

NUP214-ABL1 fusion defines a rare subtype of B-cell precursor acute lymphoblastic leukemia that could benefit from tyrosine kinase inhibitors

Nicolas Duployez,^{1,2*} Guillaume Grzych,^{1*} Benoît Duourneau,¹ Martin Alarcon Fuentes,³ Nathalie Grardel,¹ Thomas Boyer,^{1,2} Wadih Abou Chahla,³ Bénédicte Bruno,³ Brigitte Nelken,³ Emmanuelle Clappier,⁴ and Claude Preudhomme^{1,2}

*These authors equally contributed to this work.

¹CHU Lille, Biology and Pathology Center, Laboratory of Hematology, Lille; ²INSERM UMR-S 1172, Cancer Research Institute, Lille; ³CHU Lille, Jeanne de Flandre Hospital, Pediatric Hematology Department, Lille; ⁴Assistance Publique-Hôpitaux de Paris, Saint Louis Hospital, Laboratory of Hematology, Paris, France

Correspondence: nicolas.duployez@chru-lille.fr

doi:10.3324/haematol.2015.136499

Supplemental data

***NUP214-ABL1* fusion defines a rare subtype of B-cell precursor acute lymphoblastic leukaemia that could benefit from tyrosine kinase inhibitors**

Duployez N et al.

Supplementary table 1: Copy-number abnormalities found by single nucleotide polymorphism array. For each aberration, chromosome location, size, copy number state and genes involved are indicated.

Type	Chr.	Start	End	Size (kbp)	Cytoband Start	Cytoband End	CN State	Genes
Loss	1	112007833	112161863	154,03	p13.2	p13.2	1.0	<i>C1orf162, ADORA3, LOC100129269</i>
Loss	3	176777998	176912485	134,487	q26.32	q26.32	1.0	<i>TBL1XR1</i>
Loss	4	109013575	109098953	85,378	q25	q25	1.0	<i>LEF1</i>
Loss	7	50143715	50346953	203,238	p12.2	p12.2	1.0	<i>C7orf72, IKZF1</i>
Loss	7	50411817	50482012	70,195	p12.2	p12.2	1.0	<i>IKZF1</i>
Loss	9	21968561	22027094	58,533	p21.3	p21.3	1.0	<i>CDKN2A, CDKN2B</i>
Gain	9	133725766	134098448	372,682	q34.12	q34.13	3.0	<i>ABL1, QRFP, FIBCD1, LAMC3, AIF1L, NUP214</i>
Loss	12	116718607	117108280	389,673	q24.21	q24.22	1.0	<i>MIR4472-2, LINC00173, MAP1LC3B2</i>
Loss	13	44849818	44981036	131,218	q14.11	q14.11	1.0	<i>SERP2</i>
								<i>BCL7C, MIR762, CTF1, FBXL19, ORAI3, SETD1A, HSD3B7, STX1B, STX4, ZNF668, ZNF646, PRSS53, VKORC1, BCKDK, KAT8, PRSS8, PRSS36, FUS, PYCARD, TRIM72, PYDC1, ITGAM, ITGAX, ITGAD, COX6A2, ZNF843, ARMC5, TGFB1I1, SLC5A2, C16orf58, AHSP, CSDAP1, KIAA0664L3, ZNF720</i>
Loss	16	30891487	31878719	987,232	p11.2	p11.2	1.0	<i>KAT8, PRSS8, PRSS36, FUS, PYCARD, TRIM72, PYDC1, ITGAM, ITGAX, ITGAD, COX6A2, ZNF843, ARMC5, TGFB1I1, SLC5A2, C16orf58, AHSP, CSDAP1, KIAA0664L3, ZNF720</i>
Loss	20	10392115	10472593	80.478	p12.2	p12.2	1.0	<i>MKKS, C20orf94</i>
Loss	21	36284722	36415877	131,155	q22.12	q22.12	1.0	<i>RUNX1, RUNX1-IT1</i>
Loss	22	22388119	22603418	215,299	q11.22	q11.22	1.0	<i>VPREB1</i>

Supplementary Figure 1: Minimal residual disease monitoring in bone marrow as a function of time by real-time genomic quantification of the IgH rearrangement. Therapeutic interventions with the molecules used are indicated at the top of the graph. CR: complete remission; BMT: bone marrow transplantation; DLI: donor lymphocyte infusion.

