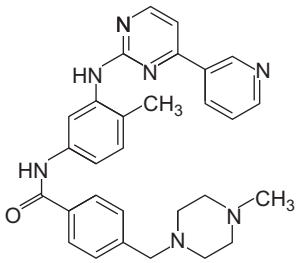
of ALL, about 20% of adult ALL cases and about 3–5% of childhood cases of ALL contain the Philadelphia chromosome.



The fact that imatinib and asparaginase are both useful drugs against ALL is revealed by the fact that both drugs have been administered to the same ALL patient, as was the case in the clinical trials of Schultz et al. (85) and Raetz et al. (86).

A chromosomal abnormality, known as the *Philadelphia chromosome*, commonly occurs in ALL. Philadelphia chromosome results from a translocation involving chromosomes 9 and 22, that is, t(9;22). As mentioned above, this abnormality is occurs in 3–5% of children and up to 20% of adults with ALL (87). The result is a fusion gene called BCR-ABL, where this fusion gene encodes a fusion protein (BCR-ABL). *BCR* means *breakpoint cluster region*, and is a gene residing on chromosome 22q11.2 (88). *ABL* means *Abelson oncogene*, and is a gene residing on chromosome 9q34. The consequence of the translocation is the BCR-ABL fusion oncogene, where expression of this gene is the BCR-ABL fusion protein.

A diagram of the event of translocation, which produces two abnormal chromosomes, is shown below (89). Collectively, both translocation products are called the *Philadelphia chromosome* (90). In translocation, the tip of the long arm of chromosome 9 is joined to the body of chromosome 22, producing the Philadelphia chromosome. Simultaneously, the distal part of the long arm of chromosome 22 is joined to the body of chromosome 9. Philadelphia chromosome contains a fusion gene that consists of the amino part of *BCR* and the carboxyl portion of ABL.

Where the patient's leukemic cells contain Philadelphia chromosome, optimal treatment requires administering a tyrosine kinase inhibitor, such as imatinib (91,92). The decision to administer imatinib is based on the diagnostic test revealing the presence of Philadelphia chromosome (Fig. 18.2).

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