Criticality and Cancer Dormancy

AMY WU, NIST, DAVID LIAO, UC San Francisco, VLADIMIR KIRLIN, CORINA TAMITA, SIMON LEVIN, JAMES STURM, ROBERT AUSTIN, Princeton University — The presence of driver mutations and subsequent clonal expansion by Darwinian evolution does not explain dormancy and re-emergence of cancer from a community of cancer and stromal cells. Dormancy appears to be a collective property of multiple cell communities including non-cancerous cells. At the simplest level, we view cancer cells interacting with stromal cells via complex, non-linear population dynamics, dynamics which can lead to very non-intuitive but perhaps deterministic and understandable progression dynamics of cancer. We explore here the dynamics of stromal-cancer cell populations in the presence of a chemotherapy drug gradient to determine to what extent the time-dependence of the populations can be quantitively understood in spite of the underlying complexity of the individual agents. The surprising result is that a basic understanding, in a quantitative and predictive manner, can be achieved. It will be intriguing to move to predictive drug dosages, the population dynamics presented here provide a model system for the clinic.