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Molecular monitoring of chronic myelogenous leukemia

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Chronic myelogenous leukemia (CML) is a myeloproliferative neoplasm that originates in an abnormal pluripotent bone marrow stem cell and is defined by the presence of t(9;22)(q34;q11)/*BCR-ABL1* (Philadelphia chromosome) at the molecular level. The clinical course of CML is generally characterized by an initial chronic phase, an ill-defined accelerated phase, and a terminal blast phase. The accelerated and blast phases are associated with disease progression, resistance to therapy and poor prognosis. During this evolution, the neoplastic cells usually acquire additional karyotypic abnormalities, most often a second copy of Philadelphia chromosome, trisomy 8, and isochromosome17q. Tyrosine kinase inhibitors (TKIs) have become the standard target therapy for CML. While most patients with chronic phase disease achieve durable complete hematologic and cytogenetic remission, response is less stable in advanced CML. The commonest mechanism of resistance to TKIs is the development of point mutations in the *ABL1* kinase domain. Early detection of mutant clones may guide decisions regarding alternative treatment options. We describe several mechanisms of resistance for TKIs in addition to *ABL1* kinase mutations. We discuss strategies for molecular monitoring of CML patients.

Biography

C Cameron Yin has received her MD from Beijing Medical University and her PhD from the University of Wisconsin-Madison. She is currently an associate professor in the Department of Hematopathology at the University of Texas MD Anderson Cancer Center. In addition to clinical responsibilities on the Leukemia, Lymphoma and Molecular Diagnostic services, Dr. Yin has been actively participating in multiple research projects in the molecular genetic abnormalities in leukemia and lymphoma, which has led to over 100 research papers and over 20 book chapters.

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