Special Issue Introduction
P tumorigenesis is a multistep process that begins with the transformation of a single cell which acquires one or more of the "hallmarks of cancer". Cells acquire these characteristics through numerous somatic mutations caused by errors of DNA replication, the action of exogenous mutagens or endogenous DNA damage, leading to the activation of oncogenes and the inactivation of tumour suppressors. Different tumours differ with respect to their mutational spectra and these patterns of mutation are often characteristic of the tumour in question. Genetic susceptibility to cancer is also conferred by inherited variants of cancer predisposition genes. As whole exome/genome sequencing becomes commonplace in a clinical genetics/personalised genomics setting, we are gradually developing an understanding of the role of both inherited and acquired variants in tumorigenesis. We plan to publish reviews of the molecular genetics of human cancers which we hope will allow the reader to compare and contrast some of the 100+ different human cancers in terms of their underlying mutational spectra.

Benefits
Rigorous mechanism in peer review: one manuscript must be reviewed by at least two relevant experts. We will endeavour to ensure high standards for the review process and subsequent publication by a team of efficient and professional reviewers and scientific editors.
No publication fee: there would be absolutely no charge for publication.
Rapid publication: we will do our best to ensure that accepted papers will be published rapidly with a high quality.
Open Access: As an author you will retain the copyright to your work. By licensing your work under the Creative Commons Attribution License, articles can be re-used and re-distributed without restriction, as long as the original work is correctly cited.
Wide promotions: Published articles will be promoted at academic conferences, through social networks for scientists and relevant indexing services.