

## Special Issue

# Tumor Heterogeneity in Breast Cancer—the “Omics” Revolution

### Message from the Guest Editors

For this Special Issue, fundamental research endeavors to describe the molecular mechanisms driving breast cancer are warranted to ultimately develop tailored therapeutic options for this devastating disease. Recent advances in genomics and transcriptomics methodologies, such as single-cell RNA-seq and spatial transcriptomics and epigenomics, allow for the study of breast cancer cell subpopulations and their complex interactions at an unprecedented temporal and spatial resolution. In this Special Issue, original research articles and reviews are welcome to be submitted. Research areas may include, but are not limited to, the following: transcriptomics analysis of primary breast tumor samples or cell lines at the bulk, single-cell, or spatial transcriptomics level; studies including the analysis of DNA regulatory landscapes with technologies such as ATAC-seq and ChIP-seq; and, finally, alternative approaches, applying other “omics” methodologies such as proteomics and metabolomics that contribute to a holistic systems-level understanding of the tumor milieu. We look forward to receiving your valuable contributions.

### Guest Editors

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### Deadline for manuscript submissions

30 June 2026



## Cancers

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## About the Journal

### Message from the Editor-in-Chief

*Cancers* is an international online journal addressing both clinical and basic science issues related to cancer research. The journal is publishing in Open Access format, which will certainly evolve to ensure that the journal takes full advantage of the rapidly changing world of information and knowledge dissemination. It publishes high-quality clinical, translational, and basic science research on cancer prevention, initiation, progression, and treatment, as well as other related topics, particularly to capture the most seminal studies in the rapidly growing area of immunology, immunotherapy, and tumor microenvironment.

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### Editor-in-Chief

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