Maximum Likelihood Analysis of Spike Trains of Interacting Nerve Cells*

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Abstract Suppose that a neuron is firing spontaneously or under the influence of other neurons. Suppose that the data available are the firing times of the neurons present. If the principal neuron's firing is modelled as occurring when an internal state variable crosses a random threshold, then one may develop maximum likelihood estimates of the unknown parameters. The resulting procedure appears useful for estimating biologically meaningful parameters of neurons, for understanding the connections present in networks of neurons and for aiding description and classification of neurons and synapses. Analyses are presented for a number of data sets collected for the sea hare, *Aplysia californica* by J. P. Segundo. The computations were carried out quite directly via the Glim statistical package. An example of a Glim program realizing the work is presented in the Appendix.

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1 Introduction

At this point in time quite a number of stochastic models have been set down to describe the firing of a neuron and the interactions of firings of neurons in groups. Various of these models are reviewed in Holden (1976). In this paper a threshold model is constructed based on firing times of influencing neurons and summation functions. A method of directly addressing the question of causal connections of possibly inter-related neurons, given data consisting of the times at which the individual neurons fired results. The new method is a maximum likelihood procedure supported by the known neurobiology. This approach has the substantial advantage that biologically interpretable parameters are estimated. The approach is an alternate to an indirect second moment based method which had been given earlier, (Brillinger , Bryant and Segundo (1976). It provides an alternate to the visual technique suggested by Gerstein *et al* (1978) with the advantage of being applicable to arbitrary sized collections of neurons. It is an alternate to a procedure presented in Borisyuk *et al* (1985) with the advantage of making use of more biology. One other work that may be cited is Nakao *et al* (1985). These researchers employed an extended Kalman filter and mean squared error fitting. The maximum likelihood method of this paper extends the one Brillinger and Segundo (1979) presented for the case of continuous input.

The computations of the paper were carried out in the statistical system known as Glim, see Baker and Nelder (1978). This system is fairly widely available. It is interactive and available for microcomputers as well as large machines.

2 Some Statistical Methodology

We begin with a few definitions taken from stochastic point process theory.

Counting Process. For points $\{\tau_j\}$ randomly scattered along a line, the counting process $N(t)$ gives the number of points realized in the time interval $(0,t]$. For

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the case of a neuron spike train, with the neuron firing at times τ_j , $N(t)$ gives the number of times the neuron fired between time 0 and time t,

$$N(t) = \#\{\tau_j \text{ with } 0 < \tau_j \leq t\}$$

History. The history, $\mathbf{H}_t$, consists of the variates determined up to and including time t that are necessary to describe the evolution of the counting process. For example for the process $N \equiv \{\tau_j\}$, one might take $\mathbf{H}_t = \{\tau_j \mid \tau_j \leq t\}$.

Conditional Intensity. For the process N and history $\mathbf{H}_t$, the conditional intensity at time t is defined as

$$\mu(t) = \lim_{h \to 0} Prob\{event\ in\ (t, t+h] \mid \mathbf{H}_t\}/h$$

For small h one has the interpretation,

$$Prob\{event\ in\ (t, t+h] \mid \mathbf{H}_t\} \approx \mu(t)h$$

Given a sufficiently rich history, the conditional intensity determines the process. If the process N is under the influence of various other processes, then their influence may be described formally by setting down a specific functional form for μ in terms of those processes.

The likelihood of a data set is defined to be the probability of obtaining the data values as a function of the unknown parameters. One may show that in the point process case, given the process realization for the time interval $(0,T]$, the likelihood may be written

$$\exp\{-\int_0^T \mu(t)dt + \int_0^T \log \mu(t)dN(t)\} = \exp\{-\int_0^T \mu(t)dt\} \prod_j \mu(\tau_j) \tag{2.1}$$

in terms of the conditional intensity function.

References to the theory of stochastic point processes include Cox and Lewis (1966), Lewis (1972) and Snyder (1975). Likelihood for point process data and the issue of causality are also addressed in Ogata *et al* (1982).

In our computational work, specifically in determining the maximum likelihood estimates via Glim, it will be convenient to approximate the point processes involved by 0-1 time series. Suppose that a small time interval Δ is taken. Define the following series

$$N_t = 1 \text{ if } there\ is\ an\ event\ in\ (t, t+\Delta]$$

$$N_t = 0\ otherwise$$

for $t = 0, \pm\Delta, \pm 2\Delta, \ldots$. The point process $N(t)$ is thus replaced by a time series N_t that is 0 most of the time and 1 occasionally. Let

$$p_t = Prob\{N_{t+\Delta}=1 \mid \mathbf{H}_t\} \tag{2.2}$$

with $H_t = \{N_u \mid u \leq t\}$, then the likelihood is given by

$$\prod_t p_t^{N_t}(1 - p_t)^{1-N_t} \tag{2.3}$$

In this paper we work with maximum likelihood estimates. These are defined as the parameter values that maximize expressions like (2.1) and (2.3) above. The asymptotic properties of such estimates in the point process case were studied formally in Sagalovsky (1982), under mixing (that is asymptotic independence) assumptions, and Dzhaparidze (1985). The specific practical

problem becomes that of finding appropriate expressions for the conditional intensity function $\mu(t)$ or the approximating p_t.

3 A Biological Description and an Associated Analytical Model

We turn to a crude description of some of the biology involved, in order to construct a conceptual model of the situation. One process by which a neuron influences the firing of a second may be described as follows. With the firing of a neuron, a transmitter is emitted from its axon terminal. If another neuron is nearby, this chemical may pass over and act on its dendrite. The permeability is changed and there is a brief current flow. This current propagates to create a change in postsynaptic potential at a trigger zone. When that potential passes an extant level, then the cell fires. Depending on the situation, the potential created by the first cell will either accelerate or slow the firing of the second. (There may be later swings in effect as well.) A further aspect of the process is that on firing, the potential at the trigger zone is reset to the extant level. It is to be noted that the potential may further be affected by the internal mechanics of the neuron itself. This last leads to spontaneous firing. (It must be remarked that but one class of neurons has been described here. Other neurons behave differently.)

The above biological description may be put into an analytic form as follows. Let the spike trains of two neurons say B and C be described by counting functions $B(t)$, $C(t)$ respectively. Here $B(t)$ gives the number of spikes of the cell B that occur in the time interval $(0,t]$. $C(t)$ has a similar meaning. Suppose one is interested in the firing of neuron C. Supposing that the neuron B fired at time τ, let $b(t-\tau)$ represent the effect on the potential at the trigger zone of the second. It will be called a summation function. If $\gamma(t)$ denotes the time elapsed at time t since the second neuron last fired, then the (membrane) potential at its trigger zone may be represented as

$$U(t) = \int_0^{\gamma(t)} b(u)\,dB(t-u) \tag{3.1}$$

assuming additivity of the effects of the individual firings. (The presence of $\gamma(t)$ introduces feedback into the system.)

Let $\theta(t)$ denote the threshold level at time t and assume that it has the form $\theta(t)=\theta+\epsilon(t)$, with $\epsilon(t)$ noise. Then the conditional intensity of the neuron C's firing is based , for h small, on

$$Prob\,\{spike\ in\ (t,t+h]\,|\,|\,\mathbf{H}_t\} = Prob\,\{U(s)\,crosses\,\theta(s)\ \text{for}\ some\ s\ in\ (t,t+h]\,|\,|\,\mathbf{H}_t\}$$

taking the history to consist of B's and C's past firings. An explicit likelihood function of a given data set may now be set down by means of expression (2.1) once an expression for the probability on the right above has been set down. In practice it may be reasonable to add a term like

$$V(t) = \theta_1\gamma(t) + \theta_2\gamma(t)^2 + \theta_3\gamma(t)^3 \tag{3.2}$$

to $U(t)$, corresponding to to C's own effect on the potential. It allows for spontaneous firings of the neuron. References presenting analytic models for neuron firing include Johannesma (1968), Gestri (1971) and Knox (1974). Holden (1976) is a general reference. One may also mention Nakao *et al* (1985) and Borisyuk *et al* (1985).

The problem of evaluating a crossing probability like (3.1) is generally a difficult one. For the case of a high threshold, approximations are sometimes available. (See for example Cuzick (1981).) In the case of a random step function (with jumps occurring at the points of a rate ν Poisson process and amplitudes

independent normals with mean 0 and variance σ^2) ten Hoopen *et al* (1963) show that (3.1) is given by

$$\nu\Phi\left\{\frac{U(t)}{\sigma}\right\} - \frac{d}{dt}\log\{1-\Phi\left\{\frac{U(t)}{\sigma}\right\}\}$$

where Φ is the standard normal cumulative. This will be approximately $\nu\Phi(g(t)/\sigma)$ for ν large or the derivative of U small.

The following discrete approximation to this model might be employed to develop analyses . Let C_t and B_t denote 0–1 valued time series corresponding to the point processes, with $t=0,\pm\Delta,\pm 2\Delta,....$ Let γ_t denote the time elapsed at t since the preceding time that $C_t=1$. Let

$$U_t = \sum_{j\Delta=0}^{\gamma_t-\Delta} b_{j\Delta} B_{t-j\Delta}$$

Then we assume $C_t = 1$ if $U_t \geq \theta+\epsilon_t$ and equals 0 in the contrary case. Further, let $\mathbf{H}_t = \{B_u, C_u \mid u \leq t\}$ denote the history. Assuming that $\epsilon_{t+\Delta}$ is statistically independent of the previous ϵ 's and Gaussian with mean 0 and variance 1, one has

$$p_t = Prob\{C_{t+\Delta}=1 \mid \mathbf{H}_t\} = Prob\{U_{t+\Delta}\geq\theta+\epsilon_{t+\Delta} \mid \mathbf{H}_t\}$$
$$= \Phi(U_{t+\Delta}-\theta)$$

This is the crucial expression providing the required p_t. The corresponding discrete approximation to the likelihood is

$$\prod_t p_t^{C_t}(1-p_t)^{1-C_t} \tag{3.3}$$

The maximum likelihood estimates of the unknowns appearing would be determined by maximizing expression (3.3) as a function of $\{b_u\}$ and θ.

Consider next the case of neuron C firing away, possibly under the influence of two neurons A and B . A model for consideration has $U(t)$ given by

$$U(t) = \int_0^{\gamma(t)} a(u)dA(t-u) + \int_0^{\gamma(t)} b(u)dB(t-u) \tag{3.4}$$

and $V(t)$ given by (3.2). For the fitting, again approximate the point processes by 0–1 time series. Suppose the time variable t is defined to take on the values $0,\pm 1,\pm 2,...$, that is $\Delta=1$. Suppose that $V(t)$ of (3.2) is present and that ϵ_t is a sequence of independent standard normal variates. The probability, p_t, of (2.2) is now given by

$$p_t = \Phi\left\{\sum_{u=0}^{\gamma_t} a_u A_{t-u} + \sum_{u=0}^{\gamma_t} b_u B_{t-u} + \theta_1\gamma_t + \theta_2\gamma_t^2 + \theta_3\gamma_t^3 - \theta_0\right\} \tag{3.5}$$

As an issue of causality it would be of interest, for example, to see if the coefficients $\{b_u\}$ are near 0. This would correspond to the neuron B not influencing the neuron C in a direct fashion. Any apparent association of neurons B and C would be due to their both being influenced by A. One could also consider the case of neuron B under the influence of neurons A and C . Once estimates of the parameters are available, estimates of the processes $U(t), V(t)$ themselves may be computed.

4 **Experimental Methodology.**

The particular experimental procedure and the electrophysiological recording techniques leading to the data analyzed are described in detail in Bryant *et al.*

(1973). Briefly, experiments were performed with identified nerve cells of the abdominal ganglion of the sea hare *(Aplysia californica)*. The ganglion, including interconnected cells, was removed from specimens. Microelectrodes were inserted into the cells in order to measure their electrical activity and on some occasions to induce activity. The presynaptic cell was stimulated with about 50-msec pulses each of which elicited one spike. In one experiment to be considered, hyperpolarizing pulses were injected to make the cell respond in an inhibitory fashion. The data collected were the successive times at which the individual neurons fired.

Data from these and similar experiments were also analyzed in Brillinger *et al* (1976) making use of a "linear" model and employing second-order moments alone.

5 **Results**

5.1 Two Cell Experiments. The first data set analyzed consisted of corresponding spike trains of the cell R15 and of an unidentified neuron in the right visceropleural connective (RVP). It is data like that of Figure 14A of Bryant *et al* (1976). RVP had the property of exciting R15. In the experiment analyzed, RVP was driven by a Poisson process. The number of spikes observed were 720 and 224 respectively. The spikes trains were replaced by 0-1 time series taking a sampling interval, Δ, of .075 seconds. This lead to series of approximately 10000 points. The unknowns were estimated by maximizing the likelihood (3.3), employing expression (3.5). Figure 1 shows the estimated summation function, $\hat{b}_u$, the estimated spontaneous potential function, $\hat{V}(t)$, and an estimate of the probability of firing as a function of the total potential. Specifically, the top graph gives $\hat{b}_u$ (and estimated $\pm$ two standard error lines.) The function $\hat{b}$ is seen to swing positive, corresponding to an excitory junction, followed by about half a second of slowing. The spontaneous function $\hat{V}(t)$, given in the second graph, is seen to be rising slowly and to be far below the threshold level $\hat{\theta}_0$. The bulk of the firing of R15 is apparently resulting from the RVP stimulation, rather than spontaneous behavior. Suppose that $\hat{\eta}$ denotes the estimated linear predictor,

$$\sum_u \hat{b}_u B_{t-u} + \hat{\theta}_1 \gamma_t + \hat{\theta}_2 \gamma_t^2 + \hat{\theta}_3 \gamma_t^3 - \hat{\theta}_0 \tag{5.1}$$

Then the bottom graph provides an estimate of p_t. Specifically points are plotted at

$$\sum_t \{C_t \text{ with } \eta - h < \hat{\eta} < \eta + h\} / \sum_t \{1 \text{ with } \eta - h < \hat{\eta} < \eta + h\}$$

for selected η and small h. The smooth graph is $\Phi(\eta)$. This graph is one means of studying the validity of the model. The variability of the final plotted points is large, there not being many cases, so the fit seems not unreasonable.

The second pair of point processes analysed corresponded to an inhibitory junction. Neuron L10 was injected with hyperpolarizing pulses. There were 531 pulses and 503 action potentials respectively. The data analyzed is like that of Figure 16A in Bryant *et al* (1976). The pulses were taken as the input process B_t and the L10 firing times as the output process C_t. Figure 2 provides the results of the analysis. The top graph makes apparent the inhibitory nature of the pulses. The second graph shows the rapid rise of $\hat{V}(t)$, necessary for the cell to fire at all. The bottom graph suggests that the model is fitting to some reasonable extent.

The computations of these and the following data sets were carried out via Glim . Because of the character of Berkeley's implementation of Glim, only 31

coefficients could be included in the analysis. In order to study longer lags, in some computer runs, the sampling interval, Δ, was taken larger than the .075 of the results presented here. Those runs suggested that the summation functions were near 0 at the longer lags. In the actual computations it seemed simplest to first create a data file via a FORTRAN program then to process that file via Glim. The data file consisted of vectors $\gamma_t, C_t, B_t, B_{t-1}, B_{t-2}, \ldots$. A Glim program for one of the three cell experiments, discussed in the next section, is given in the Appendix.

5.2 Three Cell Experiments. The first set of three spike train computations presented are mainly meant to demonstrate that the proposed technology of addressing causal connections works in a situation of known structure . In the previous section's experiment with hyperpolarizing pulses a neuron L3 was present as well. It is known that the firing of L10 inhibits the firing of L3, (see Bryant *et al* (1973).) Given that the pulses have been injected directly into L10, the only connection of the pulses and L3 should be through L10. Figure 3 presents the results of fitting the model with p_t given by expression (3.5), taking C to correspond to L3, A to correspond to the pulse train and B to correspond to the L10 train. The top graph provides $\hat{a}_t$ and $\hat{b}_t$. The solid lines provide $\pm$ 2 standard error limits. The only connection indeed appears to be with L10 and it is inhibitory as anticipated. The second graph demonstrates that L3 may be expected to fire spontaneously, fairly frequently, if left uninfluenced by L10. The bottom graph again suggests a not unreasonable fit of the model. The numbers of pulses, L10 spikes, L3 spikes were 952, 359, 735 respectively.

In the final experiment whose data analysis will be presented, cells L2, L3, L10 were observed. What is known *(ibid)* about these cells is that L10 inhibits the firing of both L2 and L3. What is not know is whether or not L2 influences L3 directly or L3 influences L2 directly. From estimating the crossintensity function, it is clear that the firings of the cells L2 and L3 are associated. A question of interest then is are L2 and L3 connected directly? The numbers of firings for the data analysed where 952, 359, 735 for cells L10, L2, L3 respectively. L10 was Poisson driven in this particular experiment. The data is like that of Figure 15DEF of Bryant *et al* (1973).

Figure 4 provides the estimates $\hat{a}_u$ and $\hat{b}_u$. On the basis of the figure and these values and estimated standard errors, it seems reasonable to infer that neuron L2 is not directly influencing neuron L3. This result is consistent with the partial coherence analysis presented in Brillinger *et al* (1976). The graph further makes it apparent that the neuron L10 is having an inhibitory effect on the firing of the neuron L3. This effect seems to last for a bit over a second.

Figure 4 also provides $\hat{V}_t$ as based on expression (3.2), with the threshold given by $\hat{\theta}_0$. This expression suggests how the neuron L3 is led to fire spontaneously, i.e. if left uninfluenced by other neurons. The last graph of the figure suggests the fit is good.

Finally Figure 5 provides the results of a maximum likelihood analysis of the question of whether neuron L3 is directly influencing neuron L2. The conclusions are as before. The direct connection is the one from L10. Apparent differences are though, that the summation function, a_u, is biphasic and the spontaneous function, V_t, grows slowly.

5.3 Spontaneous experiments. This section is provided only for appearances sake. The principal concern of the paper has been situations wherein the influence of other neurons is a principal contributor to the firing of a neuron. We remark that Sampath and Srinivasan (1977) provide discussions of various stochastic

models for spontaneous neuronal firing. Some of these models can also be fit by the method of maximum likelihood.

6 Discussion

This paper has presented a new technique for disecting the connections of networks of neurons, as well as estimating biologically interpretable parameters for those neurons, making use of extracellular data alone. It is based on an assumption of random threshold and of firing as a level crossing process. The technique produces estimates of the uncertainties and allows formal testing of hypotheses, eg. whether specified phenomena are present. The technique handles feedback that is present directly. The technique is highly flexible: an arbitrary number of neurons are allowed, phenomena such as ageing and adaptation may be incorporated, the noise distribution may be changed. The new technique may be anticipated to be more efficient than previously proposed second-order and partial likelihood procedures. It further involves no assumption re the stochastioc properties of the inputs and can estimate more parameters than the existing techniques.

Appendix

There follows a Glim program for carrying through the proposed analysis in a three cell case. Assume that there exists a data file, called "nerve.data", containing vectors made up of tt = γ_t , y1 = C_t , a = A_t , b = A_{t-1} ,..., o = B_t, p = B_{t-1} ,...

```
$units 9916    ! number of input vectors
$calc d1 = 1    ! binomial parameter
$data tt y1 a b c d e f g h i j k l m n o p q r s t u v w x y z a1 a2
! names of input values
$open 7 'nerve.data'
$dinput 7
$yvar y1 ! y1 is 1 if cell fired, 0 if not
$error b d1    ! declares binomial model
$link p $    ! declares probit link function
$calc t2 = tt*tt$
$calc t3 = t2*tt$
$fit tt+t2+t3+a+b+c+d+e+f+g+h+i+j+k+l+m+n+o+p+q+r+s+t+u+v+w
  +x+y+z+a1$
$d e$
```

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LEGENDS

Figure 1. Top graph. Summation function showing dependence of cell R15 on cell RVP. Solid lines give ± 2 standard error limits plotted around 0. level.

Middle graph. Estimated spontaneous function $V(t)$ of expression (3.2) and estimated threshold level, θ_0.

Bottom graph. Estimate of the probability of cell R15 firing as a function of the estimated linear predictor, expression (5.1).

Figure 2. Top graph. Summation function showing the effect of the directly injected hyperpolarizing pulses on the firing of L10. The solid lines provide ± 2 standard error limits.

Middle graph. As for Figure 1.

Bottom graph. As for Figure 1.

Figure 3. Top graph. Estimates of summation functions a_u, b_u of expression (3.5) with A_t corresponding to injected hyperpolarizing pulses and B_t to firing times of L10. The solid lines give estimated ± 2 standard error limits about level 0. for the pulses.

Middle graph. As for Figure 1.

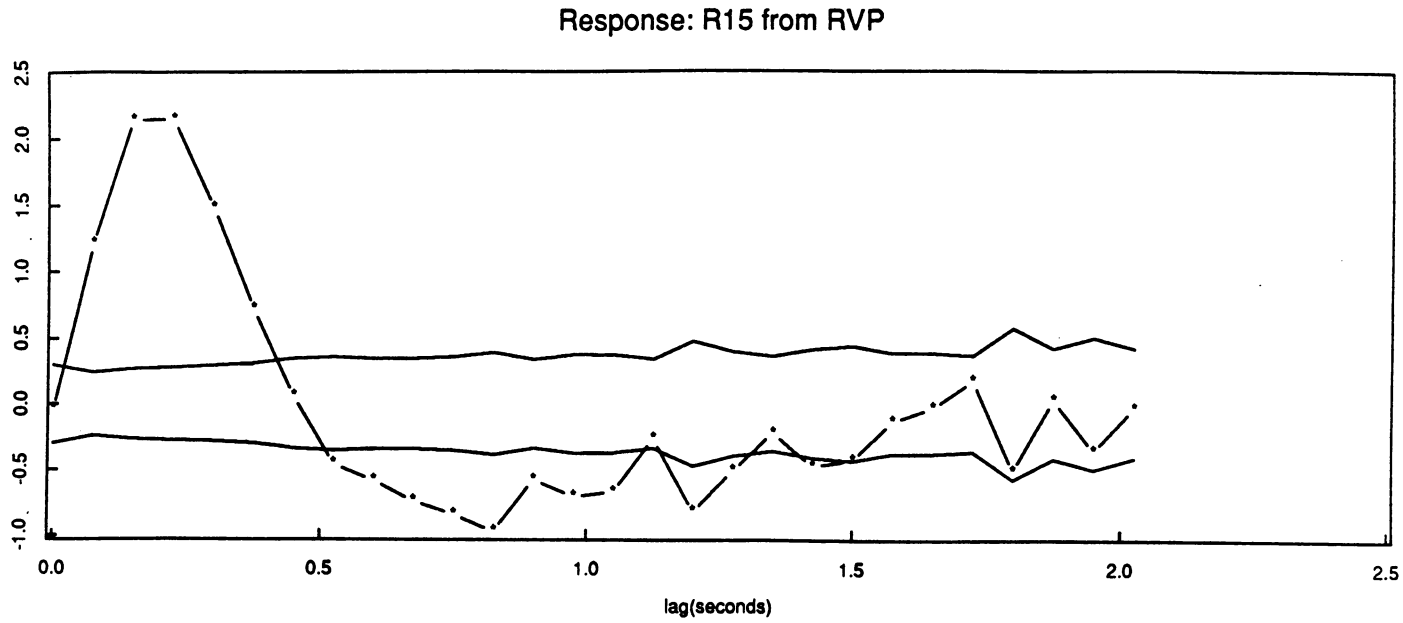
Bottom graph. As for Figure 1.

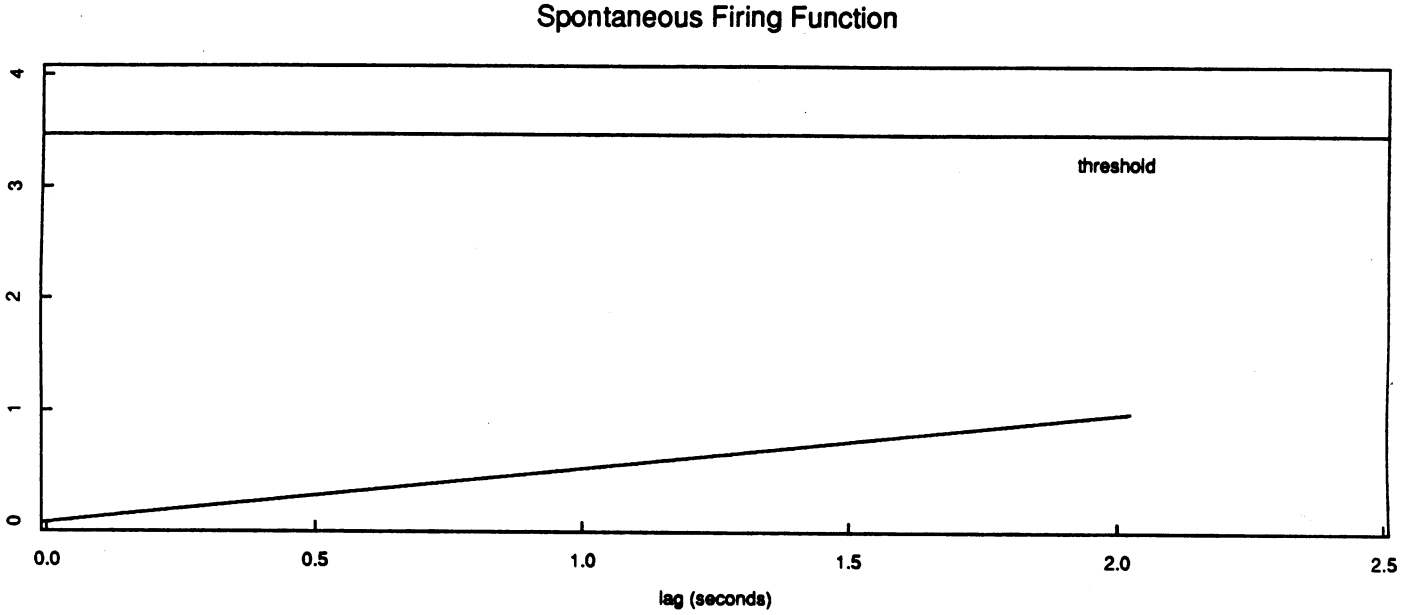
Figure 4. Top graph. Three cell experiment. Estimate of summation functions a_u, b_u of expression (3.5) describing firing of cell L3 in terms of firings of cells L2 and L10. The solid lines give estimated ± 2 standard error limits about 0. level for L2.

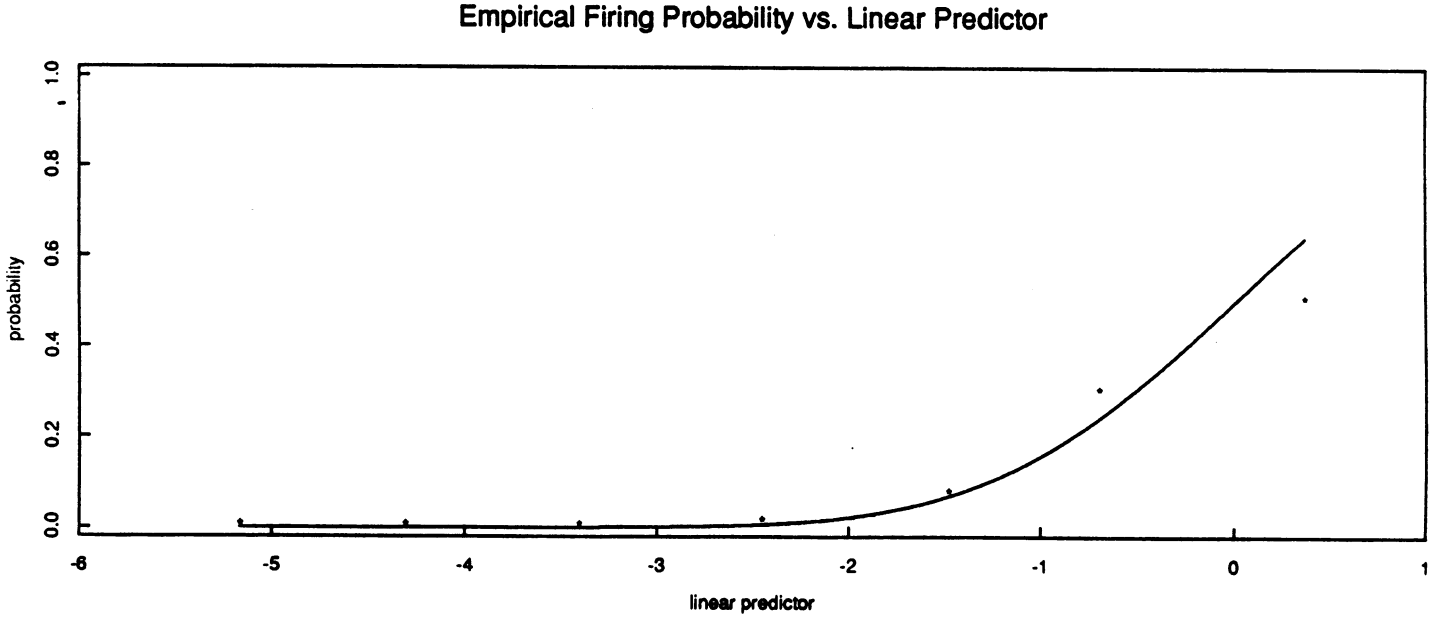
Middle graph. As for Figure 1.

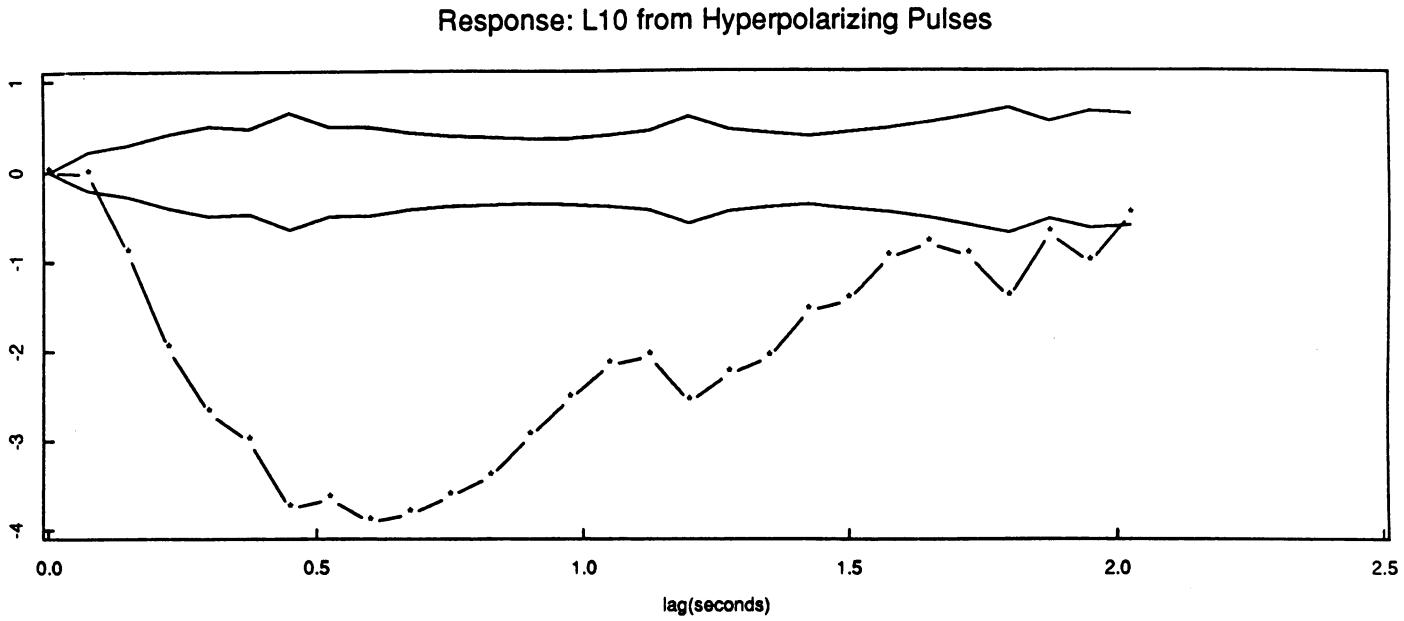
Bottom graph. As for Figure 2.

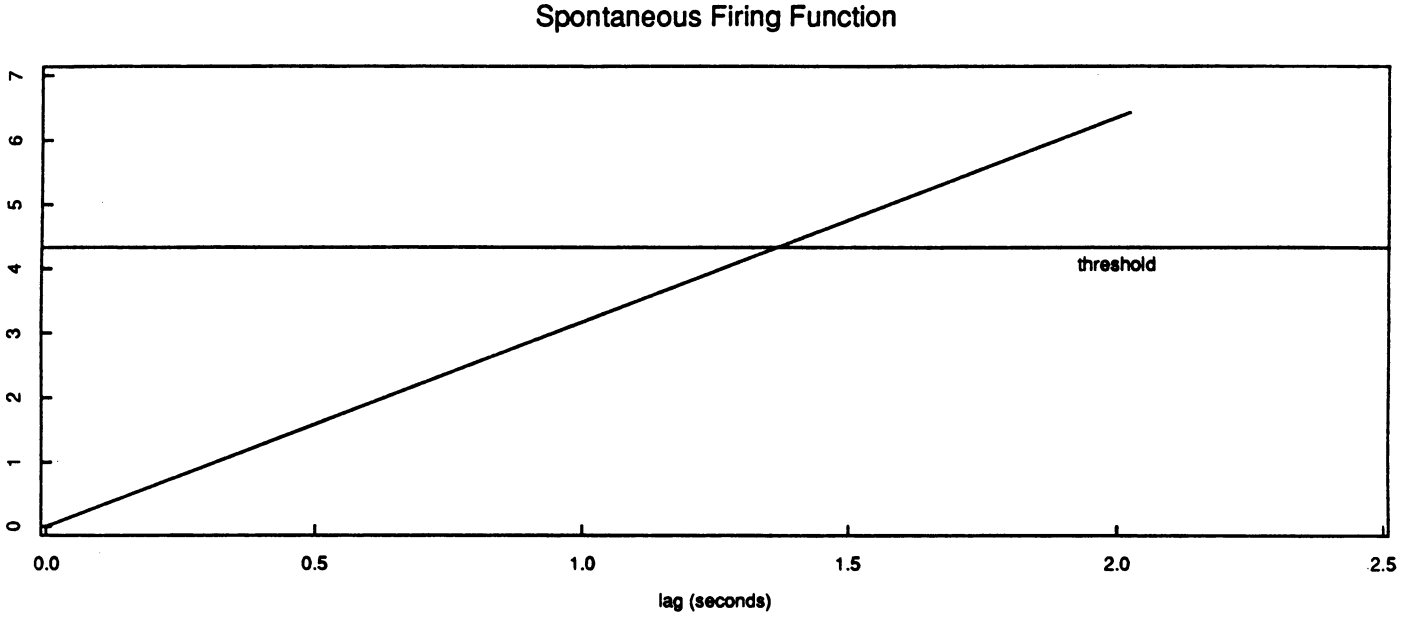
Figure 5. This is the same as Figure 4 except now the dependence of the firing of L2 as a function of L3 and L10 is studied.

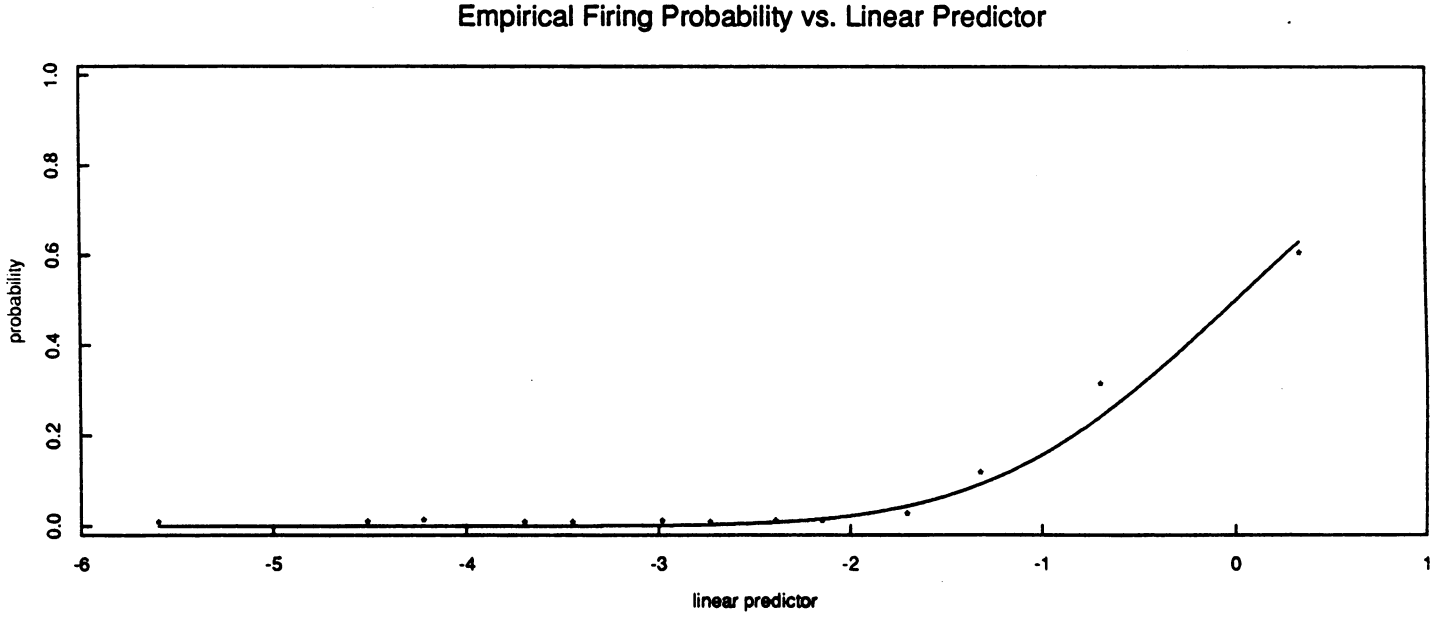

Response: R15 from RVP
2.5
2.0
1.5
1.0
0.5
0.0
-0.5
-1.0
0.0
0.5
1.0
1.5
2.0
2.5
lag(seconds)


Spontaneous Firing Function
4
3
2
1
0
threshold
0.0
0.5
1.0
1.5
2.0
2.5
lag (seconds)

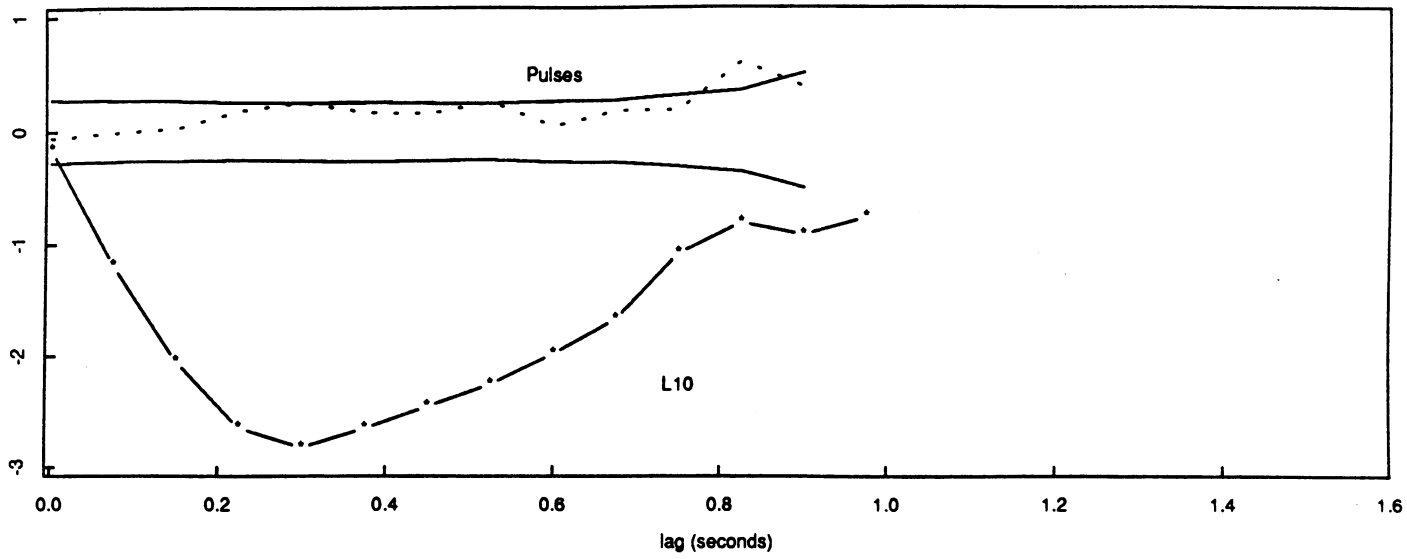

Empirical Firing Probability vs. Linear Predictor
probability
1.0
0.8
0.6
0.4
0.2
0.0
-6
-5
-4
-3
-2
-1
0
1
linear predictor


Response: L10 from Hyperpolarizing Pulses
1
0
-1
-2
-3
-4
0.0
0.5
1.0
1.5
2.0
2.5
lag(seconds)

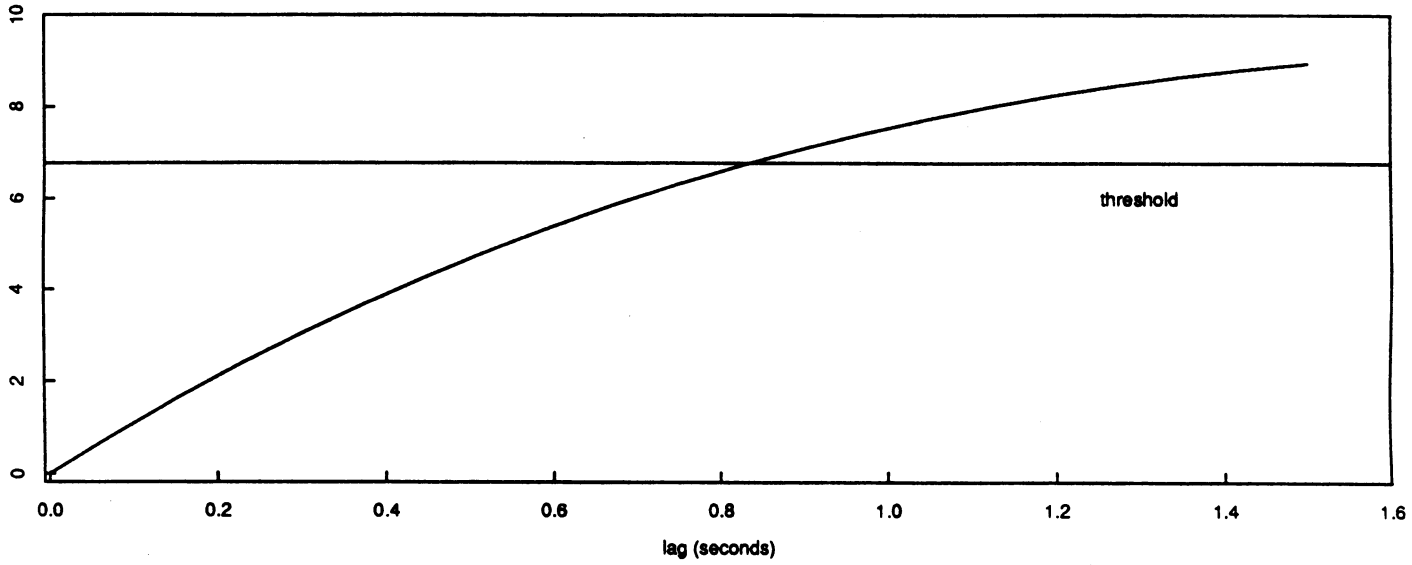

Spontaneous Firing Function
7
6
5
4
3
2
1
0
threshold
0.0
0.5
1.0
1.5
2.0
2.5
lag (seconds)


Empirical Firing Probability vs. Linear Predictor
probability
1.0
0.8
0.6
0.4
0.2
0.0
-6
-5
-4
-3
-2
-1
0
1
linear predictor

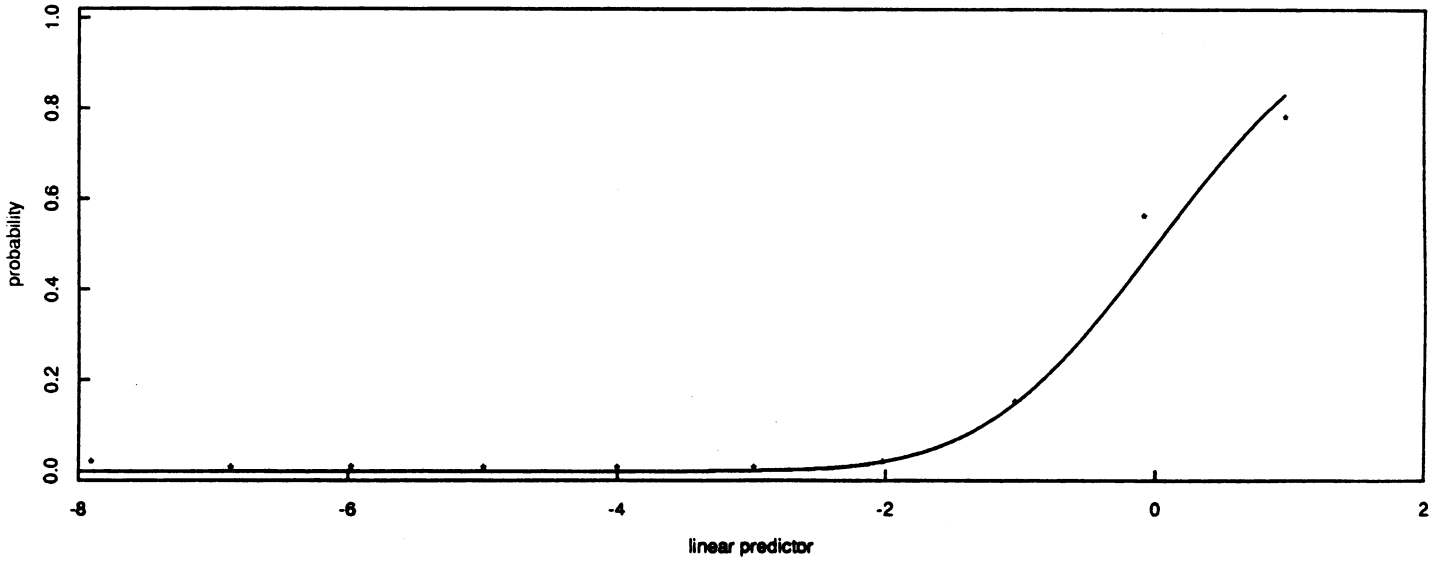
Responses: L3 from Pulses and L10



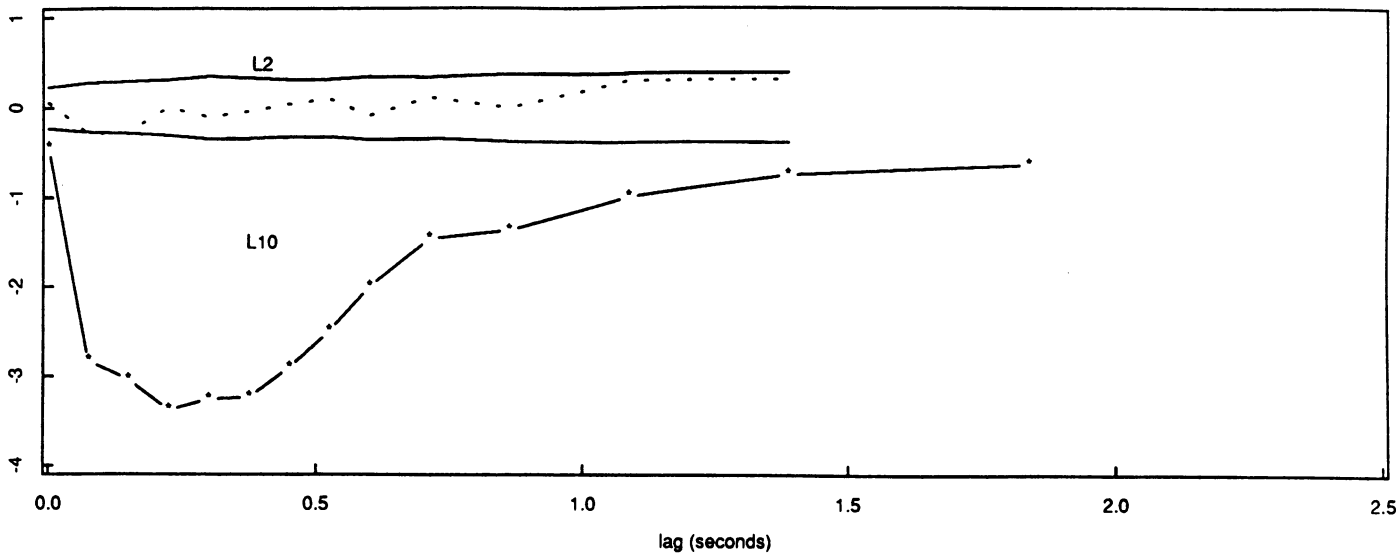
Spontaneous Firing Function



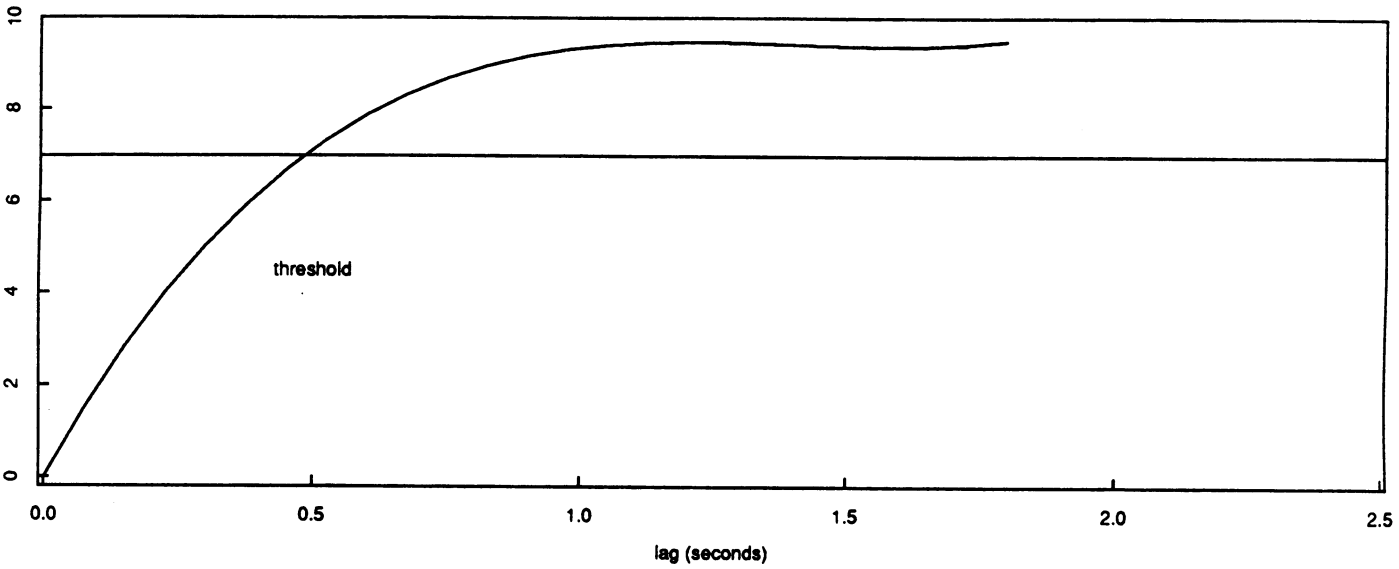
Empirical Firing Probability vs. Linear Predictor



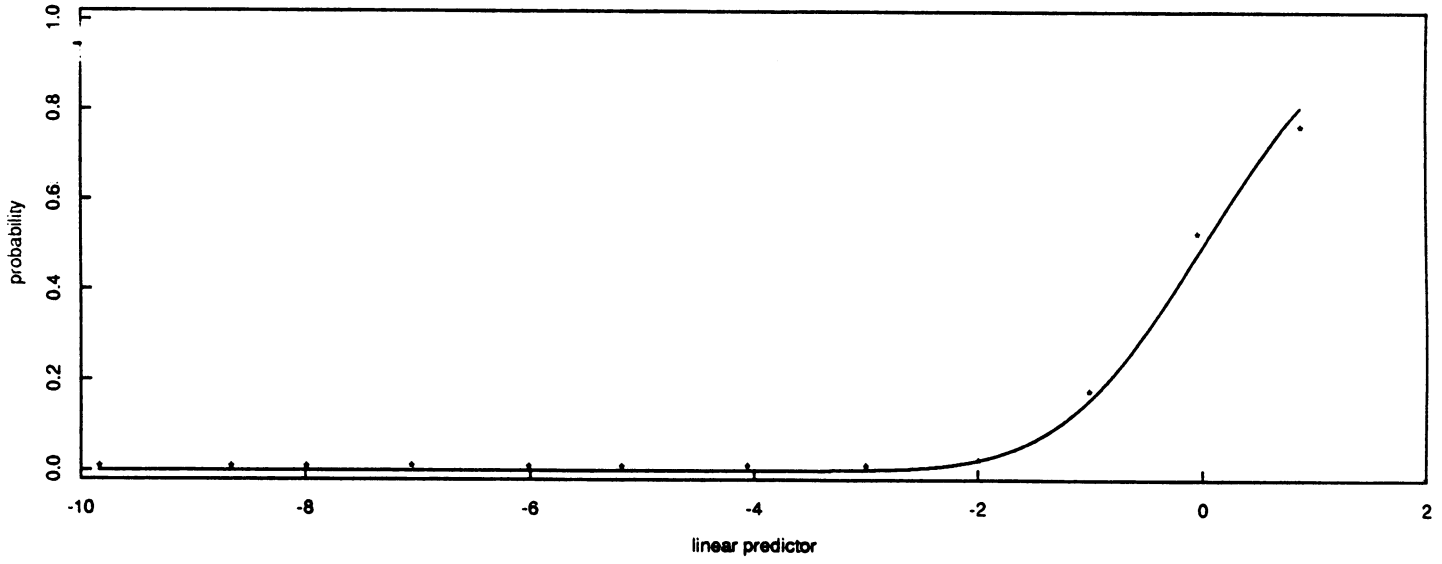
Responses: L3 from L2 and L10

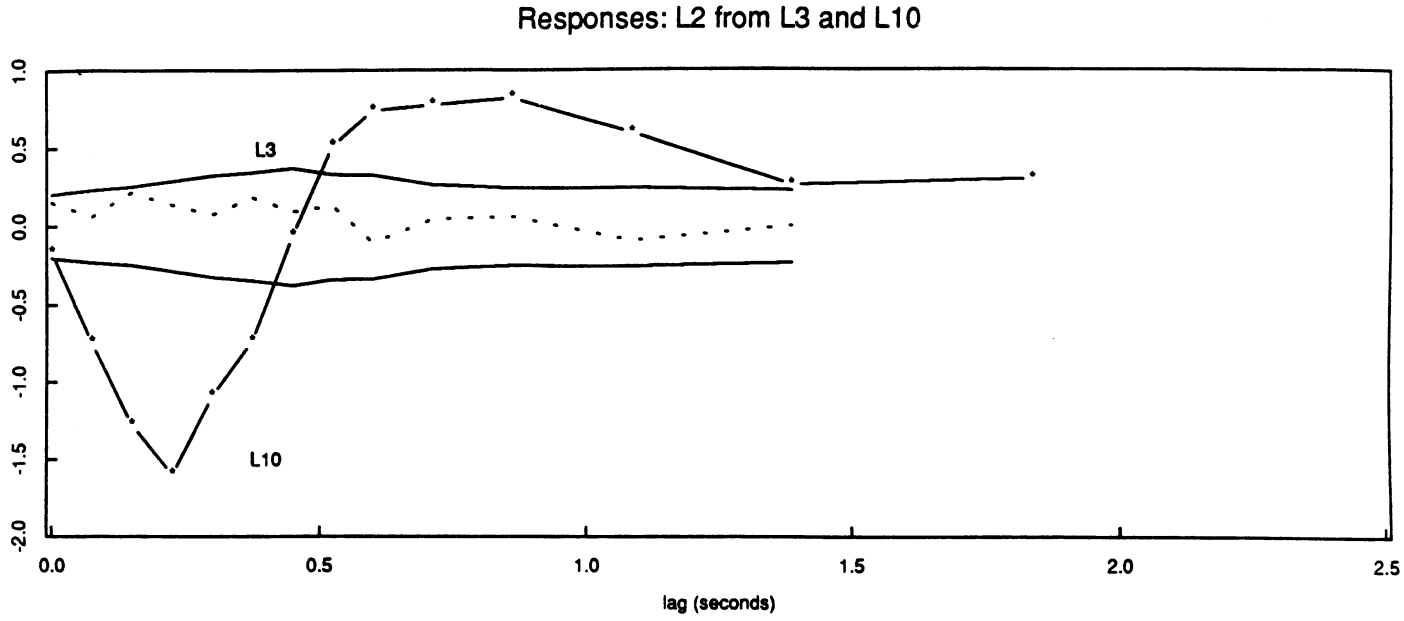


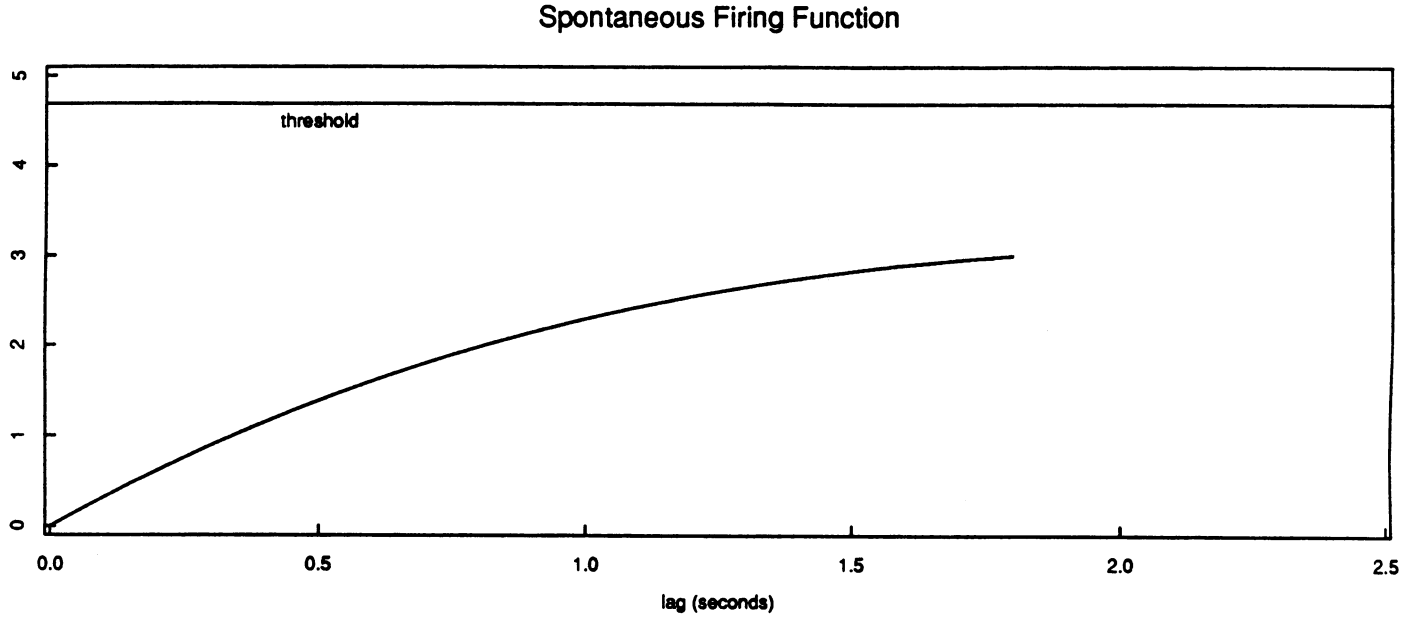
Spontaneous Firing Function

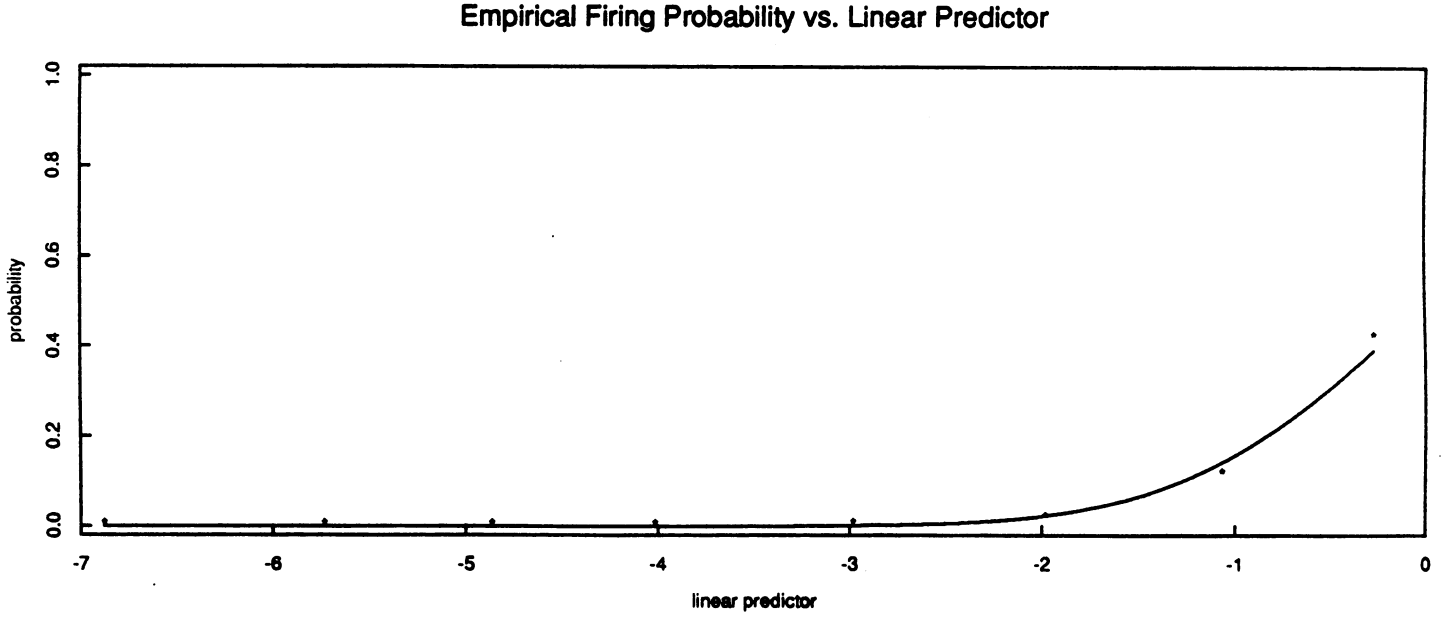


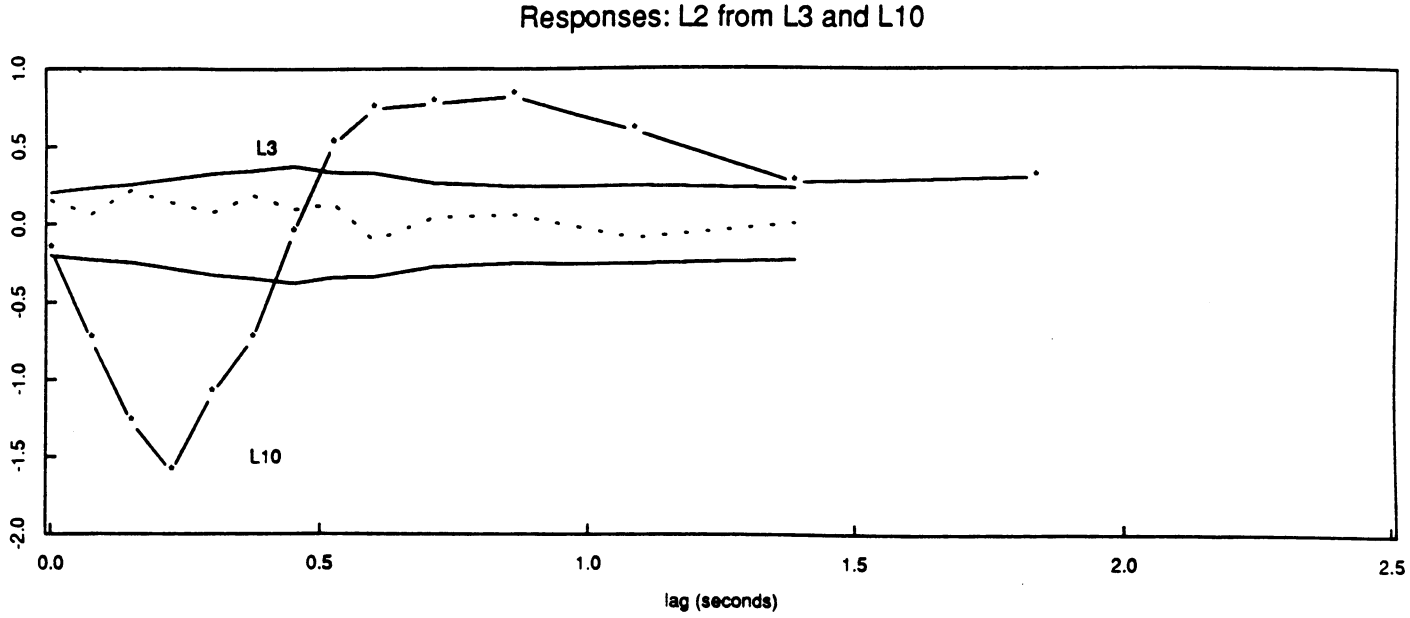
Empirical Firing Probability vs. Linear Predictor

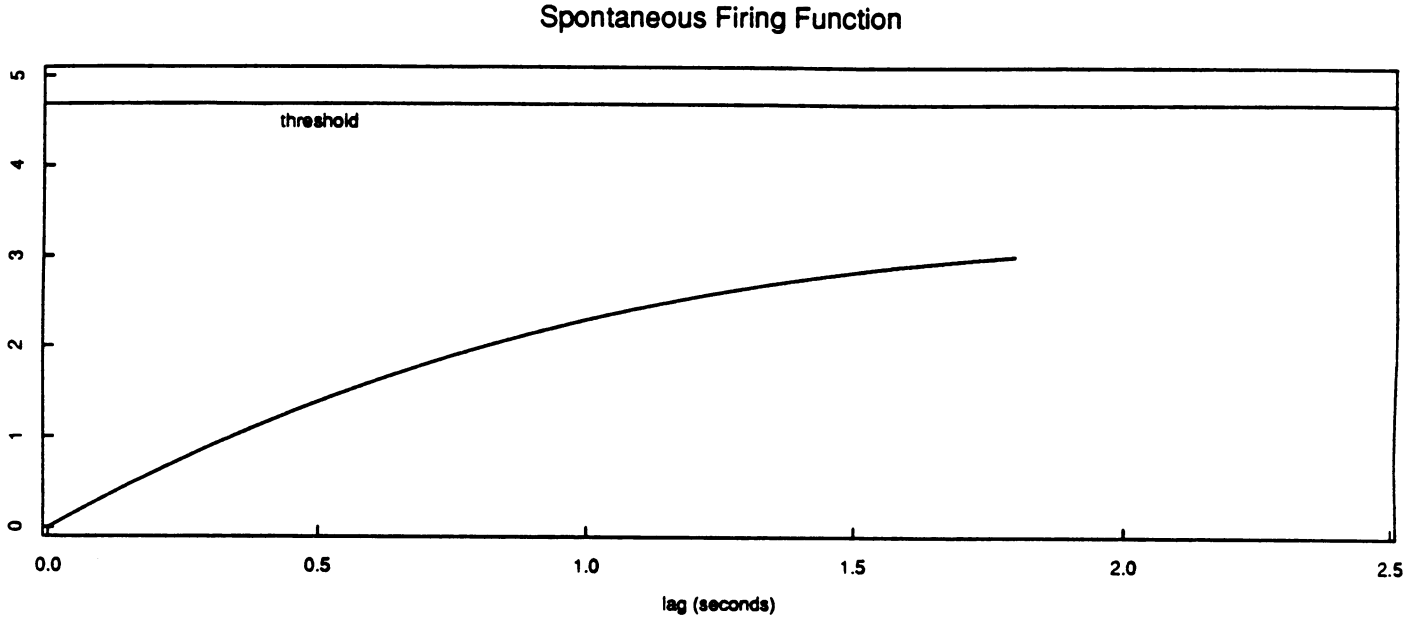


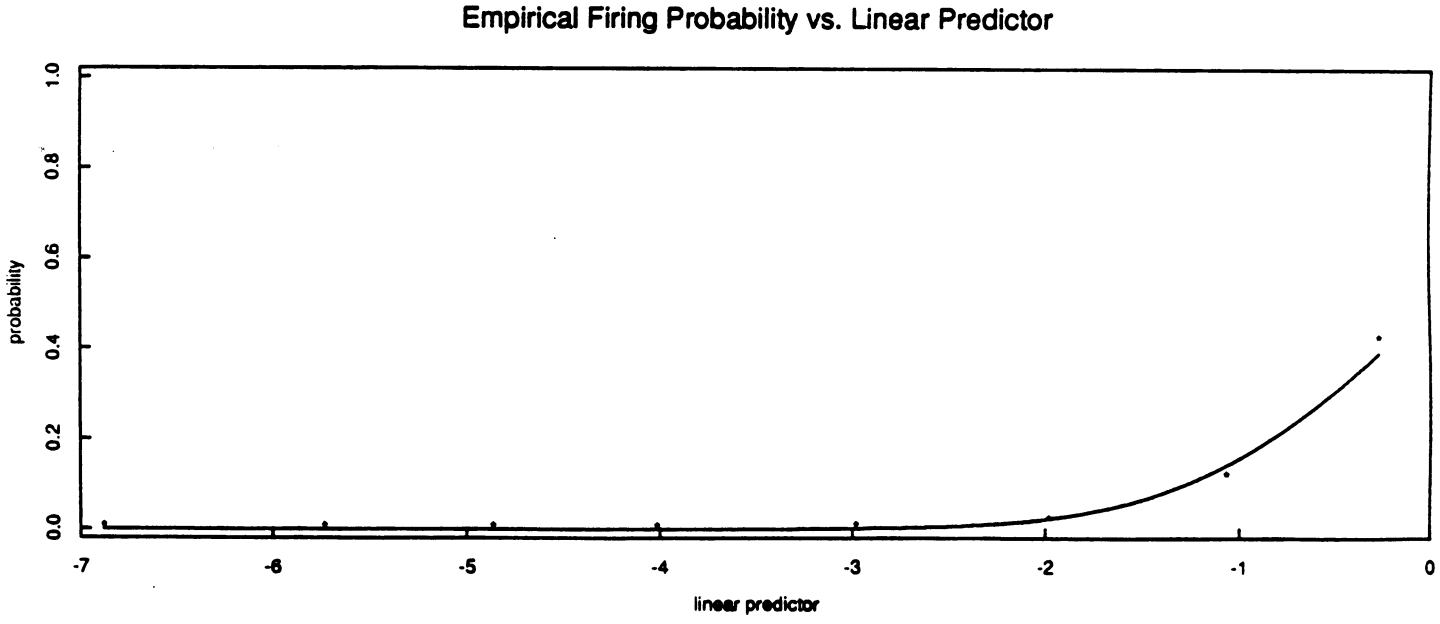

Responses: L2 from L3 and L10
L3
L10
1.0
0.5
0.0
-0.5
-1.0
-1.5
-2.0
0.0
0.5
1.0
1.5
2.0
2.5
lag (seconds)


Spontaneous Firing Function
threshold
5
4
3
2
1
0
0.0
0.5
1.0
1.5
2.0
2.5
lag (seconds)


Empirical Firing Probability vs. Linear Predictor
probability
1.0
0.8
0.6
0.4
0.2
0.0
-7
-6
-5
-4
-3
-2
-1
0
linear predictor


Responses: L2 from L3 and L10
L3
L10
1.0
0.5
0.0
-0.5
-1.0
-1.5
-2.0
0.0
0.5
1.0
1.5
2.0
2.5
lag (seconds)


Spontaneous Firing Function
threshold
5
4
3
2
1
0
0.0
0.5
1.0
1.5
2.0
2.5
lag (seconds)


Empirical Firing Probability vs. Linear Predictor
probability
1.0
0.8
0.6
0.4
0.2
0.0
-7
-6
-5
-4
-3
-2
-1
0
linear predictor