Introduction

Acute lymphoblastic leukemia (ALL), the most common subtype of childhood cancer is associated with the presence of chromosomal translocations allowing the identification of prognostically relevant subgroups ***(Marin et al., 2001)***.

Many different types of chromosomal translocations have been found in patients with acute leukemia, but only a few translocations have been frequently observed ***(Osumi et al., 2002)***. ALL is frequently associated with chromosomal translocations t(9; 22)(BCR/ABL), t(4; 11)(MLL/AF4), t(1; 19)(E2A/PBX1), and t(12; 21)(TEL/AML1) which are used to risk stratify patients in most large clinical trials ***(Nasedkina et al., 2003)***. These recurrent chromosomal translocations, which typically lead to the formation of fusion genes and their expression as chimeric transcripts and proteins, are of relevance in leukemogenesis because they influence the expression of the key regulatory genes in the hematopoietic system. It is now generally accepted that these four chromosomal translocations have therapeutic importance for risk assessment in current multiagent chemotherapy protocols, because they are predictors of prognosis ***(Siraj et al., 2002)***.

The identification of these chromosomal translocations is thus important no only for understanding the biology of ALL but also as a guide for patient management. The recognition of them has allowed precursor ALL to be subdivided into molecular entities in the recently proposed WHO classification.

The presence of t(12; 21) is associated with good response and detected in 20-25% of pediatric ALL. The presence of t(1; 19) is observed in 5-6% of ALL but constitutes 25% of pre- B subgroup and

predicts intermediate response to therapy. The other 2 translocations t(4; 11) and t(9; 22) are each observed in 5-6% , and both associated with poor prognosis, but the first occurs mostly in infant patients ***(Siraj et al., 2002)***.

The detection of chromosomal translocations by conventional cytogenetic method is not always possible because of lack of adequate metaphase, poor chromosome morphology or presence of cryptic translocation, which is only visible by using the new molecular techniques. In addition, it is time-consuming and labor-intensive method with relatively low sensitivity. Fluorescence in Situe Hybridization (FISH), Southern blotting, and RT-PCR strategies can detect these translocations. Moreover, FISH and PCR can detect minimal residual disease (MRD). However, FISH is not a very efficient technique for simultaneous detection of several different translocations. In addition it is less sensitive than PCR-based approaches ***(Nasedkina et al., 2003)***.

These considerations together with the recent availability of drugs that specifically target fusion proteins or their activity, highlight the need for a rapid and accurate identification of genetic lesions in patients with acute leukemia. In this respect, ***Pallisgaard et al., (1998)*** proposed a multiplex RT-PCR system that allows for simultaneous detection of 29 fusion genes and more than 80 breakpoints and splice variants in patients with acute leukemia. These results would be extremely laborious and time consuming to obtain by either FISH or single RT-PCR.