}^{-Au^2}\, du \tag{30}$$

$$= \frac{\sqrt{\pi} B \Omega \mathfrak{v}^{-T/\tau} \mathfrak{v}^{B^2/(4A)-C}}{2A^{3/2}} \tag{31}$$

where we have made the substitution $u = a - B/(2A)$ and used the fact that

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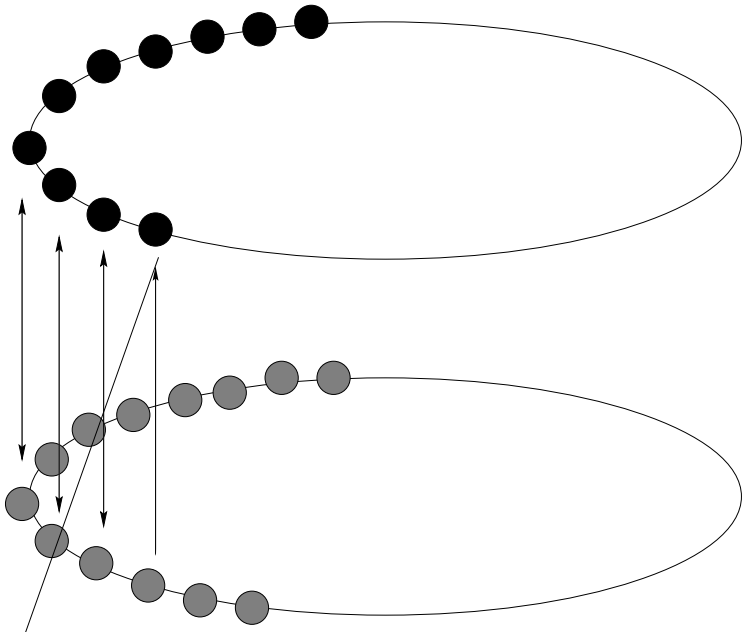
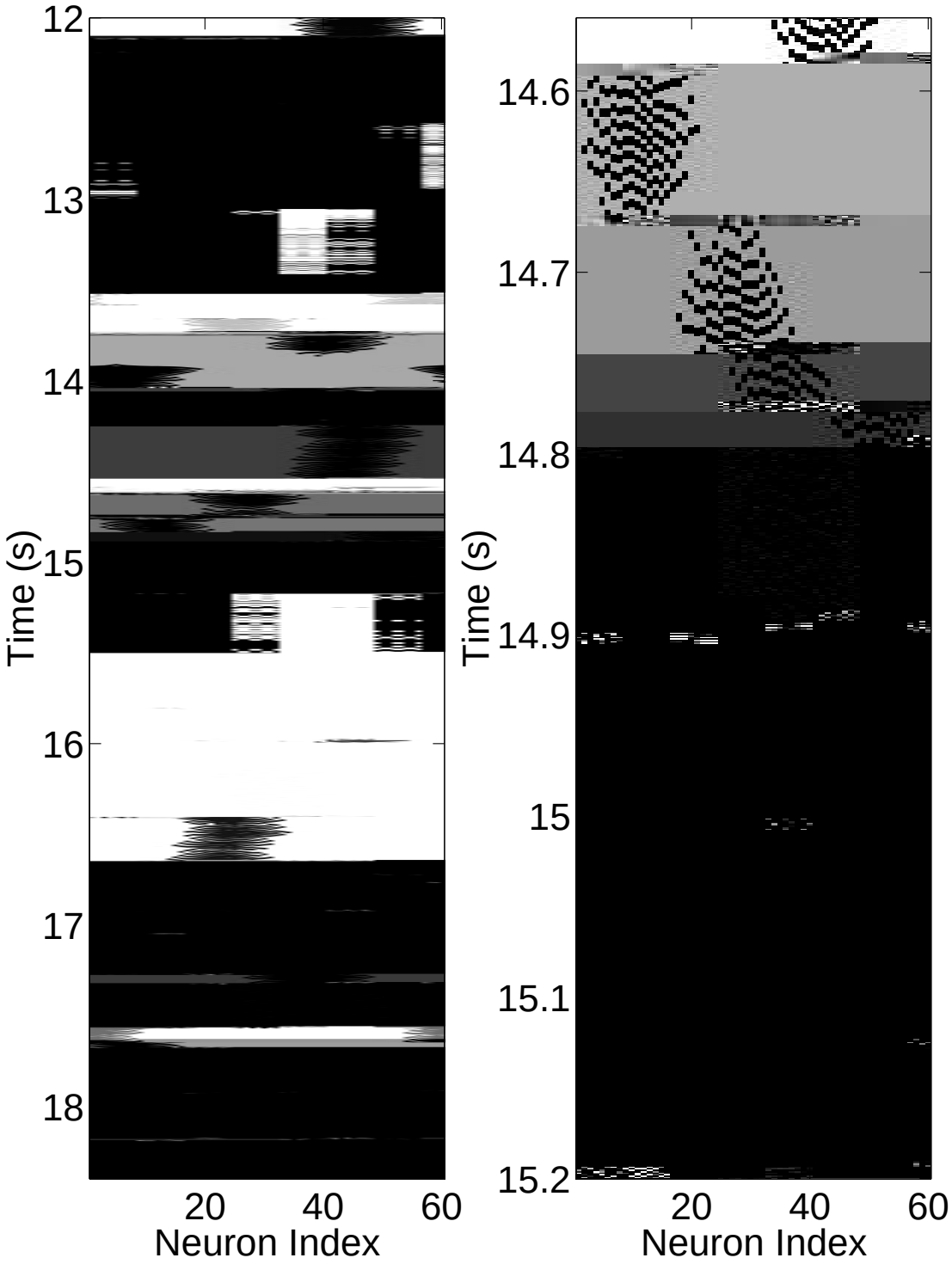
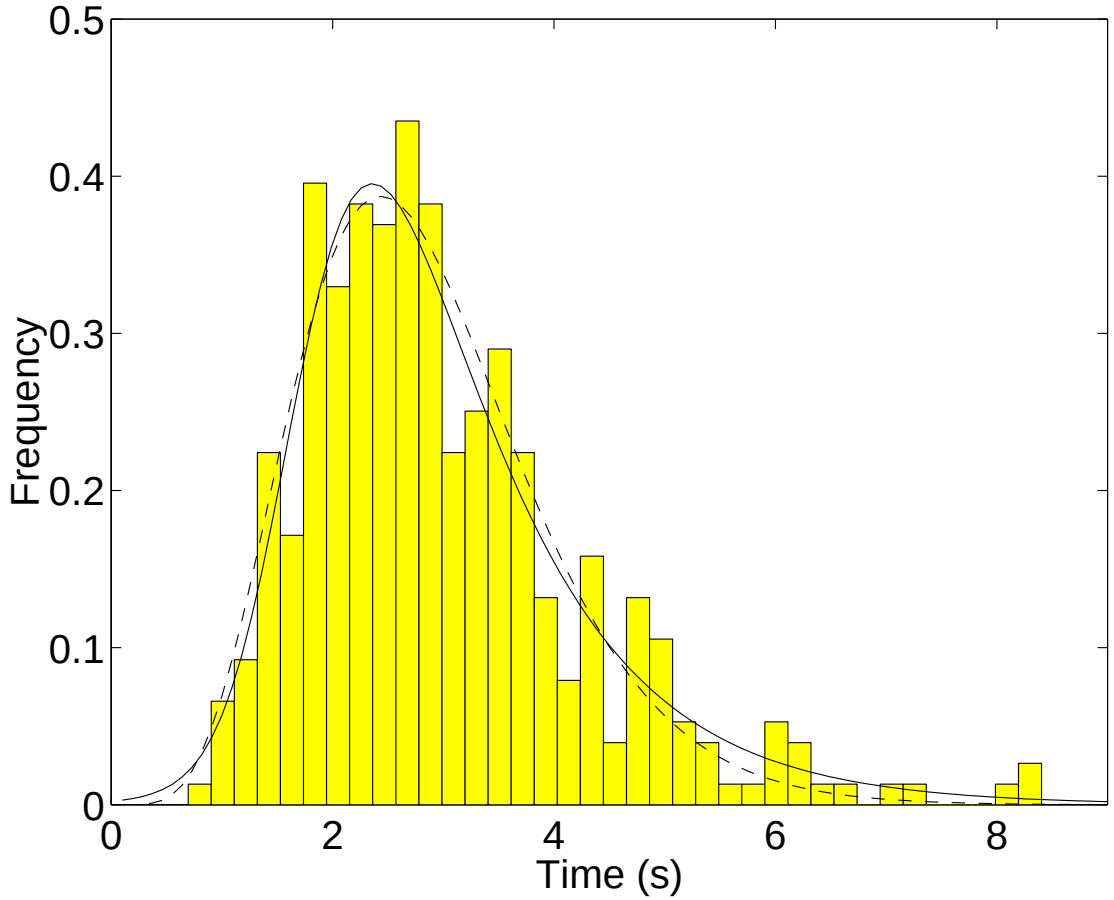
9 LAING: Solution of the reduced model (1)–(4). Parameter values are $\alpha = 0.2$, $\beta = 0.4$, $\phi_a = 0.4$, $\tau_a = 20$, $I_1 = 0.43$, $I_2 = 0.5$, $g_1 = g_2 = 1$. The top plot is u_1 and a_1, the bottom is u_2 and a_2. 33

10 LAING: Dominance durations with only adaptation considered. Top: equations (1)–(4) with $g_1 = g_2 = 1$, as given by (9). T_1 is dashed and T_2 is solid. Note

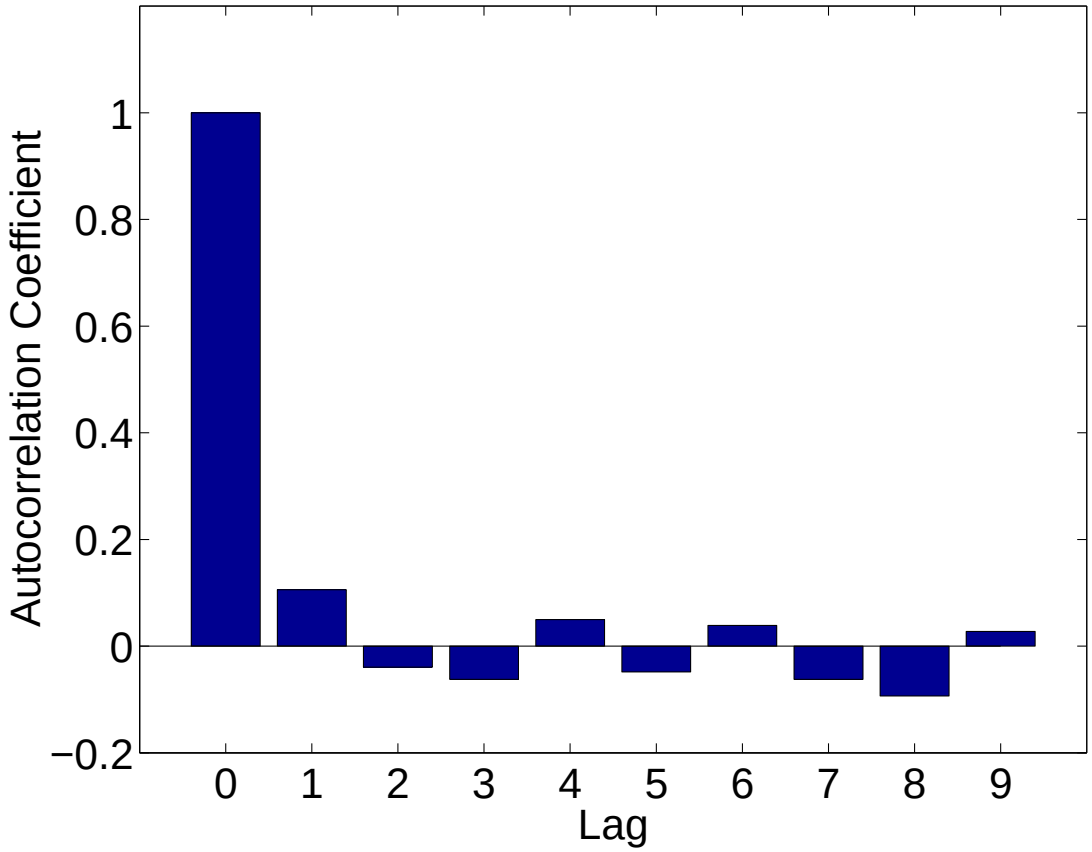




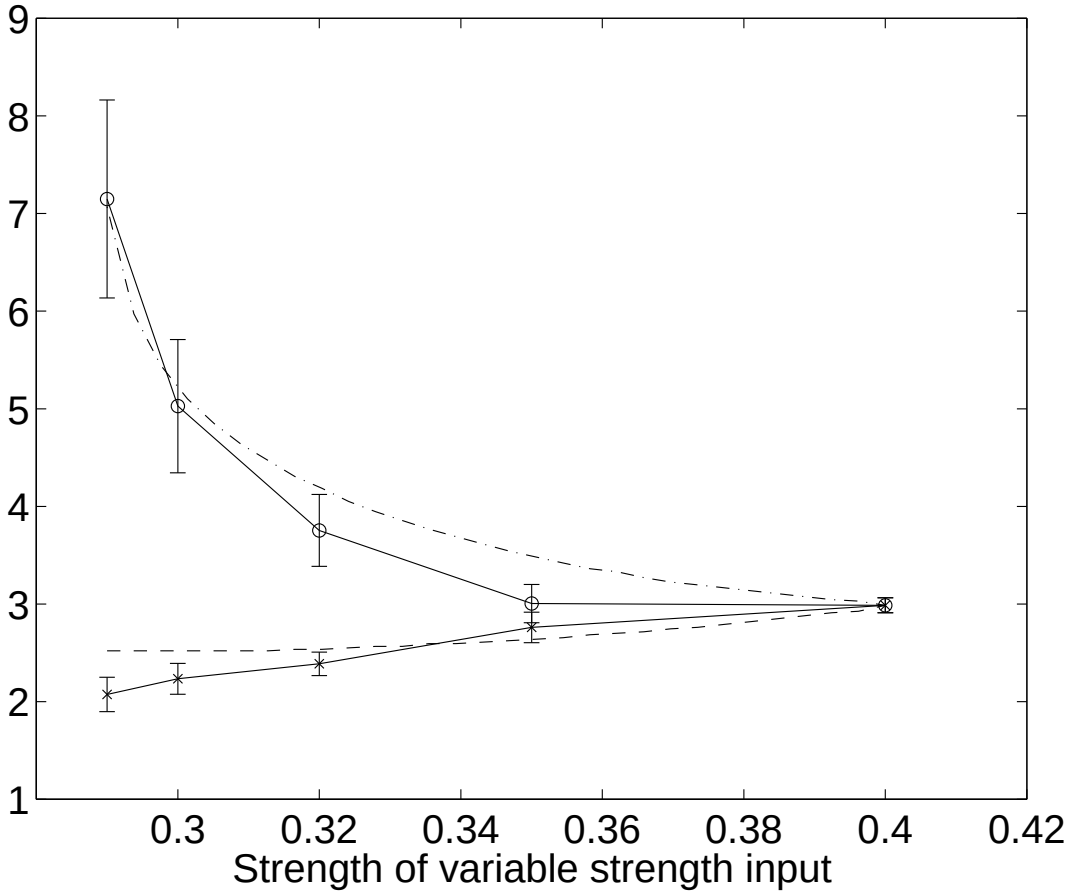
Figu e 2. LAING: Activity in the excitatory population as a function of time. The current stimuli are centered at neurons 15 and 45. The right plot shows detail of the left plot.



Figu e 4. LAING: The distribution of dominance durations for the Hodgkin–Huxley model. The solid line is equation (12) with parameters $\gamma = 0.0174$, $\eta = -0.0005$, $\kappa = 0.0782$, $\tau = 1.1389$, and the dashed is a Gamma distribution with $\lambda = 2.3593$ and $r = 6.7381$ where the Gamma distribution is $f(t) = \lambda^r/\Gamma(r)t^{r-1}\exp(-\lambda t)$.



Figu e 5. LAING: Autocorrelation coefficients for the data in Figure 4.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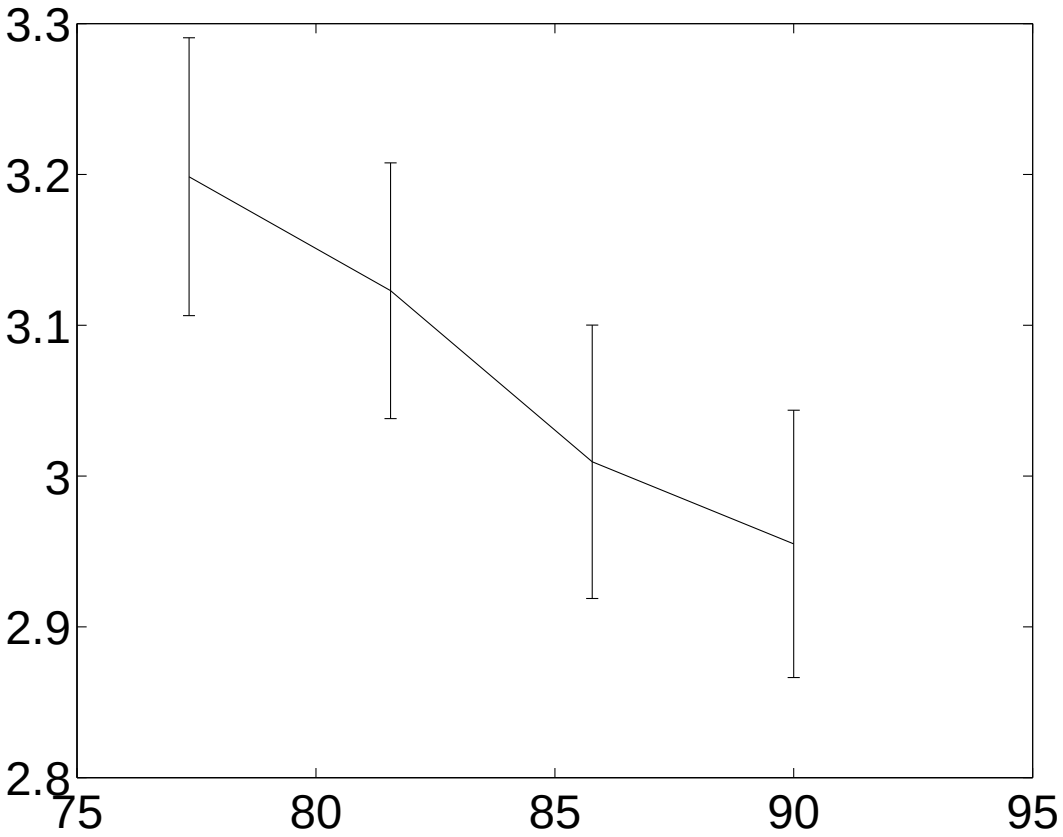
Strength of variable strength input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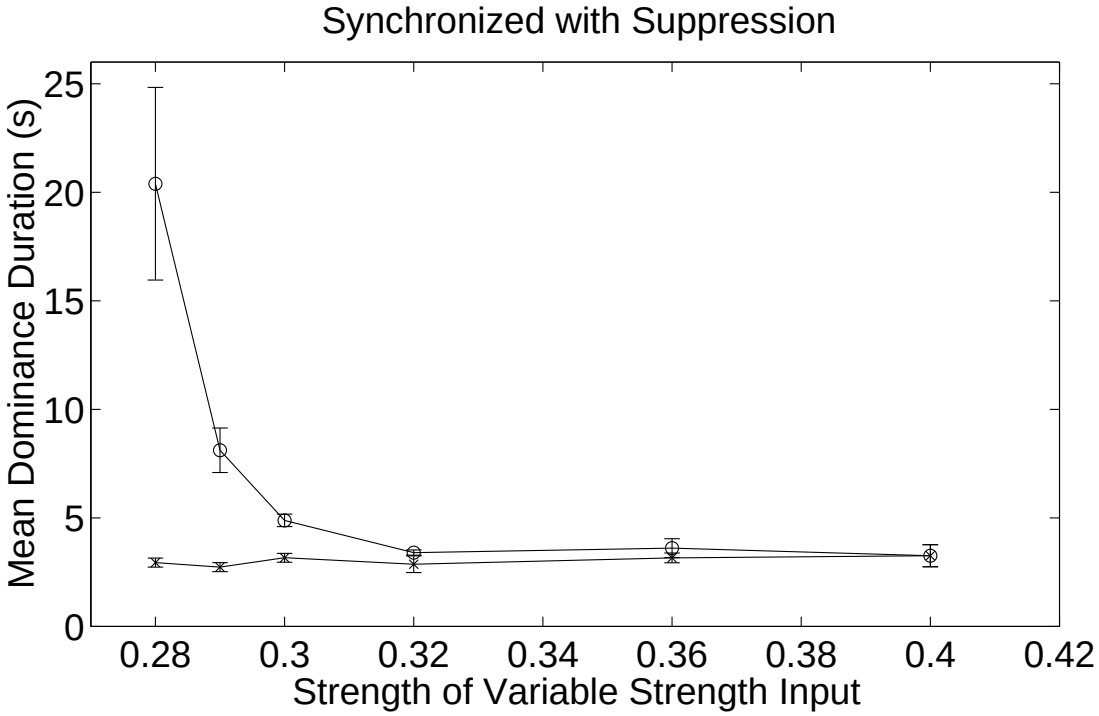
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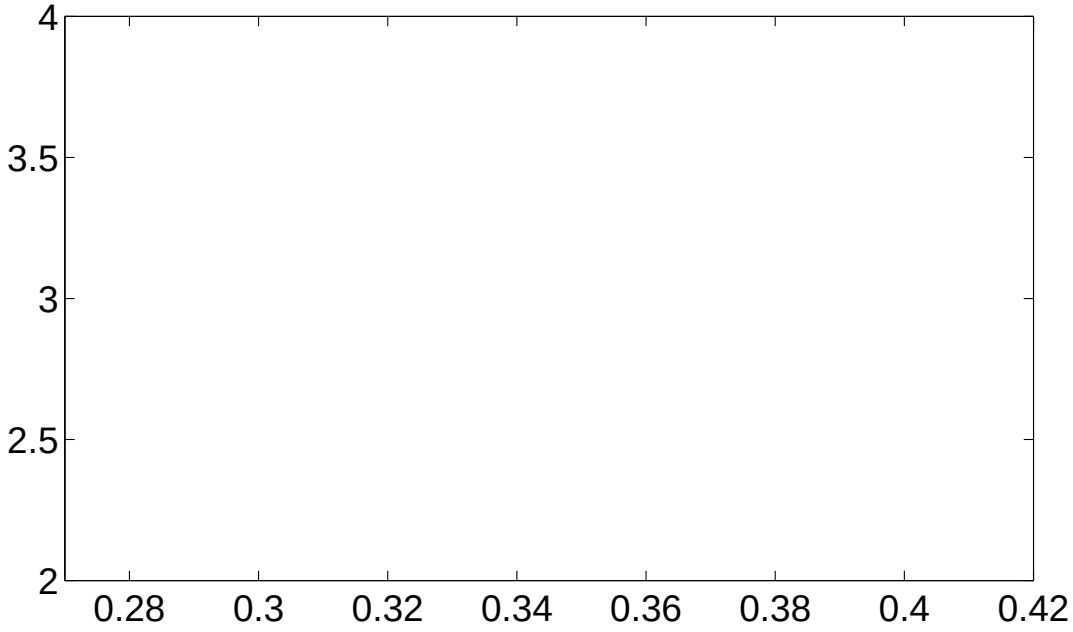
Strength of Variable Strength Input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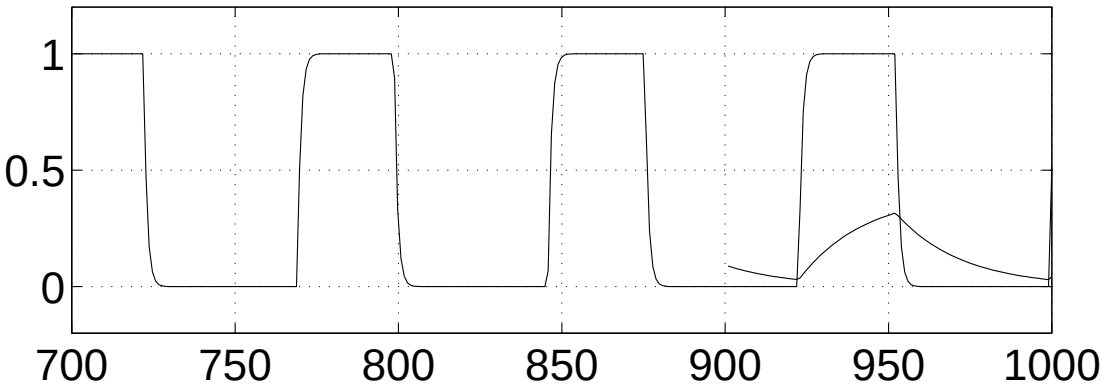
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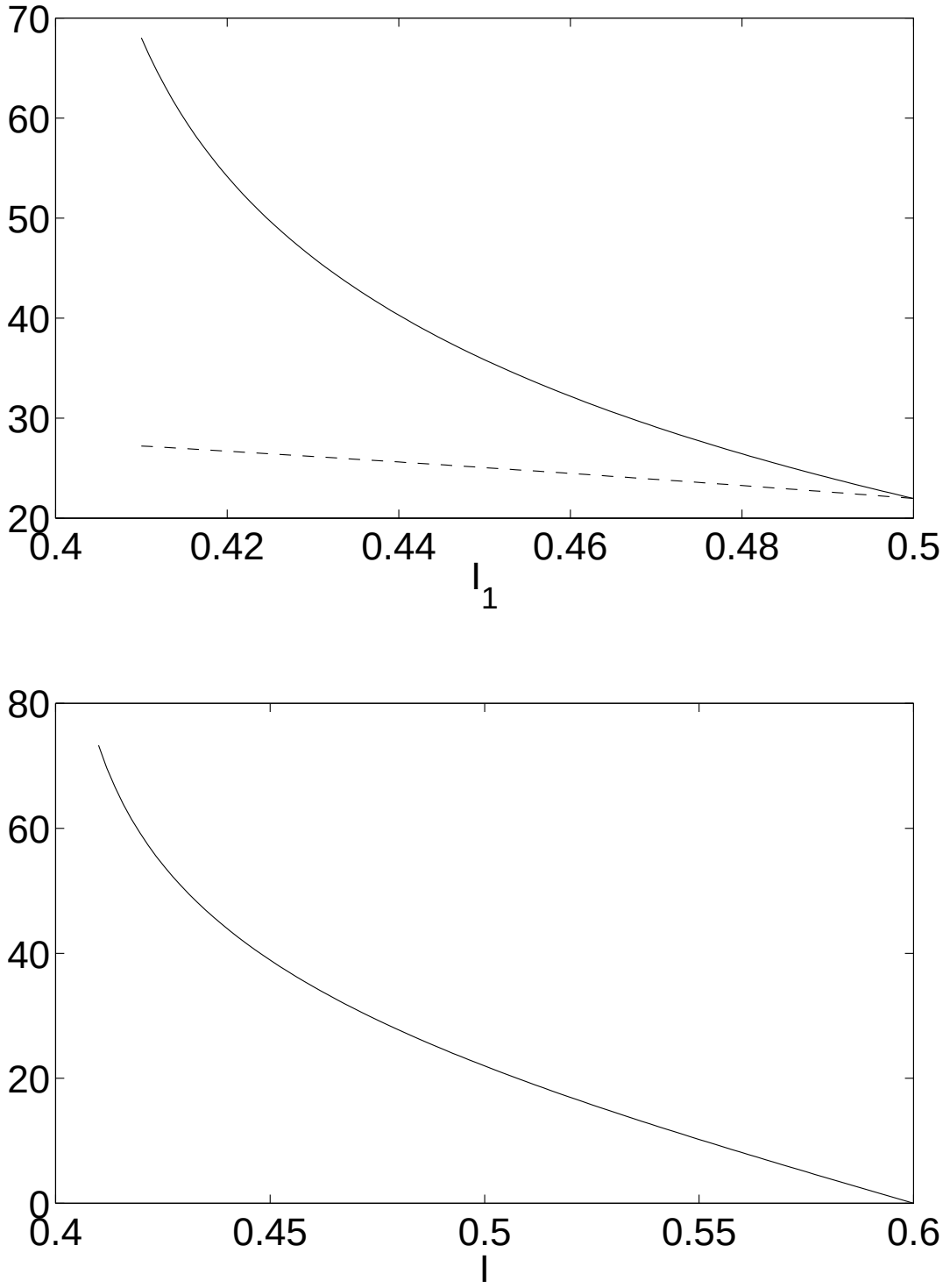
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Figure 10. L