► COVER: 'Model kit' by Lara Crow, inspired by the Review on p241, which is the first in our new series of articles on Models of cancer.

NICOLA MCCARTHY

GEMMA ALDERTON

SARAH SETON-ROGERS

MEERA SWAMI

Cancer is a complex disease that develops as a result of genetic and epigenetic changes in tumour cells and the surrounding microenvironment. Systemic changes are also likely to contribute to the progression of this disease. How should we best model these complexities? Our new article series 'Models of cancer' takes a look at the different systems that researchers use to understand cancer.
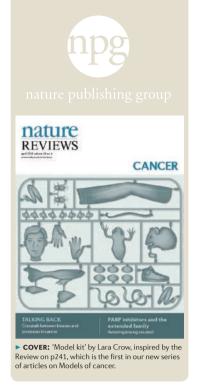
Models of cancer come in various forms from familiar ones, such as cancer cell lines in two-dimensional or three-dimensional culture and genetically modified mice, to the often overlooked ones, such as yeast, nematodes and frogs. Yeast, for example, have contributed much to our knowledge of the cell 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.

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.