KROMATID

Are Your Cells Fit?

Analytical Cytogenetics for Cell and Gene Therapy Development

Diagnostic cytogenetics was built to answer “normal or abnormal.” Cell and gene therapy needs a different question: is this cell genomically fit to become a medicine? Genomic fitness belongs in that conversation at every stage of development:



Genomic fitness belongs across every phase of development.

THE PROBLEM

“Normal” Isn’t Always the Right Question

For decades, cytogenetics has answered one question: is this normal or abnormal? Diagnostic cytogenetics does this well; it was built to characterize 20 metaphase cells and flag gross events like trisomies. But cell and gene therapy isn’t asking a diagnostic question. It’s asking a development question: *does the genomic state of this cell support the biology and function I need from it?*

That distinction matters because of sampling. Twenty cells can’t reliably detect a change present in a subset of a population, and heterogeneity, not just an aggregate result, is often what determines whether a batch performs as expected. Analytical cytogenetics closes that gap: hundreds to thousands of single cells, imaged directly and quantified against a reference population, turning genomic structure into an analytic you use throughout development, not a checkbox you clear once at the end.

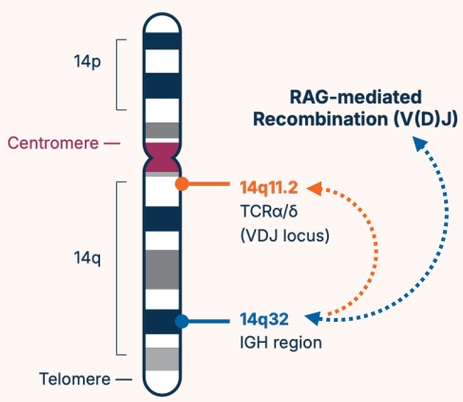
The stakes compound the longer genomic structure goes unmeasured. Two batches can pass every standard release test, viability, potency, identity, and still behave completely differently downstream: reduced potency, unstable clones, poor scalability, an unexpected IND or CTA question. These risks don’t start in manufacturing; they start with decisions made in early development, before anyone is looking at genomic structure at all. Catching it early is the only place it’s actually preventable.

THE EVIDENCE

When a Known Variant Looked Like a Red Flag

A chromosome 14q inversion observed in a clinical T-cell program was initially read as an editing-associated abnormality, and it triggered a regulatory hold.

Chromosome 14



The 14q11.2–q32 inversion: a RAG-mediated V(D)J rearrangement joining the TCRα/δ locus to the IGH region.

KROMATID’s baseline dataset tells a different story. Across 200+ healthy donors and 20,000+ metaphases analyzed, this recombination-driven rearrangement is the most frequently observed structural variant in normal T cells. Roughly a quarter of donor samples cluster into a distinct genomic profile that standard release testing never sees.

A baseline isn’t a lucky reference point; it’s what systematic characterization produces. That’s what makes a result interpretable instead of alarming, and it’s what lets process understanding compound: each new finding gets read against real biology, not treated as a fresh unknown.

See the full T-cell baseline case study, live.

WEBINAR:
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Analytical Cytogenetics for Cell and Gene Therapy | [Date] 2026

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