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## Toward an Ising model of cancer and beyond.

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### Abstract

The holy grail of tumor modeling is to formulate theoretical and computational tools that can be utilized in the clinic to predict neoplastic progression and propose individualized optimal treatment strategies to control cancer growth. In order to develop such a predictive model, one must account for the numerous complex mechanisms involved in tumor growth. Here we review the research work that we have done toward the development of an 'Ising model' of cancer. The Ising model is an idealized statistical-mechanical model of ferromagnetism that is based on simple local-interaction rules, but nonetheless leads to basic insights and features of real magnets, such as phase transitions with a critical point. The review begins with a description of a minimalist four-dimensional (three dimensions in space and one in time) cellular automaton (CA) model of cancer in which cells transition between states (proliferative, hypoxic and necrotic) according to simple local rules and their present states, which can be viewed as a stripped-down Ising model of cancer. This model is applied to study the growth of glioblastoma multiforme, the most malignant of brain cancers. This is followed by a discussion of the extension of the model to study the effect on the tumor dynamics and geometry of a mutated subpopulation. A discussion of how tumor growth is affected by chemotherapeutic treatment, including induced resistance, is then described. We then describe how to incorporate angiogenesis as well as the heterogeneous and confined environment in which a tumor grows in the CA model. The characterization of the level of organization of the invasive network around a solid tumor using spanning trees is subsequently discussed. Then, we describe open problems and future promising avenues for future research, including the need to develop better molecular-based models that incorporate the true heterogeneous environment over wide range of length and time scales (via imaging data), cell motility, oncogenes, tumor suppressor genes and cell-cell communication. A discussion about the need to bring to bear the powerful machinery of the theory of heterogeneous media to better understand the behavior of cancer in its microenvironment is presented. Finally, we propose the possibility of using optimization techniques, which have been used profitably to understand physical phenomena, in order to devise therapeutic (chemotherapy/radiation) strategies and to understand tumorigenesis itself.

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