

Projects

1. Consider the spherical brain. There is a weak sense in which we can consider the brain as a sphere with connections across hemispheres regarded as providing boundary conditions between various areas. Some nice EEG/MEG data (<http://tng.ccs.fau.edu/projects/brain-imaging/index.html>) shows that there are rotating waves during alpha rhythms. So, with this in mind, we want to understand the dynamics of arrays of oscillators on a spherical surface. In particular, can there be rotating waves. One way to approach this is via weak coupling analysis leading to:

$$\theta'_j = 1 + \sum_{k \in N(j)} H(\theta_k - \theta_j)$$

where $N(j)$ is the set of neighbors of j . Problem 1: Arrange 20 oscillators on a sphere with icosahedral symmetry such that each talks to 5 neighbors. Solve the resulting equations using $H(x) = \sin(x)$ with random initial data. For most initial data, the solutions go to synchrony. However, for a nonzero measure set of data, you will get a rotating wave. For such a rotating wave, write down the relative phases. Prove that this is a solution to the phase model. Prove that it is asymptotically stable. Replace $\sin(x)$ with $\sin(x + \beta)$ and follow the rotating wave as β changes using a bifurcation package. You may want to restate the odes in relative phase coordinates. Problem 2. For 4, 8, 12, 20 oscillators, there is a simple way to arrange the oscillators on the sphere. Consider, say 100 oscillators. Try to find a way to arrange them on the sphere as uniformly as possible. Then create an adjacency matrix for all oscillators within some radius, $r \ll 1$ of each oscillator. Create an ODE from this with the sine model and try to find rotating waves.

2. Take the standard Hodgkin-Huxley equations. Multiply the equations for the inactivation of sodium (the h equation) by $f = 1/4$ slowing it down a bit. Inject current into the model. Discover that, for example, when $I = 12$, there are mixed mode oscillations. Set $m = m_\infty$ to reduce this to a three-dimensional problem. Use the theory of canards treating V as the one fast and n, h as the slow variables to find the folded canard structure and determine the conditions when the funnel contains the singular orbit. Study this as the parameter f changes (e.g. can you find the folded canard when $f = 1$) You may want to study the paper by Jon Rubin and Martin Wechselberger.
3. Find a predator-prey model which has true limit cycles (not bogus oscillations like the Lotka Volterra). Consider a networks of such local PP systems and allow coupling between the networks. (Start with a pair and then go beyond this). The type of coupling is up to you - but should take into account, for example, different mobility between predator and prey, etc. Add some heterogeneity in the environment or possible some

weak yearly periodic forcing or correlated stochastic fluctuations. Under what conditions do the populations synchronize. (Note the latter, common fluctuations is an example of the Moran effect.) Apply the weak coupling theory to verify your results.

4. Start with the Reynolds model for inflammation in one of the bistable or tristable regimes. Imagine now that this system is distributed spatially or over multiple compartments. Devise some model equations and some methods to couple the different compartments/areas. Some possible issues are: (1) Can sepsis or overactive inflammation in one compartment drive another compartment? (2) How do the interactions depend on e.g. diffusibility of cytokines; movement of neutrophils; migration of pathogens? (3) In a spatially distributed network, is it possible to get localized sepsis or must it always spread or die out?
5. Using the Morris-Lecar model in the Class I regime, couple a pair with weak excitation (this will result in anti-phase oscillations). Now drive the pair with partially correlated noise (white and colored). Vary the strength of the noise, its color, and its correlation and study the interactions between this signal and the intrinsic coupling. In particular, explore the density of phase differences between the two neural oscillators.
6. The modification of the seashell model has interesting kinetics that have yet to be analyzed in the continuous time domain. Consider the following equation:

$$P(t) = F_e(k_e(t) * P(t)) - F_i(k_i(t) * P(t))$$

where

$$k_j(t) := 2 \exp(-t^2/\sigma_j^2)/(\sqrt{P_i}\sigma_j),$$

$$k(t) * P(t) := \int_0^\infty k(s)P(t-s) ds,$$

and

$$F_j(x) := a_j/(1 + \exp(-\nu_j(x - \theta_j))).$$

Choose a_j, θ_j such that $P = 1/2$ is a fixed point. Without loss of generality, set $\sigma_e = 1$ and treat ν_j, σ_i as parameters. Simulate this Volterra equation and find (if possible) some parameters for which there is a Hopf bifurcation. You may have to learn a bit about the error function of imaginary arguments and plot this a bit to see what it looks like.

7. The Wilson Cowan equations have the form:

$$\begin{aligned} u' &= -u + f(a_{ee}u - a_{ie}v - \theta_e) \\ \tau v' &= -v + f(a_{ei}u - a_{ii}v - \theta_i) \end{aligned}$$

$f(u) = 1/(1 + \exp(-u))$ and all parameters are non-negative. Choose parameters for this model such that the system is excitable (there are

many choices) or bistable. Introduce spatial coupling in a one-dimensional array, using spatially decaying weights, $w_{e,i}(x)$ and a convolution. Study the propagation of activity in one-dimension as you vary the ratio of the interaction distances of excitation and inhibition. Does activity always propagate? Can you reproduce the results of Dave Pinto who shows a very weak effect of the velocity on the strength of the inhibition, but a strong effect on the amount of excitation (see figure 6a in J Neurosci. 2005 Sep 7;25(36):8131-40.) Pinto also shows that waves can reflect off of regions where the density of the neurons changes abruptly. Create a model of this and try to get reflected waves. It may help to look at (1996 SIAM-J.-Appl.-Math 56: 1107-1128).

8. A former student of mine never finished his thesis but it concerned an interesting problem – optimization of an artificial liver device. The idea of the device is that blood is flowed through a tube which has a semi-permeable membrane allowing ammonia to diffuse into a packed column of liver cells. These convert the ammonia to urea which is diffused back into the flowing blood and excreted. Build up of ammonia is very dangerous and can cause all sorts of health problems. The conversion of ammonia to urea is very complicated, but we will treat it simply as a Michaelis Menten enzymatic degradation:



Build a model for this as follows. $S_o(x, t)$ is the substrate outside the liver and $P_o(x, t)$ is the product. S_i, P_i have similar definitions. The equations for S_o, P_o are

$$Z_t(x, t) = DZ_{xx} - vZ_x + d(Z_i - Z_o)$$

where Z is either P, S . Inside the tube, there is no flow so there are just ODEs based on Michaelis-Menten. For example

$$\partial_t S_i(x, t) = d(S_o(x, t) - S_i(x, t)) - R(S_i)$$

where R is the rate of degradation of the substrate. The boundary conditions are $S_o(0, t) = S_0$ and $P_o(0, t) = P_0$. and no-flux at the other end. In the steady state, this is a boundary value problem. The goal is to optimize the flow rate, v so that the maximal amount of ammonia is degraded to urea. Note that there should be such an optimum since at very slow flow rates, we complete the reactions very nicely but get little throughput, while at fast flow rates, there is little accomplished. Write the BVP and numerically solve it. Figure out what to maximize and plot this as a function of the flow rate. Use a linear approximation for the enzyme kinetics. Can you write down a closed form solution, say, as $D \rightarrow 0$? (This is kind of a Jim Keener type of problem :)