Perspective
Volume 11:01, 2025

Journal of Oncology Translational Research

ISSN: 2476-2261 Open Access

Spatial Genomics and Multiregion Biopsy: Decoding Intratumoral Heterogeneity

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Introduction

Cancer, once considered a monolithic disease driven by a handful of genetic mutations, is now understood to be a complex, dynamic ecosystem marked by profound heterogeneity. At the heart of this complexity lies intratumoral heterogeneity the existence of genetically, epigenetically, and phenotypically diverse cancer cell populations within a single tumor. ITH has emerged as a critical factor influencing tumor progression, treatment resistance, immune evasion, and disease recurrence. Traditional bulk sequencing techniques have provided invaluable insights into the genetic makeup of tumors. However, they often mask the diversity of subclonal populations by averaging signals across the entire tumor mass. In recent years, advances in spatial genomics and multiregion biopsy have opened new frontiers in the dissection of ITH, offering unprecedented resolution and context into the spatial organization and evolutionary dynamics of tumors [1,2].

Description

Multiregion biopsy involves sampling tissue from multiple spatially distinct regions of the same tumor or from primary and metastatic sites. This approach captures spatial heterogeneity more accurately than single-biopsy or bulk sequencing methods. By comparing genetic and molecular profiles across regions, researchers can reconstruct the clonal architecture and evolutionary trajectories of tumors. Early mutations shared across all regions. Later mutations specific to certain regions. Unique to individual subclones. Multiregion biopsy requires meticulous planning to avoid sampling bias. Typically, tissue samples are collected. Tissue is subjected to Whole-Exome Sequencing (WES), Whole-Genome Sequencing (WGS), or targeted gene panels, followed by bioinformatic reconstruction of phylogenetic trees and mutational landscapes [3].

While multiregion sequencing captures spatial diversity at the macro level, it lacks spatial context at the cellular resolution. Spatial genomics bridges this gap by preserving the physical location of cells and molecules within the tissue architecture during molecular analysis. Spatial genomics encompasses a suite of techniques that combine high-throughput molecular profiling with spatial information. These methods enable simultaneous mapping of gene expression, mutations, epigenetic marks, and protein levels within histological sections. This technique uses barcoded arrays or microdissection to capture mRNA transcripts from defined tissue locations, generating spatial gene expression maps. Captures transcriptomes across tissue sections with spatial barcodes. Uses metal-tagged antibodies and mass spectrometry to detect multiple proteins in tissue sections, preserving spatial relationships between cells [4].

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Received: 01 February, 2025, Manuscript No. jotr-25-168445; **Editor Assigned:** 03 February, 2025, PreQC No. P-168445; **Reviewed:** 15 February, 2025, QC No. Q-168445; **Revised:** 20 February, 2025, Manuscript No. R-168445; **Published:** 27 February, 2025, DOI: 10.37421/2476-2261. 2025.11. 297

Even multiregion biopsy may miss rare subclones or understudied regions. Tissue availability is often limited, especially in deep or metastatic tumors. Single-timepoint biopsies provide static snapshots; dynamic clonal evolution requires serial sampling or integration with longitudinal liquid biopsy. The massive volume and complexity of spatial omics data challenge current bioinformatics pipelines. Standardized frameworks for data analysis and clinical interpretation are still evolving. Multiregion sampling can pose risks and burdens to patients. Ethical considerations must balance scientific benefit with patient safety and autonomy. The field of spatial oncology is rapidly evolving, with several promising avenues for future development: Combining spatial and longitudinal data to map tumor evolution over time and space. AI-driven models to predict tumor behavior, treatment response, and recurrence risk based on spatial patterns. Assessing how spatially distributed genetic alterations influence drug distribution, efficacy, and toxicity within tumors [5].

Conclusion

Intratumoral heterogeneity is a fundamental hallmark of cancer that challenges conventional therapeutic paradigms and contributes to disease progression and resistance. Spatial genomics and multiregion biopsy represent powerful, complementary strategies to decode this heterogeneity by preserving the spatial context and evolutionary complexity of tumors. By enabling high-resolution mapping of subclonal architecture, microenvironmental niches, and cell-cell interactions, these technologies are reshaping our understanding of tumor biology. They offer critical insights for the development of precision medicine strategies, from rational drug design to immunotherapy optimization and dynamic treatment adaptation.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Melchor, L., Annamaria Brioli, C. P. Wardell and A. Murison, et al. "Single-cell genetic analysis reveals the composition of initiating clones and phylogenetic patterns of branching and parallel evolution in myeloma." *Leukemia* 28 (2014): 1705-1715.

2. Bahlis, Nizar J. "Darwinian evolution and tiding clones in multiple myeloma." *Blood* 120 (2012): 927-928.

3. Weinhold, Niels, Cody Ashby, Leo Rasche and Shweta S. Chavan, et al. "Clonal selection and double-hit events involving tumor suppressor genes underlie relapse in myeloma." *Blood* 128 (2016): 1735-1744.

4. Keats, Jonathan J., Marta Chesi, Jan B. Egan and Victoria M. Garbitt, et al. "Clonal competition with alternating dominance in multiple myeloma." *Blood* 120 (2012): 1067-1076.

5. Fox, Edward J. and Lawrence A. Loeb. "One cell at a time." *Nature* 512 (2014): 143-144.

How to cite this article: Libster, Weiss. "Spatial Genomics and Multiregion Biopsy: Decoding Intratumoral Heterogeneity." *J Oncol Transl Res* 11 (2025): 297.