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2011 Phys. Biol. 8 010301

(http://iopscience.iop.org/1478-3975/8/1/010301)

Phys. Biol. **8** (2011) 010301 (1pp) doi:10.1088/1478-3975/8/1/010301

PREFACE

Physical oncology

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Cancer is a highly heterogeneous disease whose molecular basis (genes, proteins, signaling networks, etc) has been well examined through many years of basic and clinical research. Recent advancements in high-throughput techniques now enable a new generation of large-scale omic (genomic, proteomic, interactomic, etc) studies to generate a 'data tsunami' with the hope of better understanding the disease that claimed more than 500 000 American lives in 2010. To put this data explosion into perspective, approximately 40 000 omic-related publications were generated between 1923, the first time the term 'genomic' was used, and 2000, when the first draft of the Human Genome Project was released. Between 2000 and 2005, another 50 000 were published, and nearly 100 000 since then up to the end of 2010, when the 1000 Genome Project was announced.

However, accumulating evidence suggests that understanding these biomolecular contributions alone may not be sufficient to obtain a complete picture of failures and mutations required to drive the initiation, progression and spread, or metastasis, of cancer. For instance, recent results have revealed that physical factors of the tumor microenvironment may play a key role in the initiation, establishment and development of tumors and subsequent dissemination of tumor cells to secondary sites. Physical forces underline all major steps of the metastatic cascade: increased tension in the stromal space around growing tumors promotes cellular proliferation and invasion; highly regulated contractile forces and the facile deformation of the nucleus are critical to the ability of tumor cells to negotiate and invade the 3D stromal matrix; the lower elasticity of the cytoplasm critically assists tumor cells in their subsequent intravasation and extravasation into and from the lymphatic and blood vessels; hemodynamic shear stresses, to which tumor cells are subjected during their transit in the vasculature, regulate the levels of expression and the biochemical properties of adhesion molecules and in turn mediate the re-attachment of circulating tumor cells to blood vessel walls; steric forces induced in microcapillaries and narrow lymphatic vessels onto tumor cells play a crucial role in the favored metastatisis between primary and secondary tumor sites.

This special issue of *Physical Biology* gathers together papers that examine various aspects of cancer, incorporating a novel physical/engineering perspective or unique measurement. They cover a wide range of relevant length scales (from the molecular to subcellular and cellular scales, from the multicellular to the organ scale), and describe tumor initiation, growth and metastasis in physical terms. These terms are not meant to replace omic findings but instead to augment them and perhaps connect them beyond the central dogma of biology (DNA, RNA, protein). For example, if we could think of omic findings as values on a steam table, could we borrow approaches and principles from engineering to find 'phases' of cancer? Better yet, are there 'phase transitions', or maybe even a 'triple point' of the disease, that could help explain seemingly contradictory omic data that may have unnecessarily hindered progress against cancer? We hope these papers will contribute to a new field, physical oncology, positioned at the interface of physics/engineering and cancer biology/oncology. We are hopeful that this new field will not only lead to important insights into cancer, but also generate ideas to develop new approaches to cancer prevention, diagnostics and therapy.

