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MULTISCALE MODELLING OF CANCER INVASION

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Abstract:

Recognized as one of the hallmarks of cancer, cancer cell invasion into tissue is a complex process that plays a key role in the growth and spread of cancer, culminating in metastatic spread (secondary cancers). One common aspect of all cancer progression is the secretion of matrix degrading enzymes (MDEs) by the cancer cells that modify or destroy the surrounding tissue or extracellular matrix (ECM) and support local cancer cell invasion. In conjunction with MDE activities, increased cancer cell motility due to changes in cell-adhesion properties further exacerbates the invasion. Transmembrane calcium-dependent adhesion molecules (cadherins) interact with intra-cellular proteins, such as β-catenin and give rise to adhesion junctions. Of particular importance in cancer invasion are the dynamics between the calcium-sensing receptor distribution and the calcium ions (Ca2+) from the ECM. In addition to cell-cell adhesion, the binding of various ECM ligands to cell-surface receptors (integrins) enables cell-matrix adhesion. Thus, processes occurring at a molecular (micro) scale give rise to processes occurring at the tissue (macro) scale, via processes taking place at the cellular (meso) scale. The interplay between micro-, meso- and macro-scale processes involved in cancer cell invasion are still not fully understood.

Despite recent mathematical modelling advances [1,2,3], the understanding of the biologically multiscale process of cancer invasion remains an open question. This talk will address recent advancements in multiscale modelling of cell-cell adhesion inside the tumour in conjunction with the activity of various proteolytic processes occurring along the invasive edge of the tumour. Finally, we will present computational simulations of the resulting multiscale moving boundary model and discuss a number of important fundamental properties that follow.

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