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Program#/Poster#: 761.2

Title: Diffuse structural changes modulate recurrence period in persistent neural activity

Location: San Diego Convention Center: Room 28A

Presentation Start/End Time: Wednesday, Nov 07, 2007, 8:15 AM - 8:30 AM

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Changes in neural population activity following trauma and neurodegenerative disorders are often attributed to changes in the intrinsic properties of the surviving cells. However, changes in population activity may also be due to the alterations in network architecture brought about by cell death. In this study we examine how diffuse alterations in the structure of networks affect the propagation and oscillatory characteristics of persistent activity. Simulations included networks with up to 12,800 spiking units arranged in two-dimensional excitatory and inhibitory lattices with local columnar connectivity. We examined the propensity of these networks to support persistent activity under varying probabilities of random cell deletions. In previous studies we demonstrated that diffuse deletions could result in large changes to the threshold at which networks switch from a silent to an active state characterized by persistent activity. Here we assessed changes in oscillation properties using a close return algorithm (recurrence plot) to demonstrate that these structural changes also affect the period of activity propagation in a complex manner. In homogeneous networks activity died out quickly. In networks with higher cell deletions activity became persistent and exhibited spiral wave formations with varying levers of recurrence (depending both on initial conditions and level of deletion). Networks with the highest level of deletion could no longer support complex propagating waves and showed only localized periodic oscillations. The simulations confirm that spatiotemporal properties of neuronal activity propagation in heterogeneous networks can change both quantitatively and qualitatively even if intrinsic cell properties remain constant. These changes in activity can be attributed to local change in structure but also more general principles such as percolation. The changes do not require alteration of inhibitory-excitatory ratios. These observations may be important in understanding how EEG changes following cell death in post-traumatic epilepsy as well as in neural degeneration. The connection between structural changes and changes in population activity may also help explain the increased incidents of epilepsy with aging.

Disclosures: **E.L. Ohayon**, None; **W.M. Burnham**, None; **H.C. Kwan**, None; **T.J. Sejnowski**, None; **M. Bazhenov**, None.

Support: CIHR, NIH

[Authors]. [Abstract Title]. Program No. XXX.XX. 2007 Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2007. Online.

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