Commentary

# Mutated forms of BCR-ABL gene

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Multiple regulatory mechanisms tightly controlled important cellular functions like cell cycling. To destroy the network of cellular control mechanisms, in cancer disease multiple genetic events are needed. After intensive studies of these events, many proteins playing major roles in cancer disease are identified. In an increasing number of diseases this data has led to development of medicine targeting proteins that are believed to represent the important explanation for cancer disease.

**Key Words:** *Cancer disease; Chronic myelogenous leukemia cells; Somatic cell transplantation*

## DESCRIPTION

Chronic myelogenous leukemia was the primary malignancy shown to be related to a selected cytogenetic lesion, the Philadelphia chromosomal translocation .Another 10 years went before it had been shown that the proto-oncogene Abl, normally found on chromosome 9, was translocated to the Philadelphia chromosome in CML cells. We now know that the foremost important consequence of the Philadelphia chromosome consists within the fusion of the Bcr gene to the tyrosine kinase Abl, encoding the constitutive active fusion protein Bcr-Abl, which is meant to induce all disease features of CML.

Initially, CML may be a slowly progressive disease with symptoms that sometimes develop gradually. There are three distinct clinical phases the disease progresses through - blast crisis, chronic and accelerated phase, during which the leukemic clone progressively loses its ability to differentiate. By accumulation of peripheral blood and extramedullary sites, myeloid precursors and mature cells in bone marrow, the chronic phase is characterized. Most patients are diagnosed within the chronic phase. During the chronic phase of the disease, there's massive clonal expansion of myeloid cells, which retain the power to differentiate. These are so-called accelerated phase, because these phases can last for several years before symptoms and signs of more aggressive disease appear. Within the accelerated phase the control of the proliferation becomes far more difficult. Counts rise to high levels, and therefore the risk for tissue infiltration by white blood cells are present.

Untreated the disease is fatal after a variable period of your time. Until the late nineties interferon together with cytosar, a cytostatic, has been the treatment of choice. Around 5-6 years a patient survives in average during this treatment. they typically died after a change from a chronic phase to accelerated phase or blast crisis, a terminal disease stage very almost like acute leukaemia.Transition to blast crisis is that the unavoidable outcome of CML except of patients receiving allogenic bone marrow transplantation early within the chronic phase. .

## CONCLUSION

Allogenic bone marrow or somatic cell transplantation remains the sole treatment known to cure CML. This approach is out there only to patients who have an appropriate donor and who are young enough to tolerate the procedure and therefore the subsequent toxic effect of allogenic. The goal of therapy for CML within the chronic phase is to prolong survival and minimize symptoms by achieving complete hematologic response and an entire cytogenetic response.

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