Abstract Submitted
for the MAR16 Meeting of
The American Physical Society

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 quantitive 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.

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Date submitted: 06 Nov 2015 Electronic form version 1.4