ΙΤΕ/ΕΙΧΗΜΥΘ

ΣΕΜΙΝΑΡΙΟ

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ΤΙΤΛΟΣ: **Mathematical Modeling of Avascular and Vascular Tumour Growth**

ΤΟΠΟΣ: Αίθουσα Σεμιναρίων ΙΤΕ/ΕΙΧΗΜΥΘ

ΗΜΕΡΟΜΗΝΙΑ: Τρίτη, 15 Απριλίου 2008

ΩΡΑ: 12:00

ΠΕΡΙΛΗΨΗ:

The most common cause of primary tumours is the genetic mutation of one or more cells resulting in uncontrolled proliferation. The mutated cells have a proliferative advantage over neighbouring healthy cells and are able to form a growing mass. The reason for this advantage is not necessarily an increase in the proliferation rate, but may be a decrease in the cell death rate. For example, one of the key functions of tumour suppressor genes is to induce apoptosis (programmed cell death) in damaged cells. Loss of this function allows propagation of damaged DNA. If the mutated cells remain contained within a single cluster, with a well defined boundary separating them from neighboring normal cells, the tumour is said to be benign, and surgical removal will often provide a complete cure. However, if the tumour cells are inter-mixed with normal cells and attempt to invade surrounding tissue, the growth ceases to be contained and the tumour is described as malignant.

A tumour may persist in a diffusion limited or avascular state, usually not more than 2mm in diameter, with cell proliferation balanced by cell death for many months or years. It rarely gives rise to significant damage and often goes undetected. A tumour may however emerge from dormancy by inducing the growth of new blood vessels, a process termed angiogenesis, or neovascularisation. This process allows the tumour to progress from the avascular to the vascular state. There are a large number of pro-angiogenic and anti-angiogenic factors. It is a shifting of the balance from the anti-to the pro-angionenic factors (the so-called angiogenic switch) that causes the transition from the dormant to the angiogenic phase. This switch is a highly complex process which is not fully understood, but oxygen deficiency in the tumour is thought to be an important factor stimulating the production of pro-angiogenic molecules by the tumour cells.

In this talk we shall develop generic models of both avascular and vascular tumour growth. We begin by briefly describing the underlying biochemistry and then formulate the principle characteristics necessary to the modeling of an avascular tumour. These characteristics include, cell proliferation, cell death, nutrient supply as well as internal tumour pressure. Under certain laboratory conditions the avascular tumour is known to grow as a spherical compact mass and this allows for analytical solutions to be obtained. We shall also discuss stability of growth. In the case of vascular growth the modeling process is much more complex and depends on crucial biochemical events. We shall outline the biochemistry and show how a realistic cell based model of angiogenesis can be constructed using the theory of reinforced random walks and chemical kinetics. An outline of numerical simulations will be given. Finally we shall discuss some important mathematical questions relating to the governing non-linear partial differential equations. We shall conclude with some open mathematical problems and indicate future modeling programs.