

Annals of Leukemia Research

Review Article

IKZF1 Deletions and Risk Stratification in Paediatric B-Cell Precursor Acute Lymphoblastic Leukemia

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Received: 17 April 2025; Accepted: 23 August 2025; Published: 11 December 2025

Citation of this article: Sundberg, E., Kovacs, E., Saar, K. (2025) IKZF1 Deletions and Risk Stratification in Paediatric B-Cell Precursor Acute Lymphoblastic Leukemia. Ann Leuk Res, 6(1): 04-06. doi: 10.5281/zenodo.22238140

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Summary

IKZF1 deletion is among the most reproducible adverse genetic markers in childhood leukaemia and is routinely described as an independent prognostic factor. The refined version of the marker turns out to carry almost no independent weight at all: its effect is conditional on measurable residual disease, and in good responders it disappears.

Objectives: To review the published evidence on IKZF1 deletions in paediatric B-cell precursor acute lymphoblastic leukaemia: how often they occur, what they predict, how the refined IKZF1plus profile behaves across residual disease strata, and what role the marker should play in risk stratification.

Material and Methods: Narrative review of cooperative group trial analyses, population-based cohorts, international collaborative studies and mechanistic work indexed in PubMed and the major haematology journals. Studies were eligible if they reported the frequency, prognostic effect or biological consequences of IKZF1 alteration in a defined paediatric B-cell precursor acute lymphoblastic leukaemia population. No new patient data were generated and no patients were recruited; every estimate quoted is one published by the original investigators.

Results: IKZF1 deletion occurs in approximately 15 per cent of cases. In a population-based cohort of 334 children, deletion was associated with ten-year event-free survival of 60 against 83 per cent ($p < 0.001$) and overall survival of 73 against 89 per cent ($p = 0.001$), and remained the strongest independent factor for relapse and death after adjustment for white cell count and residual disease. Screening 694 samples from a separate trial gave five-year event-free survival of 0.69 against 0.85 ($p < 0.0001$) with cumulative relapse of 0.21 against 0.10 ($p = 0.001$). The refined IKZF1plus profile behaved very differently across residual disease strata: five-year event-free survival was 94 per cent in residual disease standard-risk patients, 40 per cent in intermediate-risk and 30 per cent in high-risk ($p \leq 0.001$), with corresponding relapse incidences of 6, 60 and 60 per cent.

Conclusion: The unrefined deletion carries independent prognostic weight; the refined profile does not, behaving instead as a modifier that identifies very poor prognosis only among patients already flagged by residual disease. A child with the adverse genotype and a clean day-33 marrow has a 94 per cent event-free survival.

Abbreviations: ALL – Acute Lymphoblastic Leukaemia, BCP-ALL – B-Cell Precursor ALL, IKZF1 – IKAROS Family Zinc Finger 1, Δ IKZF1 – IKZF1 Deletion, MRD – Minimal Residual Disease, EFS – Event-Free Survival, OS – Overall Survival, RFS – Relapse-Free Survival, MLPA – Multiplex Ligation-Dependent Probe Amplification, SNP – Single Nucleotide Polymorphism, WBC – White Blood Cell Count, HR – Hazard Ratio.

Keywords: Acute Lymphoblastic Leukaemia; IKZF1; Risk Stratification; Minimal Residual Disease; Paediatric Oncology

Introduction

Survival in childhood acute lymphoblastic leukaemia now exceeds 90 per cent, and relapse affects 10 to 20 per cent of patients while remaining the leading cause of death [1,2]. Risk stratification was historically based on age and white cell count, and has been progressively replaced by cytogenetic and molecular characterisation alongside response measured as minimal residual disease [3,4].

Genome-wide copy number analysis identified deletion of IKZF1, which encodes the lymphoid transcription factor IKAROS, as one of the strongest adverse markers to emerge from that work [5,6]. Alterations are present in up to 15 per cent of cases and are heterogeneous,

comprising broad and intragenic deletions as well as sequence mutations [7,8].

The marker has been validated repeatedly across cooperative groups and national cohorts, and has been proposed for integration into treatment stratification [9,10,11]. What has proved harder to establish is whether it adds information beyond what residual disease already provides, since the two are correlated and response measurement is the dominant variable in contemporary protocols [12,13].

This review sets out the frequency and prognostic effect of IKZF1 deletion, examines how the refined IKZF1plus profile behaves once residual disease is taken into account, and considers what role the marker should have in stratification.

Material and Methods

Search strategy: PubMed and the archives of the major haematology and paediatric oncology journals were searched for studies of IKZF1 in childhood leukaemia. Search terms combined acute lymphoblastic leukaemia and paediatric with IKZF1, IKAROS, IKZF1plus, copy number alteration, minimal residual disease and risk stratification. Reference lists of retrieved trial analyses and collaborative studies were screened by hand.

Eligibility: Studies were included if they reported the frequency, prognostic significance or biological effect of IKZF1 alteration in a defined paediatric B-cell precursor acute lymphoblastic leukaemia population. Studies in Philadelphia-positive disease and in adults are cited where they inform interpretation and are identified as such.

Data extraction and handling: Design, cohort size, detection method, treatment protocol, definition of the alteration and reported survival estimates were extracted as published. Detection method is stated alongside every frequency figure, because array-based and ligation-dependent approaches differ in the deletions they resolve. No pooling or re-analysis was performed and no patient-level data were obtained. Figures 1 to 4 and Tables 1 to 3 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

Frequency and unconditional effect: A population-based cohort of 334 children treated on national protocols between 1992 and 2013, with IKZF1 status established by single nucleotide polymorphism array in 218 and by multiplex ligation-dependent probe amplification in 116, found deletion in 15 per cent [12]. Ten-year event-free survival was 60 against 83 per cent ($p < 0.001$) and overall survival 73 against 89 per cent ($p = 0.001$) (Figure 3) [12].

Adjusting for known risk factors including white cell count and residual disease, deletion remained the strongest independent factor for both relapse and death in that analysis [12]. Screening 694

diagnostic samples from a separate cooperative group trial gave five-year event-free survival of 0.69 (SE 0.05) against 0.85 (SE 0.01), $p < 0.0001$, driven mainly by higher cumulative relapse at 0.21 (SE 0.04) against 0.10 (SE 0.01), $p = 0.001$ (Figure 4) [9].

Deletion was significantly associated with the P2RY8-CRLF2 rearrangement in that cohort, but its prognostic value proved independent of that association [9]. Independent evidence from a European trial group identified deletion as an independent marker and additionally as distinguishing patients who benefit from pulses during maintenance therapy [10].

The two cohorts differ in a way worth noting. The population-based analysis covered two decades of successive national protocols, so the effect it reports is averaged across changing therapy [12]. The trial cohort applied one protocol, which isolates the marker better but describes only the treatment given [9]. That the estimates agree closely despite the difference strengthens both.

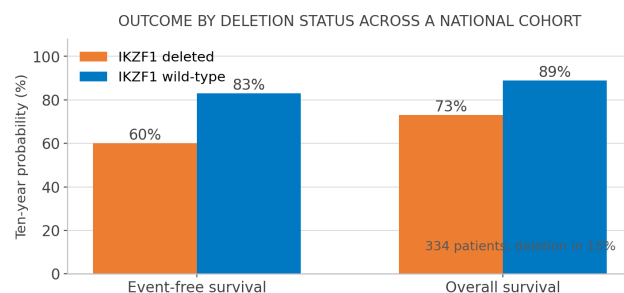


Figure 3: Ten-year outcome by IKZF1 status in a population-based cohort of 334 children [12]

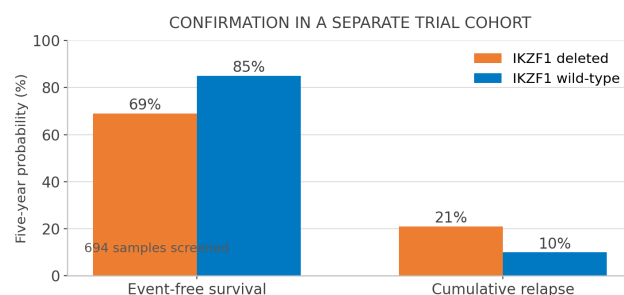


Figure 4: Five-year outcome in a separate trial cohort of 694 screened samples [9]

Study	n	Cohort	Principal reported finding
Dörge et al. 2013 [9]	694	ALL-BFM 2000	EFS 0.69 against 0.85; independent of P2RY8-CRLF2
Clappier et al. 2015 [10]	—	EORTC	Independent marker; identifies benefit from pulses
Olsson et al. 2015 [12]	334	Nordic protocols	Strongest independent factor after adjusting for MRD
Boer et al. 2016 [14]	134	International	Rare deletion variants also carry poor prognosis
Stanulla et al. 2018 [13]	Two cohorts	AIEOP-BFM 2000	IKZF1plus effect depends entirely on MRD stratum
Braun et al. 2022 [15]	—	ALL-IC BFM 2009	Relapse-free survival hazard ratio 2.72 for deletion

Table 1: Principal studies reviewed, with cohort and the finding as reported by the original investigators

Rare deletion variants deserve separate mention. Strong evidence supported the poor prognosis of deletions affecting exons 4 to 7 and 1 to 8, while the remaining third of cases carrying other variants had been little studied. An international case-control study matching each of 134 rare-variant cases to three wild-type controls on subtype, protocol, risk arm, white cell count and age found poor prognosis overall ($p < 0.001$), evident within every risk stratification arm [14]. Rare variants therefore behave like the common ones and should not be reported as indeterminate.

Refining the marker: Attempting to sharpen the prognostic signal, investigators examined co-occurring deletions and defined the IKZF1plus profile as IKZF1 deletion accompanied by deletion of CDKN2A, CDKN2B, PAX5 or the pseudoautosomal region, in the absence of ERG deletion [13]. The profile was independently prognostic on multivariable analysis and replicated in a second cohort [13].

The refinement worked in the sense that IKZF1plus identifies a worse group than IKZF1 deletion alone. It did not work in the sense the investigators expected, because the effect proved almost entirely conditional on residual disease [13].

The effect that disappears in good responders: Stratifying IKZF1plus patients by residual disease level after induction produced five-year event-free survival of 94 per cent (SE 5) in the standard-risk stratum, 40 per cent (SE 10) in the intermediate and 30 per cent (SE 14) in the high-risk stratum, with $p \leq 0.001$ across groups (Figure 1) [13]. Cumulative relapse incidence followed the same pattern at 6 per cent (SE 6), 60 (SE 10) and 60 (SE 17) (Figure

2) [13].

Those three groups share an identical adverse genotype. A child carrying IKZF1plus who is residual disease negative on days 33 and 78 has a 94 per cent event-free survival, which is better than the cohort average and far better than many children without the genotype [13,16]. The genetic marker has, in that patient, no adverse implication that survives measurement of response.

Residual disease standard risk in that protocol required negativity at two targets with sensitivity of 10^{-4} or better on both day 33 and day 78, so the good-prognosis group is defined stringently rather than loosely [13,16]. The dissociation is therefore not an artefact of a permissive response threshold.

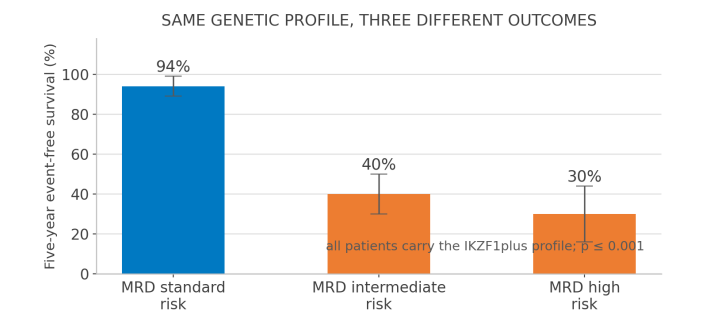


Figure 1: Five-year event-free survival among IKZF1plus patients by residual disease stratum [13]

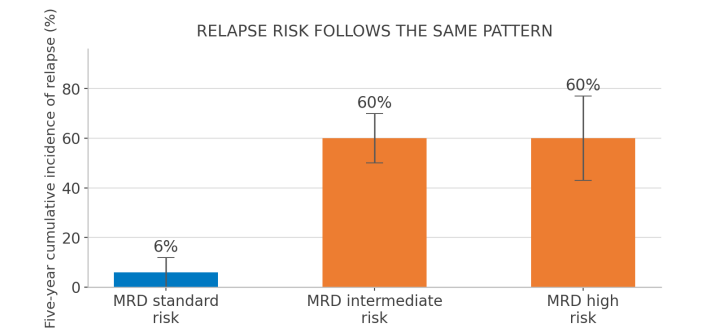


Figure 2: Cumulative relapse incidence across the same three strata [13]

How the marker relates to response: Deletion and poor response are correlated rather than independent. Residual disease above 10^{-4} at day 33 was more frequent in patients with IKZF1 deletion at 36.0 per cent and with IKZF1plus at 41.9 per cent than in wild-type patients at 24.0 per cent, approaching significance at $p = 0.051$ [15]. Patients with the IKZF1plus profile showed a tendency to slower residual disease reduction between days 15 and 33 [15].

In the same cohort, relapse-free survival was reduced with hazard ratios of 2.72 (95% CI 1.21 to 6.11) for IKZF1 deletion and 2.00 (95% CI 0.87 to 4.49) for IKZF1plus against wild-type, $p = 0.023$ [15]. That the confidence interval for IKZF1plus crosses unity in a cohort where deletion alone reaches significance is consistent with the conditional effect described above.

Marker	Effect	Depends on residual disease?
IKZF1 deletion	EFS 60% against 83% at ten years	No; independent after adjustment [12]
IKZF1 deletion	EFS 0.69 against 0.85 at five years	Independent of P2RY8-CRLF2 [9]
IKZF1plus profile	EFS 94%, 40%, 30% by MRD stratum	Yes; effect absent in good responders [13]
Rare deletion variants	Poor prognosis in every risk arm	Not separately reported [14]

Table 2: What each version of the marker predicts and whether the effect survives residual disease stratification

Where the marker sits among other genetic risk factors: IKZF1 is one of several copy number alterations identified by genome-wide analysis [6,23], and integrated classifications combining cytogenetics with genomic data have refined risk assignment beyond what either provides alone [22]. Within that framework, deletion retains prognostic value alongside a BCR-ABL1-like expression signature while high CRLF2 expression does not [11].

In Philadelphia-positive childhood disease the marker behaves differently again, and its status there has been evaluated separately [17]. Whether IKZF1 remains a poor prognostic marker under contemporary therapy has been questioned directly, which is the same conditional argument approached from a different direction [25].

What response-guided protocols already achieve: The reason the conditional finding matters is that residual disease-guided therapy is effective on its own. Randomised trials have shown that treatment can safely be reduced in residual disease-defined low-risk children [18] and that augmenting post-remission therapy benefits a residual disease-defined high-risk subgroup drawn from clinically standard-risk patients [19]. A national protocol subsequently combined both directions successfully [21].

Response measurement also carries lineage-specific subtleties that a genetic marker cannot capture: in T-lineage disease the informative timepoint is later than in B-lineage disease, and an early measurement that would condemn a B-lineage patient carries no information provided the later one is clear [20]. Any genetic marker proposed as an addition to stratification is competing against a response measure that is already doing a great deal of work.

Variable	Available at	Independent of MRD?
Age and white cell count	Diagnosis	Largely superseded [3]
Cytogenetic subtype	Diagnosis	Retains value within integrated models [22]
IKZF1 deletion	Diagnosis	Yes, on adjusted analysis [12]
IKZF1plus profile	Diagnosis	No; effect confined to poor responders [13]
Residual disease day 33 and 78	During induction	The reference variable [3,16]

Table 3: Stratifying variables in current use, when each becomes available and whether it adds to residual disease

Discussion

Two findings sit awkwardly together. IKZF1 deletion is independently prognostic after adjustment for residual disease in a population-based cohort, where it was the strongest such factor [12]. The refined IKZF1plus profile, designed to sharpen that signal, turns out to carry no adverse implication at all in patients who clear residual disease, with 94 per cent event-free survival in that group [13].

The reconciliation is probably that the two analyses are asking different questions. Adjusting for residual disease in a regression estimates an average effect across strata; stratifying by residual disease and reporting each stratum separately shows that the average conceals near total heterogeneity. Both are correct and only the second is useful at the bedside.

What this means for stratification

If the purpose of testing for IKZF1plus is to identify children needing intensified therapy, the result must be read alongside the day-33 and day-78 marrow rather than at diagnosis [13,16]. Intensifying treatment for a child with the genotype before response is known would treat a group whose expected event-free survival, if they respond well, is 94 per cent.

The corollary is more useful. Among children already identified as intermediate or high risk by residual disease, IKZF1plus identifies a subgroup with 40 and 30 per cent event-free survival and 60 per cent relapse incidence [13]. That is a group in whom transplantation or experimental therapy might reasonably be considered, and the genotype adds real information there.

Why deletion and response are not independent

IKAROS regulates lymphoid differentiation, and its loss alters signal transduction in ways that plausibly affect chemosensitivity directly [5,17]. If the deletion causes poor response rather than merely accompanying it, then residual disease is measuring the downstream consequence of the genotype and the two cannot be independent by

construction.

The data are consistent with that reading: residual disease above 10^{-4} at day 33 was substantially more common in deleted patients [15], and IKZF1plus patients cleared disease more slowly between days 15 and 33 [15]. On this account the genotype and the response measure are sequential observations of one process rather than two independent risk factors.

Study limitations

The limitations belong to the underlying literature. Detection methods differ, with array-based and ligation-dependent approaches resolving different deletion variants, and reported frequencies depend on which was used [12,14]. The IKZF1plus analysis rests on subgroups defined by two variables simultaneously, so the standard errors are wide, reaching 14 per cent in the high-risk stratum [13]. Treatment protocols differ across the cohorts compared, and the effect of a genetic marker is conditional on the therapy given, as the maintenance pulse finding illustrates [10]. Rare deletion variants comprise about a third of cases and were underexamined until recently [14]. And residual disease definitions and timepoints differ between protocols, which directly affects the stratification on which the central finding depends [13,15,16].

Two gaps deserve naming. No trial has randomised treatment intensity by IKZF1 status within residual disease strata, so the clinical utility of the genotype in good responders remains untested rather than disproved [13]. And no study reviewed here reports whether the conditional pattern seen for IKZF1plus also applies to IKZF1 deletion alone, which is the version more widely tested in practice [12,13].

Conclusion

IKZF1 deletion occurs in about 15 per cent of children with B-cell precursor acute lymphoblastic leukaemia and is associated with ten-year event-free survival of 60 against 83 per cent, remaining independently prognostic after adjustment for residual disease in population-based analysis. The refined IKZF1plus profile behaves differently. Its effect is conditional: five-year event-free survival is 94 per cent among patients who clear residual disease, 40 per cent in the intermediate stratum and 30 per cent in the high-risk stratum, with relapse incidence of 6, 60 and 60 per cent across the same three groups. A child with the adverse genotype and a clean day-33 marrow does better than the cohort average. The marker belongs in stratification, but read after the response measurement rather than instead of it.

Two questions follow. Whether the conditional pattern demonstrated for IKZF1plus also holds for IKZF1 deletion alone has not been reported, and the deletion is the version more commonly tested outside trial settings [12,13,24]. And whether the marker

retains adverse weight under contemporary response-guided protocols has been raised as an open question rather than settled [25].

Neither requires new technology. Both require existing cohorts to be reanalysed with the genotype and the response measure reported jointly rather than one adjusted for the other, which is a different and more informative presentation of data that has already been collected [12,13,15].

Acknowledgements

The authors thank the cooperative study groups and national registries whose published cohorts form the substance of this article.

Funding

No dedicated funding from public, commercial or charitable sources supported this review.

Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

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