Lecture 3. Networks, part II

Networks, part II

Continuining with the networks theme:

- Short term memory
- Stability analysis $+$ oscillations
- Wilson Cowan networks
- Fitz Hugh Nagumo
- Orientation selectivity
- Why model?

Short term memory

Long-term memories may be encoded in synaptic weights, distributed across a network.
Short-term memories may be sustained activity, independent of input.
Internal dynamics maintain signals.
e.g. Delayed match to sample task. Monkey has to remember whether green or red light was turned on (Fuster, 1995). Single neurons in prefrontal cortex show elevated activity.
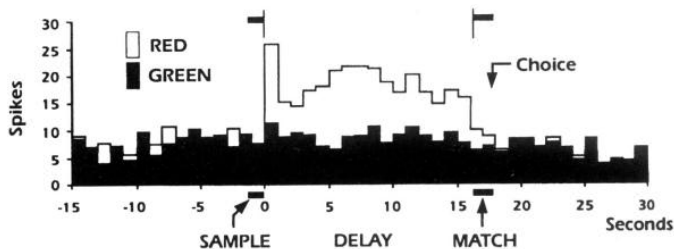
Two neuron example

Two neuron system:

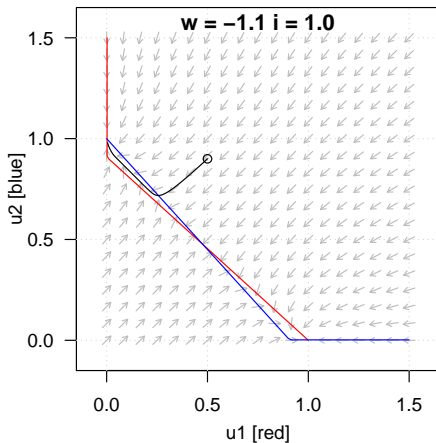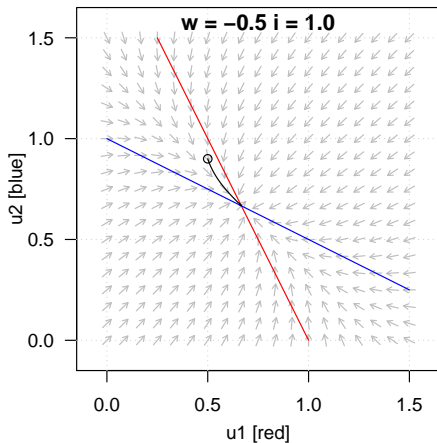$$\tau \frac{d u_1}{d t} = -u_1 + [w_2 u_2 + i_1]_+$$

$$\tau \frac{d u_2}{d t} = -u_2 + [w_1 u_1 + i_2]_+$$

$$\vdots$$

$$\tau \frac{d \mathbf{u}}{d t} = \mathbf{W} \mathbf{u} + \mathbf{i}$$

Approach: find steady-states ($\frac{d \mathbf{u}}{d t} = 0$) and then classify each point using eigenvalues.

Nullclines

Stability analysis in a nutshell

When analysing system
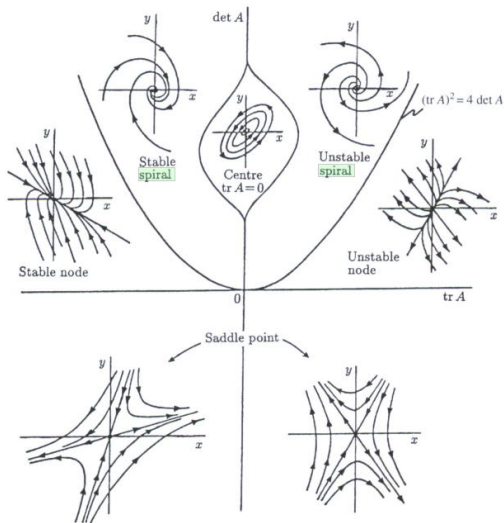
$$\frac{\mathrm{d}\mathbf{u}}{\mathrm{d}t} = F(\mathbf{u})$$

1. Find steady states (draw nullclines).
2. Linearise: find Jacobian at steady state.
3. Compute eigenvalues:
   fp is stable iff $Re(\lambda_i) < 0 \quad \forall i$
   fp is unstable otherwise

Watch out for boundary cases (e.g. zero eigenvalues): purely imaginary roots (stable centre) zero roots (higher order; Lyapunov stability).

Summary of stability criteria (Murray, p504)

For a real 2x2 matrix **A**:

Key point (2-d only): for stability det **A** > 0, tr **A** < 0.

What dynamical systems theory gets us

- Find steady states and predict stability.
- Lyapunov functions for special cases.
- Theorems for oscillations (Poincare-Bendixson; Hopf bifurcation theorem) **Can we predict when action potentials occur?**

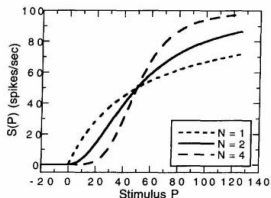## Naka-Rushton (1966) model Fig 2.4, Wilson

NR sigmoidal matches firing-rates in vision research. Same as Michelis-Menton equation in chemical kinetics:

$$s(x) = \frac{mx^n}{\sigma^n + x^n} \qquad \text{for } x > 0$$
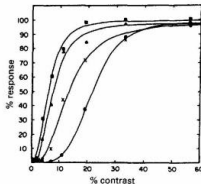
m: maximum spike rate
σ: value at which $s(x)$ reaches half-maximum.
n: typically 1–4 for cortical neurons.

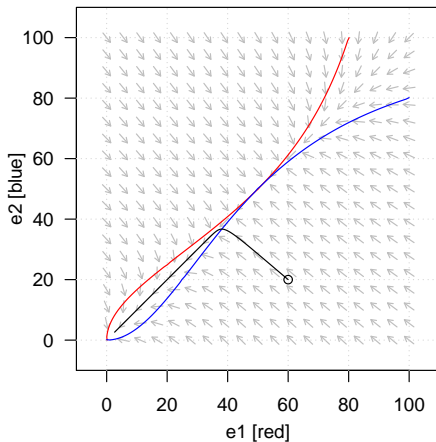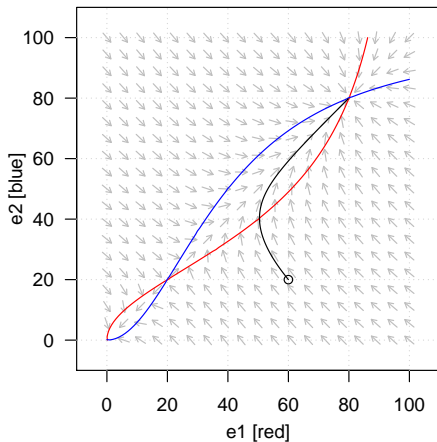Bottom plot: Good fit to range of neurons, inc four shown here (Albrecht & Hamilton, 1982).

Short-term memory circuit

Connect two NR neurons together, with input *a* times input from other neuron.

$$\tau \frac{de_1}{dt} = -e_1 + \frac{m(ae_2)^2}{\sigma^2 + (ae_2)^2}$$

$$\tau \frac{de_2}{dt} = -e_2 + \frac{m(ae_1)^2}{\sigma^2 + (ae_1)^2}$$

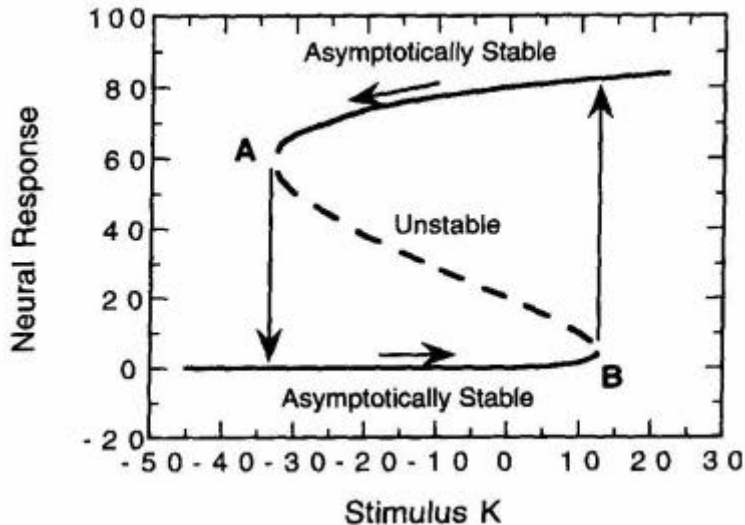Approach: draw nullclines, find steady-states and determine stability.

Nullclines

Left: m = 100; sigma = 120; a = 3; tau = 20. Right: sigma=150.
What if neurons were mutually inhibitory?

Hysteresis loops (Fig 6.5, Wilson)

$$\tau \frac{d e_1}{d t} = -e_1 + \frac{m[ae_2 + k]_+^2}{\sigma^2 + [ae_2 + k]_+^2}$$

Poincaré-Bendixson theorem

Suppose there is an annular region in an autonomous (i.e. constant coefficient) two-dimensional system that satisfies two conditions:

1. the annulus contains no equilibrium points;
2. all trajectories that cross the boundaries of that annulus enter it.

Then the annulus must contain at least one asymptotically stable limit cycle.

If there is a fixed point within a trapping region, we must ensure that it is a unstable repeller, and make a small hole around it to exclude it.

FitzHugh-Nagumo (FHN) model

Alternative simplification of HH system, for analytical tractability.
Space-clamped version (I is external input to neuron):

$$\frac{dv}{dt} = 10(v - v^3/3 - r + I) \qquad \text{Membrane voltage}$$

$$\frac{dr}{dt} = 0.8(-r + 1.25v + 1.5) \qquad \text{Recovery variable}$$
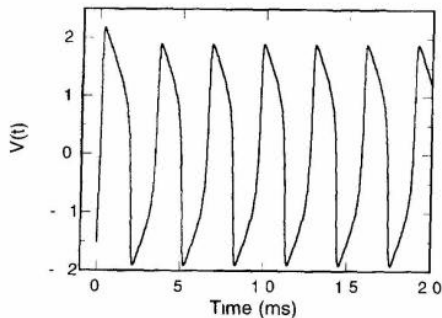
Examine nullclines and behaviour of system.

FHN model: steady states

$$\frac{\mathrm{d}v}{\mathrm{d}t} = 10(v - v^3/3 - r + I) \qquad \text{Membrane voltage}$$

$$\frac{\mathrm{d}r}{\mathrm{d}t} = 0.8(-r + 1.25v + 1.5) \qquad \text{Recovery variable}$$

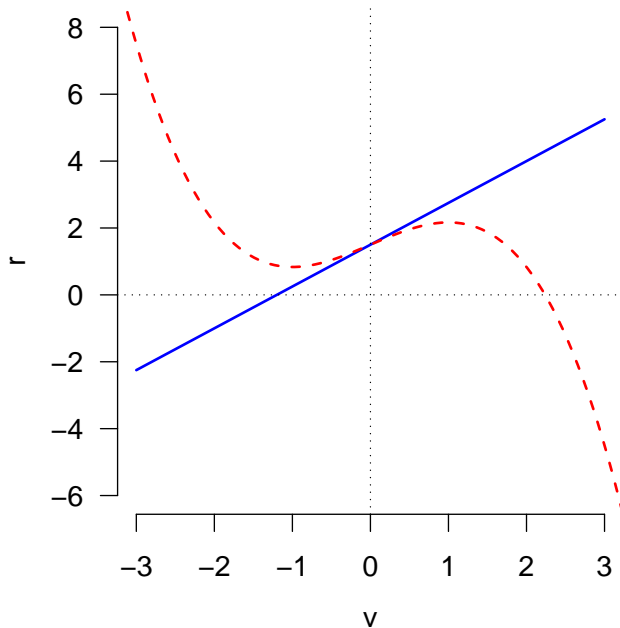$$\mathbf{J} = \begin{pmatrix} 10(1 - v^2) & -10 \\ 1 & -0.8 \end{pmatrix}$$

When $I = 0$, s.s is $(v_0, r_0) = (-1.5, -3/8)$.
Evaluate eigenvalues of $\mathbf{J}$ at s.s.: Det $=$; Trace $=$.

When $I = 1.5$, s.s. is at $(0, 1.5)$.
Evaluate eigenvalues of $\mathbf{J}$ at s.s.: Det $=$; Trace $=$.

FHN model: trapping region (see Wilson, p125)

Hopf bifurcation theorem

Poincaré-Bendixson theorem works for 2-d system only; what about higher-dimensional systems?

For an N-dimensional system, where:

$$\frac{d\mathbf{x}}{dt} = F(\mathbf{x}, \beta)$$

$\mathbf{x}_0$ is an isolated equilibrium point. Assume $\beta = \alpha$ is a critical value with properties determined from Jacobian $\mathbf{A}(\beta)$:

1. $\mathbf{x}_0$ is asymptotically stable for some finite range of values $\beta < \alpha$.

2. At $\beta = \alpha$, $\mathbf{A}(\beta)$ has one pair of eigenvalues $\pm i\omega$; all other eigenvalues have negative real part.

3. $\mathbf{x}_0$ is unstable for some range of values $\beta > \alpha$.

Then the system has an asymptotically stable limit cycle over a range $\beta > \alpha$ (supercritical/soft bifurcation) OR an unstable limit cycle over some range $\beta < \alpha$. (subcritical/hard bifurcation).

Near $\beta = \alpha$ the frequency of oscillation will be approx. $\omega/2\pi$, and the oscillation emerges with infinitesimal amplitude sufficiently close to α.

What do we gain from this?

Supercritical vs subcritical bifurcation

Typically need to do numerics around critical value α to see which side the limit cycle is located.
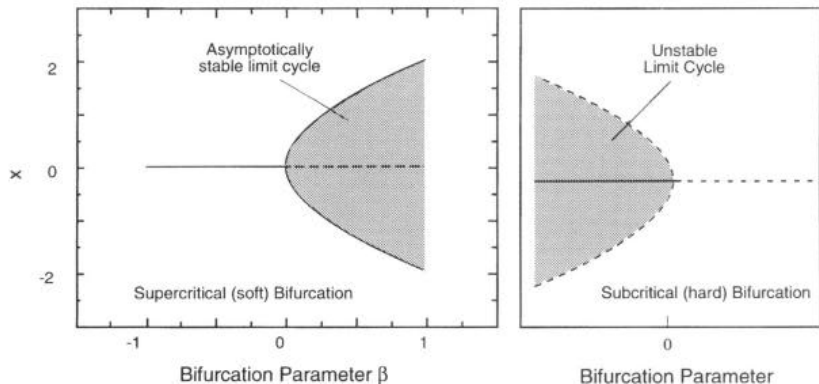
(Fig 8.9, Wilson)

Example of application of Hopf bifurcation theorem (HBT)

e.g. van der Pol equations.

$$\frac{\mathrm{d}x}{\mathrm{d}t} = y$$
$$\frac{\mathrm{d}y}{\mathrm{d}t} = -\omega^2 x + y(\beta - x^2)$$
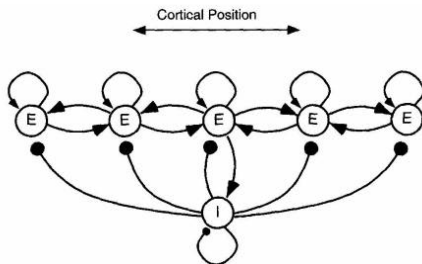
Apply HBT to determine period of limit cycle.

Wilson and Cowan (1972) networks

$$\tau \frac{dE(x)}{dt} = -E(x) + F_e\left(\sum_{x'} w_{EE}(x - x')E(x) - \sum_{x'} w_{IE}(x - x')I(x) + P(x)\right)$$

$$\tau \frac{dI(x)}{dt} = -I(x) + F_i\left(\sum_{x'} w_{EI}(x - x')E(x) - \sum_{x'} w_{II}(x - x')I(x) + Q(x)\right)$$

$$F(x) = \frac{mx^2}{\sigma^2 + x^2}$$

$$w_{ij}(d) = b_{ij} \exp(-|d|/\sigma_{ij})$$

Phenomena of Wilson and Cowan networks

Assume conditions for spatially uniform steady states to be unstable.

1. Short term memory
2. Spatially localized oscillations.
3. Travelling waves leading to visual hallucinations.
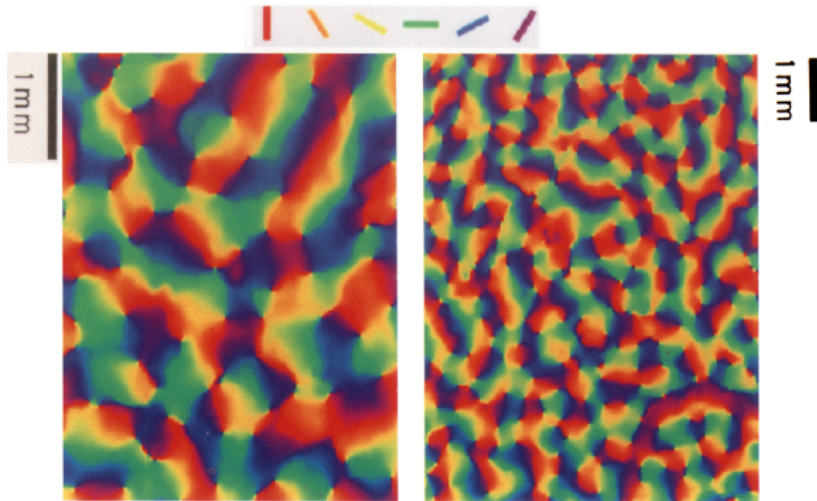
Matlab demonstrations:

1. `WCcortexSTM.m`: 10 ms pulse of 10, 100, 400 1000 μm.
2. `WCcortexWAVES.m`: 5 ms pulse (with weaker inhibition).

Visual hallucinations
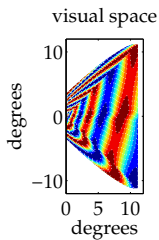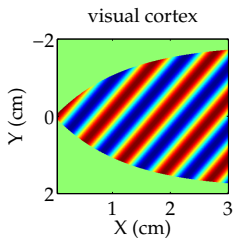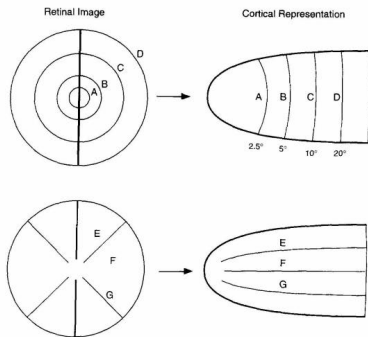
Left: retinocortical map in humans; Wilson Fig 7.16. Right: See code c2p5.m from TN.

Pinwheel maps of orientation selectivity <small>(Blasdel, 1992)</small>

Simple-cell selectivity

Is the Hubel and Wiesel feedforward model correct? Try cooling the cortex to remove effect of cortical interactions (Ferster et al., 1995). No difference in shape of tuning bands, but both seem quite wide.

Orientation tuning of cat V1 simple cell
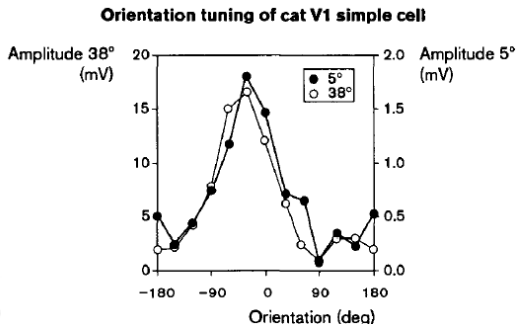
Cortical cells receive most of their input from other cortical cells; thalamic inputs only around 6% of input. Can excitatory connections do this work? Columnar organization of cortex might help.

Ben-Yishai et al. (1995)
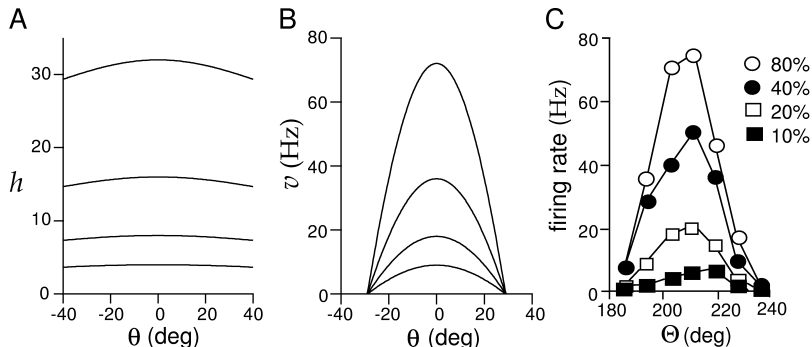
Recurrent organization of neurons.

LGN input $\qquad h(\theta) = Ac(1 - \epsilon + \epsilon \cos(2\theta))$

A controls amplitude, c is for contrast. $\epsilon = 0 \Rightarrow$ no LGN selectivity; $0.5 \Rightarrow$ maximum selectivity. OS of LGN neurons is small (e.g. $\epsilon = 0.1$).

$$\tau \frac{d v(\theta)}{d t} = -v(\theta) + \left[h(\theta) + \frac{1}{\pi} \int_{-\pi/2}^{\pi/2} (-\lambda_0 + \lambda_1 \cos(2(\theta - \theta'))) v(\theta') \, d\theta' \right]_+$$

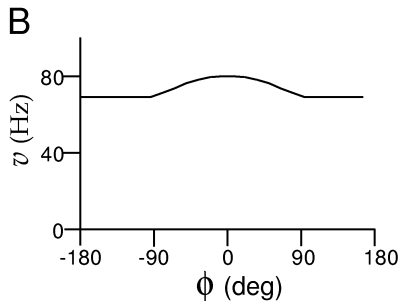# Contrast affects amplitude, not width, of orientation tuning (TN Fig 7.10)
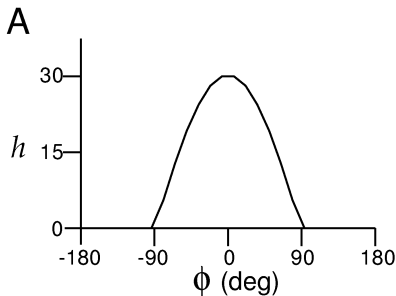

A: Weakly-tuned input ($\epsilon = 0.1$) at four contrasts. B: prediction of lateral model. C: Experimental data from Sclar and Freeman (1982).

Recurrent model also creates complex cells (TN Fig 7.11)

Chance et al. (1999) extended idea to complex cells:

$$M(\phi - \phi') = \lambda_1/(2\pi\rho_\phi)$$

$$\tau\frac{\mathrm{d}v(\theta)}{\mathrm{d}t} = -v(\phi) + \left[h(\phi) + \frac{\lambda_1}{2\pi}\int_{-\pi}^{\pi} v(\phi')\,\mathrm{d}\phi'\right]_+$$

A: simple cell inputs with different preferred phases (ϕ). B: Network response (equivalent to tuning curve of an output neuron) ($\lambda_1 = 0.95$).

Role of modelling

Marder and Abbott (1995).

- Why model?
- Confirmatory models: determine whether existing experimental data can account for observed behaviour. (e.g. conductance-based models).
- speculative models: suggest neural mechanisms underlying a behaviour; or take known mechanism and explore consequences of driving it under wide range of situations.
- Experiments test for necessity; models test for sufficiency.

Dynamic clamp

Dynamic clamp arrangement

Dynamic clamp takes membrane voltage, and simulates current that will pass through a particular channel; that channel is injected real-time, as if that channel existed on the neuron.
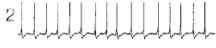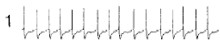This allows us to use biology to worry about all the other conductances, rather than a modeller using poor estimates of the conductances.

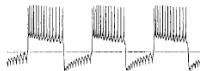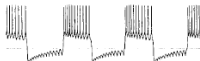# Application of dynamic clamp (Marder and Abbott, 1995, fig 2)

Can two mutually inhibitory neurons fire in synchrony?
Use dynamic clamp to "virtually connect" two neurons.

What kind of modelling is this?

Summary

- Numerical integration.
- Stability analysis (PB; HB theorems).
- Short term memory.
- Larger networks.
- Simple and complex cells revisited.

- Reading: Marder and Abbott (1995). Vogels et al. (2005); TN Chapter 7; Wilson Chapter 7,8.
- Matlab code for Chapter 7 (of Wilson) will go on web.