Research Highlight: Mathematical properties of a cancer invasion model

Work of Associate Professor Peter Pang

In a 2017 paper [1] published in the *Journal of Differential Equations*, A/P Peter Pang, together with his collaborator Yifu Wang (Beijing Institute of Technology) investigated mathematical properties of a cancer invasion model. This model describes the migration of tumour cells due to, in addition to random diffusion, two important mechanisms: (i) *chemotaxis*, which is the oriented movement of cells along concentration gradients of diffusible extracellular-matrix-degrading chemicals produced by the cells themselves or in their environment; and (ii) *haptotaxis*, which is the oriented movement of cells along a gradient of cellular adhesion, which is often facilitated by non-diffusible chemo-attractants bound in the extracellular matrix. The model investigated also allowed for the remodelling of the extracellular matrix. This model originated from Chaplain and Lolas [2,3], based on classical work of Keller and Segal [4].

In the past, such a cancer invasion model was often simplified by arguing that the diffusion rate of the matrix-degrading chemicals was often significantly higher than that of the tumour cells. This simplified model yields a system consisting of a parabolic partial differential equation, an elliptic partial differential equation and an ordinary differential equation. The remodelling process was also often neglected, citing the suspected low remodelling rate. To deal with the strongly-coupled parabolic-parabolic-ODE system in its full complexity, Profs Pang and Wang introduced new mathematical techniques. They showed that in two spatial dimensions and in the high cell proliferation regime, the model possesses a unique global solution where the density of tumour cells is uniformly bounded. Their work makes use of previous works of Tao and Winkler [5-7].

References:

[1] P.Y.H. Pang, Y. Wang, Global existence of a two-dimensional chemotaxis-haptotaxis model with remodelling of non-diffusible attractant, *J. Differential Equations* **263**(2017), 1269-1292.

[2] M.A.J. Chaplain, G. Lolas, Mathematical modelling of cancer invasion of tissue: the role of the urokinase plasminogen activation system, *Math. Models Methods Appl. Sci.* **15** (2005), 1685–1734.

[3] M.A.J. Chaplain, G. Lolas, Mathematical modelling of tissue invasion: dynamic heterogeneity, *Netw. Heterog. Media* **1** (2006), 399–439.

[4] E.F. Keller, L.A. Segel, Initiation of slime mold aggregation viewed as an instability, *J. Theoret. Biol.* **26** (1970), 399–415.

[5] Y. Tao, M. Winkler, Dominance of chemotaxis in a chemotaxis–haptotaxis model, *Nonlinearity* **27** (2014), 1225–1239.

[6] Y. Tao, M. Winkler, Boundedness and stabilization in a multi-dimensional chemotaxis–haptotaxis model, *Proc. Roy. Soc. Edinburgh Sect. A* **144** (2014), 1067–1084.

[7] Y. Tao, M. Winkler, Energy-type estimates and global solvability in a two-dimensional chemotaxis–haptotaxis model with remodeling of non-diffusible attractant, *J. Differential Equations* **257** (2014), 784–815.