Chronic myeloid leukemia (CML) is a malignant disease caused by genetic mutations of hematopoietic stem cells in the bone marrow (Apperley, 2015; Jabbour & Kantarjian, 2014). This form of leukemia affects about 1 individual per 100,000 per year and accounts for 15% of all new cases of leukemia in Western countries (Apperley, 2015). In Germany, about 1,200 patients develop CML annually (Robert Koch Institute, 2016). Until 2001, few therapeutic options were available, they caused numerous side effects, and they did not considerably ameliorate life expectancies (Baccarani et al., 2002; Guilhot et al., 1997). The introduction of tyrosine kinase inhibitors (TKIs) in 2001 heralded the start of targeted therapies in hematopoietic cancers because of their distinct impact on tyrosine kinase, encoded by the CML-pathognomonic BCR-ABL gene (Kris et al., 2010). At the same time, medication now could be administered orally and proved to be comparatively well tolerated (Hochhaus, 2011). However, the major breakthrough of TKIs concerned life expectancy; today, life spans of responding