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New Drug Provides Options for Imatinib Resistance

Results from a phase I trial involving BMS-354825 (Bristol-Myers Squibb, New York, NY) may indicate that an alternative treatment for patients with imatinib (Gleevec®, Novartis, East Hanover, NJ) resistance has been found.

Imatinib inhibits proliferation and induces apoptosis in Bcr-Abl positive cell lines. Patients who are resistant to imatinib have Bcr-Abl mutations that cannot bind to the drug. BMS-354825 binds to Bcr-Abl differently than imatinib and works with Bcr-Abl mutations.

Researchers followed 36 patients with chronic myeloid leukemia (CML) who previously were treated with imatinib. Complete hematologic remissions occurred in 86% of these patients, and 45% experienced cytogenetic remissions, most of which were complete.

Halfway through the trial, researchers amended the protocol to include 19 patients with accelerated or blast crisis CML; imatinib has been unable to treat these types of CML. Initial results indicated that 75% had hematologic responses, 35% of which were complete.

BMS-354825 is well tolerated. Some blood clots occurred, but incidences were lessened with lower doses. Researchers said that it is still too early to recognize all of the adverse effects.

This study was reported at the 46th Annual Meeting of the American Society of Hematology in December 2004.

Correction

The February 2005 *Clinical Journal of Oncology Nursing* article titled "Therapeutic Options in the Management of Colon Cancer: 2005 Update" (pp. 31–44) by Gail M. Wilkes, MS, RNC, AOCN®, reported incorrectly in Table 3 that the XELOX regimen includes cetuximab. The correct regimen is oxaliplatin 130 mg/m^2 via IV over 2 hours, day 1; and capecitabine 1,000 mg/m^2 by mouth twice a day, evening day 1–morning day 15. Repeat every 3 weeks. We apologize for the error.

Blood Tests May Replace Bone Marrow Biopsies in Patients With Leukemia or Lymphoma

The University of Texas M.D. Anderson Cancer Center has developed five blood testing methods that may replace bone marrow biopsies for diagnosing and monitoring leukemia and lymphoma. Quest Diagnostics Inc. is developing the tests based on M.D. Anderson technology and has applied for two U.S. patents.

Current diagnostic and monitoring tests for patients with leukemia or lymphoma often require painful procedures such as bone marrow biopsies that involve extraction of tissue with a bone-piercing, large-gauge needle. The new blood tests have the potential to provide a more clinically useful assessment of prognosis, disease progression, and therapeutic success while reducing the pain associated with testing. Researchers said that the blood tests show what is happening to the body as a whole, whereas biopsied tissue samples could relay information about only the specific area from which a sample was taken.

The blood tests detect certain proteins that are expressed on the surface of tumor cells and molecular targets from tumor cells. The assays will look for the CD20, CD33, and CD52 proteins and tumor-specific DNA and RNA in blood plasma.

Scientists Discover Key Genetic Factor in Determining HIV and AIDS Risk

People with more copies of a gene that helps to fight HIV are less likely to become infected with the virus or develop AIDS than those of the same geographical ancestry, such as European Americans, who have fewer copies of the gene, according to a study from the National Institute of Allergy and Infectious Diseases. Scientists believe that this discovery could lead to a screening test that identifies people who have a higher or lower susceptibility to HIV and AIDS, potentially enabling clinicians to adapt treatment regimens, vaccine trials, and other studies accordingly.

The study focused on the gene that encodes CCL3L1, a potent HIV-blocking protein that interacts with CCR5—a major receptor protein that HIV uses as a doorway to enter and infect cells. The researchers analyzed blood samples from more than 4,300 people who were HIV positive and negative and had different ancestral origins to determine the average number of CCL3L1 gene copies in each group. They found, for example, that HIV-negative African Americans had an average of four CCL3L1 copies, whereas HIV-negative European and Hispanic Americans had two and three copies, respectively.

Using the average CCL3L1 gene copy number as a reference point for each group, the researchers found that individuals with fewer CCL3L1 copies than their population's average were more susceptible to HIV infection and rapid progression to AIDS. People with more than their population's average CCL3L1 gene copies were less prone to HIV and AIDS.

Depending on the study population, each additional CCL3L1 copy lowered the risk of acquiring HIV by 4.5%–10.5%. However, below-average CCL3L1 copy numbers were associated with a 39%–260% higher risk of rapid progression to AIDS.

The researchers also studied variations in the CCR5 gene that they had linked previously to varying rates of AIDS progression. They found that individuals with low CCL3L1 copy numbers and disease-accelerating CCR5 variants had an even higher risk of HIV acquisition and rate of progression to AIDS.

The study was reported in the January 6 issue of *Science Express,* an online publication of the journal *Science.*

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