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**Monitoring treatment failure to imatinib using hsa-miR-652-3p in Chronic Myeloid Leukaemia**

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**ABSTRACT**

Chronic Myeloid Leukaemia (CML) is a myeloproliferative neoplasm with Philadelphia chromosome as the cytogenetic hallmark of the disease. It is the results of reciprocal translocation between the long arms of chromosome 9 and 22. CML is treated with tyrosine kinase inhibitor (TKI), however, failure to treatment occurs in 20% of patients. Micro RNAs (miRNAs), short non-coding RNA function as regulators of transcription and are widely studied in cancers. Thus in this study we looked at miRNA that could be linked to treatment failure in non-responder towards imatinib, a TKI. Peripheral blood (2.5ml) from adult participants namely CML patient not responding to imatinib, CML patient responding to imatinib and normal control were collected. MiRNAs were then purified using PAXgene® Blood miRNA Kit (PreAnalytiX). cDNA libraries were constructed using Illumina® TruSeq Small RNA kit (illumina) and ran on illumina Miseq, a Next Generation Sequencing instrument followed by alignment using BaseSpace (illumina). Hsa-miR-652-3p was expressed near four times more in CML patient not responding to imatinib treatment than normal control whereby almost the same in CML patient responding to imatinib treatment. These indicated increasing level of hsa-miR-652-3p represents the rising level of BCR-ABL1 transcripts and could be used as an indicator for monitoring treatment failure to imatinib in CML.