most patients who received interferon-α therapy were promptly switched to imatinib mesylate, hindering an adequate long-term prospective comparative analysis. Kantarjian *et al*., therefore, performed a retrospective comparison of imatinib mesylate and interferon-α-based regimens to determine survival advantage for imatinib mesylate.

Survival in patients with newly diagnosed Ph-positive early chronic-phase CML treated with imatinib mesylate ($n = 279$; median follow-up 42 months) was compared with survival in a historical study group treated with interferon-α-based regimens ($n = 650$; median follow-up 143 months). Patients receiving different doses of imatinib mesylate showed similar survival rates, as did patients receiving different interferon α regimens. The imatinib-mesylate-treated group had a considerable (56%) reduction in mortality compared with the interferon-α-treated group; a survival advantage for imatinib mesylate was seen in all CML prognostic risk groups. The estimated 3-year survival was 96% in the imatinib arm and 81% in the interferon-α arm ($P < 0.01$). The complete cytogenetic response rates were 87% and 28% in the imatinib mesylate-treated and interferon-treated groups, respectively. Multivariate analysis revealed imatinib to be an independent prognostic factor ($P < 0.01$).

These results demonstrate that imatinib mesylate increases the rate of major cytogenetic response and prolongs survival in patients in the chronic phase of CML. The cytogenetic response is predictive of prognosis, and in this study imatinib mesylate improved survival by improving the cytogenetic response.

Original article Kantarjian HM *et al.* (2006) Survival benefit with imatinib mesylate versus interferon-α-based regimens in newly diagnosed chronic-phase chronic myelogenous leukemia. *Blood* **108:** 1835–1840

Prognostic subclasses in karyotypically normal acute myeloid leukemia

Patients with acute myeloid leukemia (AML) and a normal karyotype comprise the largest cytogenetic group of AML. A recent study by Bullinger *et al*. identified a signature based on differential gene expression that characterized two separate subgroups of patients with cytogenetically normal AML according to their predicted outcome. Radmacher *et al*. sought to independently validate the prognostic significance of this signature in a larger group of patients.

Pretreatment samples from 64 uniformly treated adults with cytogenetically normal AML were characterized for prognostic biomarkers, including *FLT3* internal tandem duplication, and clustered into poor or good outcome groups according to the Bullinger signature; gene expression was measured using oligonucleotide microarrays. The association of the signature with patient outcome was determined using cluster analysis.

The authors confirmed significant differences in both overall survival and disease-free survival between the cluster-defined patient subgroups ($P = 0.02$ and $P = 0.05$, respectively). Furthermore, they developed a prognostic classification system derived from the gene-expression signature that could predict outcome for individual patients. A strong association was observed between the outcome classifier and *FLT3* internal tandem duplication.

Progress is being made in gene-expression profiling of AML patients with normal cytogenetics. The challenge for the future is to increase the prognostic utility of expression signatures and incorporate these biological developments into novel risk-adapted therapeutic strategies.

Original article Radmacher MD *et al.* (2006) Independent confirmation of a prognostic gene-expression signature in adult acute myeloid leukemia with a normal karyotype: a Cancer and Leukemia Group B study. *Blood* **108:** 1677–1683

Celecoxib in sporadic colorectal adenomas

Each year over a million new cases of colorectal cancer are diagnosed and half a million deaths are caused by this common malignancy. Yet, this disease is also one of the most preventable of the cancers. Recent efforts have focused on preventing and treating the adenomas that often precede this cancer. Cyclo-oxygenase 2, which mediates inflammation and tumorigenesis, is present within colorectal cancers and adenomas but not normal intestinal tissue, and has been shown in murine studies to contribute to the formation of adenomas. Two studies have now shown that the cyclo-oxygenase 2 inhibitor celecoxib has