Modelling and analysis of multi-scale networks

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Title

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Abstract

The broad goal of this thesis is to develop a deeper mathematical understanding of the processes at work in the neuronal network of a brain by rigorously examining very fundamental elements of the processes involved. Paper I considers the flow of discrete signals on a graph and Paper II the complexity arising from the interplay of simple units. The concrete probabilistic investigations of such models invariably involve very strong dependencies. Papers III and IV are thematically different in that they treat a problem from statistical physics. Apart from being interesting in their own right, these papers involve methods of dealing with strong dependencies. Methods which can be used to analytically and probabilistically describe future models.

In Paper I, the signals sent between neurons inspires a model of discrete 'particles' travelling on a graph where every edge is assigned a speed. A steady flow of particles enters on a single incoming edge of a star-graph. At the crossroads (centre node), each particle chooses an outgoing edge and proceeds along it with the corresponding constant speed. The chosen edge is that which has the greatest distance to the nearest particle. For any configuration of speeds, this gives rise to limiting cycles describing the sequence of chosen edges. For two and three outgoing directions, the behaviour is described for all possible speed configurations.

Paper II describes the dynamics of activation on a cellular automaton. Inspired by the existence of inhibitory neurons in a brain, each node (or cell) is assigned an excitatory or inhibitory type, in addition to its time-dependent activation state. Like in bootstrap percolation, the sum of a node's neighbours' activation governs its activation in the following time-step. Unlike in it, the activity of this model is highly non-monotone. Limiting (cycles of) states are examined given random initial activation. Core features of the model are identified and used to develop an understanding of the greater dynamics in certain regimes of initial activation. Since the complexity is greatly increased by some nodes inhibiting others, we suggest that inhibitory neurons provide a computational function in the brain.

Papers III and IV derive results on the Coulomb chain: particles confined to a line which experience pairwise threedimensional Coulomb interaction. We study a version where each particle only interacts with its K nearest neighbours in each direction. The inter-particle distances are random variables given by the Gibbs measure. It is shown, in Paper III for K = 2 and in Paper IV for any finite K, that the correlation between any two sets of consecutive variables decays exponentially with the number of variables separating them. This decay is used in Paper III to prove, in the case when K = 2, a Berry-Esseen type central limit theorem for the dependent random variables.

Key words

Cellular Automata, Correlation Decay, Coulomb Interaction, Dependent Central Limit Theorem, Dynamical Systems, Non-Monotone Bootstrap Percolation, Limit Sets, Probability Theory

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