

MAR16-2015-020022

Abstract for an Invited Paper
for the MAR16 Meeting of
the American Physical Society

Geometric phase transition in the cellular network of the pancreatic islets may underlie the onset of type 1 diabetes.

XUJING WANG, NHLBI, NIH

Living systems are characterized by complexity in structure and emergent dynamic orders. In many aspects the onset of a chronic disease resembles phase transition in a dynamic system: quantitative changes accumulate largely unnoticed until a critical threshold is reached, which causes abrupt qualitative changes of the system. In this study we investigate this idea in a real example, the insulin-producing pancreatic islet β -cells and the onset of type 1 diabetes. Within each islet, the β -cells are electrically coupled to each other, and function as a network with synchronized actions. Using percolation theory we show how normal islet function is intrinsically linked to network connectivity, and the critical point where the islet cellular network loses site percolation, is consistent with laboratory and clinical observations of the threshold β -cell loss that causes islet functional failure. Numerical simulations confirm that the islet cellular network needs to be percolated for β -cells to synchronize. Furthermore, the interplay between site percolation and bond strength predicts the existence of a transient phase of islet functional recovery after disease onset and introduction of treatment, potentially explaining a long time mystery in the clinical study of type 1 diabetes: the honeymoon phenomenon. Based on these results, we hypothesized that the onset of T1D may be the result of a phase transition of the islet β -cell network. We further discuss the potential applications in identifying disease-driving factors, and the critical parameters that are predictive of disease onset.