Many agents and mechanisms have been implicated in the genesis of malignant tumours as a result of intensive research in this area. A chromosomal basis for malignant transformation was suggested in 1914 by Boveri, while Bauer (1928) was the first to elaborate fully a more inclusive somatic mutation theory. In 1963, Burch proposed that for the induction of a malignant neoplastic growth four specific mutations are needed, which may include not only point mutations but also detectable chromosomal aberrations. He added that some of these mutations may be inherited while the others may be acquired during life. The observations that ionising radiations, certain chemicals and some viruses
are able to produce both mutations and malignant transformations do not contradict Burch's hypothesis. Sandberg and Yamada (1965) reported that the genesis of cancer cells may be a process dependent on modified DNA structure and function, which may or may not be microscopically detectable. Thus, the chromosomal aberrations are found in the genetic material which are large enough to be detected by the usual microscopic examination, while the absence of detectable chromosomal aberrations will not contradict the mutation theory of carcinogenesis and their presence may contribute to our understanding of the dynamics of malignant transformation and growth.

Direct evidence concerning the cause of a neoplastic transformation which may have occurred in a single cell is extremely difficult to obtain because by the time a tumour has reached a stage where clinical diagnosis is possible, the neoplastic cells have undergone numerous divisions and in any one of these, a change in the chromosome constitution might have occurred. In the altered tumour environment a change in the karyotype may confer a selective advantage on the chromosomally distinct cell line. The development of a primary neoplasm can, therefore, be divided into 2 phases: first the
neoplastic transformation resulting in a cell capable of unlimited growth and possessing invasive properties, second the progression of a tumour from a single cell to a multicellular tissue, the cells of which very frequently contain a chromosomal abnormality. Makino (1957) put forward for the first time the stem line theory of tumour progression in which the basic or stem line in a tumour is clonal in origin and contains a particular but constant chromosomal abnormality. From this stem line, cells containing other chromosome complements arise as a result of abnormal divisions. These may, if they have sufficient selective advantage, overgrow the original stem line and become the basic stem line for the tumour. On this hypothesis, the tumour is considered as a natural population of cells in which the chromosome abnormalities interact with selective forces and may be eliminated or favoured. An in vitro parallel is the transformation from a senile degenerating diploid cell strain with a finite life span to a heteroploid cell line with an infinite life span. A study of this process and in particular the loss of contact inhibition (Abercrombie and Heaysman, 1954; Abercrombie, 1962; Levine et al., 1967) which prevents the overgrowth of diploid strain might throw a considerable light on
neoplastic transformation which occurs in vivo.

Cytogenetic findings in malignant tumours can become of diagnostic significance and may help in choosing appropriate therapeutic measures. They may also contribute to the evaluation of these measures and the possibility to pinpoint high susceptibility cases.

As a result of standardization of improved techniques a large number of reports have come in and a relating specific chromosomal abnormality (Philadelphia chromosome) was discovered in chronic myeloid leukemia (Dowell and Hungerford, 1960a) and investigations of possible chromosomal anomalies in a variety of haematological disorders, particularly leukemia and allied diseases have been made with renewed interest. The consistent appearance of Ph' chromosome in a great majority of cases of chronic myeloid leukemia has since been confirmed by various workers (Baikie et al., 1960a; Speed and Lawler, 1964 and so on). This consistency and specificity of Ph' chromosome in chronic myeloid leukemia have imparted more than a diagnostic significance to it.

Suns et al. (1962) described a deletion of the short arm of one chromosome in 821 pair and designated this as Christchurch chromosome in chronic lymphocytic leukemia cases.
But this finding has so far been limited to only a few instances and so it still remains to evaluate the role of chromosomes in chronic lymphocytic leukemia.

In contrast to the chronic leukemias, the chromosomal findings in acute leukemias have been inconsistent and variable remarkably. Chromosomal aberrations in acute leukemias have been variably characterized by aneuploidy (hypo and hyper diploidy), pseudodiploidy, abnormal karyotype and a few instances of structural abnormalities such as deletion, translocation, chromatid breaks and marker chromosomes. The diversity of chromosomal changes in acute leukemias are reflected in the findings that chromosomes involved in these include almost all the groups. These have consequently offered no diagnostic aid but the genetic significance remains to be evaluated.

So the interpretation of chromosomal abnormalities in leukemia remains open to discussion. It has been postulated that chromosomal aberrations represent genetic changes causally related to the genesis of these disorders. The chromosomal changes, morphological and phenotypical and changes in the nuclear structure as well may be incidental to the development of leukemia.
Inspired by these facts, it was decided to investigate the karyotypes in different kinds of leukemia to evaluate the genetic significance of chromosomal changes.