THE GENESIS OF RESIDUAL STRESSES AND VASCULAR COLLAPSE IN SOLID TUMORS

ROBYN P. ARAUJO* AND D.L. SEAN MCELWAIN †

* Center for Applied Proteomics and Molecular Medicine
George Mason University
Manassas, VA 20110, USA
raraujo@gmu.edu

† School of Mathematical Sciences
Queensland University of Technology
Brisbane, Q. 4001, Australia

Several classic experiments performed during the latter half of the twentieth century have laid a foundation for our understanding of the physiology of solid, vascular tumors. In the experiments reported by Goldacre and Sylven [1], for example, a harmless green dye was injected into the tail veins of mice with transplanted tumors, giving rise to a deep green coloration of the whole animal with the exception of the brain (due to the blood-brain barrier) and the central regions of many of the solid tumors. The investigators concluded that tumors often 'contain substantial regions which cannot readily be reached by blood-borne substances' due to 'some kind of (vascular) collapse'. Importantly, the investigators also reported that 'patent vessels containing bright red blood with intact blood cells' could often be observed in these central regions, suggesting that the vessels in these regions are open.

Another important observation made by Goldacre and Sylven was that, for each type of tumor used in their experiments, 'the critical factor causing differences in the distribution of dye was mainly the age and to some extent the size of the tumors'. While young tumors were instantaneously colored throughout with the green dye, the development of green peripheries enclosing white centers was observed to occur at a critical age, suggesting that vascular collapse occurs after a critical period of growth.

Clearly, this phenomenon of vascular collapse is a key aspect of tumor physiology since it represents a significant barrier to the delivery of anti-cancer agents. This presentation will discuss a linear-elastic model of anisotropic growth which attempts to elucidate this complex phenomenon, and offers interesting insights into the roles of both vascular collapse and host-tumor interactions in the growth dynamics of the tumor.

References

[1] R.J. Goldacre and B. Sylven, "On the Access of Blood-Borne Dyes to Various Tumour Regions," *British Journal of Cancer*, **16**(2), 306–321, 1962.

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