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► **COVER:** 'Unstable' by Lara Crow, inspired by the Review on p102 in which Robert Benezra and colleagues discuss the function of genomic instability in cancer.


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As our knowledge of tumour development grows, our interpretations of the best methods for treating this heterogeneous disease also evolve. This is undoubtedly true for the diagnosis and treatment of oesophageal adenocarcinoma. In this month's issue, Brian Reid and colleagues (p87) reflect on the fact that monitoring and treating patients with Barrett's oesophagus, which was thought to be a pre-cancerous lesion for oesophageal adenocarcinoma, has had little impact on reducing the high mortality rate for oesophageal cancer. Recent findings from both the laboratory and the clinic indicate that a new set of biomarkers that includes cell cycle regulatory genes and markers of genomic instability might help to identify patients with Barrett's oesophagus and other patients with increased risk factors who are likely to develop adenocarcinoma.

The role of genomic instability in cancer initiation and development is still an area of ferocious debate. Does genomic instability drive tumour progression or is it simply a reflection of the high levels of mutation that occur in many solid tumours? On page 102, Robert Benezra and colleagues argue that new mouse models let us begin to pinpoint the reasons for genomic instability in cancer and understand how this drives development and progression. Hopefully, in the not too distant future we will understand how the mutation of oncogenes and tumour suppressor genes in specific tissues results in genomic instability.

Many oncogenes are kinases, reflecting the fact that kinase inhibitors are the largest class of new cancer drugs. However, results from the clinic indicate that targeting multiple kinases is likely to be a more effective approach, and on page 130 Kevan Shokat and colleagues discuss how to best identify new therapeutic combinations of kinase inhibitors.


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Printed in Wales by Cambrian Printers
on acid-free paper