etoposide ($n=67$), etoposide and cyclophosphamide ($n=11$) or etoposide and busulfan ($n=1$). At transplantation, 62% of patients ($n=49$) were in first complete remission (CR1) and 38% ($n=30$) were beyond CR1, respectively. Estimated overall survival rates were 54% and 29% for patients in CR1 or beyond CR1, respectively ($P=0.01$). Event-free survival rates were 48% and 26% for these respective groups ($P=0.02$). Nonrelapse mortality rates were higher for patients beyond CR1 than in CR1 (54% versus 31%; $P=0.05$), but both groups had a similar incidence of relapse (28% versus 41%, respectively; $P=0.28$). The median time to relapse was prolonged in individuals beyond CR1 compared with those in CR1 (12 months versus 9 months). Univariate analysis showed that overall and event-free survival were affected by white blood cell count at diagnosis, disease status at transplantation and grades 2–4 acute graft-versus-host disease.

These results showed that fractionated total body irradiation and etoposide induction therapy conferred long-term remission in patients with Ph^+ ALL.

Original article Laport GG *et al.* (2008) Long-term remission of Philadelphia chromosome positive acute lymphoblastic leukemia after allogeneic hematopoietic cell transplantation from matched sibling donors: a 20-year experience with the fractionated total body irradiation etoposide regimen. *Blood* **112:** 903–909

KRAS mutations do not predict response to 5-fluorouracil chemotherapy

The treatment of choice for advanced colorectal cancer is 5-fluorouracil, but combinations of anti-EGFR therapies with fluoropyrimidine chemotherapy have shown promise. Since *KRAS* mutations strongly predict resistance to antibodies that target the EGFR, Etienne-Grimaldi *et al.* carried out a study to investigate whether *KRAS* mutations influence treatment outcome in patients with stage IV colorectal cancer who received 5-fluorouracil treatment.

These authors previously reported that mutations in *KRAS* and p53 were not associated with outcome in 56 patients treated with 5-fluorouracil. Their current study included 93 patients with stage IV colorectal cancer and unresectable liver metastases, who were treated with 5-fluorouracil and leucovorin. Tumor samples were assessed for mutations in codons 12 and 13 of *KRAS*, polymorphisms in codon 72 of p53, and additional tumor variables.

KRAS mutations in codons 12 and 13 were present in 36 of 93 metastases. Perfect concordance of *KRAS* mutations between primary tumor and liver metastases was observed in 16 of 48 samples with such mutations. The presence of *KRAS* mutations was not significantly associated with levels of p53, thymidylate synthase, folypolyglutamate synthetase, or dihydropyrimidine activity. The response rate in patients with mutated *KRAS* metastases was 44.4%, compared with 32.1% in those with wild-type metastases. *KRAS* status had no influence on survival.

The authors conclude that *KRAS* mutational status only predicts response to the anti-EGFR element of combination therapies; tumors with *KRAS* mutations can still respond to 5-fluorouracil-based therapy.

Original article Etienne-Grimaldi MC *et al.* K-Ras mutations and treatment outcome in colorectal cancer patients receiving exclusive fluoropyrimidine therapy. *Clin Cancer Res* **14:** 4830–4835