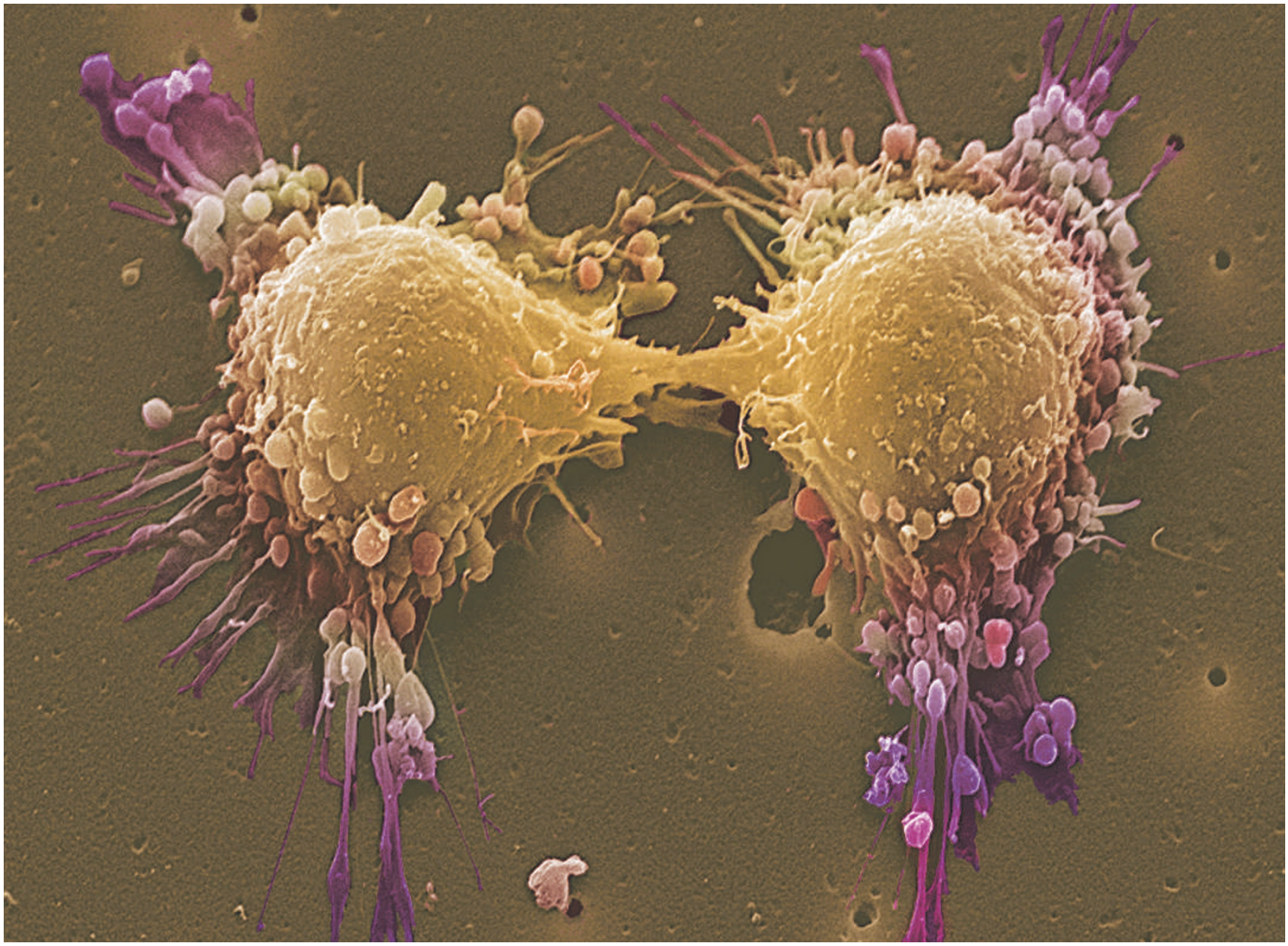

S. GSCHMEISSNER/SPL

CANCER
New fronts in an old war

Like so many wars, the war on cancer can cause despair, with final victory sometimes seeming inconceivable. In the 35 years since President Richard Nixon declared his battle on everybody's enemy, the immense progress in scientific understanding of the disease has yet to deliver the crushing blow that was hoped for.

Indeed, the problem seems to have become less tractable, not more so. The concept of cancer as a single disease has been replaced by a view of it as many different ones, each with its own molecular process, each requiring its own therapeutic approach. Thanks to advances in molecular biology, therapies targeted at specific disease processes are continually being developed. But many believe they are likely to roll back the cancer mortality figures slowly, rather than making a quick and dramatic impact.

If we leave the military analogies aside, we can see that there are possibilities for progress — not for final victories, but for saving lives in the fairly near future, and developing new approaches to stubborn problems. This special section of *Nature* looks at three of these new angles on cancer research and what they promise to contribute.

One area where science could improve survival rates is by finding ways to detect cancers early. As Laura Spinney explains on page 736, existing treatments are far more effective when delivered early, and technologies to detect disease earlier than ever before are becoming available. The problem is choosing the right signs and understanding what they mean.

Even if we catch cancers early, are the drugs targeting them correctly? A new theory suggests that they could be missing something, says Alison Abbott (see page 742). A small subset of cells, called cancer stem cells, could re-seed tumours after treatment. Researchers are trying to develop drugs aimed at cancer stem cells, in the hope of preventing the recurrence of tumours — the leading cause of cancer deaths.

New drugs need to be tested. One long-standing criticism of cancer research is that about nine out of ten drugs that look good in preclinical tests fail to fulfil that promise in human trials. Many point the finger at the mouse, the stalwart animal model of cancer, for being too mouse-like to be a good model for humans. But that is changing, says Carina Dennis (see page 739), as researchers find ever-more ingenious ways to engineer mice to be more like us. ■

For more on cancer see the Editorial on page 720, and visit our web focus at
www.nature.com/nature/focus/cancerhorizons