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These days our approach to cancer management usually has a biological rationale. But, although initially successful, a flipside often emerges in the clinic.

For instance, for years radiotherapy and chemotherapy schedules have been devised to allow recovery of normal tissues, limiting toxicity. However, surviving cancer stem cells can proliferate between treatments and repopulate the tumour. John Kim and Ian Tannock (p516) suggest ways of overcoming this.

Jerry Spivak (on p543) focuses on anaemia, a complication of cancer for which there is a specific therapy, erythropoietin. However, tumour cells have erythropoietin receptors and so erythropoietin can potentially promote tumour growth. Clinical trials must therefore focus on tumour biology so that we can learn how to use recombinant erythropoietin effectively.

Cancer cells depend on a few overactive signalling pathways for their survival, as Shoshana Klein, Frank McCormick and Alexander Levitzki discuss on p573, and drugs that successfully exploit this weakness are in the clinic. However, cancer cells quickly find a way round the block on their signal-transduction pathways and become resistant to the inhibitor. Consequently, the focus is now on developing less selective signal-transduction inhibitors and to give them in combination, not as single agents.

Early in cancer management, prognostic factors can also be misleading. Vascular endothelial growth factor (VEGF) is highly expressed during angiogenesis, and is used as an indicator of poor prognosis. However, as discussed in a Research Highlight on p500, circulating VEGF does not enhance tumour growth, so might not be as reliable a prognostic factor as first thought.

So, as we apply our knowledge of the intricate workings of cancer, tumour biology continues to confound us, making us constantly rethink our approaches to cancer management.


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