

**DETECTION OF BCR-ABL GENE AND ITS
EXPRESSED PROTEIN (P210) IN PERIPHERAL
BLOOD CELLS OF DIFFERENT PHASES OF CML**

Thesis

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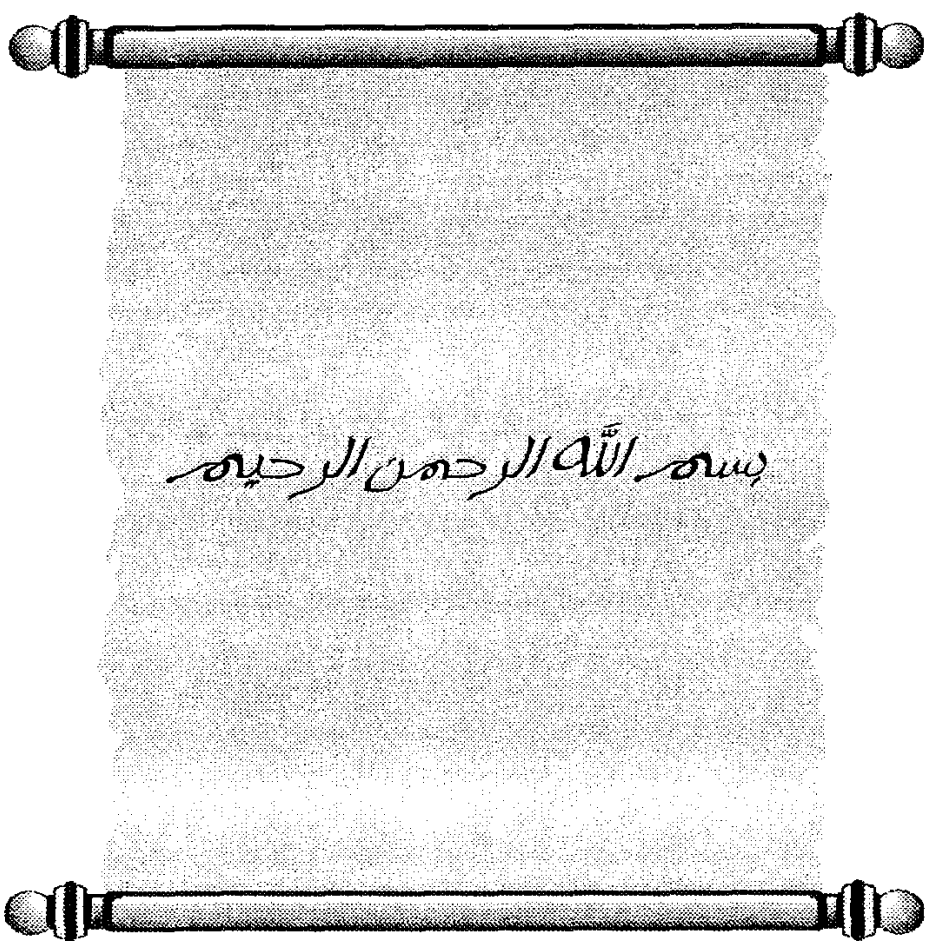
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LIST OF ABBREVIATIONS

<i>A</i>	Adenine
<i>ABL</i>	Abelson Leukemia
<i>ALL</i>	Acute Lymphoblastic Leukemia
<i>AML</i>	Acute Myeloblastic Leukemia
<i>APS</i>	Amonium Persulphate
<i>bcr</i>	Breakpoint Cluster Region
<i>Mbcr</i>	Major Breakpoint Cluster Region
<i>mbcr</i>	Minor Breakpoint Cluster Region
<i>μbcr</i>	Mu Breakpoint Cluster Region
<i>BCR</i>	BCR gene
<i>BM</i>	Bone Marrow
<i>BMT</i>	Bone Marrow Transplantation
<i>BSA</i>	Bovine Serum Albumin
<i>C</i>	Cytosine
<i>CBC</i>	Complete Blood Count
<i>cDNA</i>	Complementary Deoxyribonucleic acid
<i>CFU-E</i>	Colony Forming Unit-Erythroid
<i>CML</i>	Chronic Myelogenous Leukemia
<i>CML-N</i>	Chronic Neutrophilic Leukemia
<i>CMML</i>	Chronic Myelomonocytic Leukemia
<i>CSF</i>	Colony Stimulating Factor
<i>DNA</i>	Deoxyribonucleic acid
<i>dsDNA</i>	Double Starnded Deoxyribonucleic acid
<i>ssDNA</i>	Single Stranded Deoxyribonucleic acid
<i>EDTA</i>	Ethyline Diamine Tetra-acetic Acid
<i>EL</i>	Eosinophilic Leukemia
<i>FACS</i>	Fluoresence Activated Cell Sorting
<i>FISH</i>	Fluorescence in Situ Hybridization
<i>G</i>	Guanine
<i>G6PD</i>	Glucose 6 Phospharte Dehydrogenase

<i>J-CML</i>	Juvenile Chronic Myeloid Leukemia
<i>kb</i>	Kilobase
<i>Kd</i>	Kilo Dalton
<i>LM</i>	Light Microscope
<i>MUD</i>	Matched Unrelated Donor
<i>PB</i>	Peripheral Blood
<i>PBS</i>	Phosphate Buffered Saline
<i>Ph`</i>	Philadelphia
<i>PSCT</i>	Potential Stem Cell Transplantation
<i>RBCs</i>	Red Blood Cells
<i>RNA</i>	Ribonucleic Acid
<i>mRNA</i>	Messenger Ribonucleic Acid
<i>Rt-PCR</i>	Reverse Transcriptase Polymerase Chain Reaction
<i>T</i>	Tyrosine
<i>U</i>	Uracil
<i>WBCs</i>	White Blood Cells

ABSTRACT

Chronic myelogenous leukemia (CML) is a disease characterized by the presence of a unique molecular marker; i.e., BCR/ABL fusion gene and its mRNA (*Santini et al., 1996*).

The hallmark of the disease is the Ph⁺ chromosome which is generated by reciprocal translocation of 9,22 fusing the ABL oncogene of q arm of chromosome 9 to the breakpoint cluster region within the BCR gene on chromosome 22. The BCR/ABL gene is transcribed into a BCR/ABL specific mRNA that is translated into P210 kd protein possessing enhanced tyrosine kinase activity as compared to that of normal protein (*Melo, 1996*).

A Western blotting as well as dot blot assays for the BCR/ABL fusion protein of circulating WBCs, in addition to Rt-PCR amplification for BCR/ABL gene were performed for 27/33 patients in different phases of the Ph⁺ positive CML patients to assess their clinical utility in diagnosis and monitoring the disease.

Forty five blood samples were collected from 33 patients in different phases of the disease, at different times in addition to two control samples from patients with high total leukocytic count. However, only 27 of them gave a reproducible results.

We observed that, all studied cases 27/27 showed a positive result by dot blot assay in the form of bluish colored dots with different intensities corresponding to different phases of the disease.

On the other hand, Western blotting assay for chronic phase patients revealed a major band for
