

# The Significance of Cellular Hashes in Acute Leukemia

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## DESCRIPTION

Following decades in which cytarabine plus anthracycline was the exclusive treatment for acute myeloblastic leukemia, targeted medicines have emerged. These include anti-CD123 and anti-CD52 monoclonal antibodies, as well as particular inhibitors of genetic mutations (such anti-*IDH*, *IDH2*, or *FLT3*).

What role should these treatment choices play in light of the tumor heterogeneity that comes with diagnosing leukemia and the potential for clonal drift in this kind of tumor. For targeted medications, it would be necessary to analyze the different therapeutic targets at the individual cell level rather than within the population as a whole.

In fact, a particular molecular target's prognostic significance and therapeutic relevance varies depending on whether it is a stem cell with robust proliferative and self-renewal capabilities or a cell in terminal differentiation with limited proliferative ability. This cell-by-cell examination is not without its challenges, though. The first one is scientific because, despite the various methods used to standardize the data, the comparison of two distinct single-cell analysis procedures is subtle. The second trap is a practical one because every single cell experiment is quite expensive both materially and in terms of time. Acute Myeloid Leukemia (AML) is a genetically, epigenetically, and clinically heterogeneous disease that is typified by proliferative, clonal, abnormally differentiated, and occasionally poorly differentiated cells of the hematopoietic system infiltrating the bone marrow, blood, and other tissues, leading to the failure of normal hematopoiesis. AML causes a disproportionate amount of cancer-related deaths, is more prevalent among the elderly, and is linked to serious consequences and high mortality. Cure rates have risen to 15% for the people over 60 and almost 40% for those under 60 due to

recent advancements in disease management. In example, the treatment of acute leukemia's and other hematological malignancies has advanced significantly thanks to molecular analysis tools. Tumor escape following chemotherapy and immunotherapy is caused by a variety of causes. Of interest are the identification of escape metabolic pathways and the expression of resistance markers throughout the population. Nevertheless, since it is unable to identify the cell populations that are affected by these expressions, the information offered by this total population analysis is insufficient. If a resistance factor impacts a leukemic stem cell with strong mitogenic capacity or a terminally differentiated cell with low proliferation potential, its expression is anticipated to have a distinct prognostic value, albeit this has not been confirmed. Flow cytometry techniques have addressed this limitation by enabling the analysis of a high number of markers on the same cell, albeit typically less than 20 markers are employed in regular tests, and the number cannot exceed 50 markers. The analysis of slightly more than 100 parameters is possible with mass spectrometry. Despite the fact that this number of markers appears high already, it is completely disproportionate to the roughly 20,000 genes that a cell has the potential to produce. The methods that are currently available for single cell RNA analysis permit this approach. In biological systems with cellular heterogeneity, single-cell RNA sequencing, or scRNA-seq, is an effective technique for examining the intricate cellular transcriptome at the single-cell level. Classical single-cell RNA analysis is a highly strong instrument, but it has a number of shortcomings. The first is undoubtedly the expense of conducting such an analysis, which is challenging to implement in regular analysis. The inability to compare two samples from distinct studies, even after the data have been standardized and normalized, is the second drawback. In order to get over these two drawbacks, the cell hashing technique looks promising.

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**Received:** 05-Jan-2024, Manuscript No. JLU-24-29387; **Editor assigned:** 08-Jan-2024, PreQC No. JLU-24-29387 (PQ); **Reviewed:** 26-Jan-2024, QC No. JLU-24-29387; **Revised:** 02-Feb-2024, Manuscript No. JLU-24-29387 (R); **Published:** 09-Feb-2024, DOI: 10.35248/2329-6917.24.12.360

**Citation:** Marco V (2024) The Significance of Cellular Hashes in Acute Leukemia. J Leuk. 11:360.

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