ÉCOLE POLYTECHNIQUE
FÉDÉRALE DE LAUSANNE

Institut de mathématiques des sciences computationnelles et ingénierie
MATHICSE

SEMINAIRE D'ANALYSE NUMERIQUE

- ***MARDI 01 Juin 2010 à 16h15 à la salle MA A110***

Prof. Mark ***CHAPLAIN*** *(University of Dundee, UK)* donnera une conférence intitulée:

"MULTISCALE MATHEMATICAL MODELLING OF CANCER GROWTH AND SPREAD"

The ability to invade tissue is one of the hallmarks of cancer. Cancer cells achieve this through the secretion of matrix degrading enzymes, cell proliferation, loss of cell-cell adhesion, enhanced cell-matrix adhesion and active migration. Invasion of tissue by the cancer cells is one of the key components in the metastatic cascade, whereby cancer cells spread to distant parts of the host and initiate the growth of secondary tumours (metastases). A better understanding of the complex processes involved in cancer invasion may ultimately lead to treatments being developed which can localise cancer and prevent metastasis. In this talk we present two mathematical models of cancer cell invasion – a discrete, cell-based model and a continuum PDE model.

The discrete model focuses on how cell adhesion may be regulated by interactions between E-cadherin and beta-catenin and how the control of cell adhesion may be related to cell migration, to the epithelial-mesenchymal transition and to invasion in populations of eukaryotic cells. E-cadherin mediates cell-cell adhesion and plays a critical role in the formation and maintenance of junctional contacts between cells. Loss of E-cadherin-mediated adhesion is a key feature of the epithelial-mesenchymal transition. Beta-catenin is an intracellular protein associated with the actin cytoskeleton of a cell. Within a mathematical individual-based multiscale model, we are able to explain experimentally observed patterns solely by a variation of cell-cell adhesive interactions. Implications for cell migration and cancer invasion are also discussed.

The continuum model of cancer cell invasion of tissue explicitly incorporates the important biological processes of cell-cell and cell-matrix adhesion using non-local (integral) terms in a system of partial differential equations. The cells use a so-called "sensing radius" R to detect their environment. We show that in the limit as $R \to 0$, the non-local model converges to a related system of reaction-diffusion-taxis equations. A numerical exploration of this model using computational simulations shows that it can form the basis for future models incorporating more details of the invasion process.

Lausanne, le 28 mai 2010
Prof. Assyr Abdulle
VL

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Les séminaires qui ont lieu à la Section de Mathématiques sont annoncés sur Internet à l'adresse WWW (http://memento.epfl.ch/cgi-bin/memento/select?memento=MATHS)