CALL FOR EXPRESSIONS OF INTEREST

African Collaboration in Cancer Chemoimmunotherapy, Biophysics and Mathematical Modeling

Advanced multidisciplinary manuscript | Building a future African cancer-research consortium

APPLICATION DEADLINE	22 July 2026 at 11:55 PM East Africa Time (EAT)

African researchers are invited to express interest in a multidisciplinary collaboration examining biological and biophysical determinants of chemoimmunotherapy response in non-small-cell lung cancer. The study integrates transcriptomics, spatial omics, drug-transport biophysics, and tumor-effector mathematical modeling.

Who should express interest?

- Researchers with expertise in chemotherapy, immunotherapy, tumor immunology, tumor-microenvironment biology, or translational oncology.
- Researchers in cancer biophysics, drug delivery, systems oncology, bioinformatics, or mathematical and computational modeling.
- Applicants based at the Ocean Road Cancer Institute or another cancer-dedicated center are particularly encouraged to apply.
- The call is open across Africa, with priority to applicants from sub-Saharan Africa and those with relevant grant or collaborative experience.

Nature of the collaboration

- Core analyses and an initial manuscript have been completed.
- The remaining work is primarily editorial and scholarly, including biological interpretation, clinical framing, methodological refinement, and critical manuscript review.
- No major new primary analysis is currently anticipated.
- The collaboration also aims to establish a multidisciplinary African consortium for future cancer and immunology funding opportunities.

Authorship and contributorship will reflect substantive intellectual input and follow the target journal's criteria.

How to express interest

- A brief expression of interest describing the expertise you would contribute.
- A short curriculum vitae.
- Up to two to three selected publications relevant to the call.
- A concise summary of relevant grant, consortium, or collaborative experience.

Send expressions of interest to

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STUDY ABSTRACT

Omics-informed tumor-effector modeling reveals delivery, trafficking, resistance, and cytotoxic-synapse modules of chemoimmunotherapy response

Background: Chemoimmunotherapy has improved outcomes in non-small-cell lung cancer (NSCLC), but response remains heterogeneous because therapeutic efficacy emerges from coupled processes involving tissue access, intracellular drug handling, tumor resistance, antigen presentation, immune-cell recruitment, and cytotoxic killing.

Methods: We integrated public bulk RNA sequencing, single-cell RNA sequencing, peripheral immune and T-cell receptor data, and spatial transcriptomics within a delivery-trafficking-resistance-killing (DTR-K) framework. Six biological modules were mapped to a transport-coupled tumor-effector model: drug access and tumor-microenvironment barriers, trafficking and lysosomal handling, ABC-transporter resistance, antigen processing and HLA visibility, T/NK cytotoxic synapse, and suppressive microenvironment and exhaustion. Module scoring, compartment-resolved analysis, spatial archetyping, model-parameter mapping, transient simulation, and counterfactual decomposition were performed.

Results: In bulk tumors, the T/NK cytotoxic-synapse module showed the largest responder-associated effect (Cohen's d = 0.998), while antigen/HLA showed a moderate-to-large effect (d = 0.795). Single-cell and peripheral immune analyses localized DTR-K signals to CD8 T-cell, macrophage, endothelial-cell, and circulating effector compartments. Spatial analysis classified 127 regions of interest into immune-recognition-high, drug-handling/resistance-high, barrier/access-high, suppressive-microenvironment-high, and mixed-low-signal states. Integrated mapping ranked antigen/HLA and the T/NK cytotoxic synapse as the dominant c1-linked immune-recognition axis. Across 288 transport-coupled simulations, c1 and treatment-induced immune recruitment (b3) reduced terminal tumor burden in all evaluated contexts, whereas transport formulation altered the predicted depth of control.

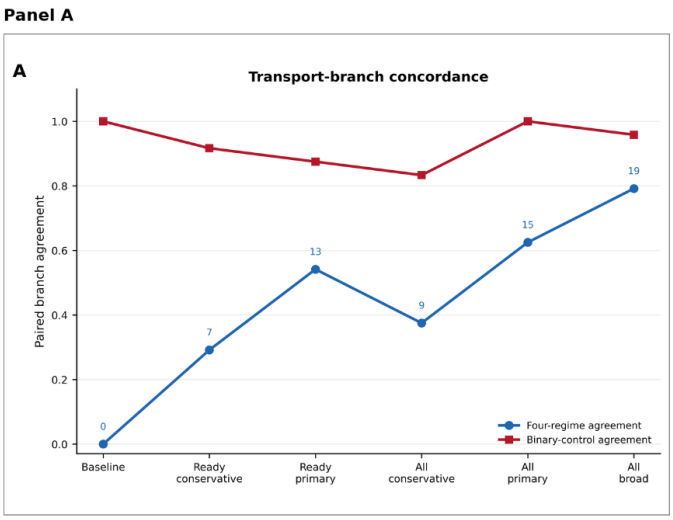
Conclusions: Integrating omics, biophysical transport, and mathematical modeling exposes a coordinated chemoimmunotherapy response architecture in which tumor visibility, effector competence, delivery, intracellular handling, and resistance jointly shape tumor control. The framework provides biologically organized targets for model calibration, experimental validation, and intervention design.

Keywords: chemoimmunotherapy; non-small-cell lung cancer; tumor microenvironment; single-cell RNA sequencing; spatial transcriptomics; biophysical transport; mathematical modeling; drug resistance

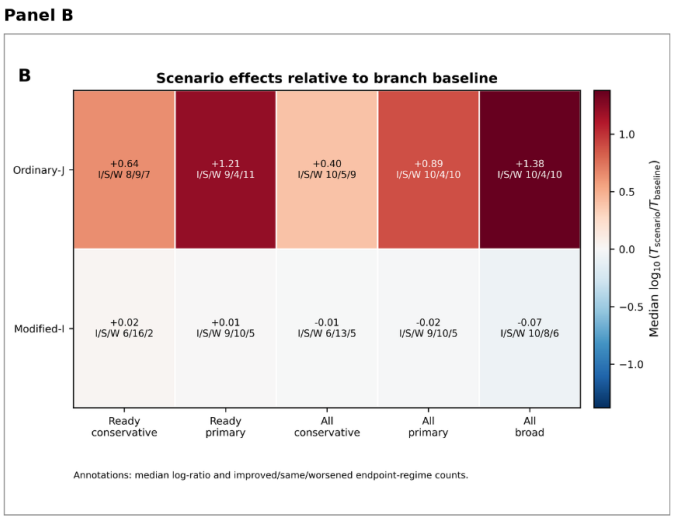
INTERDISCIPLINARY STUDY FIGURE

Omics, transport biophysics, tumor-effector dynamics, and response modeling

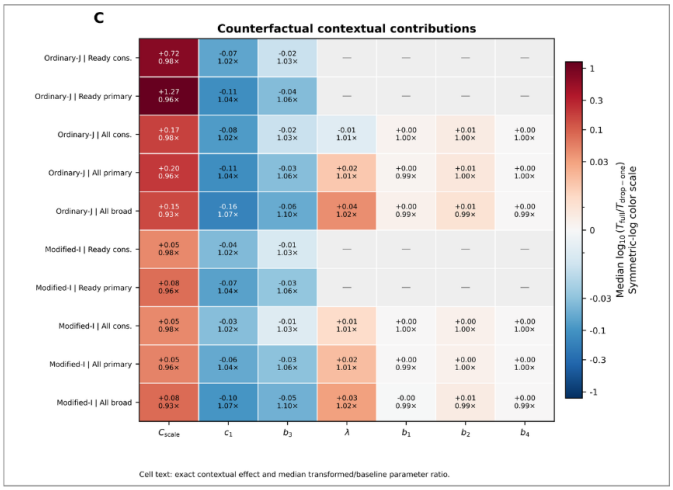
Panel A



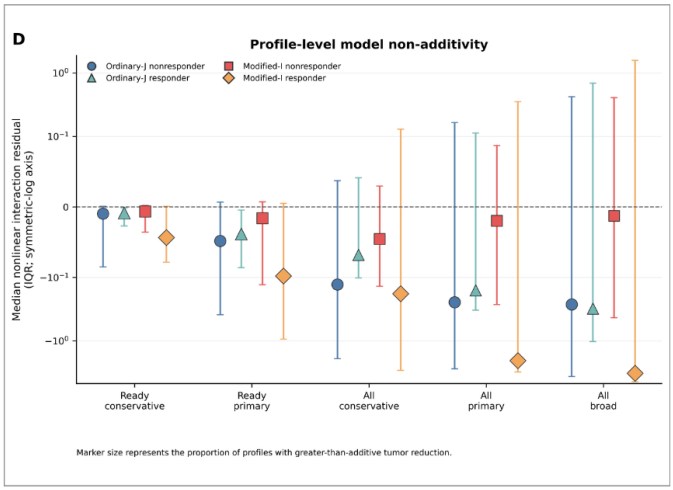
Panel B



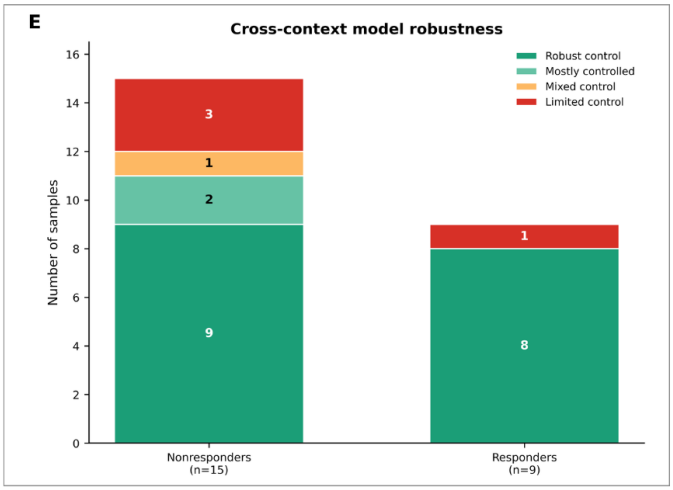
Panel C



Panel D



Panel E



Panel F

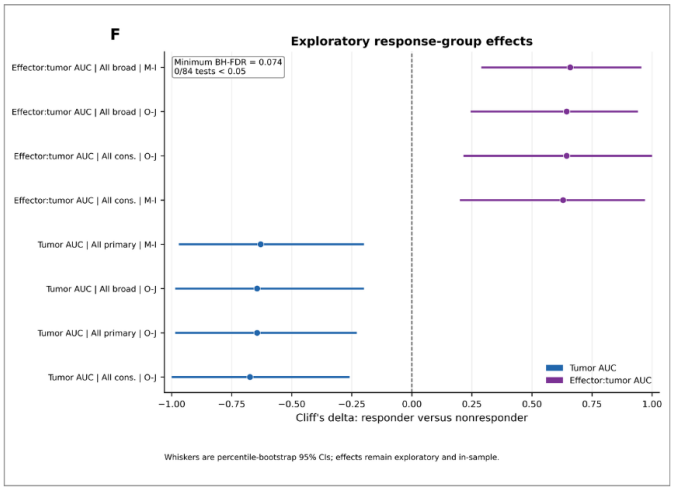


Figure. The panels integrate omics-informed parameter perturbation, drug-transport biophysics, tumor-effector dynamics, counterfactual decomposition, model robustness, and response-group patterns to examine chemoimmunotherapy response heterogeneity.