Chronic myeloid leukemia (CML) is a clonal, myeloproliferative disorder of hematologic stem cells and accounts for 15% of adult leukemias in the United States (Jemal et al., 2007). CML progression usually occurs in three phases, including a chronic phase (CP) that most often is asymptomatic, an accelerated phase (AP), and a terminal blast phase (BP) (Sawyers, 1999). If CML is left untreated, progression from CP to BP usually occurs in three to five years (Sawyers). CML is characterized by a genetic translocation between chromosomes 9 and 22 (the Philadelphia chromosome). The translocation results in an abnormal fusion gene that encodes for the constitutively active BCR-ABL tyrosine kinase, the known causative agent underlying CML pathogenesis (Daley, Van Etten, & Baltimore, 1990; Faderl et al., 1999). The identification of this protein has made it an ideal target for therapeutic intervention.

The tyrosine kinase inhibitor (TKI) imatinib was the first BCR-ABL–targeted agent approved in 2001 for the treatment of patients with CML and has revolutionized the treatment of the disease. Unfortunately, resistance to imatinib has become a clinically significant problem that limits the long-term benefits of the drug in many patients with CML (Oestreicher, 2007a). The mechanisms that underlie imatinib resistance are multifactorial and should be considered carefully when healthcare professionals are choosing second-line treatment. This article discusses the mechanisms and identification of resistance and treatment options for when resistance occurs, as well as nursing implications.