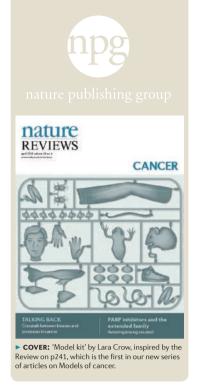
## FROM THE FDITORS











Models of cancer come in various forms from familiar ones, such as cancer cell lines in two-dimensional or three-dimensional culture and genetically modified mice, to the often overlooked ones, such as yeast, nematodes and frogs. Yeast, for example, have contributed much to our knowledge of the cell cycle and the maintenance of cell polarity, whereas egg extracts from *Xenopus laevis* have helped our comprehension of biochemical pathways and protein modifications that are essential for normal cellular function. The mapping of the fate of every cell in *Caenorhabditis elegans* was crucial for our grasp of the genetic pathway that regulates programmed cell

death, and Danio rerio are proving to be useful models of angiogenesis and metastasis. *Drosophila melanogaster* is another genetically tractable organism that has helped to identify pathways that are deregulated during

cancer formation, including the Hippo tumour suppressor pathway.

ancer is a complex disease that develops as a result of genetic

and epigenetic changes in tumour cells and the surrounding

contribute to the progression of this disease. How should we

microenvironment. Systemic changes are also likely to

best model these complexities? Our new article series 'Models of cancer' takes a look at the different systems that researchers use to understand

Whether results are generated by cell lines, yeast or mouse orthotopic tumour models, the cumulative information derived from these models should help us to understand the subtleties of cancer formation in greater detail. All models have their limitations, which can lead to arguments about what makes a good or bad model. However, as long as we use the right models to address our hypotheses, we should move closer to more efficacious treatments for cancer.

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