

SUPPLEMENTAL TABLES

Supplemental Table 1. List of DNA gene mutations tested in diagnostic tumor samples of our ALL cohort

OncoPlus assay alone (17 genes)	Shared between OncoPlus and Foundation One Heme assays (161 genes)						
<i>CBLB</i>	<i>ABL1</i>	<i>CCND3</i>	<i>EPHA3</i>	<i>IKZF1</i>	<i>MYC</i>	<i>PTCH1</i>	<i>STK11</i>
<i>DDX41</i>	<i>AKT1</i>	<i>CCNE1</i>	<i>EPHA5</i>	<i>IL7R</i>	<i>MYCN</i>	<i>PTEN</i>	<i>TCF3</i>
<i>DICER1</i>	<i>ALK</i>	<i>CD274</i>	<i>ERBB2</i>	<i>JAK1</i>	<i>MYD88</i>	<i>PTPN11</i>	<i>TET2</i>
<i>ELOC</i>	<i>APC</i>	<i>CDH1</i>	<i>ERBB3</i>	<i>JAK2</i>	<i>NF1</i>	<i>RAD21</i>	<i>TP53</i>
<i>ERCC3</i>	<i>ARID1A</i>	<i>CDK12</i>	<i>ERBB4</i>	<i>JAK3</i>	<i>NF2</i>	<i>RAD50</i>	<i>TSC1</i>
<i>FAT3</i>	<i>ARID2</i>	<i>CDK4</i>	<i>ESR1</i>	<i>KMT2D</i>	<i>NFE2L2</i>	<i>RAD51</i>	<i>TSC2</i>
<i>FH</i>	<i>ASXL1</i>	<i>CDK6</i>	<i>ETV6</i>	<i>KDM6A</i>	<i>NOTCH1</i>	<i>RB1</i>	<i>U2AF1</i>
<i>H3-3A</i>	<i>ATM</i>	<i>CDKN2A</i>	<i>EZH2</i>	<i>KDR</i>	<i>NOTCH2</i>	<i>RET</i>	<i>VHL</i>
<i>H3C2</i>	<i>ATR</i>	<i>CEBPA</i>	<i>FANCA</i>	<i>KEAP1</i>	<i>NPM1</i>	<i>ROS1</i>	<i>WT1</i>
<i>H3C3</i>	<i>ATRX</i>	<i>CHEK1</i>	<i>FBXW7</i>	<i>KIT</i>	<i>NRAS</i>	<i>RUNX1</i>	<i>ZNF384</i>
<i>ITPKB</i>	<i>AXL</i>	<i>CHEK2</i>	<i>FGFR1</i>	<i>KMT2A</i>	<i>NTRK1</i>	<i>SDHB</i>	<i>ZRSR2</i>
<i>MLH3</i>	<i>B2M</i>	<i>CREBBP</i>	<i>FGFR2</i>	<i>KRAS</i>	<i>NTRK2</i>	<i>SDHC</i>	
<i>NBN</i>	<i>BAP1</i>	<i>CSF1R</i>	<i>FGFR3</i>	<i>MAP2K1</i>	<i>NTRK3</i>	<i>SDHD</i>	
<i>PIK3CB</i>	<i>BCOR</i>	<i>CSF3R</i>	<i>FLT3</i>	<i>MAPK1</i>	<i>NUP98</i>	<i>SETBP1</i>	
<i>POLE</i>	<i>BCORL1</i>	<i>CTCF</i>	<i>FOXL2</i>	<i>MDM2</i>	<i>PALB2</i>	<i>SETD2</i>	
<i>TERT</i>	<i>BIRC3</i>	<i>CTNNA1</i>	<i>GATA1</i>	<i>MEF2C</i>	<i>PAX5</i>	<i>SF3B1</i>	
<i>TRAF7</i>	<i>BLM</i>	<i>CTNNB1</i>	<i>GATA2</i>	<i>MEN1</i>	<i>PBRM1</i>	<i>SMAD4</i>	
	<i>BRAF</i>	<i>CUX1</i>	<i>GNA11</i>	<i>MET</i>	<i>PDGFRA</i>	<i>SMARCB1</i>	
	<i>BRCA1</i>	<i>CXCR4</i>	<i>GNAQ</i>	<i>MLH1</i>	<i>PDGFRB</i>	<i>SMC1A</i>	
	<i>BRCA2</i>	<i>DAXX</i>	<i>GNAS</i>	<i>MPL</i>	<i>PHF6</i>	<i>SMC3</i>	
	<i>BTK</i>	<i>DDR2</i>	<i>GRIN2A</i>	<i>MRE11</i>	<i>PIK3CA</i>	<i>SMO</i>	
	<i>CALR</i>	<i>DDX3X</i>	<i>HNF1A</i>	<i>MSH2</i>	<i>PIK3R1</i>	<i>SRSF2</i>	
	<i>CBL</i>	<i>DNMT3A</i>	<i>HRAS</i>	<i>MSH6</i>	<i>PLCG2</i>	<i>STAG2</i>	
	<i>CCND1</i>	<i>EGFR</i>	<i>IDH1</i>	<i>MTOR</i>	<i>POT1</i>	<i>STAT3</i>	
	<i>CCND2</i>	<i>EP300</i>	<i>IDH2</i>	<i>MUTYH</i>	<i>PPP2R1A</i>	<i>STAT5B</i>	

Both OncoPlus and Foundation Heme assays are designed to detect somatic mutations in tumor samples. However, these panels also include genes that are frequently mutated in hereditary hematopoietic malignancies. When patients were found to have a variant suspicious for germline mutation, they were further evaluated with skin biopsy and/or serial NGS analysis at the time of remission (see Supplemental Figure 4).

Supplemental Table 2. List of 80 genes with RNA sequencing coverage

<i>ABL1</i>	<i>EP300</i>	<i>JAK3</i>	<i>PAX5</i>
<i>ABL2</i>	<i>EPOR</i>	<i>KMT2A</i>	<i>PCM1</i>
<i>ALK</i>	<i>ERBB2</i>	<i>LMO1</i>	<i>PDGFRA</i>
<i>BCL11A</i>	<i>ERG</i>	<i>LMO2</i>	<i>PDGFRB</i>
<i>BCL11B</i>	<i>ETV1</i>	<i>LYL1</i>	<i>PICALM</i>
<i>BCL2</i>	<i>ETV6</i>	<i>MLLT1</i>	<i>RET</i>
<i>BCL6</i>	<i>EWSR1</i>	<i>MLLT10</i>	<i>ROS1</i>
<i>BCR</i>	<i>FGFR1</i>	<i>MLLT3</i>	<i>RUNX1</i>
<i>BTG1</i>	<i>FGFR2</i>	<i>MLLT4</i>	<i>SNX29</i>
<i>CIITA</i>	<i>FGFR3</i>	<i>MLLT6</i>	<i>TAL1</i>
<i>COL1A1</i>	<i>HOXA11</i>	<i>MYB</i>	<i>TAL2</i>
<i>CREBBP</i>	<i>HOXA13</i>	<i>MYC</i>	<i>TCF3</i>
<i>CRLF2</i>	<i>HOXA3</i>	<i>NCOA2</i>	<i>TCL1A</i>
<i>CSF1R</i>	<i>HOXA9</i>	<i>NOTCH1</i>	<i>TEC</i>
<i>DEK</i>	<i>IGH</i>	<i>NTRK1</i>	<i>TFE3</i>
<i>DUSP22</i>	<i>IGK</i>	<i>NTRK2</i>	<i>TFEB</i>
<i>EGFR</i>	<i>IGL</i>	<i>NTRK3</i>	<i>TLX1</i>
<i>ELL</i>	<i>IL3</i>	<i>NUP214</i>	<i>TLX3</i>
<i>ELN</i>	<i>JAK1</i>	<i>NUP98</i>	<i>WHSC1</i>
<i>EML4</i>	<i>JAK2</i>	<i>P2RY8</i>	<i>ZNF384</i>

Fusions involving these genes were detected with the RNA sequencing analysis performed at the University of Chicago and Moffitt Cancer Center (uses Foundation One Heme Assay).

Supplemental Table 3. Demographics of 400 adult ALL cases

	B-ALL (n= 329)	T-ALL (n= 57)	ETP-ALL (n= 14)
Age at diagnosis, median (range)	49 (18 – 84)	38 (18 – 88)	31.5 (18 – 72)
Age group, n (%)			
<40 yrs	122 (37.1)	30 (52.6)	9 (64.3)
≥40 yrs	207 (62.9)	27 (47.4)	5 (35.7)
Gender, n (%)			
Female	162 (49.2)	16 (28.1)	5 (35.7)
Male	167 (50.8)	41 (71.9)	9 (64.3)
Race/ethnicity, n (%)			
Non-Hispanic White	65 (19.8)	11 (19.3)	4 (28.6)
Hispanic	29 (8.8)	1 (1.8)	1 (7.1)
Non-Hispanic Black	15 (4.6)	5 (8.8)	4 (28.6)
Asian	5 (1.5)	0	1 (7.1)
Unknown	215 (65.3)	40 (70.1)	4 (28.6)
Therapy-related ALL, n (%)			
Yes	45 (13.7)	2 (3.5)	1 (7.1)
No	248 (75.4)	51 (89.5)	13 (92.9)
Unknown	36 (10.9)	4 (7)	0
Myeloid mutations, n (%)			
<i>TP53</i> -mutated ALL	60 (18.2)	9 (15.8)	1 (7.1)
non- <i>TP53</i> myeloid CH mutations	44 (13.4)	13 (22.8)	6 (42.9)
No myeloid CH mutations	225 (68.4)	35 (61.4)	7 (50)

CH, clonal hematopoiesis

Supplemental Table 4. List of first-line (initial) therapies in Ph-positive B-ALL, Ph-negative B-ALL, and T-lineage ALL patients

	Ph+ B-ALL (n= 94)	Ph-neg B-ALL (n= 235)	T-lineage ALL (n= 71)
Hyper-CVAD + TKI	43 (45.7)	–	–
A10701	21 (22.3)	–	–
Other chemotherapy + TKI	20 (21.3)	–	–
A10403 + TKI	5 (5.3)	–	–
Inotuzumab + TKI	4 (4.3)	–	–
Blinatumomab + TKI	1 (1.1)	–	–
A10403-based	–	97 (41.3)	33 (46.5)
Hyper-CVAD	–	57 (24.3)	14 (19.7)
Other chemotherapy	–	34 (14.5)	5 (7.1)
Inotuzumab and/or blinatumomab	–	11 (4.7)	–
A41501	–	10 (4.2)	–
Unknown	–	26 (11)	10 (14.1)
ALL 0434	–	–	9 (12.6)

TKI (tyrosine kinase inhibitor) therapy included dasatinib (n=87) or ponatinib (n= 7), only for patients with Ph+ B-ALL.

Hyper-CVAD regimen involves part A (cyclophosphamide, vincristine, adriamycin, dexamethasone) and part B (high-dose methotrexate, cytarabine) given in alternating cycles(1).

A10701 regimen consists of induction therapy with dasatinib and dexamethasone, followed by allogeneic hematopoietic cell transplant (HCT), autologous HCT or chemotherapy(2).

A10403 regimen is a pediatric-inspired protocol that consists of four blocks of intensive therapy (induction, consolidation, interim maintenance, delayed intensification) followed by 2-years of maintenance therapy for patients age <50 years(3). At University of Chicago, older patients (age >50 years) were also treated with modified A10403 (reduced-doses of asparaginase and prednisone)(4) at physician discretion.

A41501 regimen incorporates inotuzumab to the A10403 backbone.

ALL 0434 regimen incorporates nelarabine to the A10403-based regimen for patients with T-lineage ALL(5).

Other chemotherapy group included chemotherapy regimens outside of what is described above, namely CVD (cyclophosphamide, vincristine, dexamethasone)(6), mini-hyperCVD with venetoclax(7), European Working Group on Adult ALL (EWALL) regimen or other lower intensity chemotherapy regimens(8).

Supplemental Table 5. Baseline gene rearrangements and structural variants of 329 molecularly characterized adult B-ALL patients

B-ALL subtype per WHO22 and ICC	Number of patients (%)	Gene rearrangements (number of patients)	Myeloid gene mutations (number of patients)
<i>BCR::ABL1</i>	94 (28.6)	<i>BCR::ABL1</i> (94) Other: <i>PAX5::MLLT3</i> (2), <i>IGH::REREP3</i> (2), <i>IGH::IRF8</i> (2), <i>IGH::CEBPD</i> (1), <i>P2RYX-CD99</i> (1), <i>ARHGAP26::NR3C1</i> (1), high hyperdiploidy (6)	<i>ASXL1</i> (6), <i>RUNX1</i> (6), <i>TET2</i> (4), <i>DNMT3A</i> (4), <i>CUX1</i> (3), <i>BCORL1</i> (2), <i>SRSF2</i> (2), <i>TP53</i> (2), <i>IDH1</i> (1), <i>IDH2</i> (1), <i>CBL</i> (1)
<i>BCR::ABL1</i> -like (Ph-like)	54 (16.4)	<i>IGH::CRLF2</i> (34), <i>P2RY8::CRLF2</i> (5), <i>PCM1::JAK2</i> (3), <i>BCR::JAK2</i> (2), <i>EBF1::JAK2</i> (1), <i>SNX29::JAK2</i> (1), <i>NUP214::ABL1</i> (1), <i>FOXP1::ABL1</i> (1), <i>ETV6::ABL1</i> (1), <i>DGKI::ROS1</i> (1), <i>MYO18A::FGFR1</i> (1), <i>CHST15::FGFR2</i> (1), <i>EBF1::PDGFRB</i> (1), <i>SSBP2::CSF1R</i> (1), Other: <i>PAX5::ZCCHC7</i> (1), other <i>PAX5</i> alterations (5)	<i>DNMT3A</i> (2), <i>TET2</i> (2), <i>BCORL1</i> (2), <i>SMC3</i> (1), <i>CBL</i> (1)
Low hypodiploid/near haploid	32 (9.7)	<i>RHEBL1::KMT2D</i> (1), <i>FBX011::MSH6</i> (1), <i>SSBP2::CHD1</i> (1), <i>PAX5</i> alteration (1)	<i>TP53</i> (26), <i>DNMT3A</i> (4), <i>TET2</i> (1), <i>IDH2</i> (1), <i>CUX1</i> (1), <i>SF3B1</i> (1), <i>ZRSR2</i> (1)
<i>KMT2A</i> -rearranged	14 (4.3)	<i>KMT2A::AFF1</i> (9), <i>KMT2A::MLLT3</i> (2), <i>KMT2A::MLLT1</i> (2), <i>KMT2A::ELL</i> (1)	<i>TP53</i> (5)
High hyperdiploid	11 (3.3)	<i>MYST3::ANK1</i> (1), <i>IGH::ZEB2</i> (1), <i>IGH::MIR125B1</i> (1)	<i>ASXL1</i> (1), <i>U2AF1</i> (1), <i>CUX1</i> (1), <i>SMC1A</i> (1)
<i>ZNF384</i> -rearranged	8 (2.5)	<i>EP300::ZNF384</i> (5), <i>TCF3::ZNF384</i> (1), <i>TAF15::ZNF384</i> (1), <i>CREBBP::ZNF384</i> (1)	None
<i>TCF3::PBX1</i>	4 (1.2)	<i>TCF3::PBX1</i> (4)	None
<i>BCL/MYC</i>	4 (1.2)	<i>IGH::BCL2</i> (2), <i>IGH::MYC</i> (1), <i>IGH::BCL3</i> (1), <i>IGH::ID4</i> (1)	<i>TP53</i> (2)
<i>CEBP</i> fusion	3 (0.9)	<i>IGH::CEBPE</i> (1), <i>IGH::CEBPA</i> (1), <i>IGH::CEBPB</i> (1)	None
<i>PAX5</i> , non-P80R	3 (0.9)	<i>PAX5::ETV6</i> (1)	None
<i>PAX5</i> , P80R	2 (0.6)	No fusions	None
iAMP21	2 (0.6)	No fusions	None
<i>ETV6::RUNX1</i>	1 (0.3)	<i>ETV6::RUNX1</i> (1)	None
B-other	97 (29.5)	<i>IGH::ID4</i> (2), <i>IGH::IRF8</i> (1), <i>IGH::ATRX</i> (1), <i>RHEBL1::KMT2D</i> (1), <i>IGF1R-CHD2</i> (1), <i>CCDC88C::ETV6</i> (1), <i>HOOK3::MYST3</i> (1)	<i>TP53</i> (25), <i>TET2</i> (4), <i>ASXL1</i> (1), <i>CBL</i> (1), <i>IDH2</i> (1), <i>SF3B1</i> (1), <i>U2AF1</i> (1), <i>RUNX1</i> (1), <i>CUX1</i> (1), <i>BCOR</i> (1), <i>BCORL1</i> (1)

Supplemental Table 6. Baseline gene rearrangements and structural variants of 71 molecularly characterized adult T-lineage ALL patients

T-ALL subtype per WHO22 and ICC	Number of patients (%)	Gene rearrangements (number of patients)	Myeloid gene mutations (number of patients)
<i>HOX</i> -dysregulated	17 (23.9)	<i>PICALM::MLLT10</i> (8), <i>SET::NUP214</i> (3), <i>NUP214::ABL1</i> (3), <i>NUP98::HOXD13</i> (1), <i>DDX3X::MLLT10</i> (1), <i>KMT2A::AFDN</i> (1)	<i>TP53</i> (3), <i>DNMT3A</i> (2), <i>TET2</i> (2), <i>U2AF1</i> (2), <i>RUNX1</i> (1), <i>IDH2</i> (1)
<i>TAL1</i> -rearranged	11 (15.5)	<i>STIL::TAL1</i> (9), <i>TRA::TAL1</i> (2)	<i>DNMT3A</i> (2), <i>TP53</i> (1), <i>BCOR</i> (1), <i>RUNX1</i> (1), <i>IDH1</i> (1)
<i>TLX1</i> -rearranged	4 (5.7)	<i>TRB::TLX1</i> (2), <i>TRD::TLX1</i> (2)	<i>DNMT3A</i> (1), <i>SF3B1</i> (1), <i>ASXL1</i> (1), <i>BCOR</i> (1), <i>BCORL1</i> (1)
<i>LMO2</i> -rearranged	3 (4.2)	<i>TRB::LMO2</i> (2), <i>TRA::LMO2</i> (1)	<i>IDH2</i> (1), <i>TP53</i> (1)
<i>TLX3</i> -rearranged	2 (2.8)	<i>TRB::TLX3</i> (1), <i>BCL11B::TLX3</i> (1)	None
<i>NKX2</i> -rearranged	1 (1.4)	<i>TRA::NKX2-1</i> (1) and <i>LEF1</i> loss	None
<i>BHLHB1</i> -rearranged	1 (1.4)	<i>TRA::BHLHB1</i> (1)	None
Unknown	8 (11.3)	Not tested with RNA-sequencing	<i>ASXL1</i> (1), <i>DNMT3A</i> (1), <i>U2AF1</i> (1)
T-other	24 (33.8)	<i>PVT1::PDCD1LG2</i> (1), <i>NOSIP::NOTCH1</i> (1), <i>RUNX1::BFAR</i> (1), <i>TRA::NOTCH1</i> (1)	<i>TP53</i> (5), <i>IDH2</i> (3), <i>ASXL1</i> (3), <i>DNMT3A</i> (2), <i>TET2</i> (2), <i>RUNX1</i> (2), <i>U2AF1</i> (1)

Supplemental Table 7. List of pathogenic/likely pathogenic germline variants in our ALL cohort

Patient ID	Gene	Variant
ALL24	<i>CHEK2</i>	p.I200T
ALL55	<i>CHEK2</i>	p.S465Vfs*15
ALL57	<i>CHEK2</i>	p.T410Mfs*15
ALL69	<i>MUTYH</i>	p.Y179C
ALL94	<i>CHEK2</i>	p.T410Mfs*15
ALL103	<i>BRCA2</i>	p.V3365fs
ALL104	<i>BRCA2</i>	c.3720_3721delGT
ALL144	<i>CHEK2</i>	p.T410Mfs*15
ALL161	<i>MEN1</i>	p.T215fs*13
ALL163	<i>FANCC</i>	p.E327FS*47
ALL200	<i>RET</i>	p.E511K
ALL222	<i>CHEK2</i>	c.444+1G>A
ALL263	<i>CHEK2</i>	p.R160G
ALL296	<i>CHEK2</i>	p.I200T
ALL328	<i>VHL</i>	p.R200W
ALL341	<i>MUTYH</i>	p.G382D
ALL342	<i>MUTYH</i>	p.G382D
ALL355	<i>ATM</i>	p.S2855_V2856delinsRI
ALL396	<i>TP53</i>	p.E339*

Supplementary Table 8. Multivariable analysis of overall survival (OS) in B-ALL patients

	Hazard ratio (95% CI)	p value
Age, continuous	1.02 (1 – 1.03)	0.0014
Therapy related vs <i>de novo</i> B-ALL	0.86 (0.49 – 1.48)	0.59
Low hypodiploidy	1.06 (0.53 – 2.13)	0.85
<i>BCL/MYC</i>	2.86 (0.63 – 12.9)	0.17
<i>TP53</i> -mutated	2.19 (1.25 – 3.83)	0.005
Myeloid mutations	1.88 (1.08 – 3.26)	0.024
<i>RB1</i> -mutated	1.56 (0.73 – 3.32)	0.24
First-line therapy		
Pediatric regimen	0.73 (0.34 – 1.56)	0.42
HyperCVAD	1.41 (0.71 – 2.81)	0.32
Other therapy	0.75 (0.37 – 1.5)	0.42

Supplemental Table 9. Clinical characteristics of patients studied in scDNA + protein sequencing experiments

	<i>De novo vs therapy-related</i>	Age	Sex	Blast percentage	ALL subtype
B-ALL1	Therapy-related	66	F	67%	Low hypodiploid
B-ALL3	Therapy-related	65	M	72%	Low hypodiploid
ETP-ALL1	De novo	65	M	63%	T-other
ETP-ALL2	De novo	61	M	81%	T-other
T-ALL2	De novo	56	M	85%	Hyperdiploid

F, female; M, male

Supplemental Table 10. Clinical characteristics of patients studied in scRNA-seq experiment

	Age	Sex	Blast percentage	B-ALL subtype	Gene mutations
CH-01	65	F	NA	NA	<i>TP53</i> (R273H at 7%)
CH-02	71	F	NA	NA	<i>DNMT3A</i> (C494Afs* at 9%)
B-ALL with MyM-1	63	F	54%	B-other	<i>TET2</i> (R1214Q at 43%), <i>NRAS</i> (G12D at 7%)
B-ALL with MyM-2	56	M	41%	Low hypodiploid	<i>TP53</i> (C176Y at 24%)
B-ALL with MyM-3	71	M	85%	Low hypodiploid	<i>TP53</i> (G244C at 15%), <i>RB1</i> (c.1961-2A>G at 11%)
B-ALL with MyM-4	74	F	84%	Ph-like	<i>DNMT3A</i> (E774E at 27%), <i>JAK2</i> (R683G at 24%), <i>CDKN2A</i> loss
B-ALL with MyM-5	60	F	65%	Low hypodiploid	<i>DNMT3A</i> (R882H at 33%), <i>TP53</i> (M237I at 52%)
B-ALL without MyM-1	61	F	90%	B-other	Normal karyotype, no mutations
B-ALL without MyM-2	59	F	49%	B-other	Normal karyotype, no mutations

F, female; M, male; MyM, myeloid mutations; NA, not applicable

Supplemental Table 11. Ingenuity pathway analysis comparing B-lymphoblasts from B-ALL with MyM vs B-ALL without MyM

	Z score	(-logp value)	Number of genes
Cell survival	1.089	30	144
Invasion of cells	3.073	19	101
Tumor growth	2.16	16	90
Homeostasis of blood cells	-2.32	15	58
Apoptosis of tumor cells	-2.58	12	32
Glucocorticoid receptor signaling	NA	9.46	37
Sirtuin signaling pathway	-1.213	8.428	24
EIF2 signaling	-3.15	8.38	21
Cytotoxicity of lymphoid cells	-2.24	8	19
PD1, PDL1 cancer immunotherapy	3.64	7.72	14
IL-8 signaling	1.29	6.76	18
Rho GTPase signaling	1.6	5.863	19
Actin cytoskeleton signaling	1.6	5.81	18
Oxidative phosphorylation	3.46	5.75	12
ERK/MAPK signaling	-0.905	4.65	15
Protein kinase A signaling	0.243	4.217	21

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