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Preface

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The past two decades have witnessed enormous advances in our understanding of the molecular basis of cell structure and function. All scientists recognize the spectacular success of the human genome project and the consequent burgeoning interest in the related field of proteomics. Biochemists and cell biologists have made similarly impressive strides in elucidating the mechanisms mediating cell signalling and its consequences for the control of cell proliferation, motility and gene expression. In spite of all these advances, cancer remains a very difficult disease to treat. It is still one of the major causes of death in the world (particularly the developed world), with approximately 11 million people diagnosed and around 7 million people dying each year. The World Health Organisation predicts that current trends point to these figures continuing to increase, with around 9 million predicted to die in 2015, with the number rising to 11.5 million in 2030. Perhaps a major reason for these statistics is that cancer growth is a complicated complex phenomenon involving many inter-related processes across a wide range of spatial and temporal scales. New approaches are necessary if further progress in curing the disease is to be made, including quantitative, predictive mathematical modelling.

Over the past two decades or so, there have also been many advances in the mathematical modelling of cancer growth and development, and a comprehensive overview of this activity may be found in [1]. In the last decade especially, much research, both experimental and theoretical has been shaped by the influential review paper of Hanahan and Weinberg, "*The Hallmarks of Cancer*" [2], where six key traits (hallmarks) of cancer growth were identified: evading apoptosis, self-sufficiency in growth signals, insensitivity to anti-growth signals, limitless replication potential, sustained angiogenesis, and tissue invasion and metastasis.

Hanahan and Weinberg have up-dated their overview in a very recent paper "*Hallmarks of Cancer: The Next Generation*" [3], where they discuss the multiscale nature of cancer growth and development, and the interactions between the cancer and its "microenvironment". Other emerging

hallmarks are identified (deregulating cellular energetics and avoiding immune destruction) and two "enabling characteristics" of cancer growth are discussed (genome instability and mutation, tumor-promoting inflammation). Perhaps this special issue of *MMNP* devoted to cancer modelling can be thought of as the modelling equivalent to [3]. The papers to appear in this issue illustrate the range of activity currently going on at present in the cancer modelling community which will form the basis of "next generation" models, and there is an impressive range of scales of "hallmarks" being modelled: gene regulatory networks, stem cell models, the immune response to cancer, invasion models, angiogenesis, metastasis and treatment models. In short, the state-of-the-art in multiscale modelling of cancer. Looking to the future, the development of quantitative, predictive models (based on sound biological evidence and underpinned and parameterised by biological data) will no doubt have a positive impact on patients suffering from the disease through improved clinical treatment.

References

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