Of 349 patients treated between 1996 and 2001, 33 reported statin use. At the time of surgery (4–7 weeks post-chemoradiation), no statin patients were found to have metastatic disease, compared with 23 (7%) of the nonstatin patients. Univariate analysis of pathologic complete response showed that only tumor stage was statistically significant (lower stage yielding a better response; adjusted odds ratio 6.1 for stage T1 versus T4). After adjusting for nonsteroidal anti-inflammatory drug use, clinical stage and type of chemotherapy, however, the odds ratio for statin use on pathologic complete response was 4.2 (95% CI 1.7–12.1, $P=0.003$).

Acknowledging the small patient numbers and the retrospective study design, the authors cautioned against the drawing of definite conclusions. The study does, however, raise a new hypothesis to be explored through prospective studies using different endpoints, such as relapse-free or overall survival, and controlled dose and duration of statin use. Should statins be conclusively shown to improve response to neoadjuvant therapy, they might prove to be an accessible, safe, new treatment agent for nonmetastatic rectal cancer.

Rebecca Doherty

Original article Katz MS *et al.* (2005) Association of statin use with a pathologic complete response to neoadjuvant chemoradiation for rectal cancer. *Int J Radiat Oncol Biol Phys* **62:** 1363–1370

Detection of recurrent disease in prostate cancer

2-[^{18}F]fluoro-2-deoxyglucose positron emission tomography (FDG-PET) is useful in the detection of a variety of solid tumors, but the value of this imaging technique in prostate cancer is unproven. Image artefacts in the lower pelvis—caused by accumulation of excreted FDG tracer in the bladder—have been a significant problem in this setting. The recent development of newer, iterative image-reconstruction techniques, however, has allowed improved interpretation of FDG-PET images. In their recent study, Schöder and colleagues investigated the use of FDG-PET plus optimal image reconstruction in patients with biochemical recurrence following prostatectomy for prostate cancer.

This retrospective analysis included 91 patients, all of whom had undergone radical retropubic prostatectomy and had subsequently suffered prostate-specific antigen relapse (prostate-specific antigen >0.1 ng/ml in three consecutive measurements at least 2 weeks apart). Local and/or metastatic lesions were shown by FDG-PET in 31 of the patients. Comparison with bone scan, MRI and CT results showed three of the cases to probably be false positives; in addition, two further cases that were missed by FDG-PET, one of local recurrence and one of metastatic disease, were identified by other imaging devices. The remainder of the false-negative results by FDG-PET, however, were also negative using other imaging techniques.

The study showed that the probability of detecting recurrent disease by FDG-PET increased with rising PSA levels. Noting this fact, Schöder *et al.* suggest that this method might be appropriate only in those with PSA levels of over 2.4 ng/ml or a PSA doubling time of above 1.3 ng/ml/year.

Ruth Kirby

Original article Schöder H *et al.* (2005) 2-[^{18}F]Fluoro-2-deoxyglucose positron emission tomography for the detection of disease in patients with prostate-specific antigen relapse after radical prostatectomy. *Clin Cancer Res* **11:** 4761–4769

PDGFRA mutations and imatinib sensitivity in GISTs

The vast majority of gastrointestinal stromal tumors (GISTs) express the KIT tyrosine kinase, and oncogenic mutations in the *KIT* gene are thought to occur in up to 80% of these cases. Of the remainder, some bear mutations in the gene encoding platelet-derived growth factor receptor alpha (PDGFRA), a homologous kinase. A recent study by Corless *et al.* sought to determine the *KIT* and *PDGFRA* mutation status of over 1,000 GIST specimens, and to investigate the sensitivity of the various *PDGFRA* isoforms to the kinase inhibitor imatinib.

The researchers screened genomic DNA samples from 1,105 paraffin-embedded, unique GISTs. Mutations in the *PDGFRA* gene were found in 80 cases (7.2%) and most of these were within exon 18, in the region corresponding to the kinase activation loop (located in the second tyrosine kinase domain). *In vitro* expression of the *PDGFRA* mutant isoforms demonstrated that most mutations involving codon D842 (e.g. D842V) were resistant to