

Conclusion: We describe here the molecular details of how signals from an oncogenic tyrosine kinase become converted into changes in gene expression that define the B-lineage leukaemia subtype Ph+B-ALL. Our work highlights the complexity of these changes by acknowledging the role of gene regulatory elements/enhancers in this process and further describe for the first time that B-ALL subtypes can be distinguished by their enhancer usage and promoter-enhancer interactions. Uncovering the precise enhancer regions for the genes deregulated in Ph+B-ALL and the TFs recruited to them may open new windows for therapeutic intervention.

Disclosures No relevant conflicts of interest to declare.

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