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Although mouse models have been recently maligned for their inability to accurately predict the clinical efficacy of anticancer therapeutics, and for how their oncogenic mechanisms differ from humans, it might not be time to set them free from their cages just yet. Three articles in this issue remind us of their usefulness in studying cancer development and the response to therapy.

On page 821, Hugues deThé and colleagues describe how mouse models of acute promyelocytic leukaemia (APL) have been instrumental in forming our understanding of the pathogenesis of this disease. Some mouse models develop a form of APL that is very similar to the human disease. The cancer can also be driven into remission with the same therapeutics in both mice and humans. These mice have therefore been a valuable tool in understanding mechanisms of disease progression as well as mechanisms of therapeutic agents.

On page 807, Vladimir Anisimov *et al.* discuss the similarities and differences between cancer risk in humans and mice. Rodents have many epidemiological trends in cancer incidence that are similar to those of humans, such as a high susceptibility to mammary tumours, a decreasing risk of cancer with advanced age, and differences in cancer incidence between the sexes. Studies in mice could therefore be used to identify the underlying basis of some of these trends.

Finally, as detailed by Robert Hoffman (page 796), experiments using fluorescent markers in mice allow us to study and manipulate many aspects of tumour progression in real time. So, although studies in mice might not provide a perfect representation of human cancer, there is still much we can learn from them about tumour formation, spread, incidence and treatment.