# Introduction

Acute lymphoblastic leukaemias are characterized by clonal proliferation, accumulation and tissue infiltration by neoplastic cells. They are mainly regarded as childhood diseases, with an early incidence peak at 2 to 5 years of age. The age adjusted incidence of ALL in adults amounts to about one third of that in children. However, ALL has a bimodal distribution, with a second peak around old age and a low but steady rise in incidence with increasing age ***(Faderl et al., 1998).***

A number of medications, or chemotherapy agents are known to be effective against ALL. However, the best combination of medicines or the best treatment schedule is still not known. Because there are so many different medicines, dosing schedules and combinations , regimens can also vary based on characteristics such as the age of patient, total number of white blood cells, or certain genetic factors found in some patients with ALL ***(Linker et al., 2002).***

Sixty to eighty percent (60-80%) of adults with acute lymphoblastic leukaemia (ALL) can be expected to attain complete remission status following appropriate induction therapy. Approximately 35% to 40% of adults with ALL can be expected to survive 2 years with aggressive induction combination chemotherapy and effective supportive care during induction therapy. A few studies that use intensive multiagent approaches suggest that a 50% 3-year survival is achievable in selected patients but, these results must be verified by other investigations ***(Zhang et al., 2002).***

The majority of cases of ALL demonstrate an abnormal karyo type, either in chromosome number (ploidy) or as structureal changes such as translocation, inversion or deletion ***(Gleissner and Thiel, 2003).***

BCR-ABL fusion gene is the molecular equivalent of the Philadelphia translocation. The Philadelphia chromosome is the most frequent genetic abnormality known in human leukaemia. It can be detected in 20-40% of adults with ALL and has been confirmed as the most important negative prognostic factor ***(Gleissner et al.,2002)*** with a correlation to older age and higher leucocyte counts, as well as an extremely low proability of long term survival after conventional chemotherapy ***(Dombret et al., 2002).*** In this reciprocal translocation, referred to as t(9;22) (q34;q11), the ABL proto-oncogene from chromosome 9 is brought under the control of the 5` portion of BCR sequences on chromosome 22. Fusion is performed in a head-to-tail fashion and creates the BCR-ABL fusion gene ***(Annino et al., 2002).***