BIOMATH SEMINAR

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Harris 4119

Dynamics and complexity of neural spike correlations

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Abstract: Correlations among neural spike times are found widely in the brain; they can be used to modulate or limit information in population coding, and open the possibility for cooperative coding of sensory inputs across neural populations. Correlations also introduce a daunting complexity; when every neuron is potentially correlated with every other, the amount of information needed to represent spiking activity grows exponentially with the number of cells.

In this talk I discuss recent work towards understanding how the structure and transfer of correlated activity is affected by both intrinsic neuron dynamics and network architecture. I first present an interesting and non-intuitive result about how the phase space structure of neural models - specifically the bifurcation that mediates their transition from rest to firing - affects their ability to transmit common signals. Second, I analyze the ability of pairwise maximum entropy models - a technique borrowed from statistical mechanics for representing spiking activity in a simpler way - to perform on a broad class of feedforward circuits. This study provides an explanation for the surprising finding that responses in primate retinal ganglion cells are well-described by this model, even in cases where the circuit architecture seems likely to create a richer set of outputs (Shlens et al., J Neurosci, 2006; 2009; Schneidman et al., Nature, 2006), and identifies pathways by which specific circuit mechanisms influence the complexity of correlation structure.