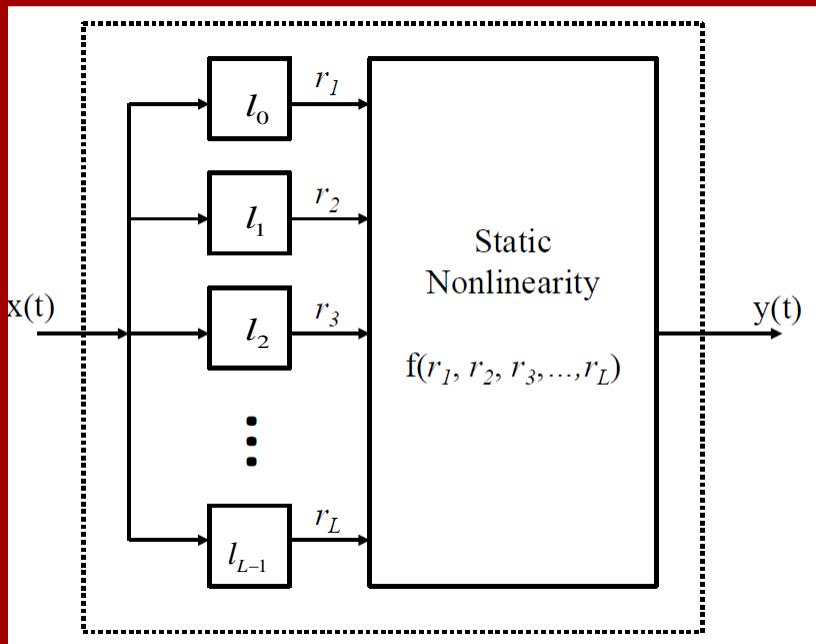


2nd Order Volterra Systems



$$y(n) = k_0 + \sum_m k_1(m) x(n-m) + \sum_{m_1} \sum_{m_2} k_2(m_1, m_2) x(n-m_1) x(n-m_2)$$

The Volterra kernels $k(\cdot)$ are expanded on a complete set of discrete "L" orthogonal Laguerre functions " $l(\cdot)$ " such that the 1st and 2nd order Volterra kernels are:

$$k_1(\tau) = \overline{\sum_{j=0}^{L-1} c_1(j) l_j(\tau)}$$

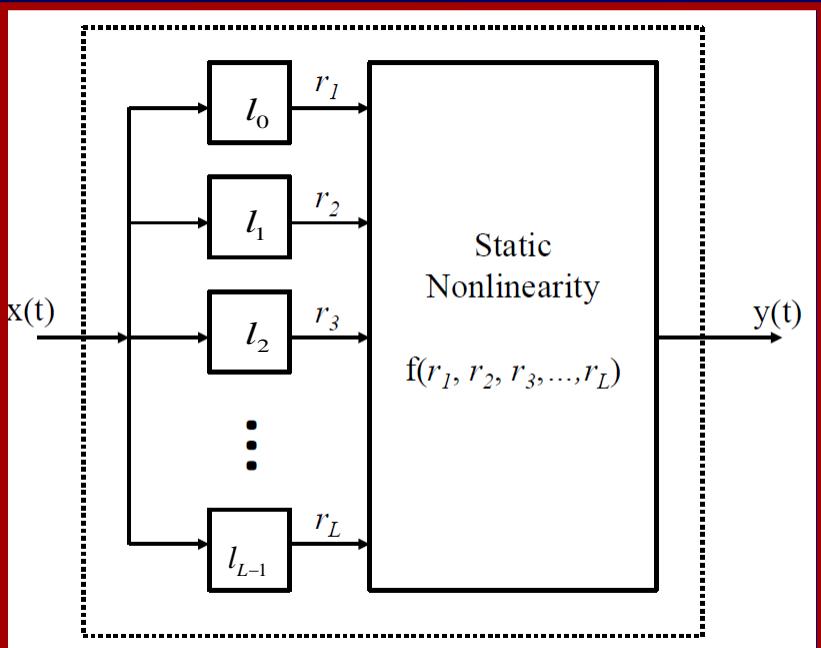
$$k_2(\tau_1, \tau_2) = \overline{\sum_{j_1=0}^{L-1} \sum_{j_2=0}^{L-1} c_2(j_1, j_2) l_{j_1}(\tau_1) l_{j_2}(\tau_2)}$$

$$y(n) = c_0 + \overline{\sum_{j=0}^{L-1} c_1(j) r_j(n)} + \overline{\sum_{j_1=0}^{L-1} \sum_{j_2=0}^{L-1} c_2(j_1, j_2) r_{j_1}(n) r_{j_2}(n)}$$

where

$$r_j(n) = \sum_{\tau=0}^M l_j(\tau) x(n-\tau)$$

2nd Order Volterra Systems



Both $r(\cdot)$ and $v(\cdot)$ are used to represent the outputs of the Laguerre filters

$$y(n) = c_0 + \sum_{j=0}^{L-1} c_1(j)r_j(n) + \sum_{j_1=0}^{L-1} \sum_{j_2=0}^{L-1} c_2(j_1, j_2)r_{j_1}(n)r_{j_2}(n)$$

where

$$r_j(n) = \sum_{\tau=0}^M l_j(\tau)x(n-\tau)$$

Neuronal Modes

For a second order Volterra System,
form the symmetric Coefficient matrix
 \tilde{C} using the estimated coefficients c_0 ,
 $\{c_1\}$, and $\{c_2\}$ of the Laguerre
expansions of the system kernels.

$$\tilde{C} = \begin{bmatrix} c_0 & c_1(1)/2 & c_1(2)/2 & \dots & c_1(L)/2 \\ c_1(1)/2 & c_2(1,1) & c_2(1,2) & \dots & c_2(1,L) \\ c_1(2)/2 & c_2(2,1) & c_2(2,2) & \dots & c_2(2,L) \\ \vdots & \vdots & \vdots & & \vdots \\ c_1(L)/2 & c_2(L,1) & c_2(L,2) & \dots & c_2(L,L) \end{bmatrix}$$

where L is the number of Laguerre fns.

$$\text{Let } \tilde{v}^T = [1 \ v_1 \ v_2 \ \dots \ v_L]^T$$

$$\text{then } y(n) = \tilde{v}^T(n) \tilde{C} \tilde{v}(n)$$

$$\text{where } n = 1, \dots, N$$

But $\tilde{\Sigma} = \tilde{M}^T \tilde{\Lambda} \tilde{M}$
 where \tilde{M} is the matrix of eigen vectors
 and $\tilde{\Lambda}$ is the $\text{diag}(\lambda_0, \lambda_1, \dots, \lambda_L)$
 λ_j is j th eigen value ; $j=0, 1, \dots, L$

Hence,

$$\begin{aligned} y &= \tilde{v}^T \tilde{M}^T \tilde{\Lambda} \tilde{M} \tilde{v} \\ &= (\tilde{M} \tilde{v})^T \tilde{\Lambda} (\tilde{M} \tilde{v}) \\ &= \hat{v}^T \tilde{\Lambda} \hat{v} \end{aligned}$$

$$\hat{v}_j = m_{j,0} + m_{j,1} v_1 + m_{j,2} v_2 + \dots + m_{j,L} v_L$$

$[m_{j,0} \ m_{j,1} \ m_{j,2} \ \dots \ m_{j,L}]^T$ is the j th eigen vector of $\tilde{\Sigma}$.

Select K dominant eigen values such that $K \leq L$. They correspond to the K dominant Neuronal modes m_1, m_2, \dots, m_K .

Choose

$$\frac{|\lambda_i|}{\sum_j |\lambda_j|} \geq \epsilon$$

$$\epsilon = 0.01 - 0.05$$

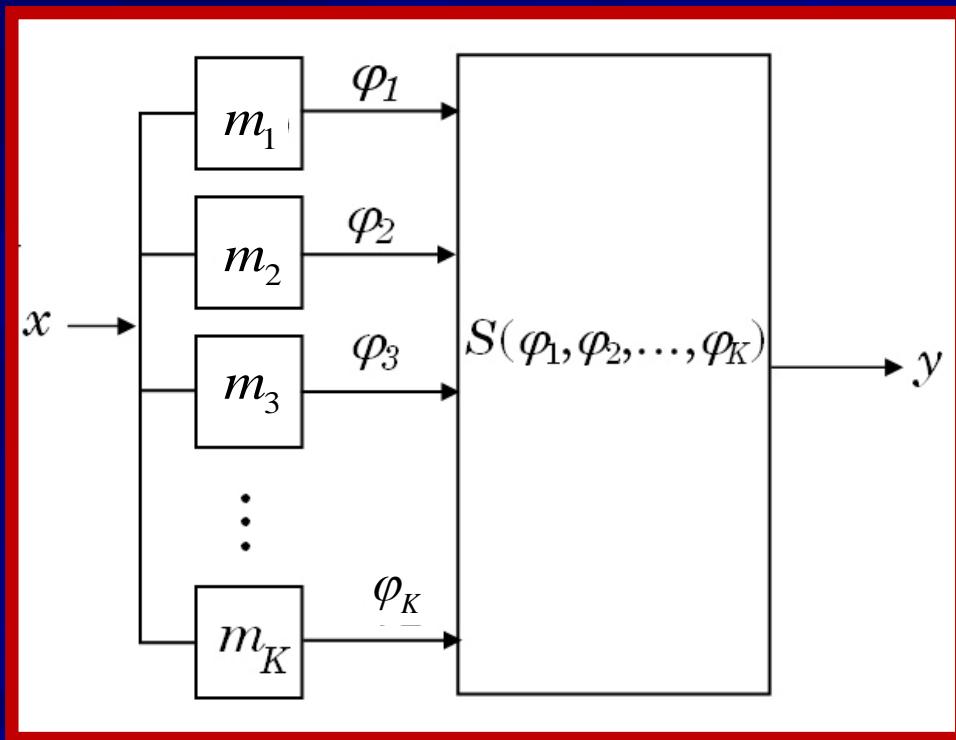
$$m_j = m_{j,0} + m_{j,1} b_1 + m_{j,2} b_2 + \dots + m_{j,L} b_L$$
$$j = 1, \dots, K$$

$K \triangleq$ Number of Neuronal Modes.

$L \triangleq$ Number of Laguerre Functions.

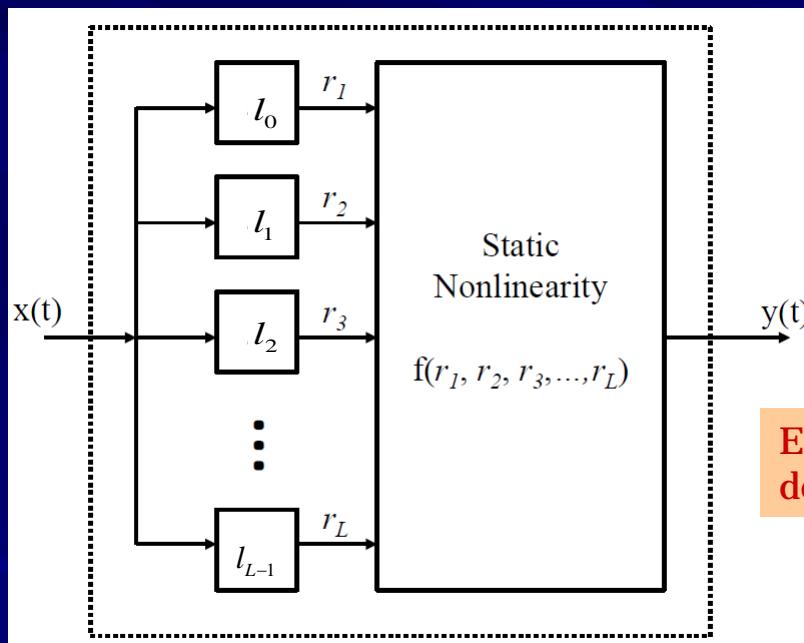
$b_i \triangleq (i-1)^{\text{th}}$ Discrete Laguerre Function, b_{i-1} .

Modular Volterra Models

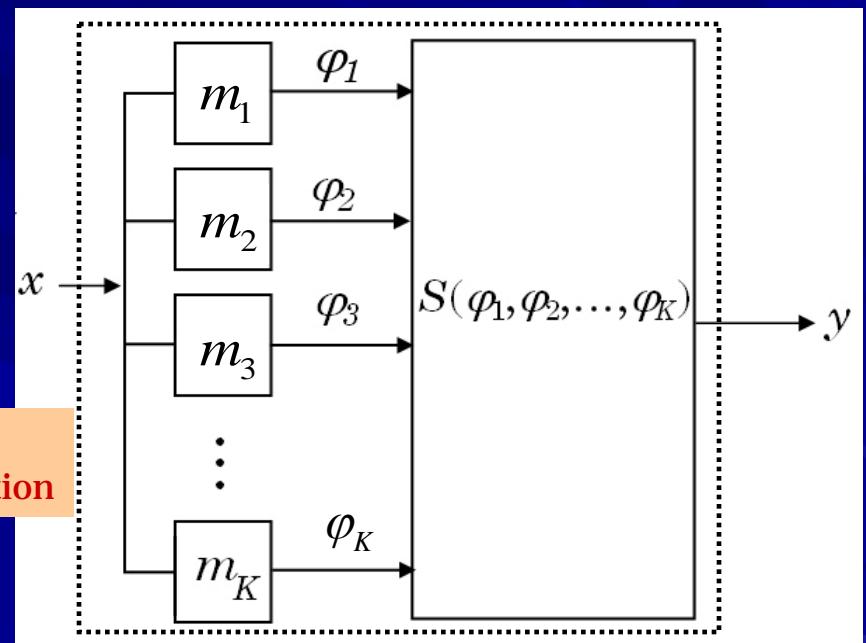


Wiener - Bose Model

Modular Volterra Model

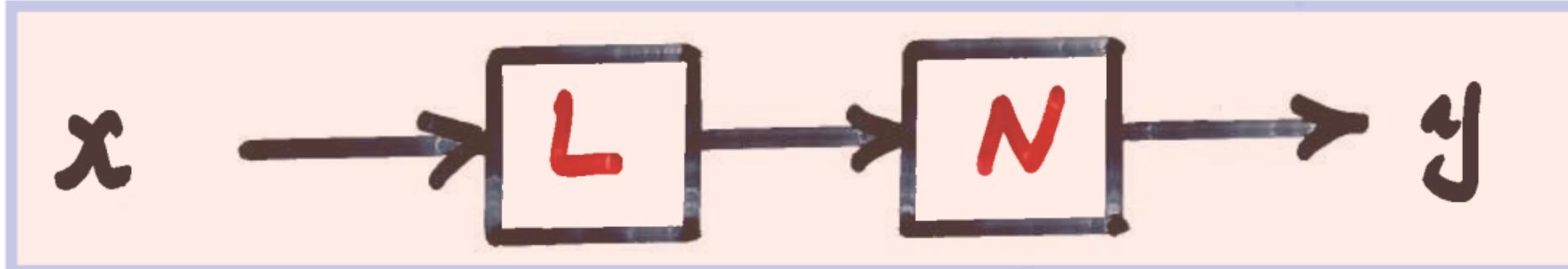


Eigen-decomposition

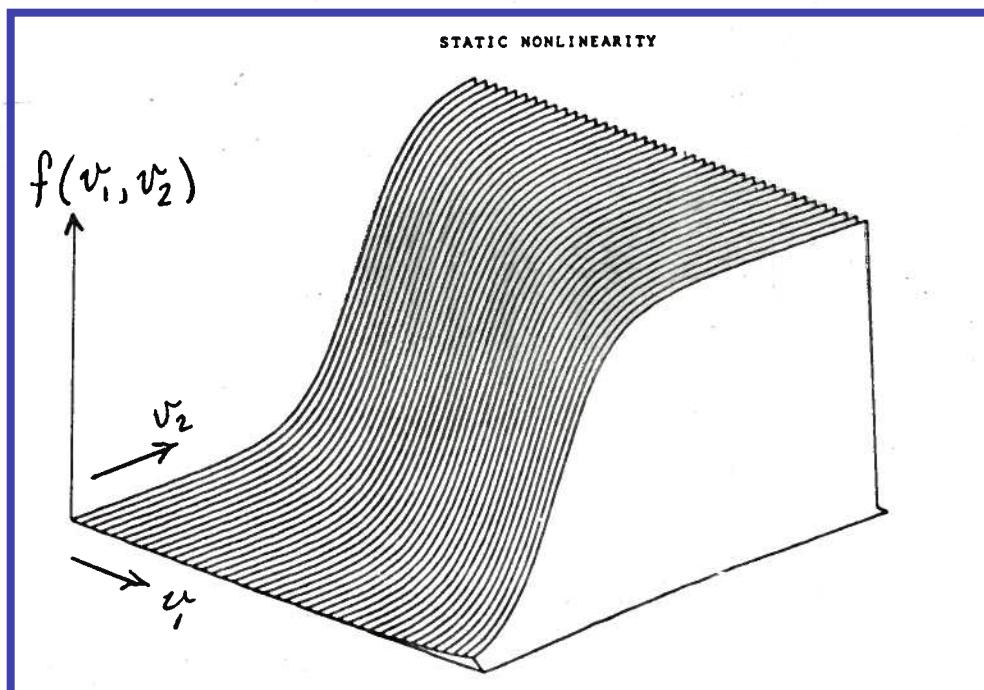
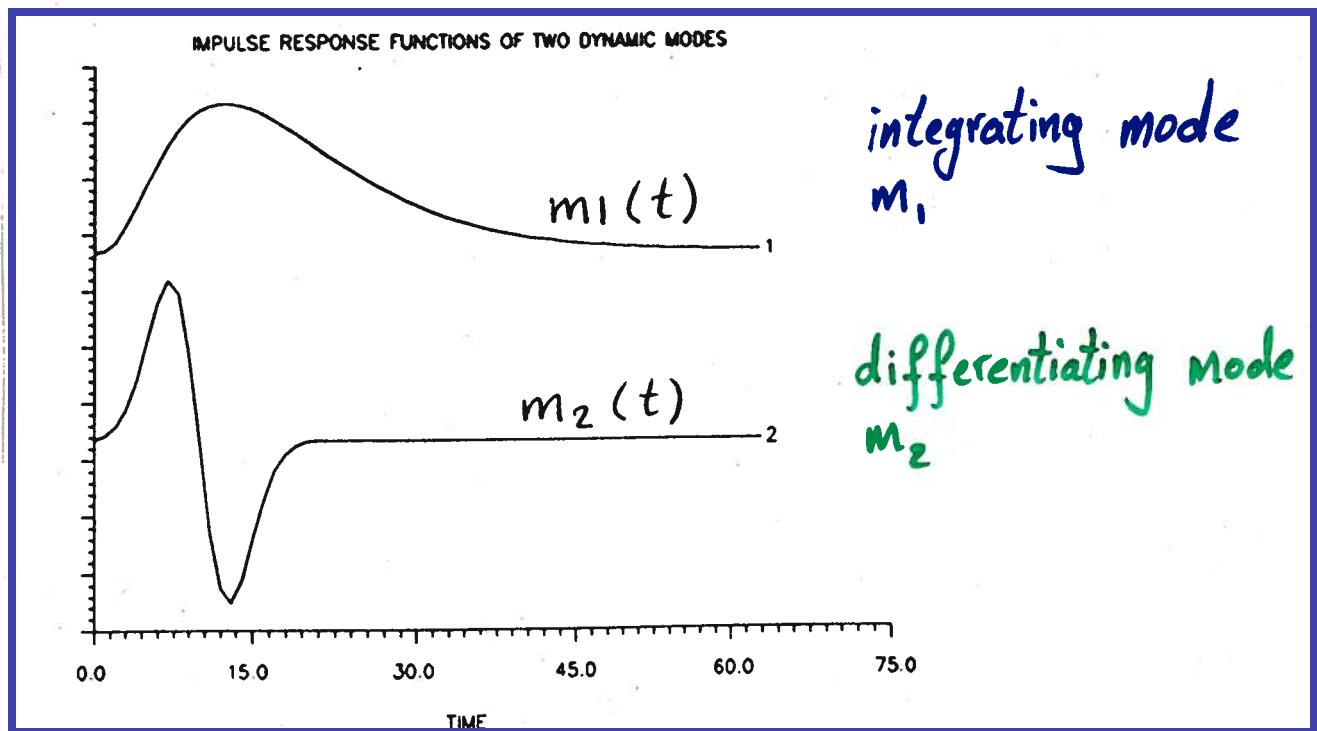
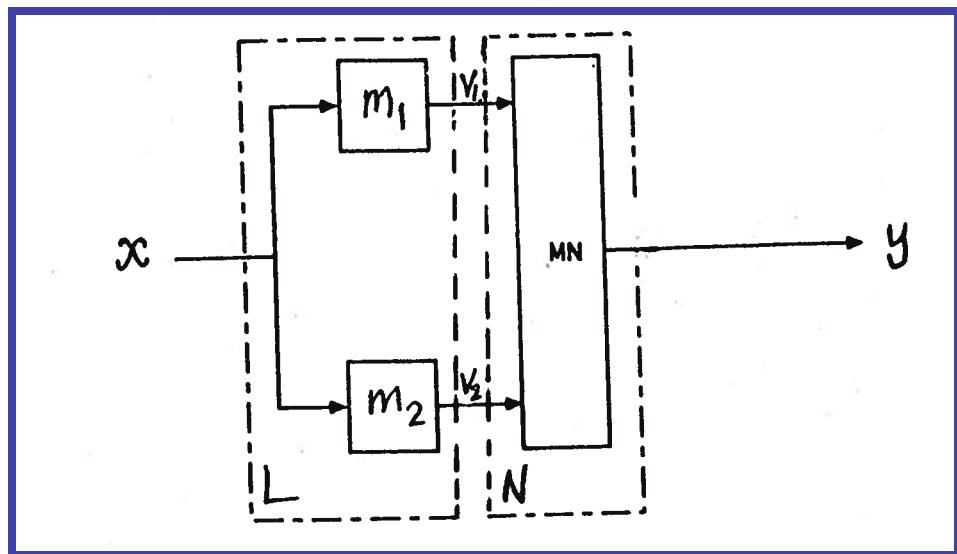


Signal Transformation in a Neural Unit

Example



Modular Volterra Model



$$m_1(t) = \frac{t^2}{12} [e^{-\frac{t}{4}} - e^{-\frac{t}{8}}] \quad (\text{integrating mode})$$

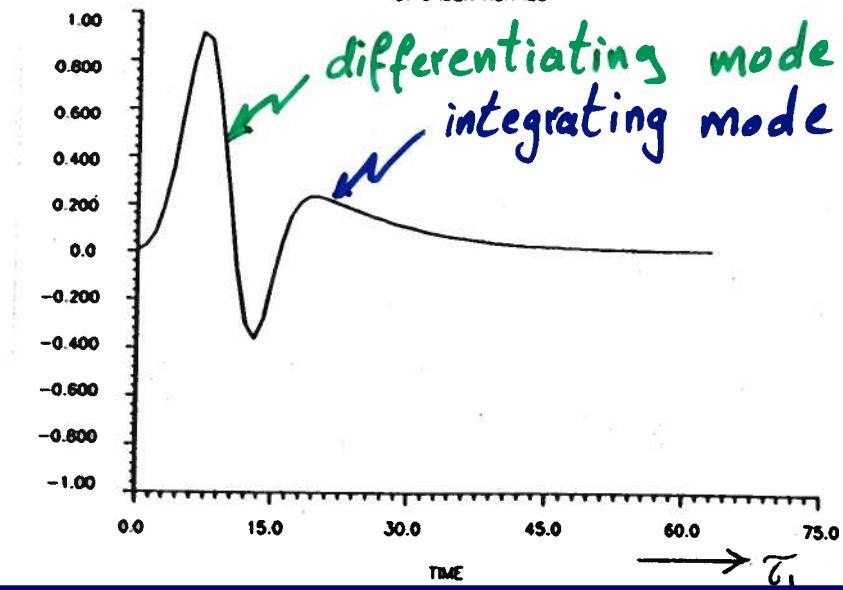
$$m_2(t) = -[t-10] \left[e^{-\frac{(t-10)^2}{16}} \right] \quad (\text{differentiating mode})$$

The Multi-Input Nonlinearity (MN) is

$$f(v_1, v_2) = \left[1 + 0.4 e^{-0.2(v_1+2v_2)} \right]^{-1}$$

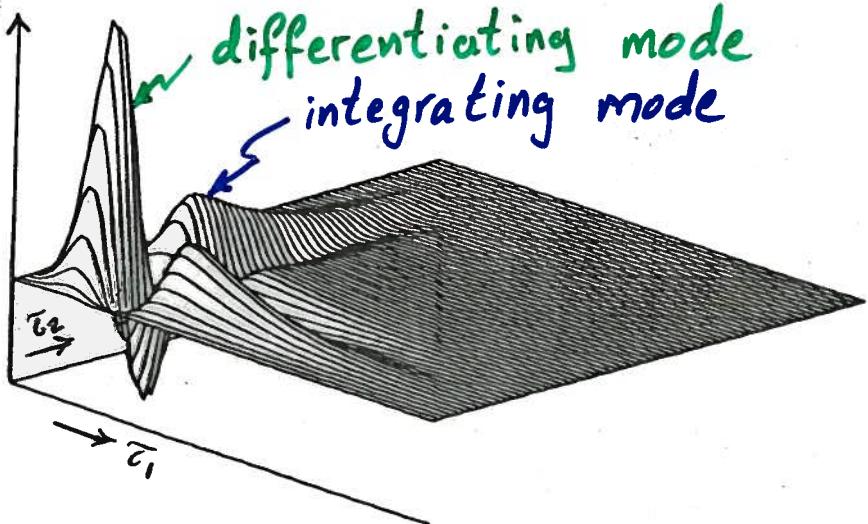
$h_1(\bar{\tau}_1)$

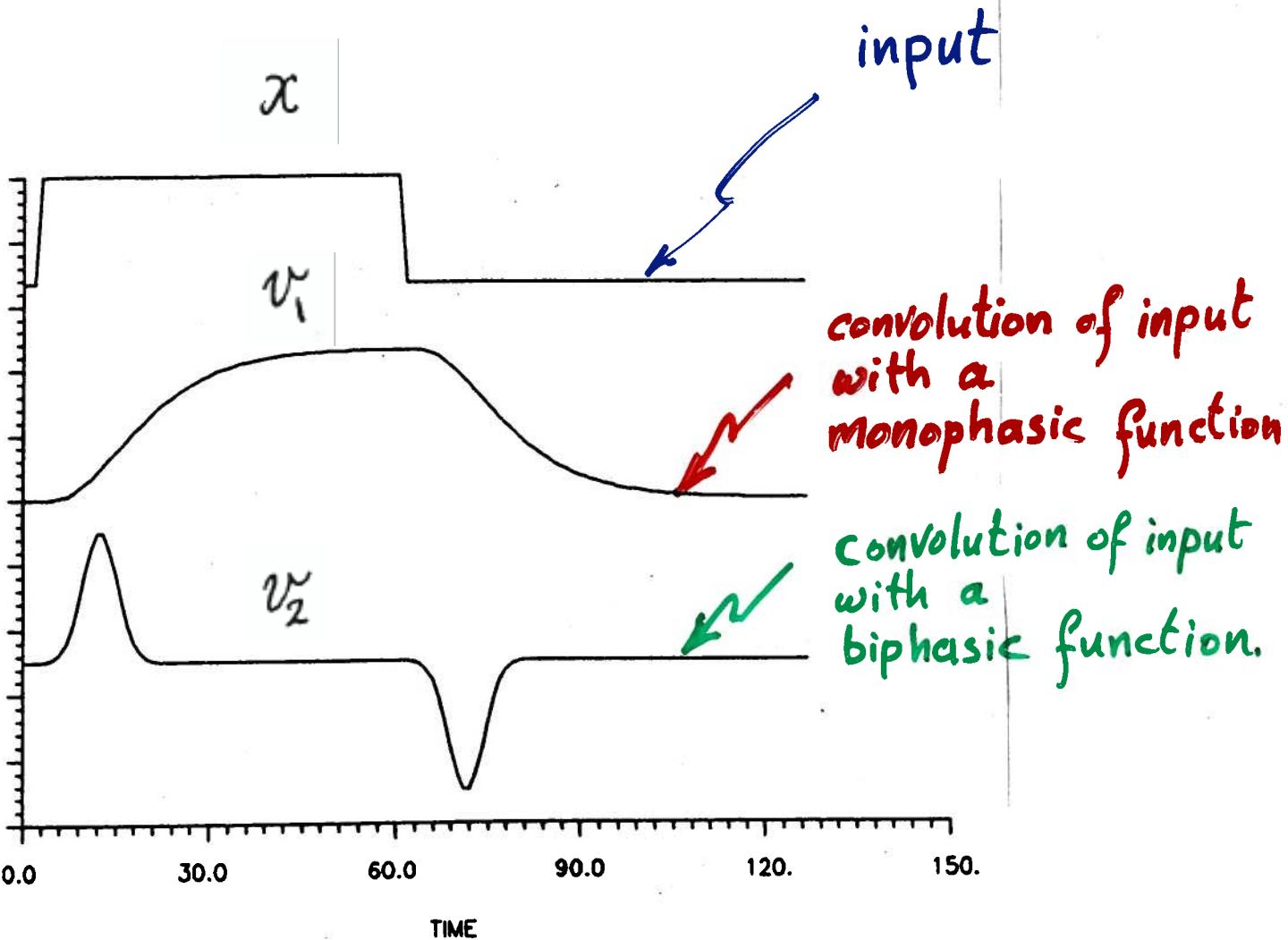
FIRST ORDER KERNEL



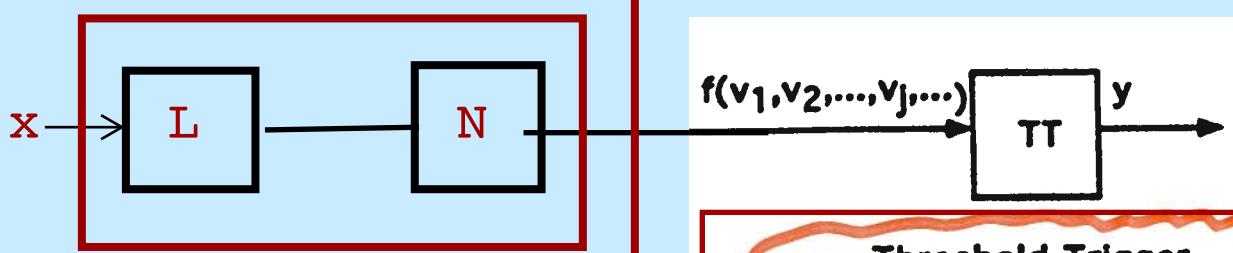
$h_2(\bar{\tau}_1, \bar{\tau}_2)$

SECOND ORDER KERNEL



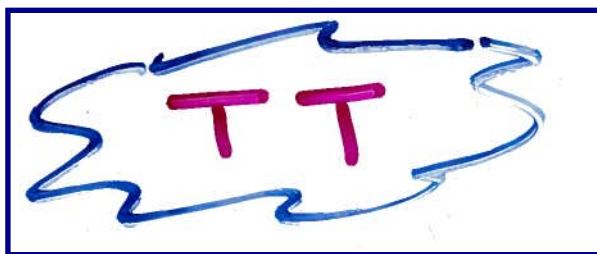


Block-structured model of a "spiking" system



Threshold-Trigger
A spike is produced whenever $f(v_i, \dots, v_j, \dots) \geq \theta$
Threshold

$TT \triangleq$ Threshold Trigger



$$y = \frac{1}{2} + \frac{1}{2} \operatorname{sgn} [f(v_1, v_2, \dots) - \theta]$$

↑
signum
↑
threshold

To include refractoriness :

If $y(n) = 1$, then

$$y(n+1) = 0$$

⋮

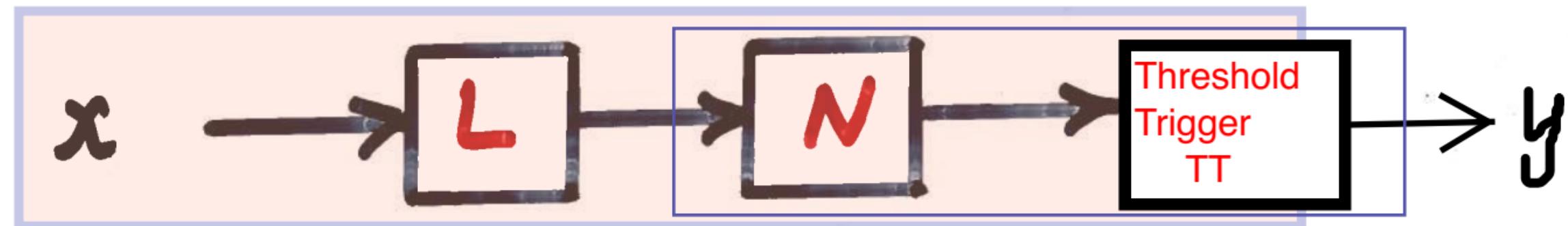
$$y(n+t_r) = 0$$

where $t_r \equiv$ refractory period

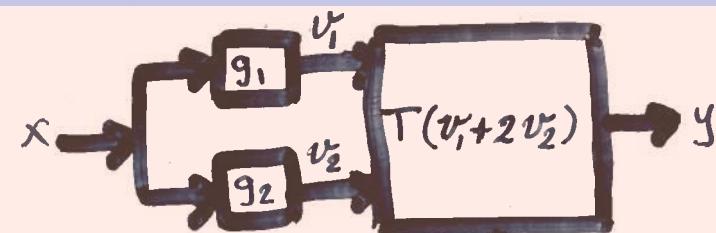
♪ This is a hard-threshold nonlinearity with refractoriness.

Signal Transformation and Coding in a Neural Unit

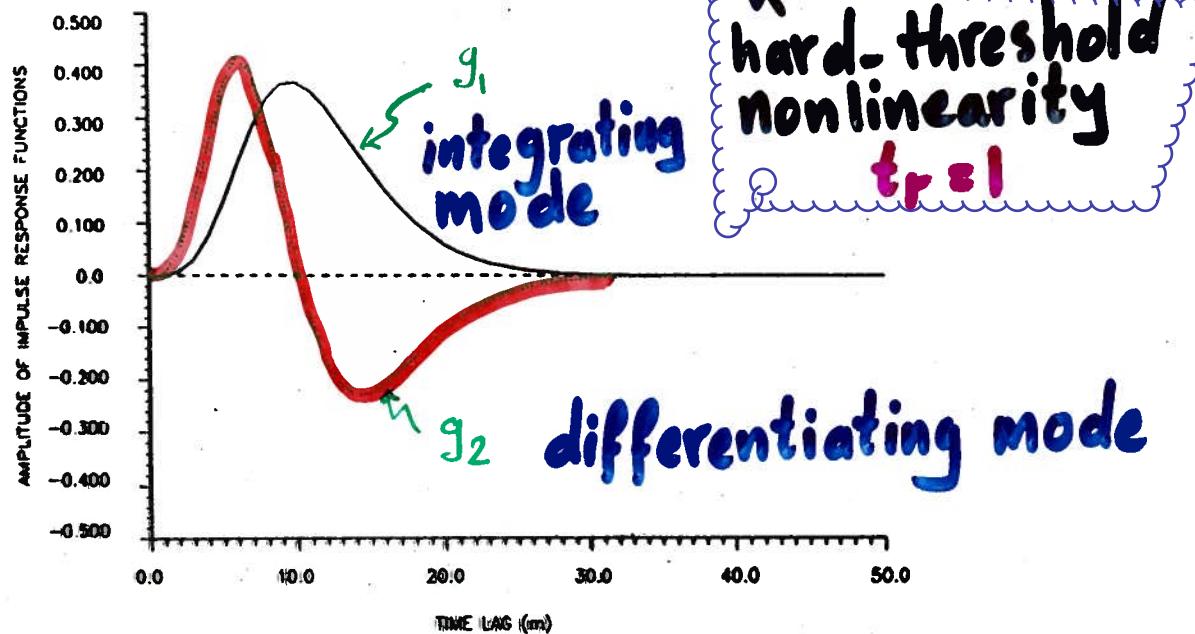
Example



Example :



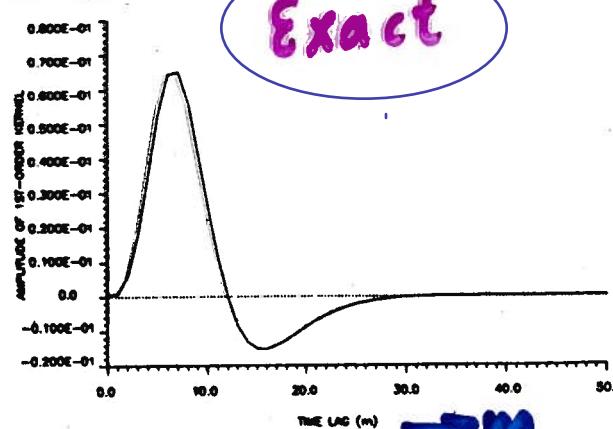
Modular
Volterra
Model



hard-threshold
nonlinearity
 $T_r = 1$

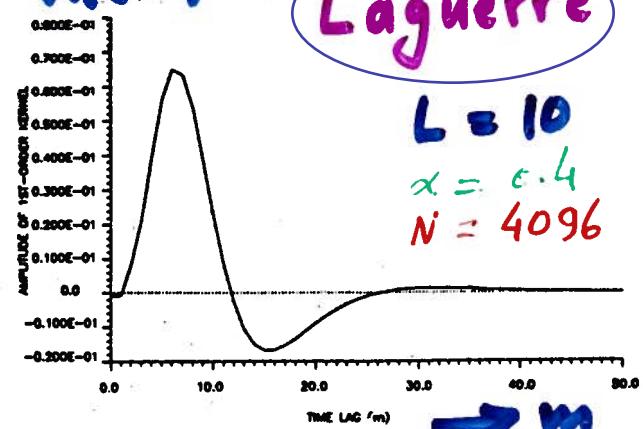
differentiating mode

$R_1(m)$



Exact

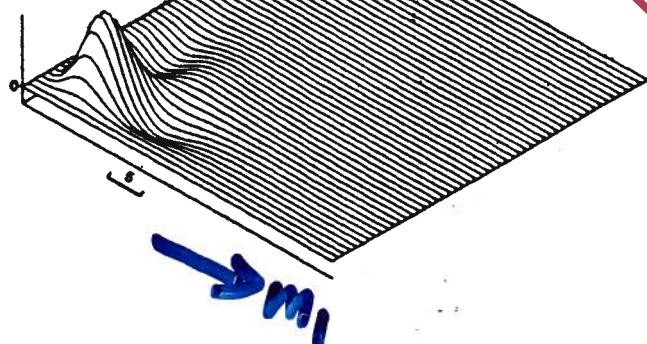
$R_1(m)$



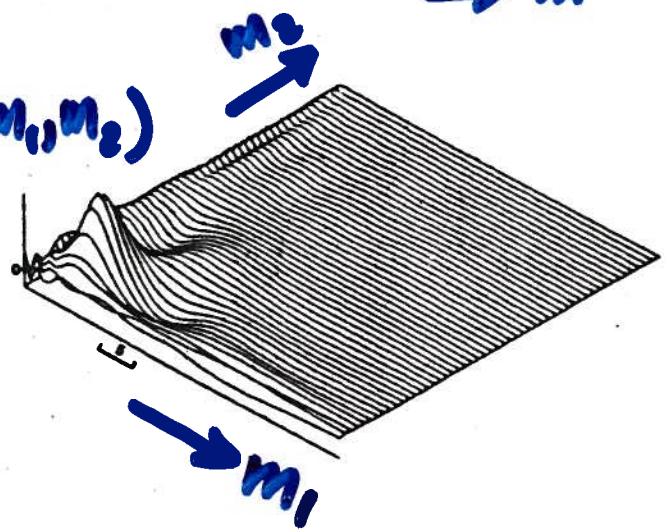
Laguerre

$$L = 10 \\ \alpha = 0.4 \\ N = 4096$$

$k_2(m_1, m_2)$



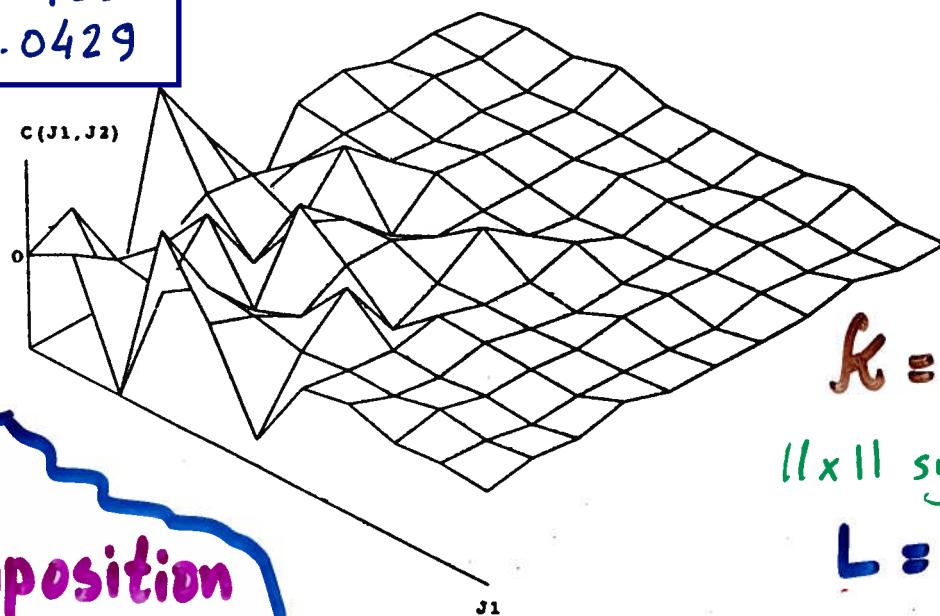
$R_2(m_1, m_2)$



A 3D Perspective for ζ

$$\zeta_{\max} = 0.0433$$

$$\zeta_{\min} = -0.0429$$



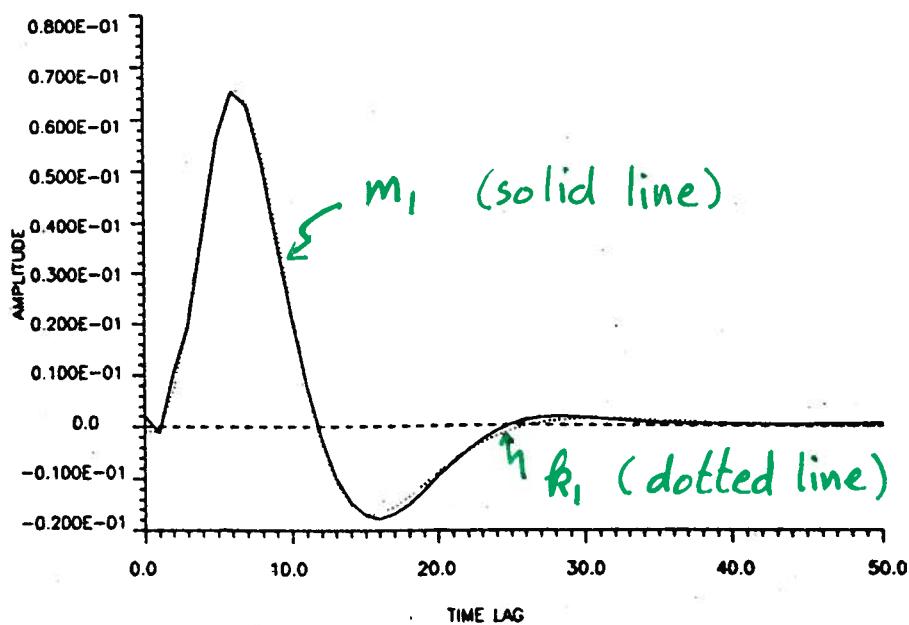
no. of distinct
Laguerre
coefficients
 \nwarrow
 $k = 55$

11×11 symmetric matrix

$$L = 10$$

Eigen
Decomposition

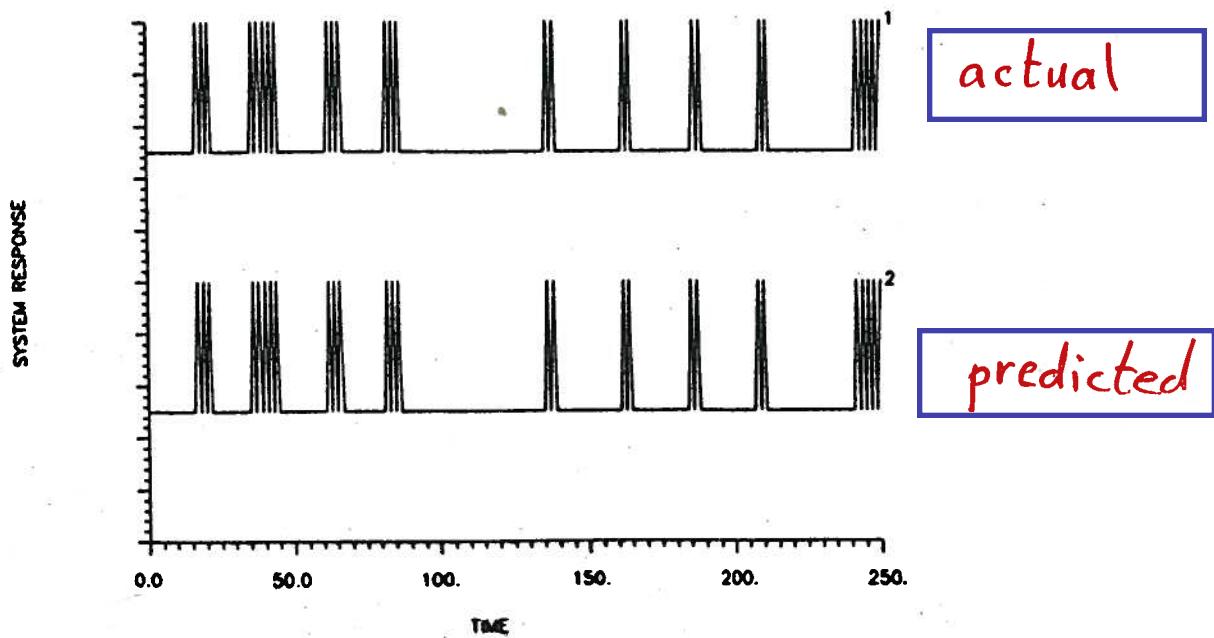
Only one dominant eigen value
 \Rightarrow One neuronal mode m_1 .



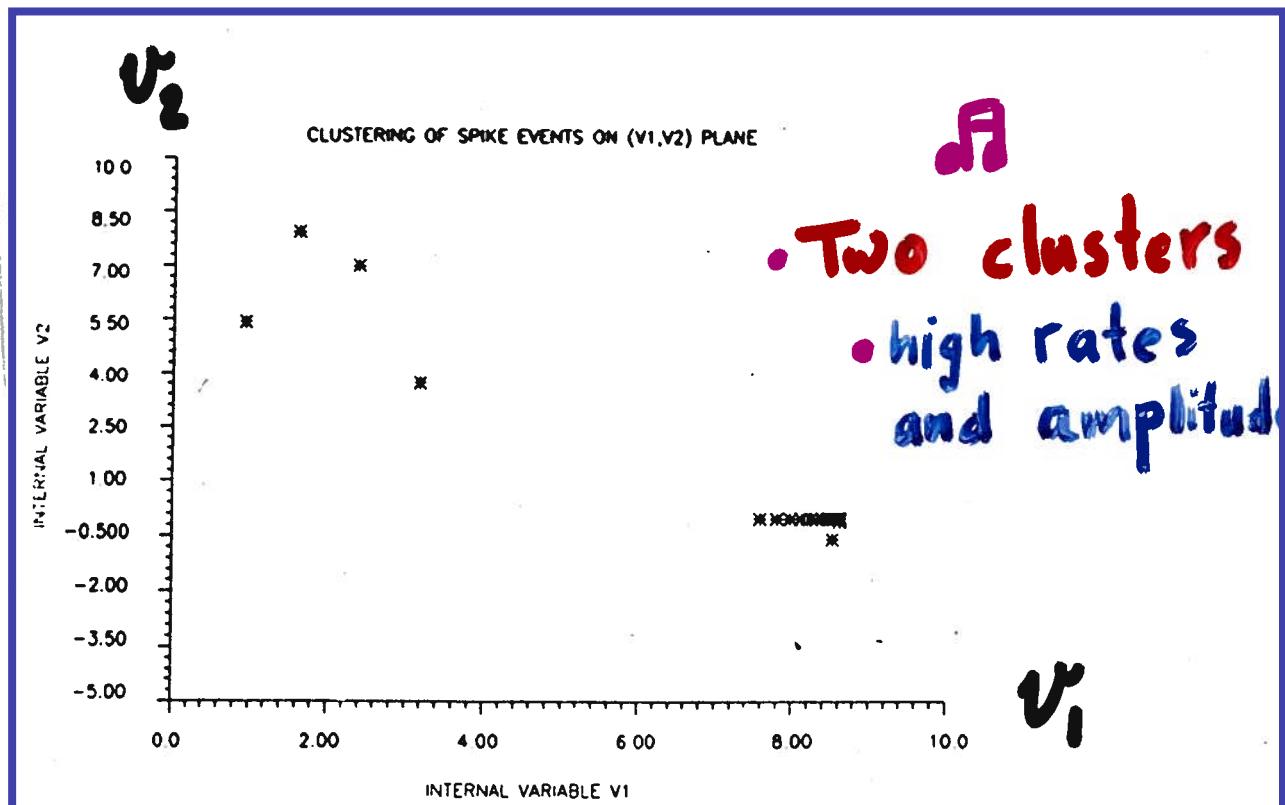
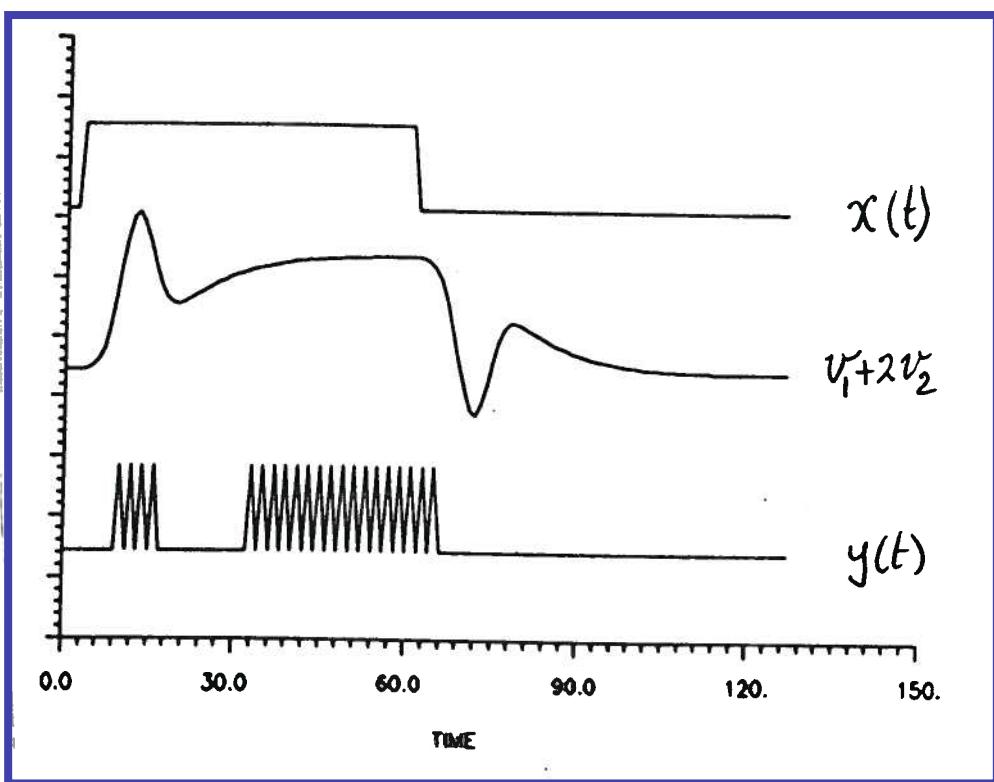
m_1 is in agreement with R_1 .

Spike-train responses

Threshold $\theta = 0.12$



- * Actual response obtained from original model (with g_1 and g_2) .
- * Predicted response obtained from derived model (with m_1) .
- * Response to a random stimulus different from the one used for model estimation .



Spike output codes the onset of the event and its duration.

2nd Order Volterra Systems

$$\begin{aligned}y(n) = & k_0 + \sum_m k_1(m) x(n - m) \\& + \sum_{m_1} \sum_{m_2} k_2(m_1, m_2) x(n - m_1) x(n - m_2)\end{aligned}$$

Principal Dynamic Modes (PDMs)

$$y_2(n) = \mathbf{x}^T(n) \mathbf{Q} \mathbf{x}(n) \quad \text{Output in terms of 1st & 2nd order Volterra kernels}$$

$$\mathbf{Q} = \begin{bmatrix} k_0 & \frac{1}{2}k_1(0) & \frac{1}{2}k_1(1) & \cdots & \frac{1}{2}k_1(M) \\ \frac{1}{2}k_1(0) & k_2(0, 0) & k_2(0, 1) & \cdots & k_2(0, M) \\ \frac{1}{2}k_1(1) & k_2(1, 0) & k_2(1, 1) & \cdots & k_2(1, M) \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ \frac{1}{2}k_1(M) & k_2(M, 0) & k_2(M, 1) & \cdots & k_2(M, M) \end{bmatrix} = \begin{bmatrix} k_0 & \frac{1}{2}\mathbf{k}_1^T \\ \frac{1}{2}\mathbf{k}_1 & \mathbf{k}_2 \end{bmatrix}$$

\mathbf{k}_1 and \mathbf{k}_2 are matrices for 1st & 2nd order Volterra kernels

$$\mathbf{Q} = \mathbf{R}^T \Lambda \mathbf{R}$$

\mathbf{R} is the eigen vector matrix, and Λ is the diagonal eigen value matrix

sth PDM

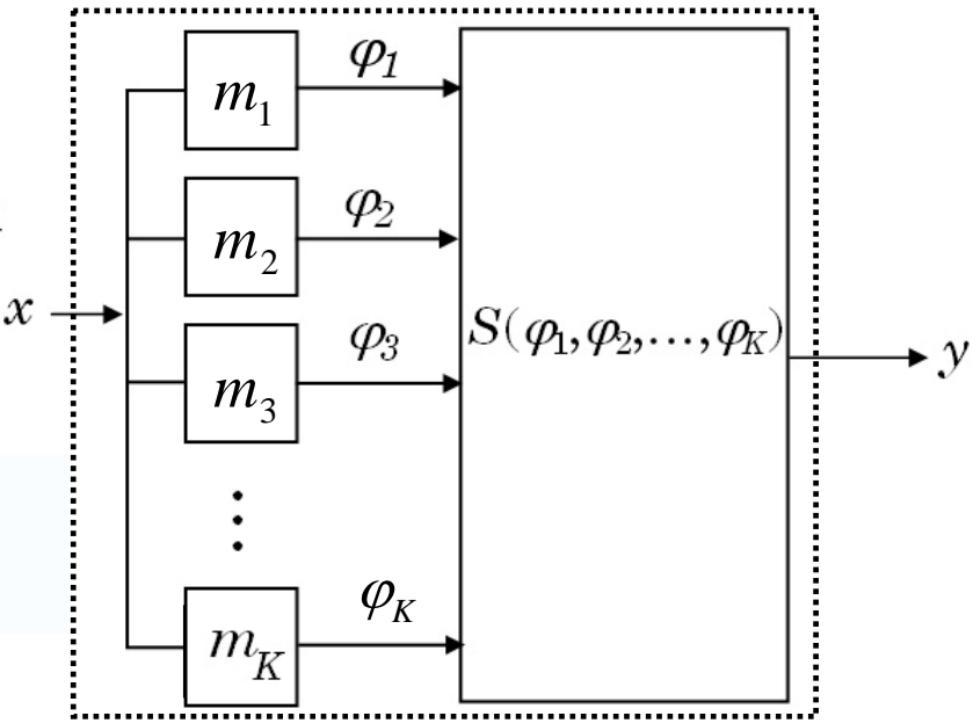
$$m_s(n) = \sum_{r=1}^{M+1} \mu_{s,r} \delta(n-r+1)$$

where $\mu_{s,r}$ is the eigen vector, $\delta(\cdot)$ is the Kronecker delta and λ_s is the eigen value

$$\chi \leq \frac{|\lambda_s|}{\sum_i |\lambda_i|}, \chi \text{ was chosen to be 0.01}$$

$$y_2(n) = \sum_{s=1}^K \lambda_s [m_s(n) * x(n) + \mu_{s,0}]^2 = \sum_{s=1}^K \lambda_s [\varphi_s + \mu_{s,0}]^2$$

Output in terms of PDMs

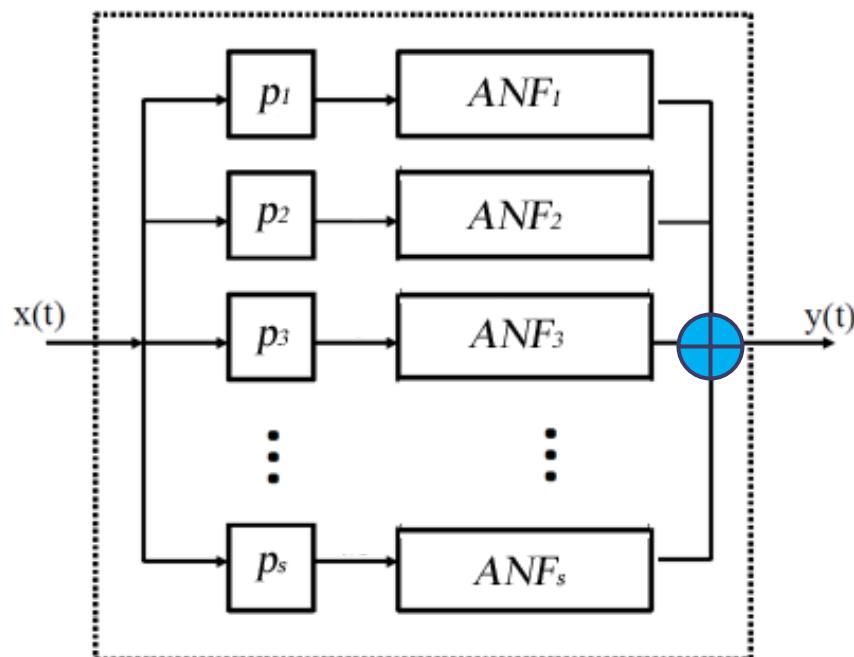


Principal dynamic modes

$$K \leq L$$

Higher Order Models

Parallel LN Cascades



ANF ≡ Associated Nonlinear Function

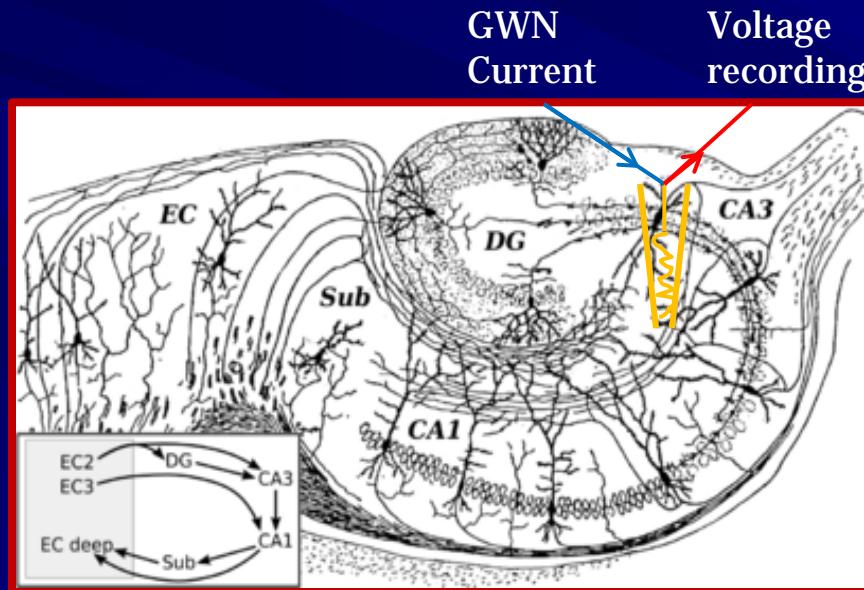
$$y(n) = a_0 + \sum_{m=1}^M \sum_{s=1}^S a_{m,s} (p_s(n) * x(n))^m$$

Both $p_s(n)$ and $m_s(n)$ represent the same PDM.

Estimate the power series coefficients $a(m,s)$ to better fit $y(n)$ to the measured response to GWN.

PDM computed from 2nd order but model order is not limited to 2nd order system. Higher order static nonlinearity can be used to model higher order systems.

Data Acquisition

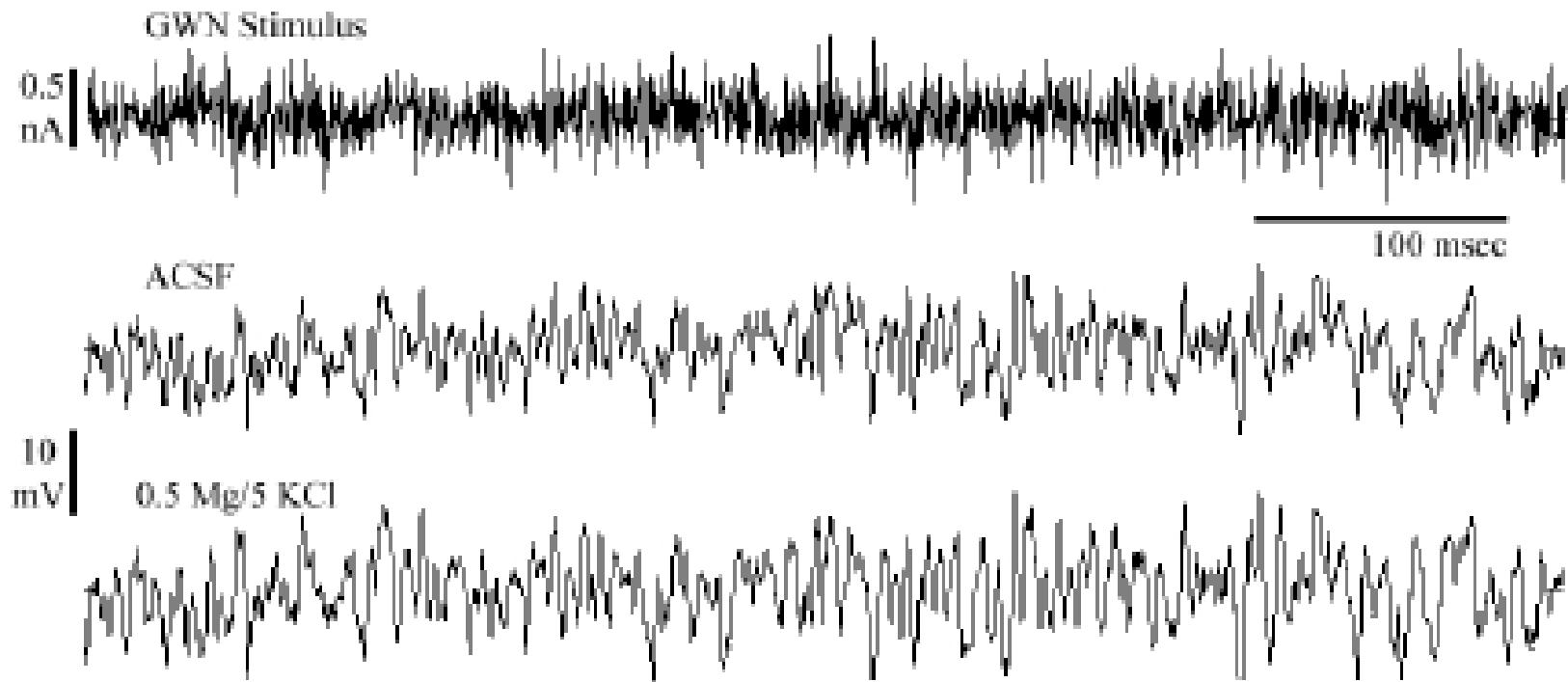


■ Whole cell patch on CA3 stratum pyramidale of mouse whole hippocampus

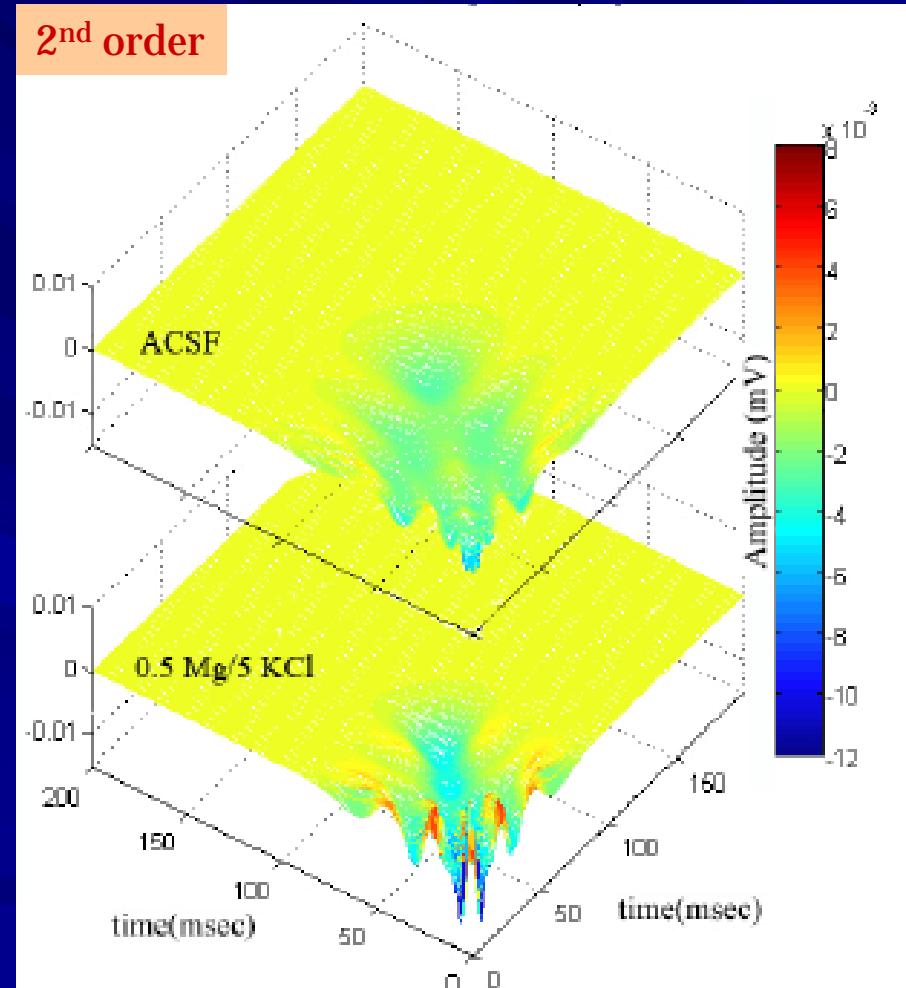
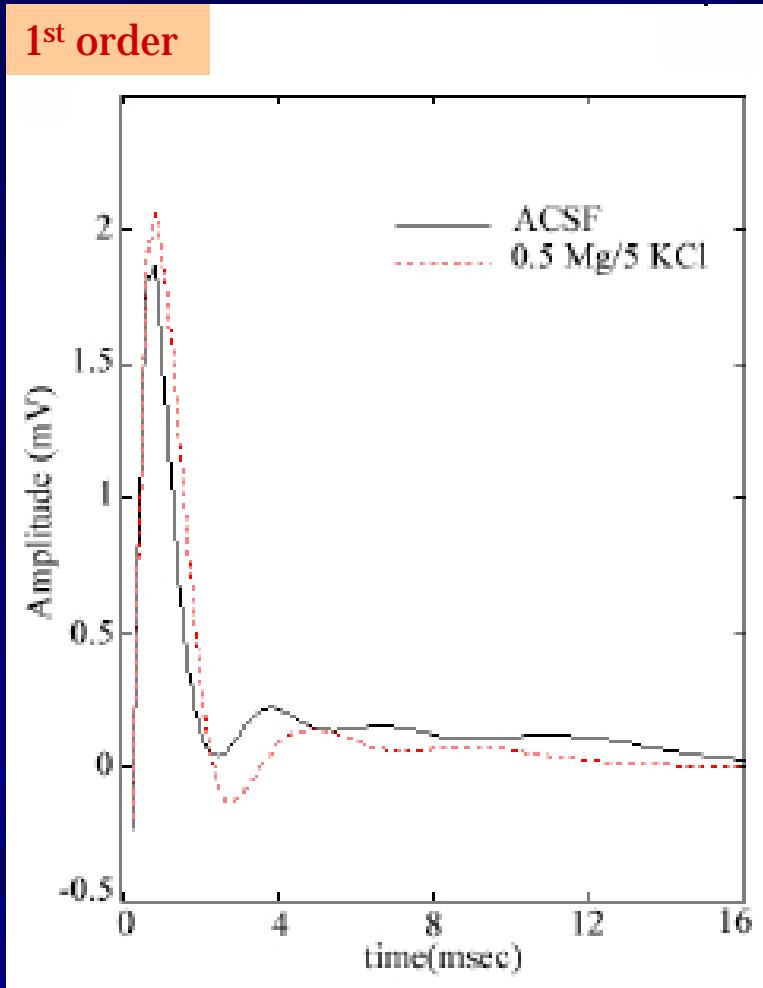
- Recording and stimulation using same patch clamp electrode
- Gaussian white noise (GWN) input current stimulation
- Output voltage recording

■ Epileptogenic condition: Low-Mg²⁺/High-K⁺ perfusion of various concentrations

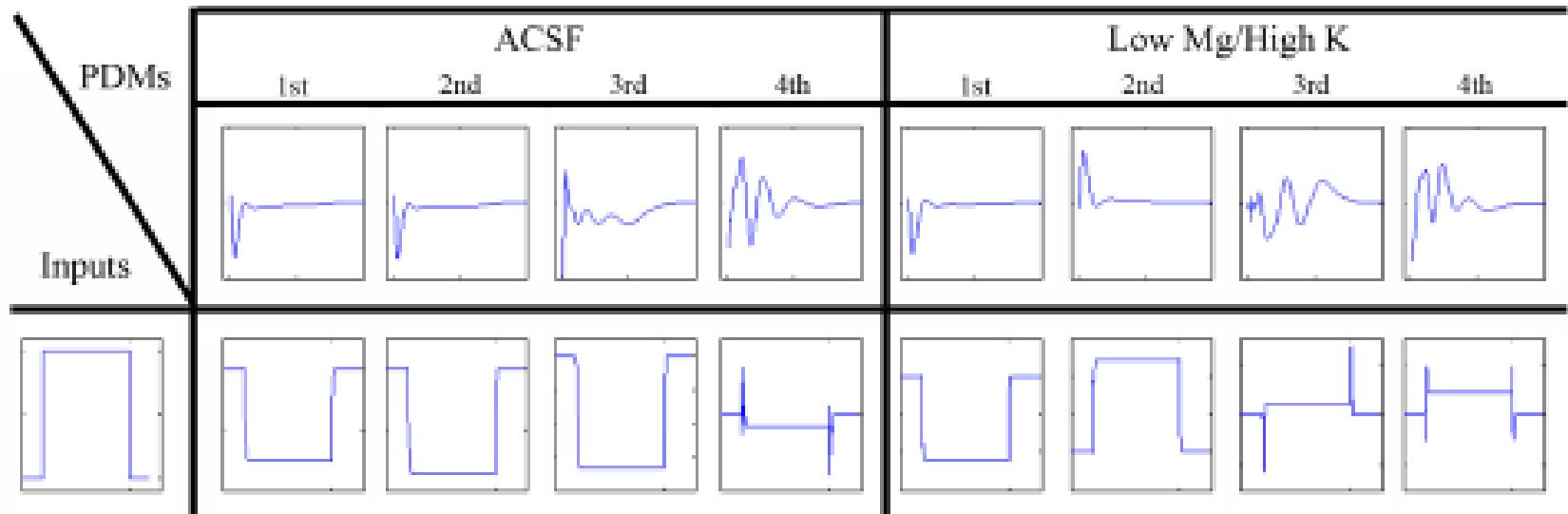
Voltage Response to GWN Current Stimulus



Measured Volterra Kernels



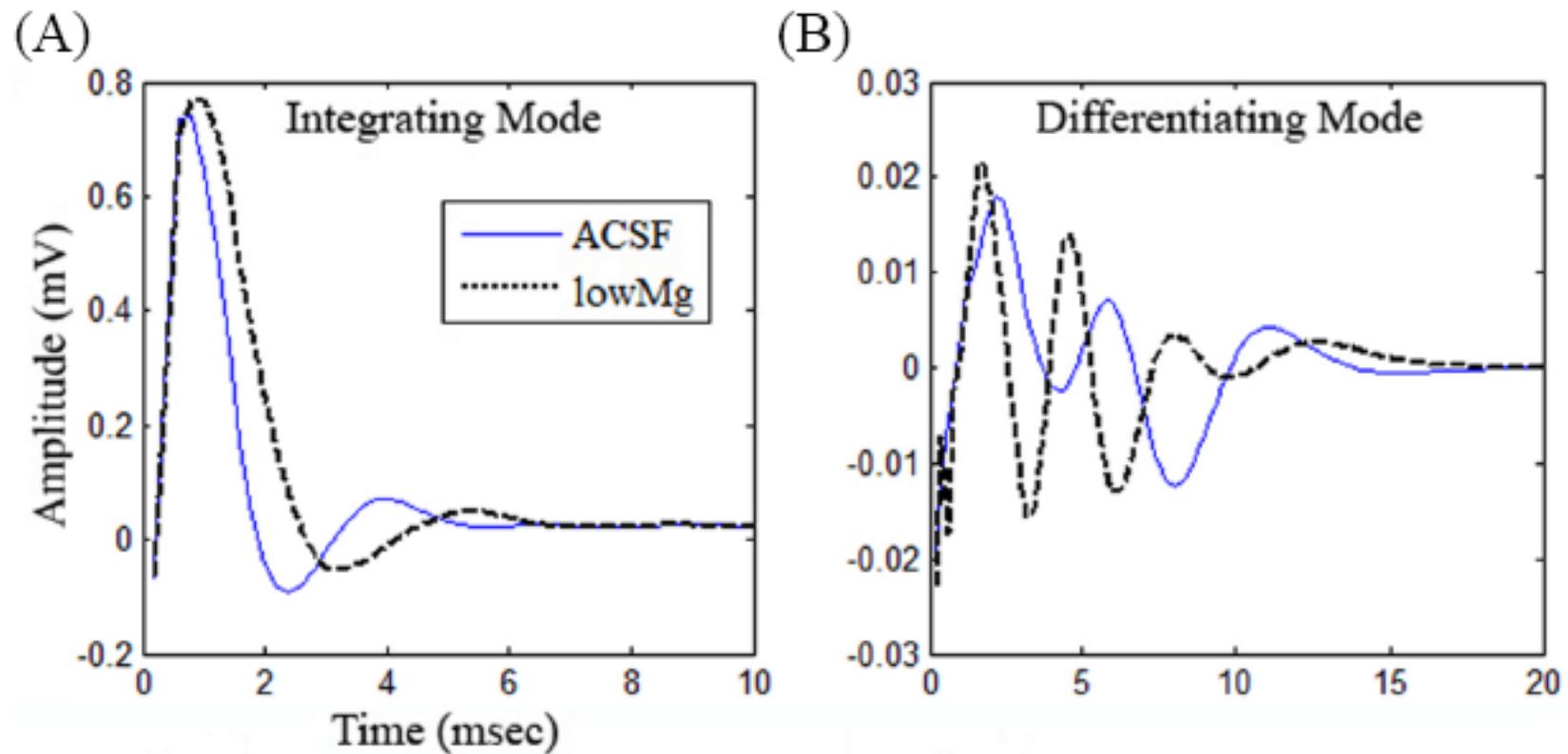
Pre- and post-epileptogenic treatment PDMs and their responses to a step function.



PDMs characterized by convolving with a step function

- **Integrating PDM:** scales with the magnitude of the input (i.e. a step response to a step function)
- **Differentiating PDM:** sensitive to the rate of change of the amplitude (i.e. sharp spikes at the start and stop edges)

Principal Dynamic Modes (PDMs)



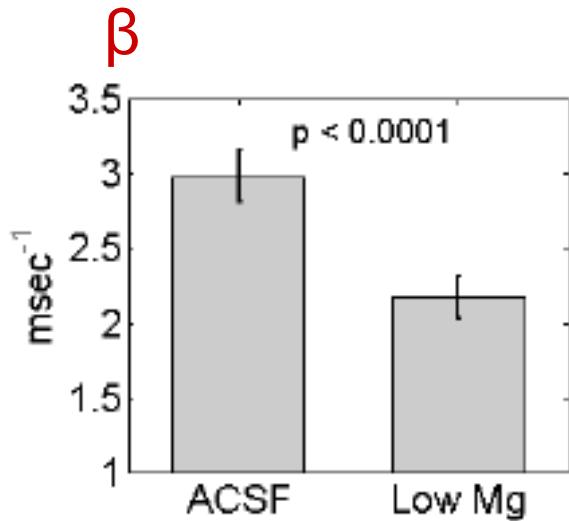
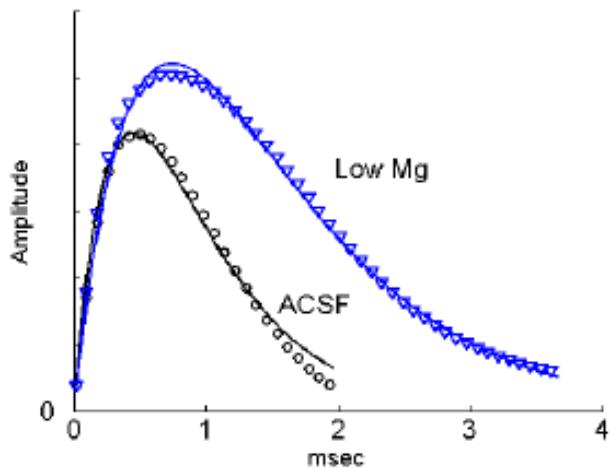
Typical sample traces of integrating and differentiating PDMS under normal ACSF and low-magnesium/high-potassium perfusion.

A functional parameter β representing decay time of integrating PDM

The value of β is determined by nonlinear least-squares fitting of the ACSF and low Mg data to the function

$$m_1(t) = H \beta t e^{(-\beta t)}$$

where H is fitted to the height.



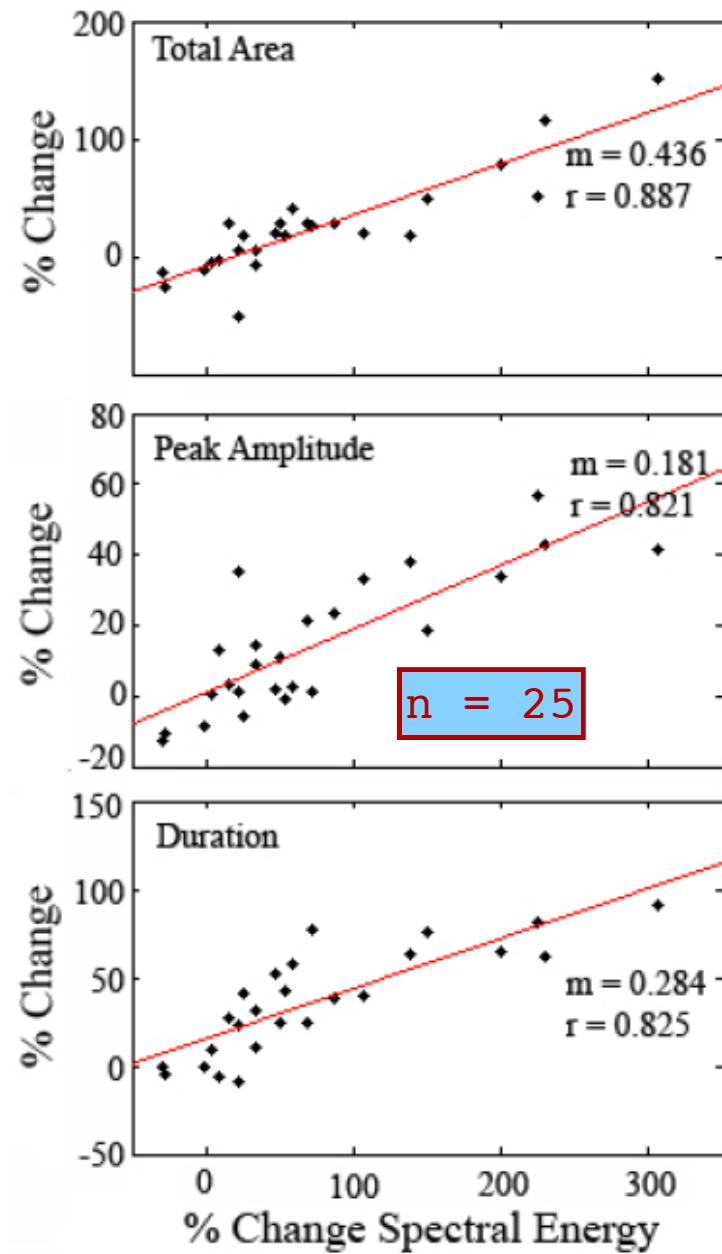
n = 25

Integrating PDMs and Neuronal Excitability

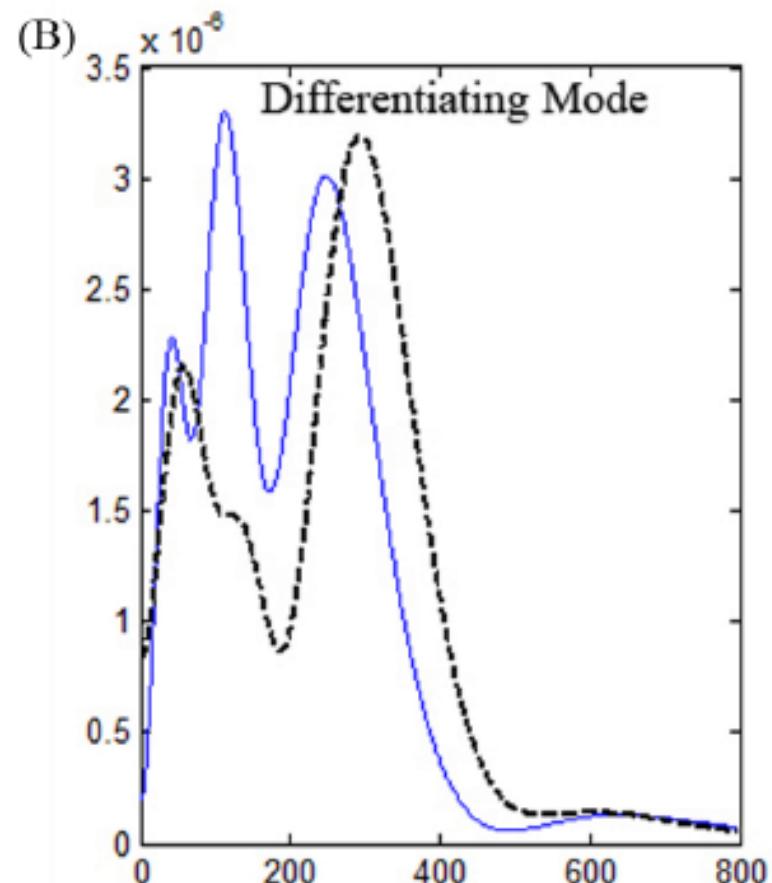
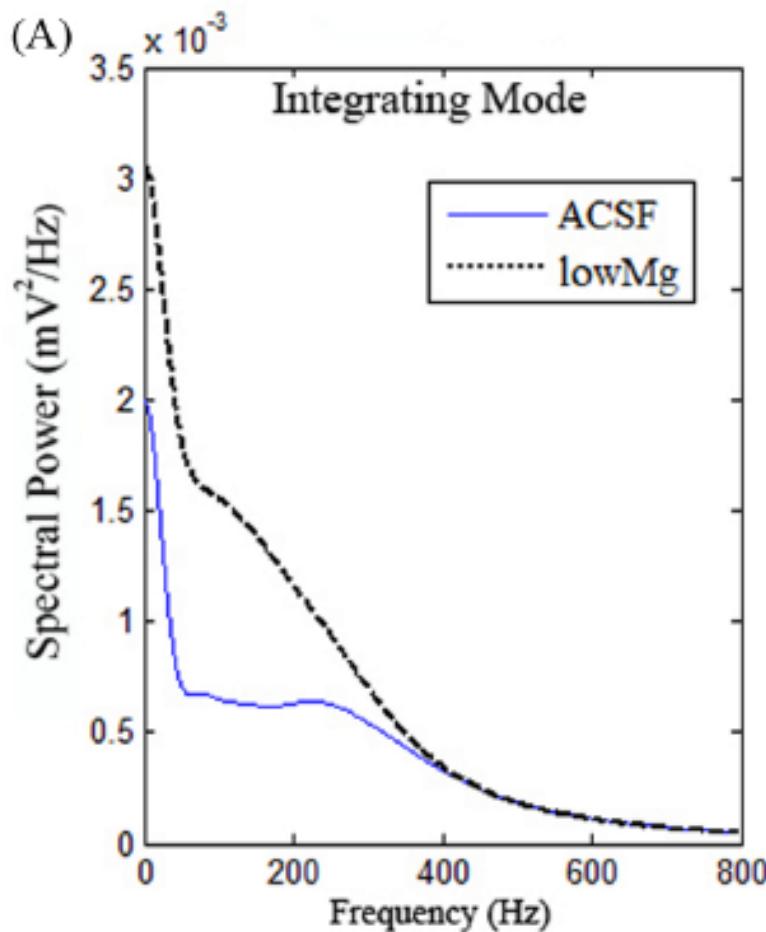
Percent change of features of integrative PDMs relative to control versus the percent change of the spectral energy relative to control.

m is the slope of the linear fit and r is the Pearson correlation coefficient.

Changes of the integrating PDM features showed strong positive correlation with the change of the level of excitation (spectral energy).



Average Power Spectrum of PDMs



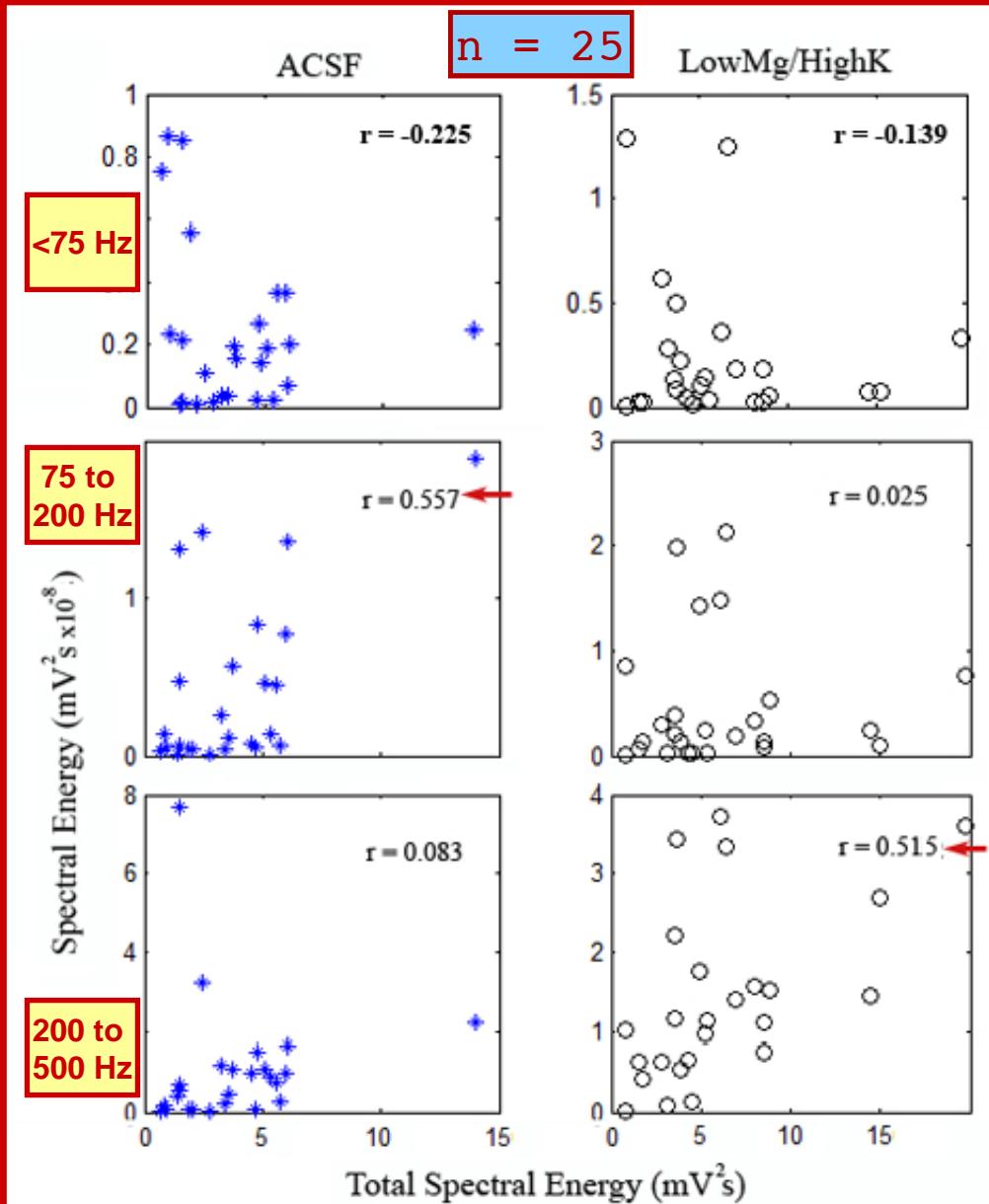
n = 25

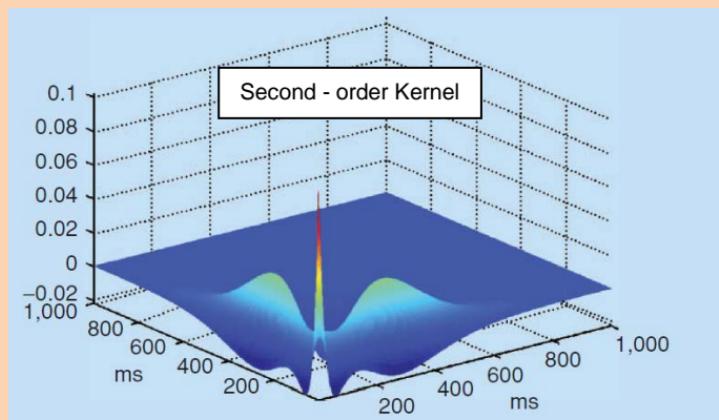
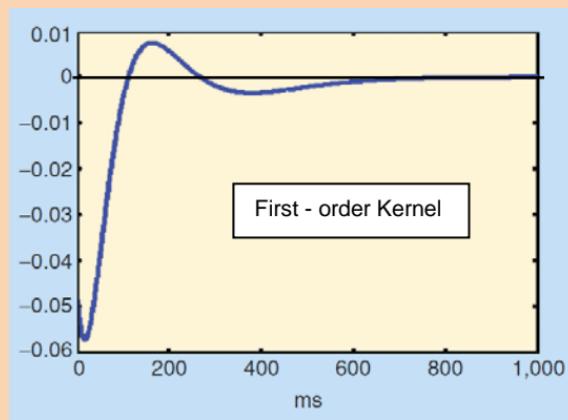
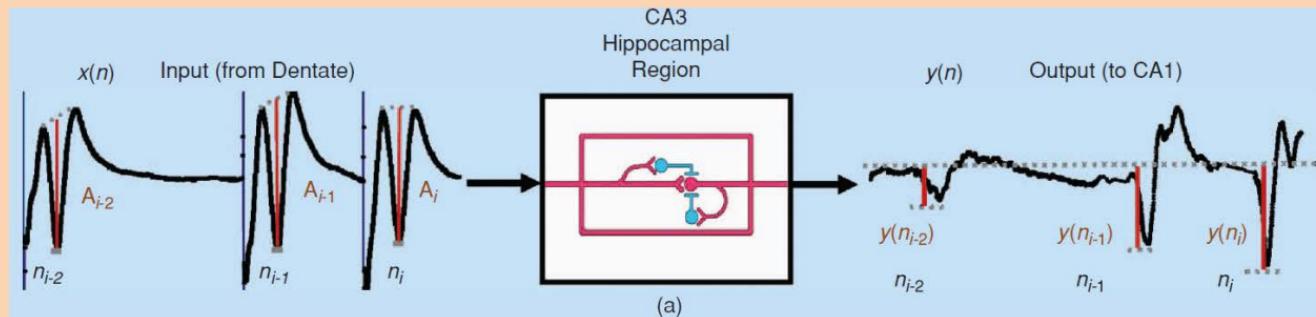
Differentiating PDMs and Neuronal Excitability

Percent Area under the curve of the various frequency components of the differentiating PDMs.

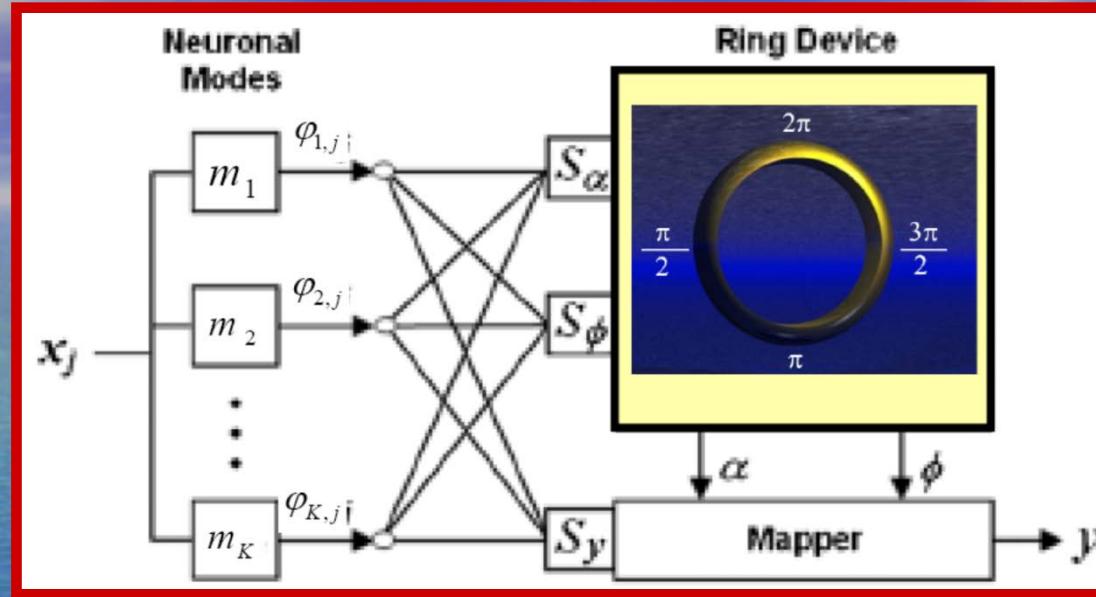
The red arrows indicate frequency ranges with the largest Pearson correlation coefficient “r”.

Under epileptogenic conditions, differentiating PDMs shifted their dominant frequencies from ripple activities (75 - 200 Hz) to fast ripple activities (200 - 500 Hz).



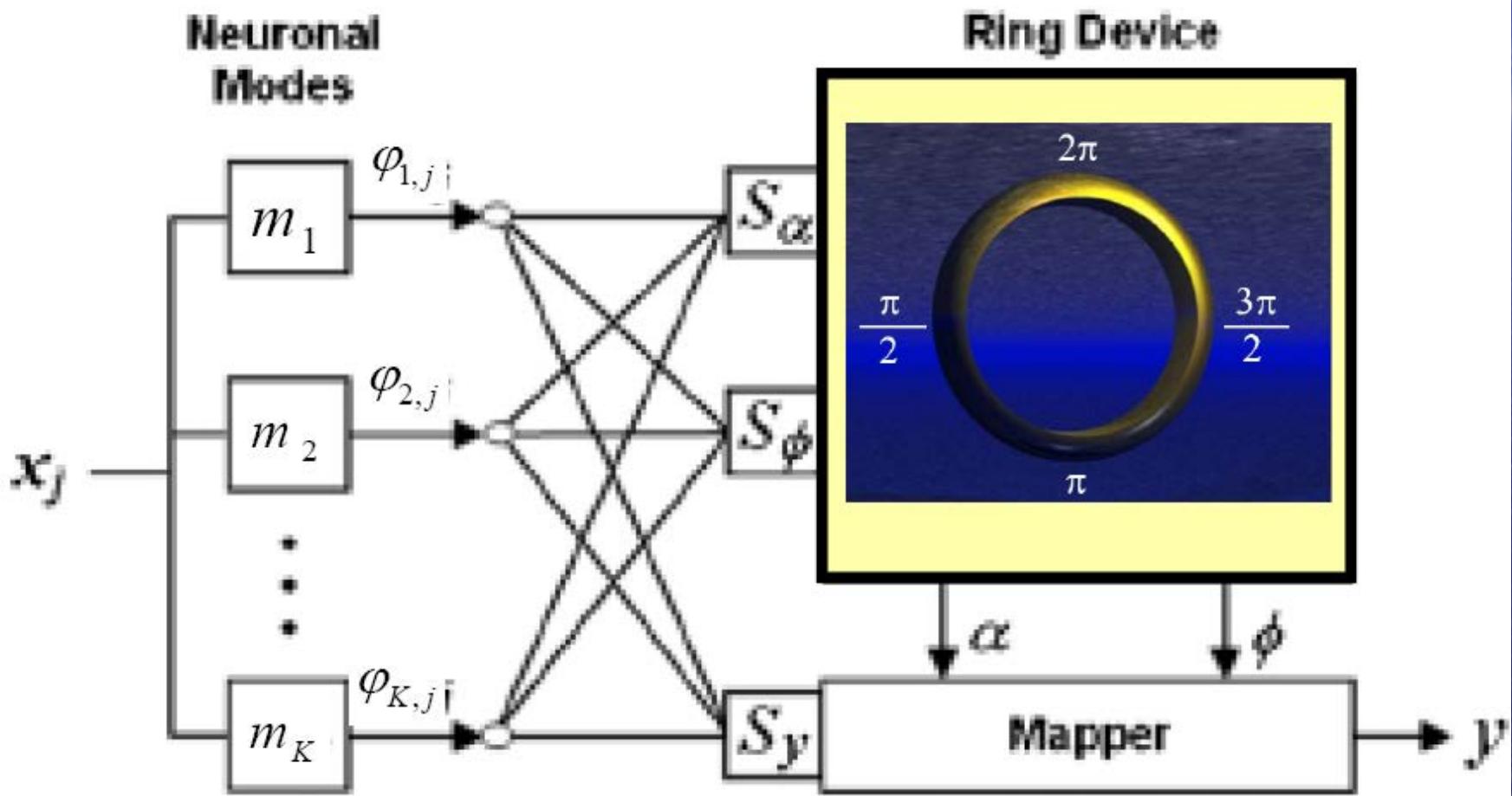


Cognitive Rhythm Generators (CRGs)



- The **cognitive aspect** of the **CRG** derives from input decoding performed by an orthogonal set of neuronal modes representing the system kernels. **Cognition** involves both **perception** and **information processing**, so the modes provide the medium through which the **CRG** perceives its environment and performs an initial transformation of the incoming signals.
- The rhythm generator takes the mode-transformed signals and encodes them into amplitude and phase variables that are then mapped to an observable output by a static nonlinearity.

Cognitive Rhythm Generator (CRG) Model



Proposed as an archetype for **neural coding** that is based on **Winfree's ring devices**. It is a general representation of neuronal assemblies and their functionality.

$$m_k(t) = a_k t \exp(1 - a_k t) \{ \cos((k-1)a_k t) + b_k \}$$

$$\varphi_{k,j}(t) = \int_0^\infty m_k(\tau) \cdot x_j(t-\tau) d\tau$$

$$S_u = \varepsilon_u \sum_k \sum_j \mu_{k,j} \varphi_{k,j}$$

for $u = \{\alpha, \phi, y\}$

Modular Volterra

clock

$$\frac{d\alpha}{dt} = k_\alpha \alpha (1 + R_\alpha(\phi) S_\alpha - \alpha^2),$$

labile clock

$$\frac{d\alpha}{dt} = k_\alpha \alpha^{1/3} (R_\alpha(\phi) V(S_\alpha) - \alpha),$$

$$\frac{d\phi}{dt} = \omega (1 + R_\phi(\phi) S_\phi)$$

$R_\alpha(\cdot)$ is a refractory function

$R_\phi(\cdot)$ is a refractory function

$V(\cdot)$ is a threshold function controlling activation
of the labile clock

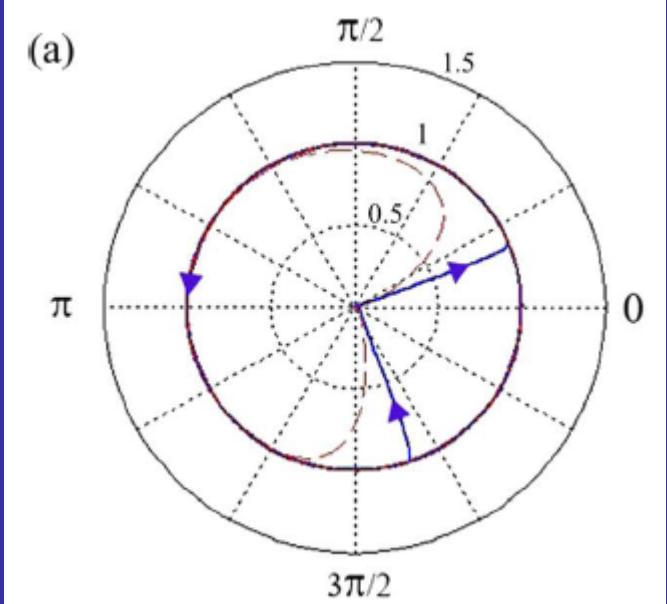
The implicit ring device functions $R_\phi(\cdot)$, $R_\alpha(\cdot)$ and $V(\cdot)$ are all sigmoids of the form

$$s(u) = \frac{1}{1 + \exp(-c_1(u - c_2))}$$

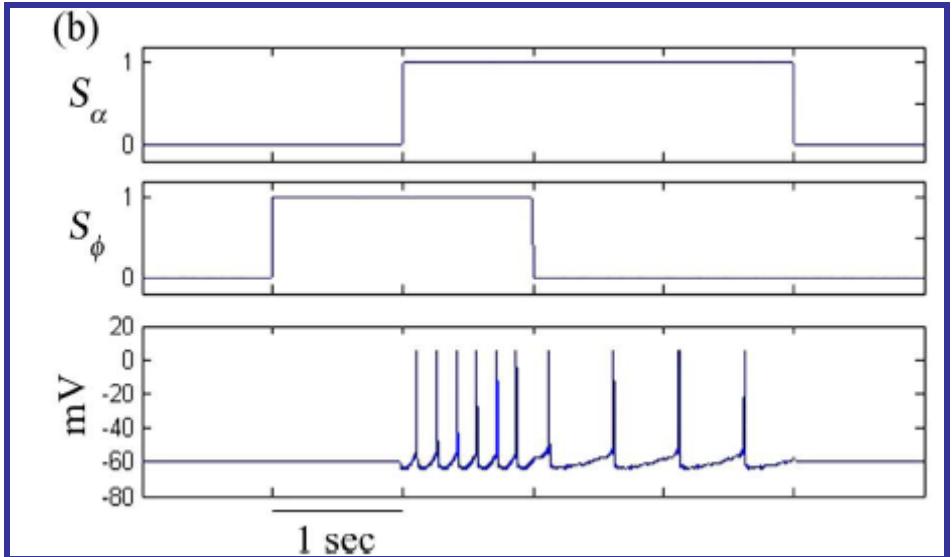
$$y = S_y + \alpha W(\phi)$$

Ring Device

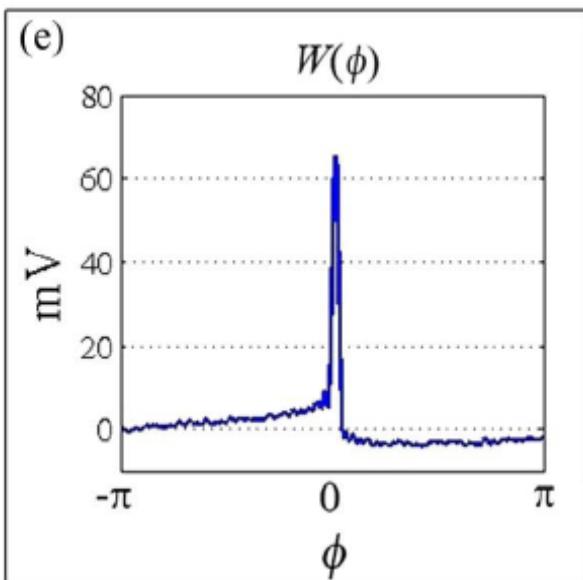
(a) Labile clock ring dynamics: a suprathreshold alpha stimulus (red segmented line) forces the state trajectory (blue solid line) away from the origin and toward its active limit cycle (the threshold is 0.5). The radius of the limit cycle is not determined by the stimulus magnitude but by the threshold function. When the stimulus is removed, the state dot returns to its orbit near the origin.



(b) Stimuli S_{α} and S_{ϕ} are administered in staggered fashion. The frequency-modifying effect of the phase stimulus is not registered unless the labile clock is in its active state, which is brought about by the suprathreshold amplitude step.

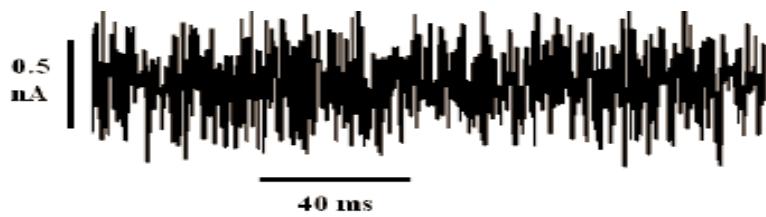


(e) The intrinsic waveform of the static nonlinearity is the recorded biological source waveform normalized in phase over one cycle.

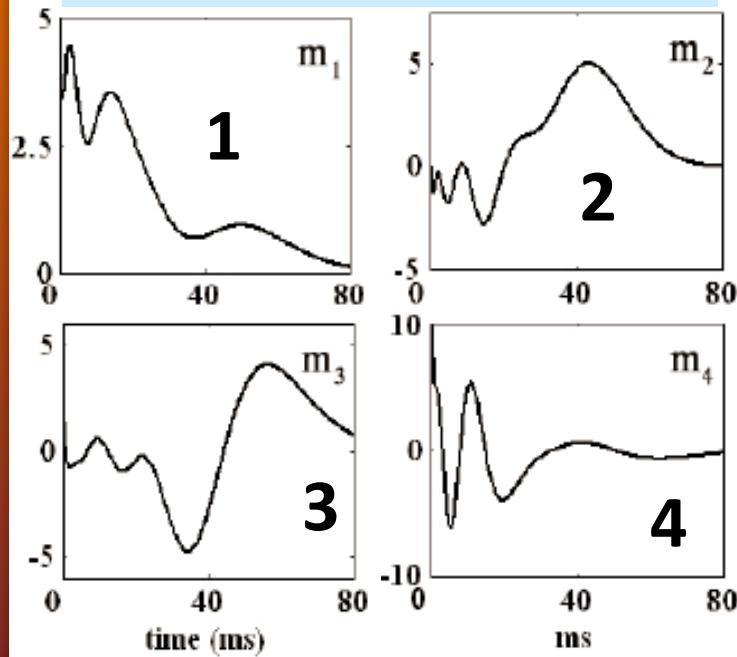


Neural Coding with CRGs

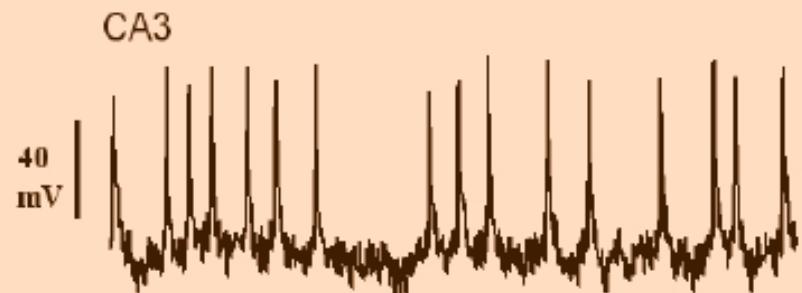
Bandlimited Gaussian white noise stimulus



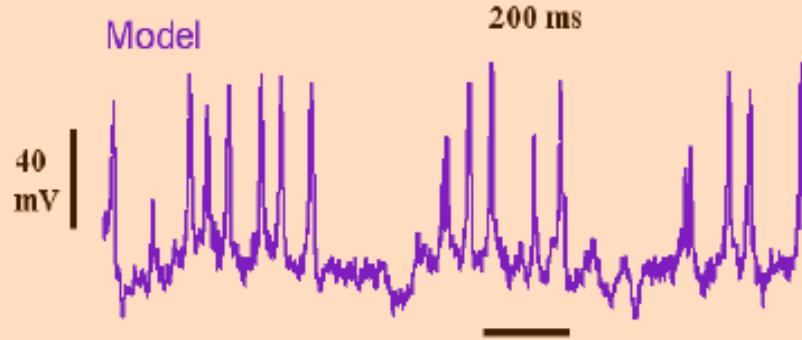
Measured Neuronal Modes (in order of decreasing dominance):

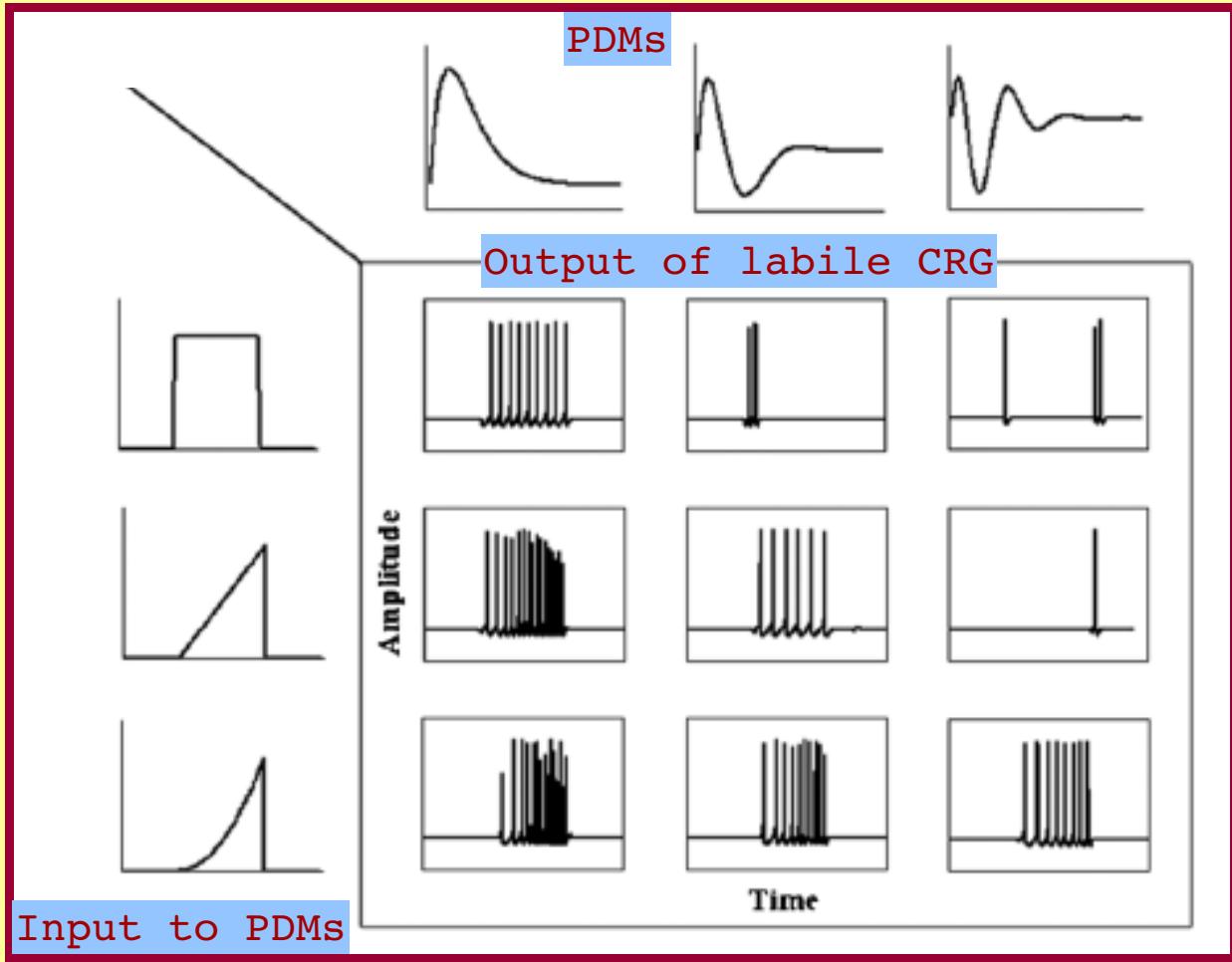


CA3
pyramidal
neuron
response



Simulated
CRG
response

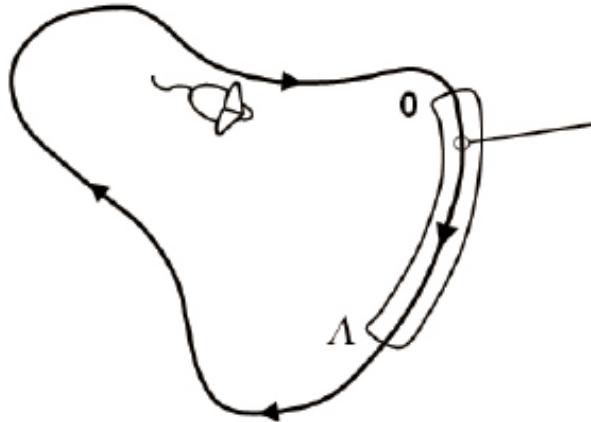




Rate encoding in a labile CRG. The response matrix shows the rate-varying spike output from the mapper as it corresponds to the input-mode pairings

Theta-Gamma Coding with CRGs

Theta Phase Precession

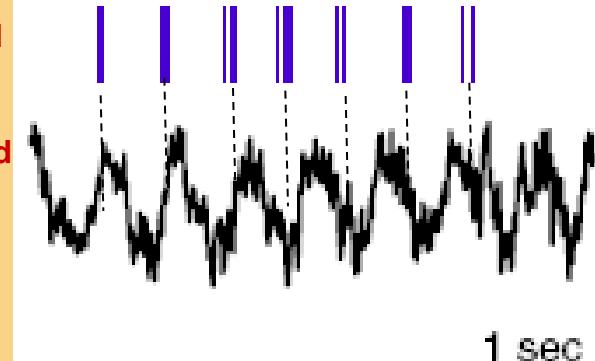


Place
Field

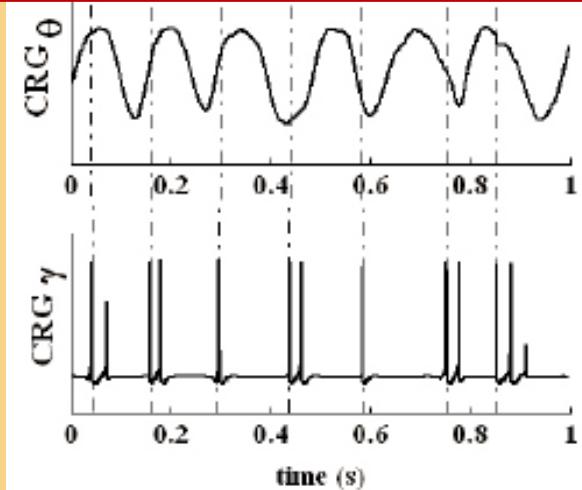
Fast (gamma) spikes of place cell **precess** with respect to slow (theta) population rhythm in navigating rodent

Rat (Geisler et al., 2010)

Place Cell
Theta field potential



CRG (Zalay and Bardakjian, 2009)

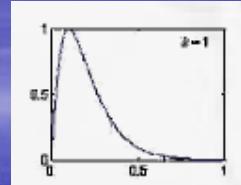
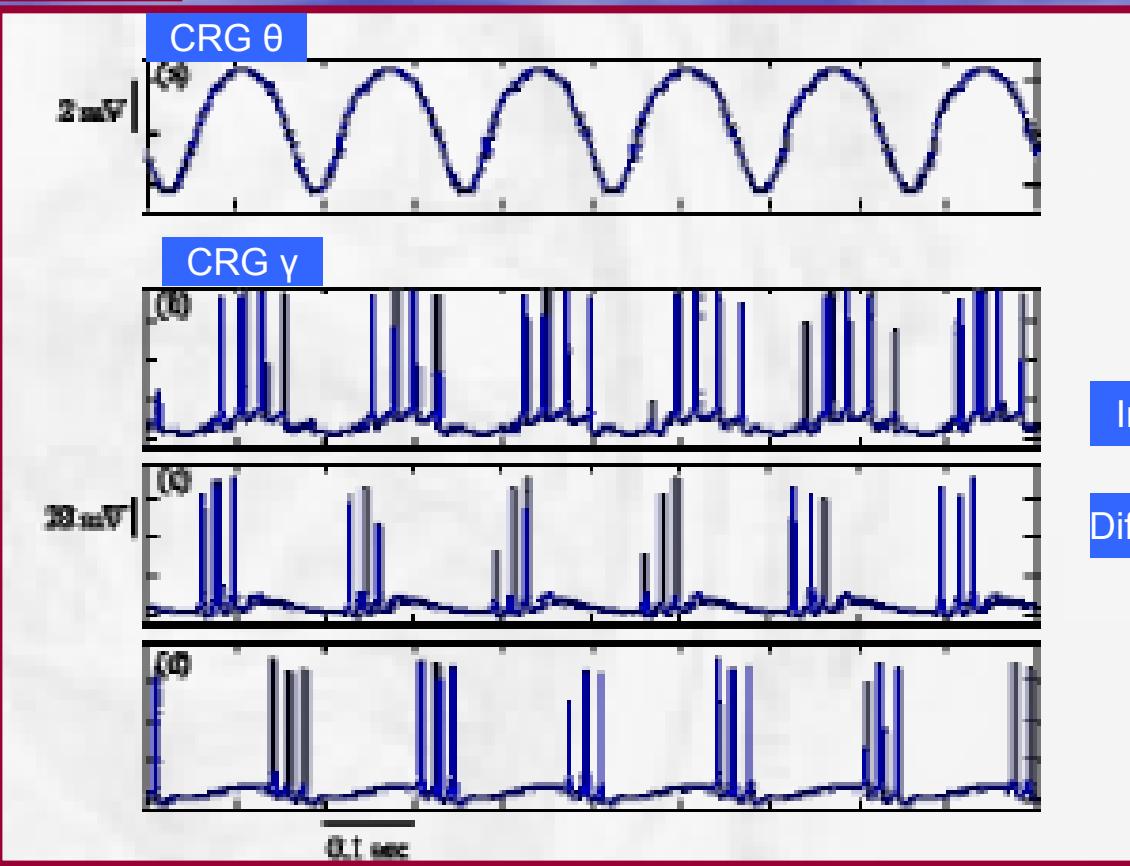
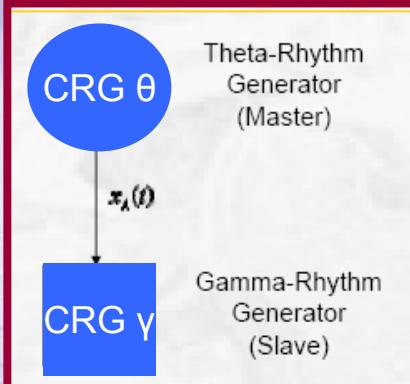


CRG 1
(θ)

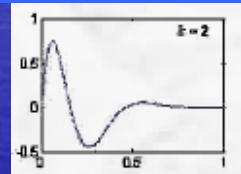


CRG 2
(γ)

Theta-Gamma Code



Integrating PDM



Differentiating PDM

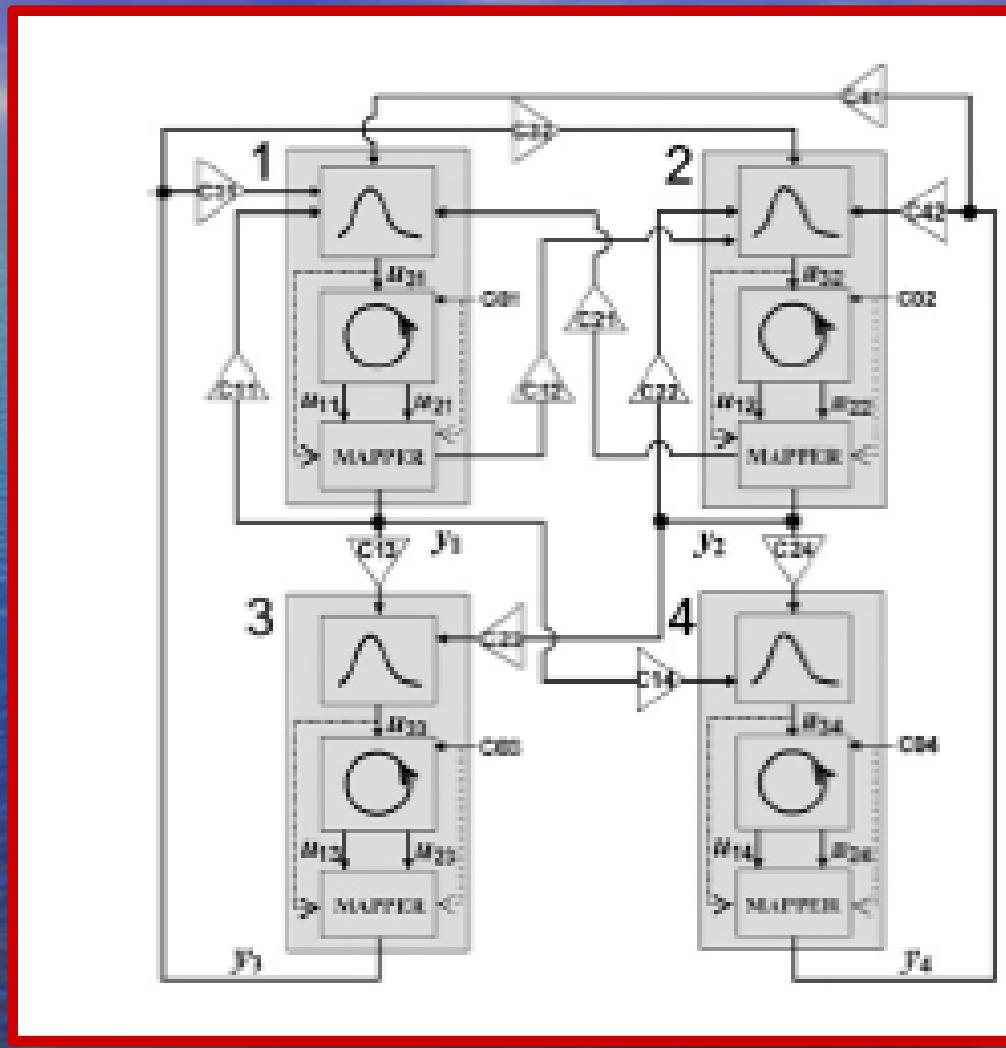
- CRG θ generates a theta oscillation autonomously that controls the gamma-rhythm activity produced by CRG γ . The phase of the gamma-activity in relation to the theta-rhythm (e.g. rising phase, crest, descending phase) depends on the dominant neuronal mode:
- Gain = 0.1 for integrating PDM.
- Gain = 1 for 1st order differentiating PDM.
- Gain = -1 for 1st order differentiating PDM.

System characterization of neuronal excitability in the hippocampus and its relevance to observed dynamics of spontaneous seizure-like transitions

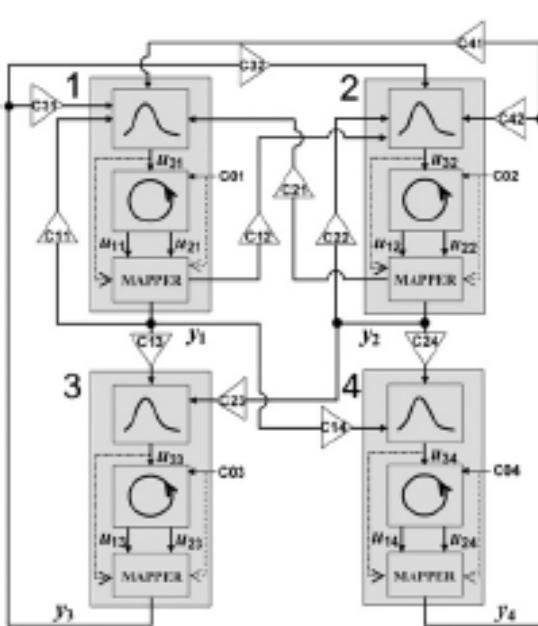
Osbert C Zalay¹, Demitre Serletis^{1,2,3}, Peter L Carlen^{1,2,3} and
Berj L Bardakjian^{1,4}



Model of spontaneous SLEs using 4 Coupled CRGs



Cognitive Rhythm Generator (CRG) Model of SLEs



Model Parameters

c_{mn} = coupling parameters

k_n = modulatory gain

β_n = decay constant of integrating mode

Cartesian form of Ring Devices

$$\dot{u}_{1n} = \omega_n \{ u_{2n}(1 + S_{\phi,n}) + u_{1n}(1 + S_{\alpha,n} - u_{1n}^2 - u_{2n}^2) \}$$

$$\dot{u}_{2n} = \omega_n \{ -u_{1n}(1 + S_{\phi,n}) + u_{2n}(1 + S_{\alpha,n} - u_{1n}^2 - u_{2n}^2) \}$$

$$\dot{u}_{3n} = u_{4n}$$

$$\dot{u}_{4n} = \beta_n F_n - 2\beta_n u_{4n} - \beta_n^2 u_{3n}$$

Outputs of
Integration PDMs

$$F_n = \sum_{m=1}^M c_{mn} y_m + x_n(t)$$

Input to
Integration PDMs

$$y_n = c_{0n} + u_{3n} + \sqrt{u_{1n}^2 + u_{2n}^2} W \left(\arctan \frac{u_{2n}}{u_{1n}} \right)$$

Outputs
of CRGs

$$S_{\phi,n} = c_{0n} + k_n u_{3n}$$

$$S_{\alpha,n} = 0$$

$$u_1 = \propto \sin \phi$$

$$u_2 = \propto \cos \phi$$

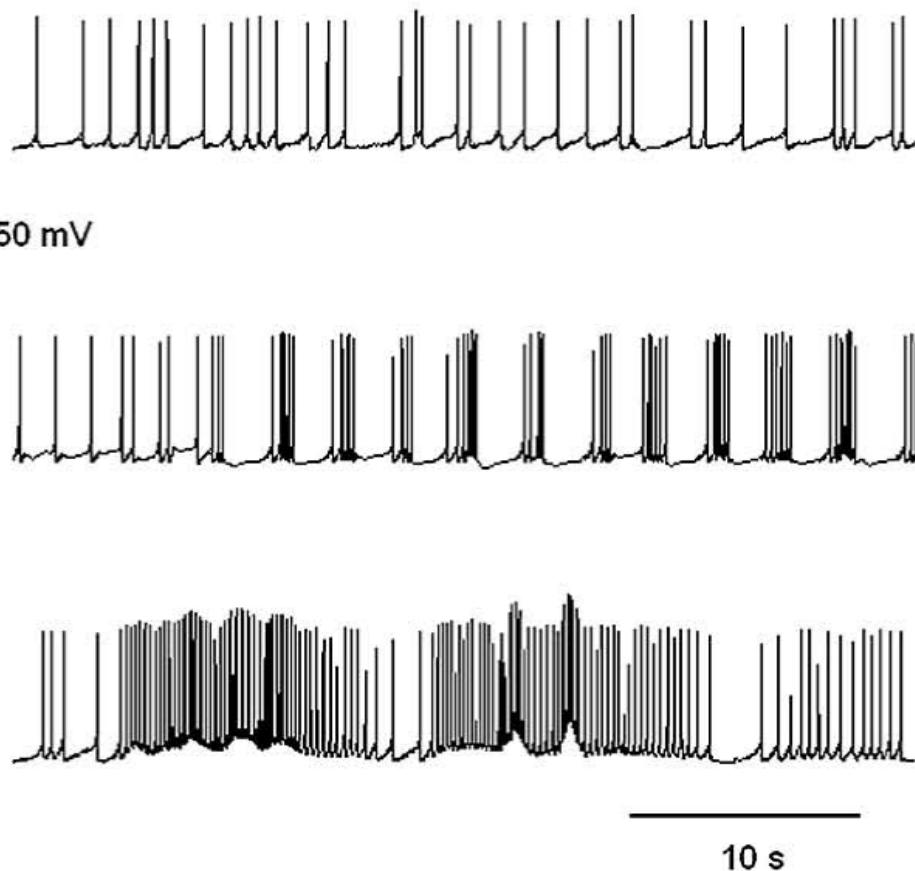
$$\text{neuronal mode} \equiv m_1(t) = \beta t e^{-\beta t} \Rightarrow$$

$$\begin{cases} \dot{u}_{3n} \\ \dot{u}_{4n} \end{cases}$$

Integration
PDMs

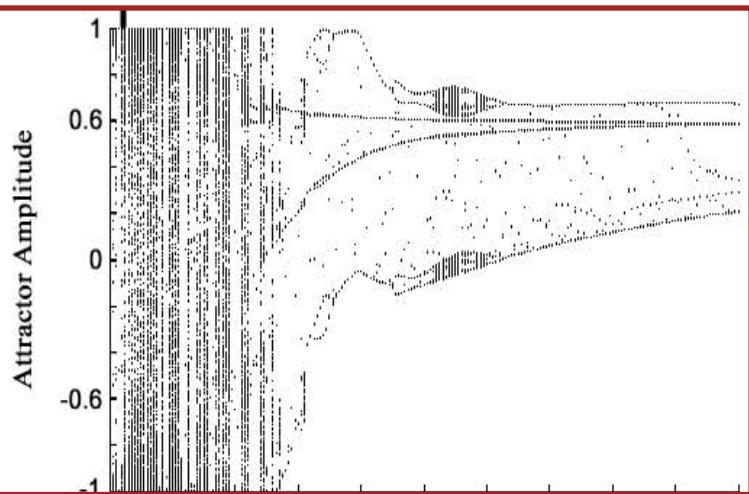
CRG Model of Spontaneous SLEs

Decreasing Mode Decay Rate (*increasing excitability*)

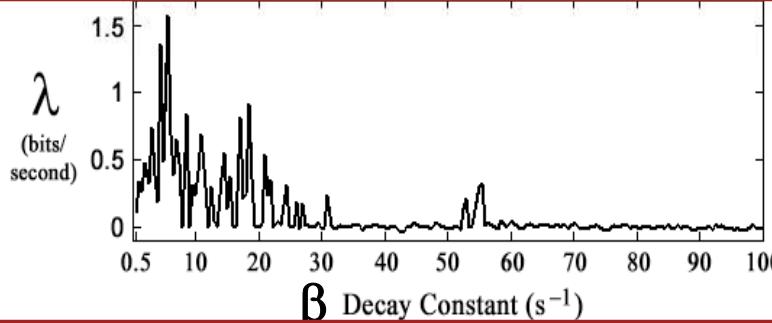


β as a Bifurcation Parameter

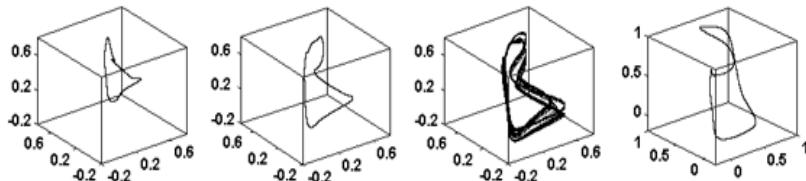
(a)



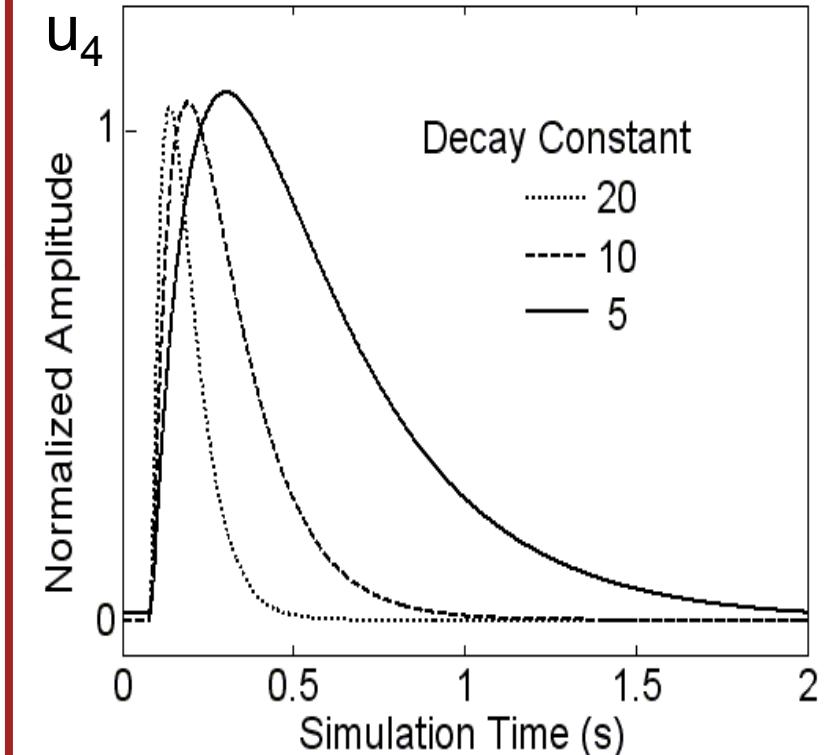
(b)



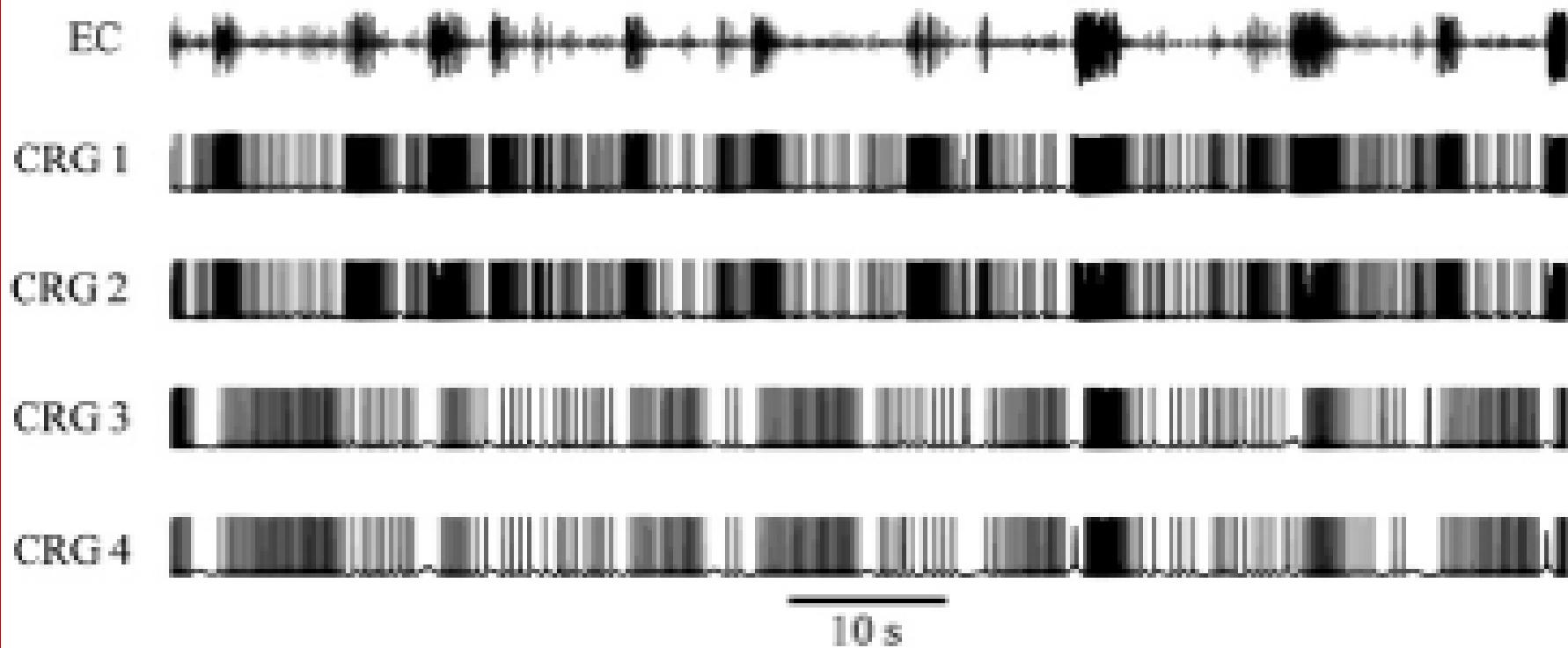
(c)

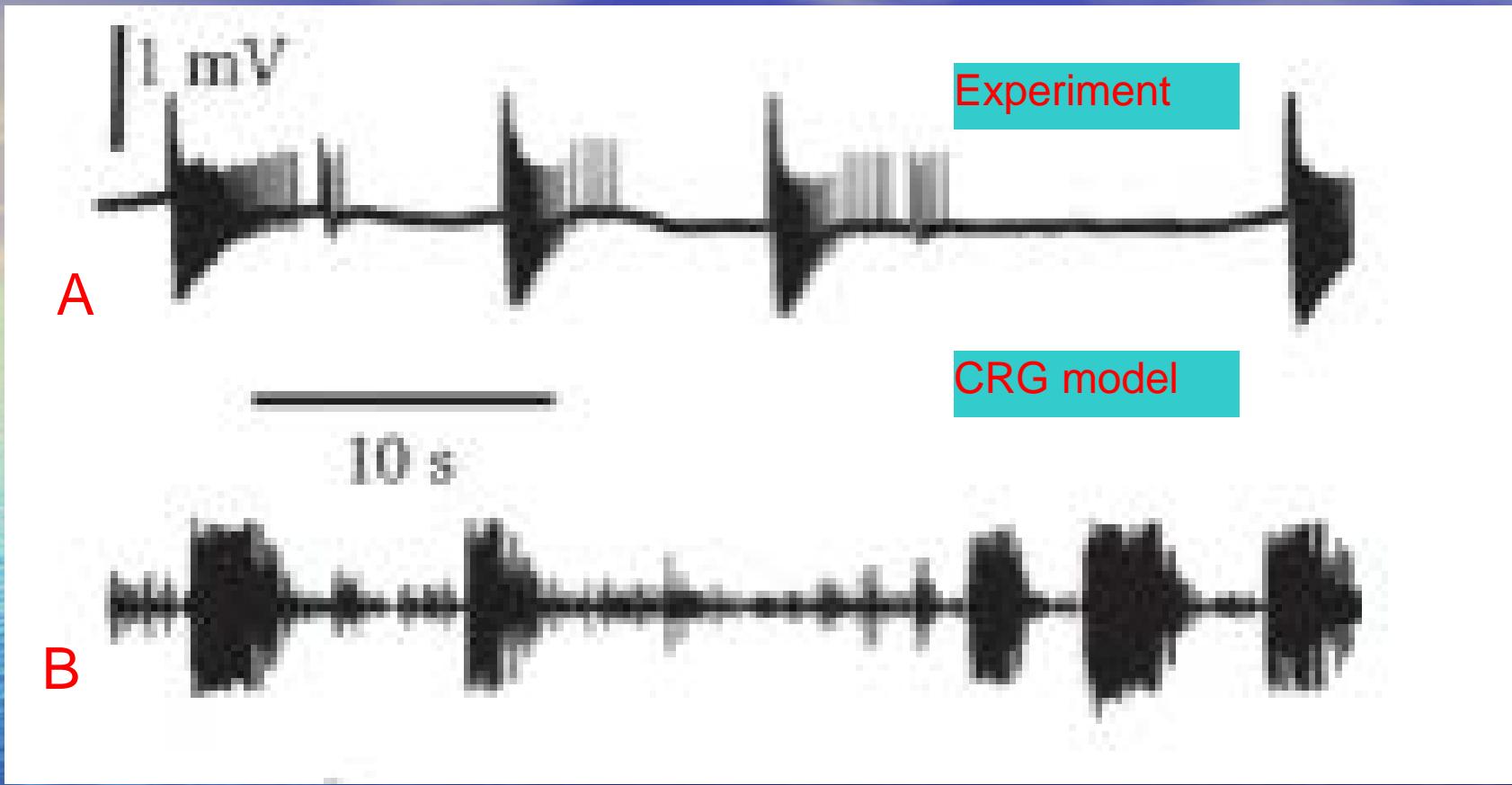


Decreasing Decay Constant, β



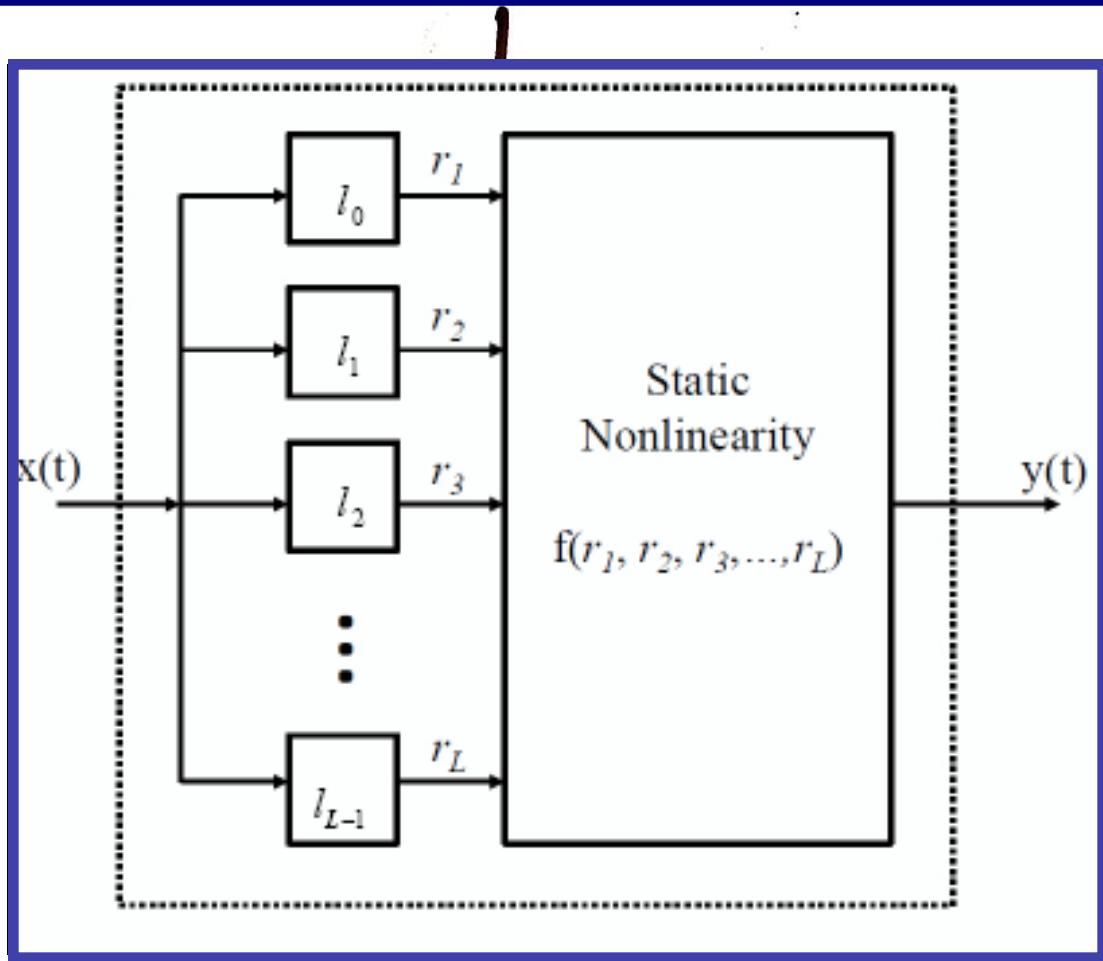
$$\beta = 2$$





Extracellular field recording showing recurrent SLEs from (A) *stratum pyramidale* in a rat hippocampal slice, and (B) CRG model. The experimental trace was de-trended.

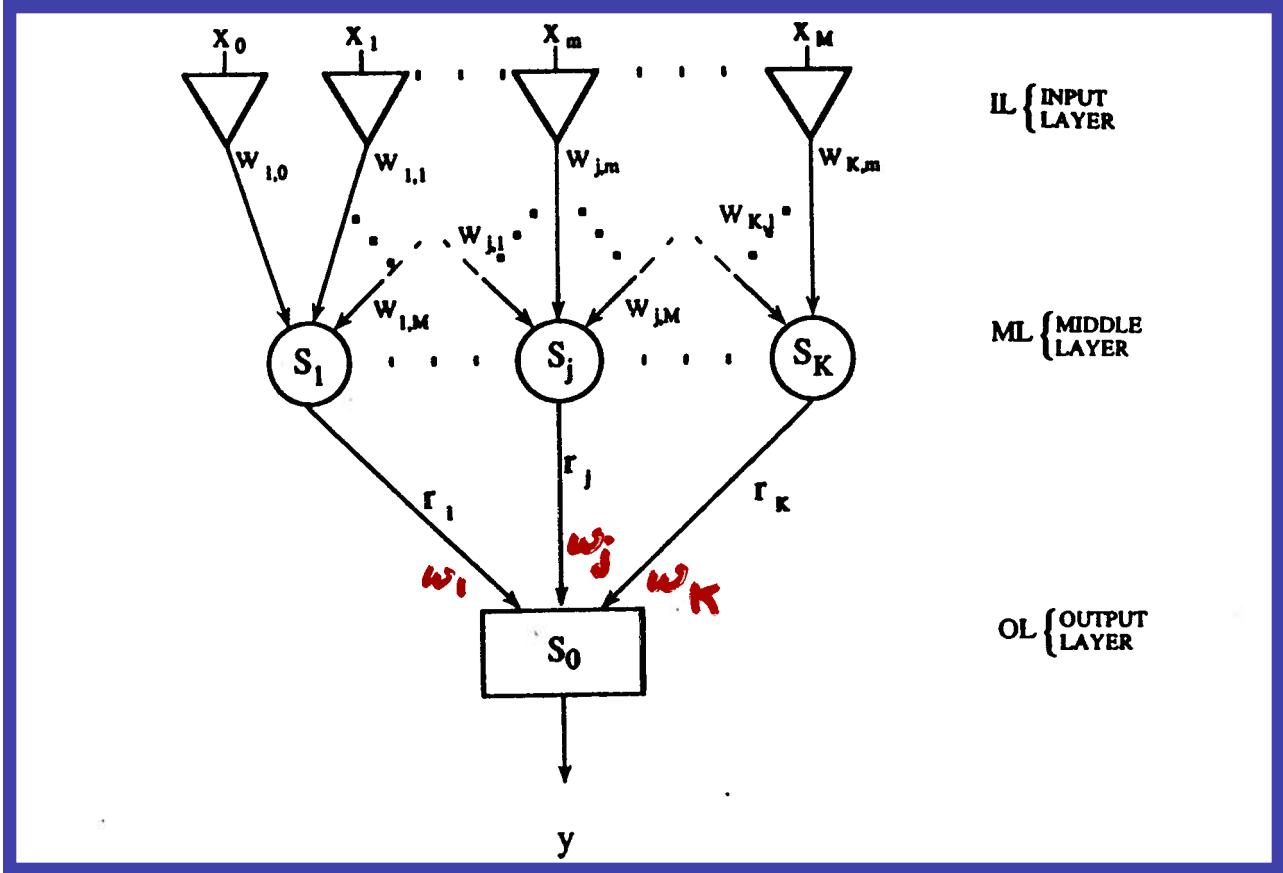
Wiener-Bose Model



Modular
Volterra
Models

Connectionist
Volterra
Models

Connectionist Volterra Models



- Each unit of the middle and output layer performs a nonlinear transform of a weighted sum of respective inputs.

$$r_j(n) = S_j \left(\sum_{m=0}^M w_{j,m} x(n-m) \right)$$

$$y(n) = S_0 \left(\sum_{j=1}^K w_j r_j(n) \right)$$

$$y(n) = S_0 \left[\sum_{j=1}^K w_j S_j \underbrace{\left\{ \sum_{m=0}^M w_{j,m} x(n-m) \right\}}_{u_j} \right]$$

* For a Volterra Series

$$y(n) = \sum_{i=1}^{\infty} \sum_{m_1, \dots, m_i} [k_i(m_1, \dots, m_i)$$

$$x(n-m_1) \dots x(n-m_i)]$$

* Both expansions represent
input-output maps of the
same inputs and outputs.

Kernel Estimation via ANN Training

Let each activation function be represented by a Taylor series expansion

$$S_j(u) = \alpha_{0,j} + \alpha_{1,j} u_j + \dots + \alpha_{i,j} u_j^i + \dots$$

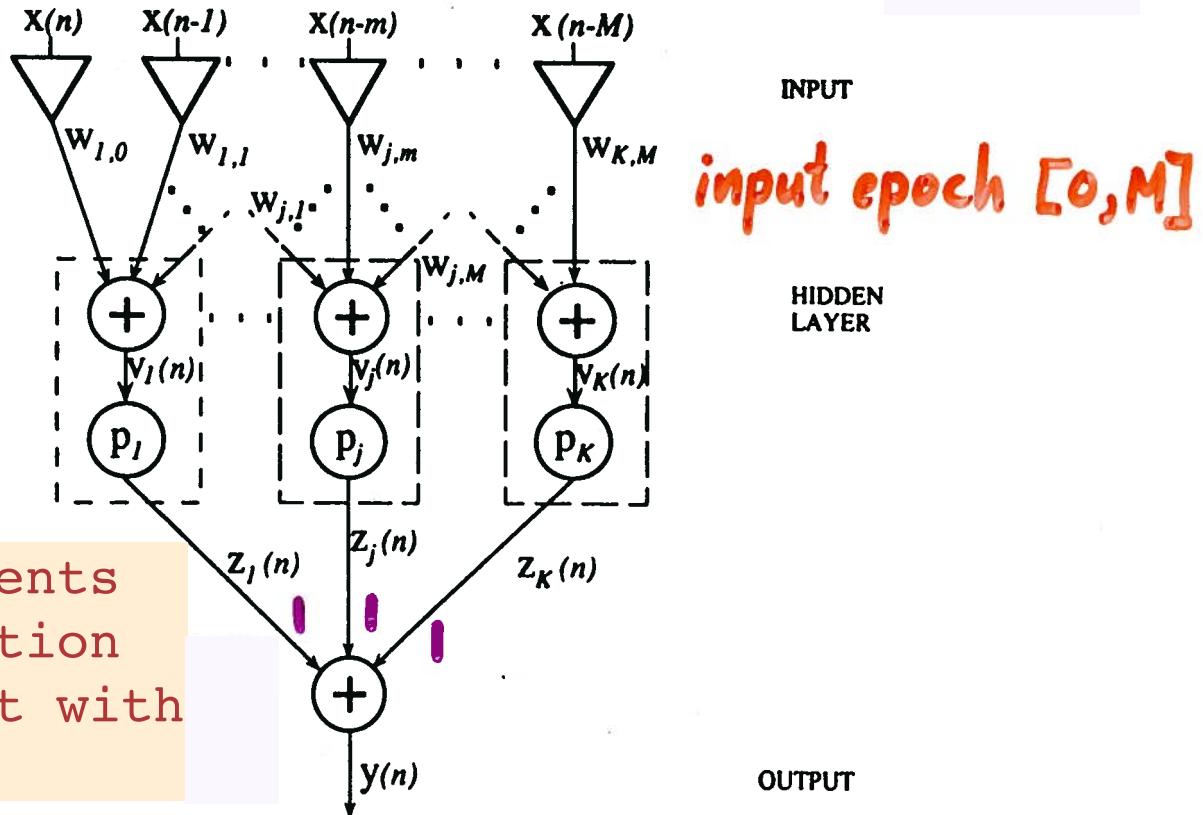
Hence, equating this expansion of $y(n)$ with the Volterra Series Expansion of $y(n)$ leads to the general expression for the Volterra Kernels of the ANN

$$k_i(m_1, \dots, m_i) = \sum_{j=1}^K \alpha_{i,j} w_j W_{j,m_1} \dots W_{j,m_i}$$

Thus, the Volterra Kernels of any order can be estimated from the sets of weights $\{w_j\}$ and $\{W_{j,m}\}$, as well as the coefficients $\{\alpha_{i,j}\}$ of the activation fnz. These are obtained by training the ANN.

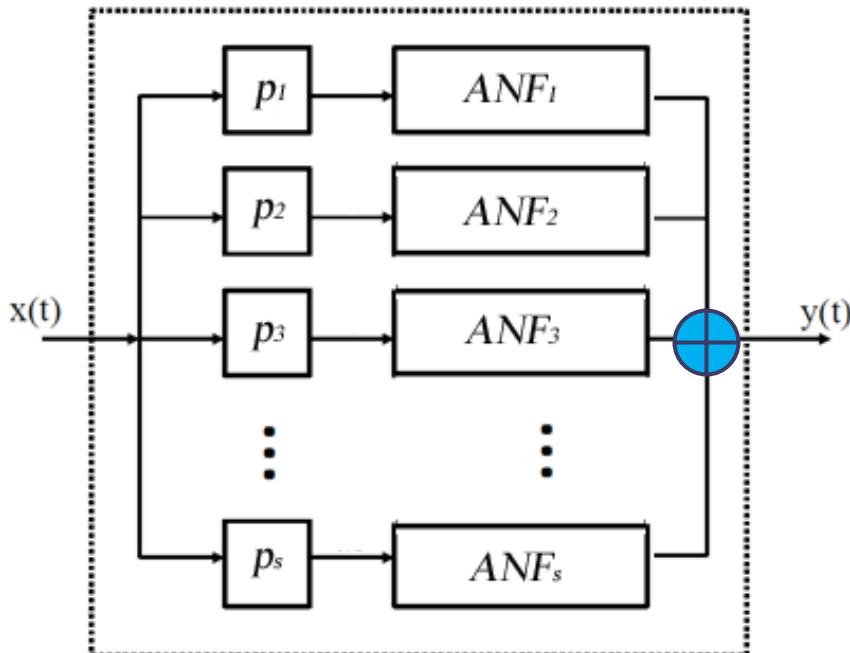
Polynomial Activation Functions

Let $K \triangleq$ Number of principal dynamic modes



If the activation function $p_j(v_j(n))$ is assumed to be a polynomial, then the multi-input nonlinearity $f(v_1, \dots, v_K)$ can be decomposed into the sum of univariate nonlinearities corresponding to each principal dynamic mode.

Modular Volterra Model



ANF ≡ Associated Nonlinear Function

$$y(n) = a_0 + \sum_{m=1}^M \sum_{s=1}^S a_{m,s} (p_s(n) * x(n))^m$$

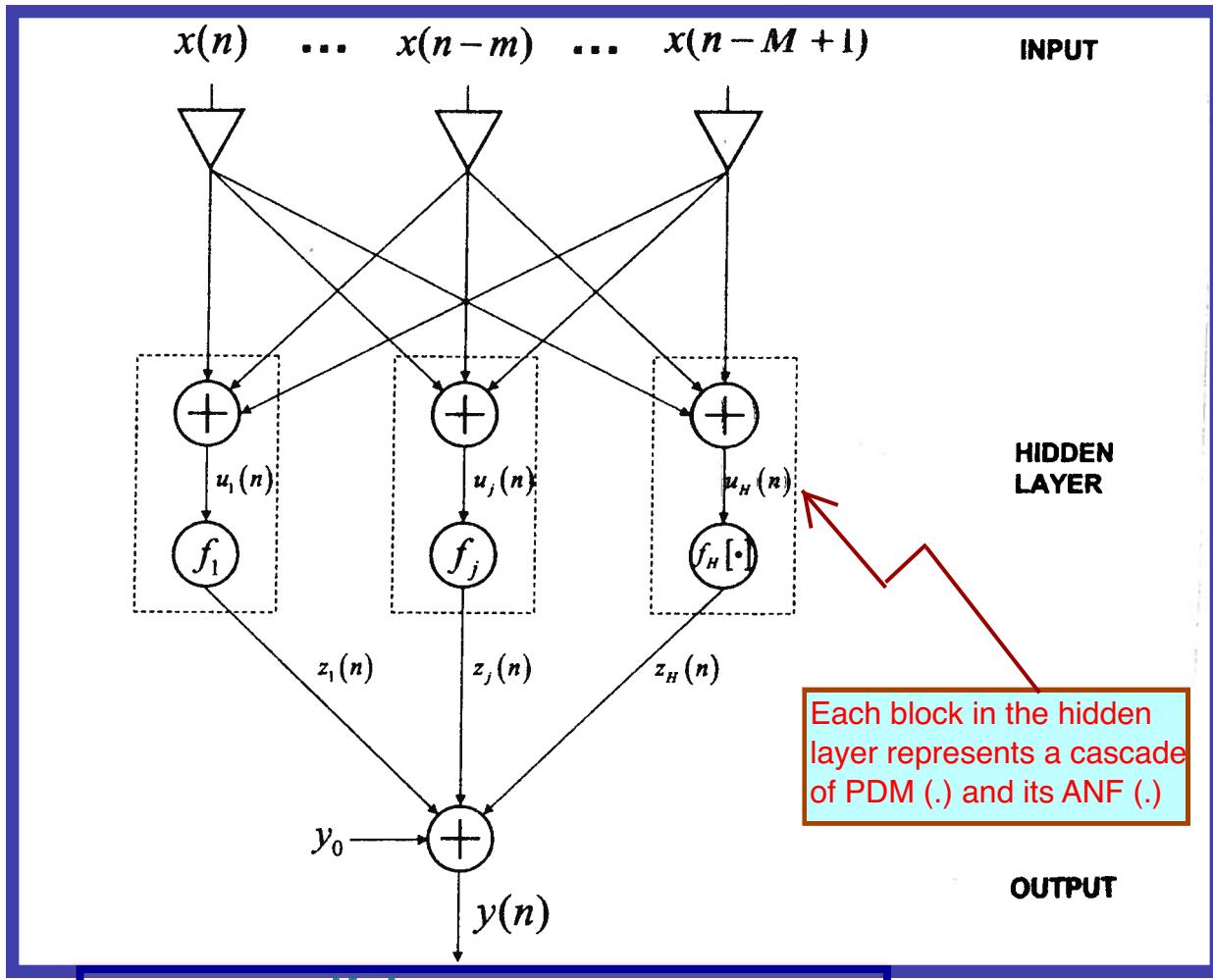
Both $p_s(n)$ and $m_s(n)$ represent the same PDM.

Estimate the power series coefficients $a(m,s)$ to better fit $y(n)$ to the measured response to GWN.

PDM computed from 2nd order but model order is not limited to 2nd order system. Higher order static nonlinearity can be used to model higher order systems.

Separable Volterra Network

PANN



$$u_j(n) = \sum_{m=0}^{M-1} w_{j,m} x(n-m)$$

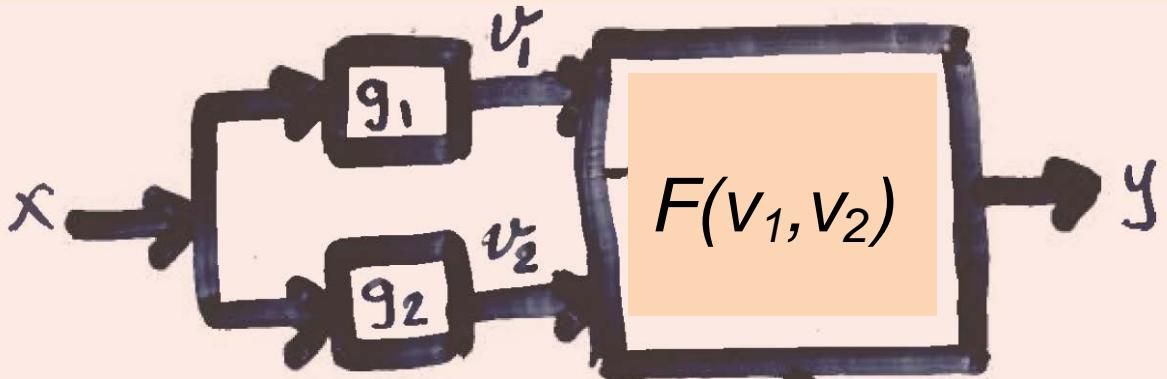
PDM

$$f_j(u_j) = \sum_{q=1}^Q c_{j,q} u_j^q$$

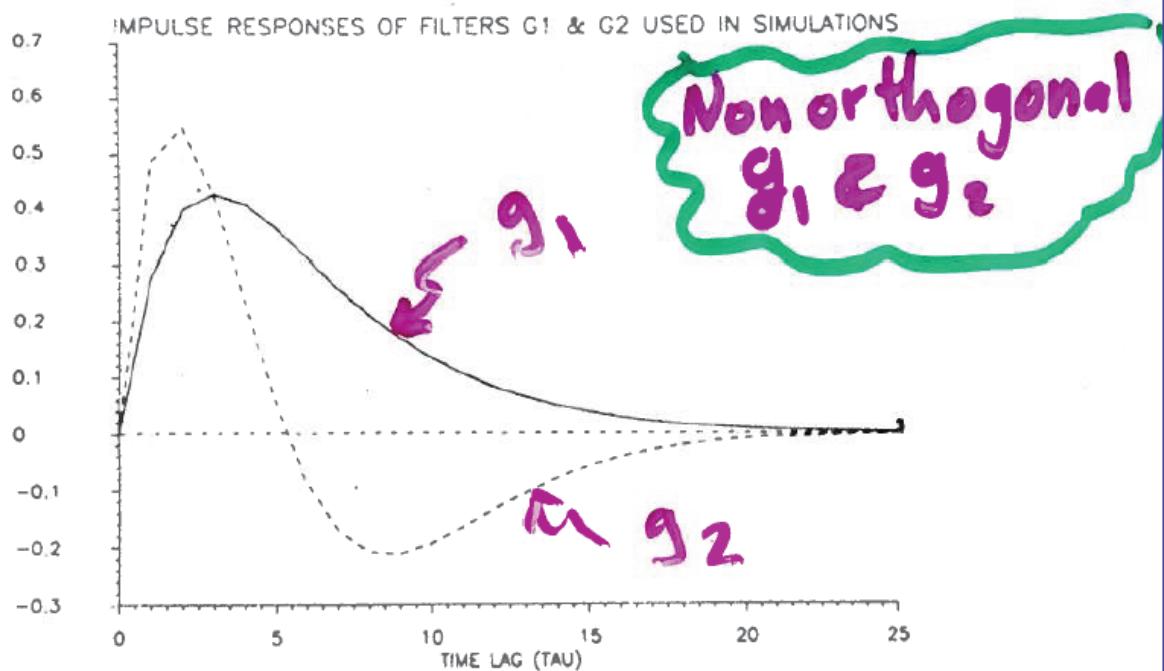
ANF

$$k_q(m_1, \dots, m_q) = \sum_{j=1}^H c_{j,q} w_{j,m_1} \cdots w_{j,m_q}$$

Example



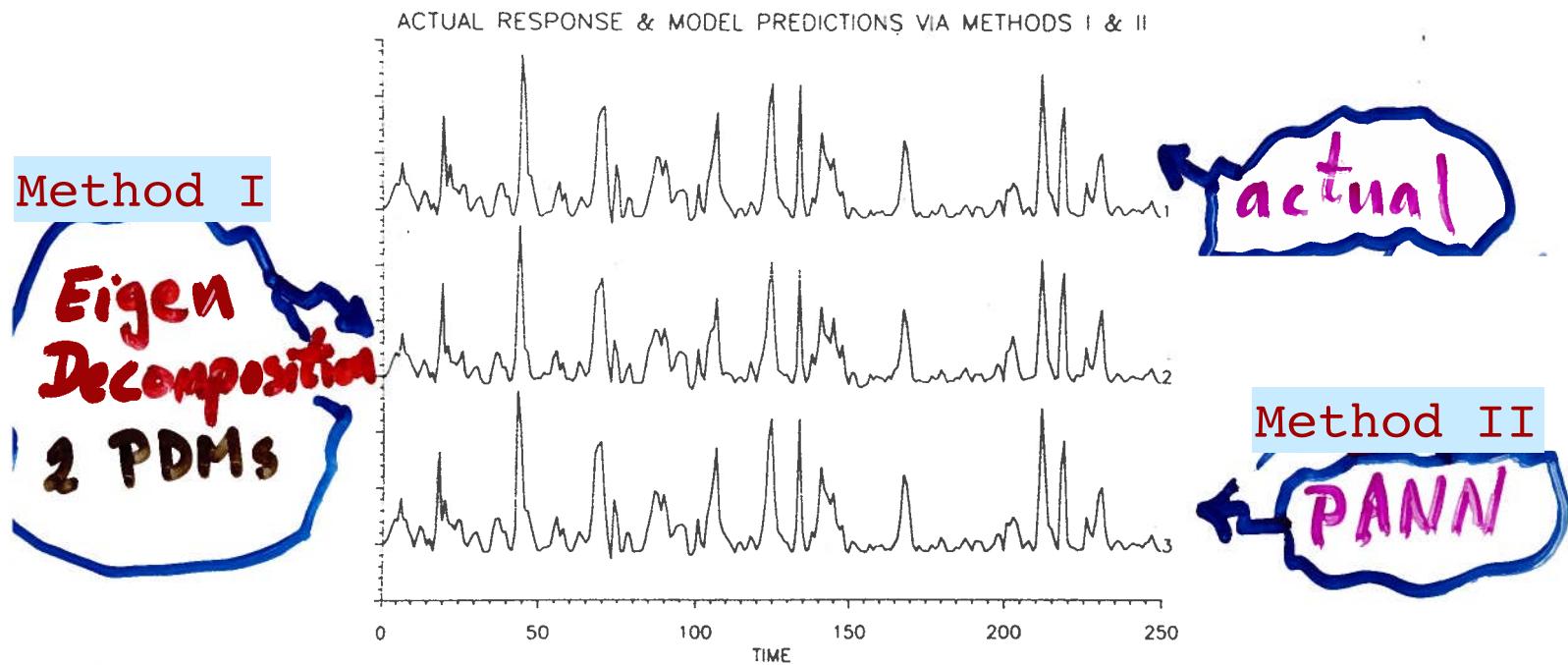
$v_i \triangleq$ convolution of GWN
with g_i for $i=1,2.$



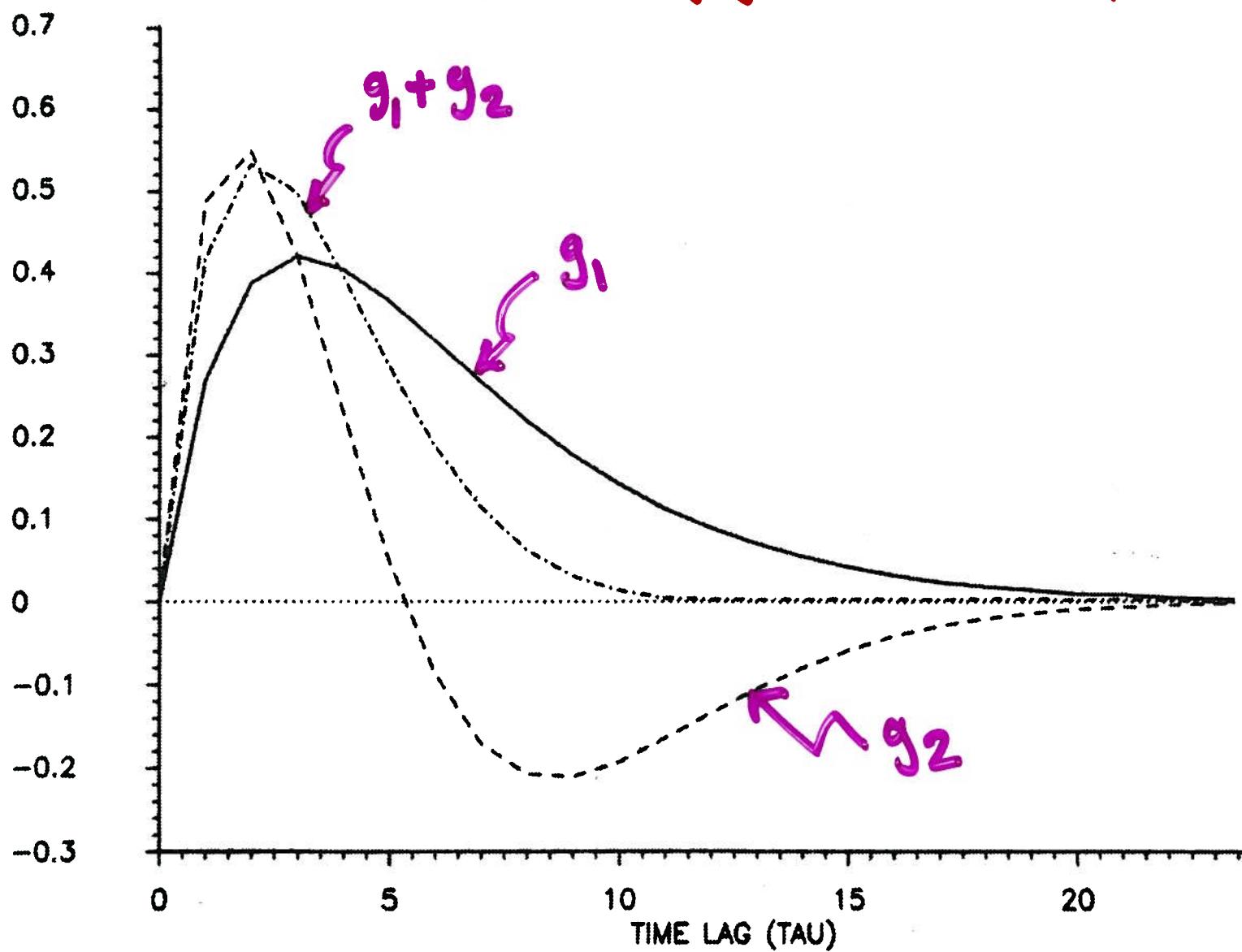
$$y = v_1 + v_2 + v_1 v_2 - \frac{1}{3} v_1^3 + \frac{1}{4} v_2^4$$

* Not feasible to estimate all of the system kernels.

- * Fourth-order PANN with four hidden units.
- * Eigen values = {2.38, -0.65, 0.14, -0.12}



- Estimated PDMs for the PANN of the fourth order system
- Three nonorthogonal PDMs are obtained, the fourth hidden unit had negligible activation function



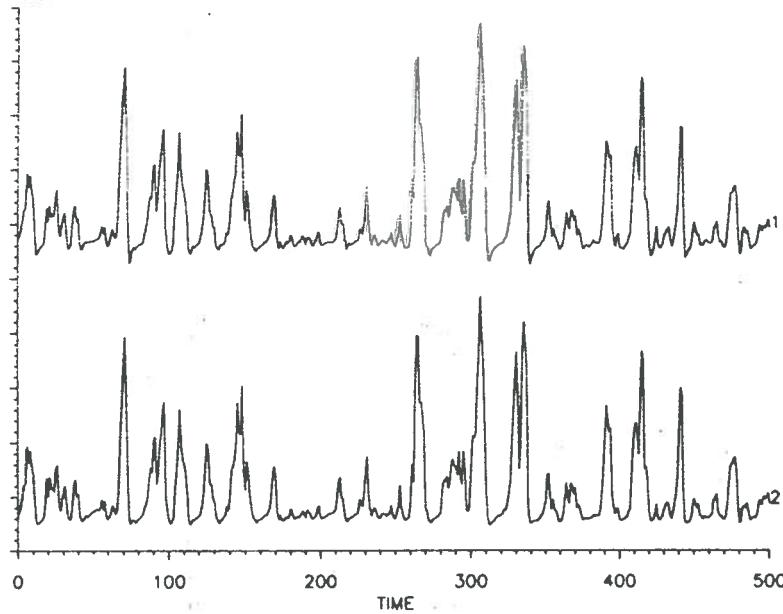
- The three modes correspond to g_1, g_2 & (g_1+g_2) .
- The fourth mode had polynomial coefficients on the order of 10^{-4} .

Predictive Accuracy For Highly Nonlinear Systems

$$y = \exp[v_1] \sin[0.5(v_1 + v_2)]$$

⇒ Infinite-order Volterra system

SYSTEM RESPONSE & MODEL PREDICTION FOR HIGH-ORDER SYSTEM



Actual
infinite
order

fifth-order
PANN
with two
PDMS

Annals of Biomedical Engineering, Vol. 25, pp. 239-251, 1997
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Modeling Methodology for Nonlinear Physiological Systems

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IOP PUBLISHING JOURNAL OF NEURAL ENGINEERING
J. Neural Eng. 9 (2012) 056004 (11pp) doi:10.1088/1741-2560/9/5/056004

Markers of pathological excitability derived from principal dynamic modes of hippocampal neurons

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Biol Cybern (2007) 96:113–127

DOI 10.1007/s00422-006-0108-2

Principal dynamic mode analysis of action potential firing in a spider mechanoreceptor

Georgios D. Mitsis · Andrew S. French · Ulli Höger · Spiros Courellis · Vasilis Z. Marmarelis

IEEE TRANSACTIONS ON NEURAL NETWORKS, VOL. 8, NO. 6, NOVEMBER 1997 pages1421-1433

Volterra Models and Three-Layer Perceptrons

Vasilis Z. Marmarelis, *Fellow, IEEE*, and Xiao Zhao, *Member, IEEE*