



## ampli set BCR-ABL<sup>CE IVD</sup>

45 tests

cat 1403

detection of translocation t(9;22)

PCR analysis of fusion genes is based on the design of oligonucleotide primers at opposite sides of the breakpoint fusion regions so that the PCR product contains the tumour specific fusion sequences. The fusion gene is transcribed into fusion mRNA, which can serve as the PCR target after reverse-transcription (RT) into cDNA.

The Philadelphia chromosome (Ph), besides being the hallmark of CML, also occurs in approximately 2-10% of childhood ALL and in 20-50% of adult ALL with an incidence progressively increasing with age. Ph translocation always results in the joining of 3' sequences of the tyrosine kinase c-ABL proto-oncogene on chromosome 9 to the 5' sequences of the BCR gene on chromosome 22. The result of this translocation is the formation of hybrid c-abl mRNA and of a hybrid protein which amino acid sequence overlaps a part of the bcr gene in the N-terminus and a part of the abl gene in the C-terminus.

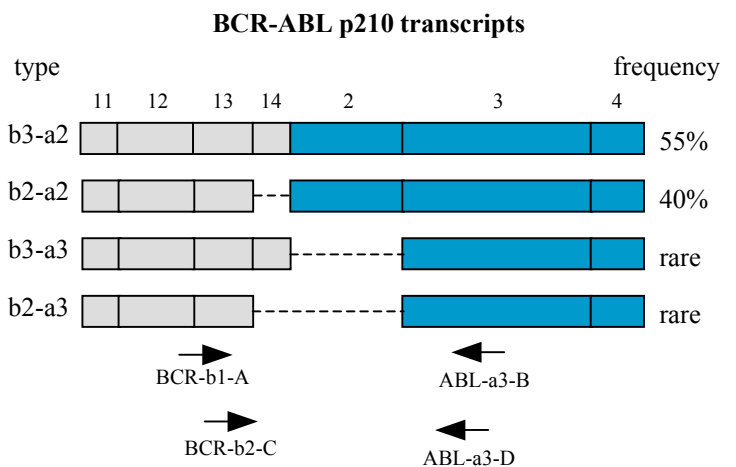
In CML the fusion transcripts encode a BCR-ABL protein of 210 Kda, called p210BCR/ABL. Approximately 40% of Ph+ ALL show the same molecular rearrangements as in CML; the remaining 60% of Ph+ ALL show a BCR/ABL protein of 190Kda, called p190BCR/ABL. Polymerase Chain Reaction (RT-PCR) of fusion genes is based on the design of oligonucleotide primers at opposite sides of the breakpoint fusion regions so that the PCR product contains the tumour specific fusion sequences.

**Principle of method:** A) extraction of genomic DNA; B) reverse transcription; C) amplification; C) detection on agarose gel

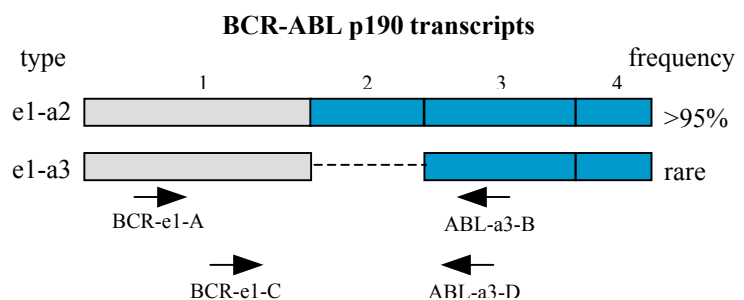
**Applicability:** on extracted and purified RNA

### ANALYSIS OF RESULTS

BCR-ABL p210	PCR product Size (bp)	
	I PCR	Nested PCR
p210 b3-a2	417	360
p210 b2-a2	342	285
p210 b3-a3	243	186
p210 b2-a3	168	111



BCR-ABL p190	PCR product Size (bp)	
	I PCR	I PCR
p190 e1-a2	521	381
p190 e1-a3	347	207



The size of the PCR products could be different due to variable breakpoint positions in the genes

### REFERENCES

*Leukemia* 13:1901-38 (1999) *Leukemia* 5:448-51 (1991)