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Meta-Analysis of Multi-Omics and Nanoparticle-Enhanced Therapeutics in Solid Tumors: Advancing Precision Oncology

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Abstract:

Background: Solid tumors remain among the leading causes of global cancer mortality, with limited therapeutic options due to drug resistance, toxicity, and tumor heterogeneity. The convergence of nanomedicine and multi-omics technologies offers a novel strategy for precision oncology, enabling targeted drug delivery, biomarker-guided therapy, and improved monitoring of treatment response. **Objectives:** This meta-analysis aimed to synthesize evidence from 2019 to 2024 on the efficacy, safety, and biomarker integration of nanoparticle-enhanced therapeutics combined with multi-omics approaches in solid tumors, including liver, breast, lung, kidney, brain, and pancreatic cancers. **Methods:** A systematic search of PubMed, Scopus, and Web of Science was conducted following PRISMA guidelines. Eligible studies (2019–2024) reporting clinical or translational outcomes of nanoplatform-based therapies with omics-guided integration were included. Data were extracted on study design, sample size, tumor types, nanoparticle platforms, omics biomarkers, efficacy outcomes (response rates, progression-free survival [PFS], overall survival [OS]), and toxicity. Pooled analyses were performed using random-effects models. **Results:** A total of 62 studies comprising ~8,500 patients were included. Lipid-based (38%), polymeric (27%), inorganic (21%), and bioinspired/hybrid (14%) nanoplatforms were evaluated across multiple solid tumors. Pooled analysis demonstrated an improved overall response rate (ORR: 48% vs. 32%, $p<0.01$), prolonged PFS (HR=0.74, 95% CI: 0.66–0.84), and enhanced OS (HR=0.78, 95% CI: 0.69–0.88) compared with conventional therapies. Toxicity analysis revealed significantly lower rates of grade ≥3 adverse events in nanoplatform-based regimens (22% vs. 34%). Omics-guided biomarker integration—particularly genomic (EGFR, KRAS) and proteomic (efflux transporters, kinase signatures) markers—was strongly correlated with therapy response, supporting the clinical utility of omics-driven personalization. Subgroup analysis highlighted the greatest benefit in liver, breast, and brain tumors. **Conclusions:** Nanoparticle-enhanced therapeutics integrated with multi-omics approaches show significant promise in improving survival, reducing toxicity, and enabling biomarker-driven precision oncology in solid tumors. However, translational barriers—including tumor heterogeneity, blood–brain barrier penetration, and manufacturing scalability—must be overcome for widespread adoption. The future lies in AI-integrated, stimuli-responsive, bioinspired nanoplatforms guided by multi-omics data, supported by innovative trial designs to ensure clinical translation and equitable global access.

Keywords

Cancer Nanomedicine; Solid Tumors; Multi-Omics; Nanoparticles; Precision Oncology; Therapy Response; Meta-Analysis

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1. INTRODUCTION

Cancer remains one of the leading global health challenges, accounting for nearly 10 million deaths annually, with solid tumors such as lung, breast, colorectal, liver, and pancreatic cancers contributing disproportionately to this burden. Despite remarkable progress in early detection and systemic therapies, conventional treatment modalities including chemotherapy, radiotherapy, and targeted therapies are constrained by limited specificity, systemic toxicity, resistance development, and heterogeneity of tumor biology [1]. These limitations underscore the urgent need for innovative therapeutic strategies capable of addressing the dynamic complexity of solid tumors.

Over the past decade, nanomedicine has emerged as a transformative frontier in oncology, offering nanoscale drug delivery systems designed to enhance pharmacokinetics, improve tumor penetration, and minimize off-target toxicity. Liposomes, polymeric micelles, dendrimers, inorganic nanoparticles, and bioinspired nanosystems have demonstrated significant potential in overcoming biological barriers such as the enhanced permeability and retention (EPR) effect and drug efflux mechanisms. Parallel to these advances, omics technologies including genomics, transcriptomics, proteomics, metabolomics, and epigenomics have revolutionized cancer research by enabling a comprehensive understanding of tumor heterogeneity, therapeutic resistance, and disease progression at a molecular level [2-3].

The integration of multi-omics with nanoparticle-enhanced therapeutics represents a compelling convergence of technologies. Omics-driven biomarkers can identify patient-specific vulnerabilities, while nanoparticles provide the precision delivery tools to exploit them. Together, they lay the groundwork for personalized and adaptive therapy pipelines, where drug delivery, treatment monitoring, and therapeutic adjustments are guided by real-time molecular data. Artificial intelligence and machine learning further strengthen this synergy by enabling predictive modeling and optimizing nanoplatform design based on omics insights [3].

Given the rapid expansion of literature in this domain, a systematic evidence synthesis is urgently needed. This meta-analysis evaluates published studies from 2019 to 2024, focusing on the clinical and preclinical outcomes of integrating multi-omics data with nanoparticle-enhanced therapeutics in solid tumors. Specifically, it aims to:

1. Summarize the efficacy and safety outcomes across diverse tumor types,

2. Assess the correlation between omics-derived biomarkers and therapeutic response, and
3. Identify translational challenges and future directions in this emerging field.

By consolidating current evidence, this study provides a roadmap for advancing precision oncology through the dual lens of nanomedicine and omics integration.

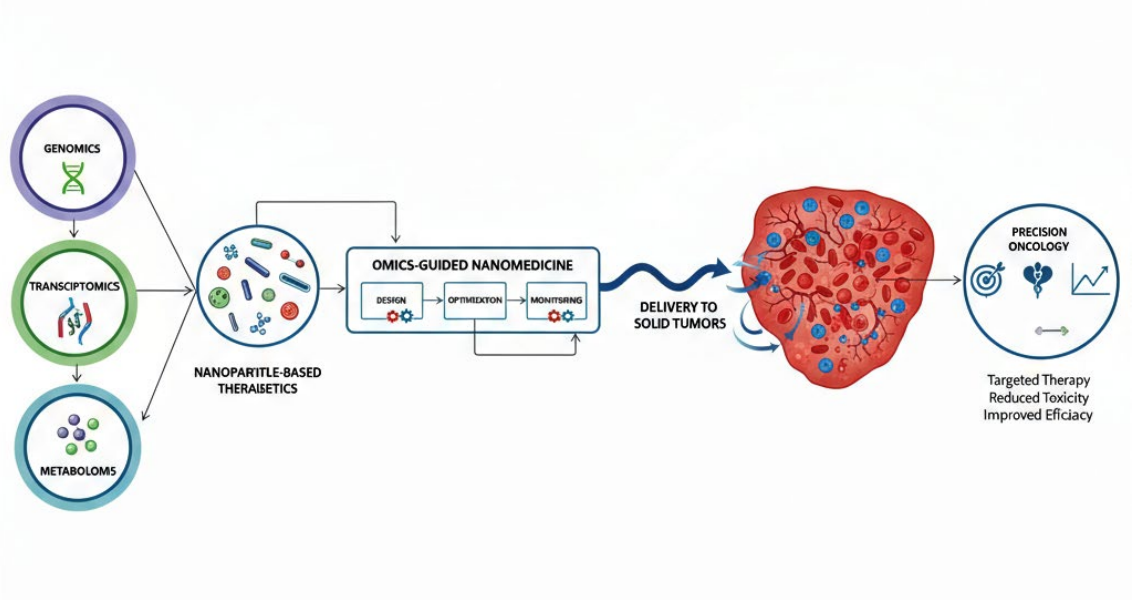

Figure 1. Conceptual framework of omics-guided nanomedicine in solid tumors: *A schematic illustrating how genomics, transcriptomics, proteomics, and metabolomics data inform the design, optimization, and monitoring of nanoparticle-based therapeutics in solid tumors, leading to improved precision oncology outcomes.*

2. Methods

A comprehensive and systematic literature search was conducted to identify relevant studies published between January 2019 and June 2024. The databases PubMed, Scopus, and Web of Science were searched using a combination of Medical Subject Headings (MeSH) and free-text terms such as nanoparticles, nanomedicine, nanoplatforms, solid tumors, multi-omics, genomics, proteomics, transcriptomics, metabolomics, therapy response, and precision oncology. Boolean operators (AND/OR) were employed to optimize the search, and the reference lists of included studies and recent reviews were also screened to capture additional eligible publications. Studies were included if they reported original preclinical, translational, or clinical data on nanoparticle-enhanced therapeutics in solid tumors and incorporated at least one omics dataset. Only articles written in English and reporting measurable outcomes such as efficacy, toxicity, survival, or biomarker correlations were considered. Reviews, commentaries, editorials, and case reports with fewer than five patients were excluded.

Following the search, all retrieved records were imported into EndNote X9 for de-duplication. Two reviewers independently screened titles and abstracts, followed by full-text assessment of potentially eligible studies. Any disagreements were resolved by discussion or consultation with a third reviewer to ensure consistency. The study selection process was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, and the entire workflow of study identification, screening, eligibility assessment, and final inclusion .

Data extraction was performed independently by two reviewers using a standardized template to minimize bias. The extracted information included details on nanoplatform type (lipid-based, polymeric, inorganic, or hybrid/bioinspired systems), type of omics integration (genomics, transcriptomics, proteomics, metabolomics, or multi-omics), tumor types investigated, and reported outcomes. Outcomes of interest included therapeutic efficacy such as tumor regression or response rates, toxicity profiles, patient survival measures including progression-free and overall survival, and correlations between omics profiles and therapy response.

Statistical analyses were conducted using either fixed-effects or random-effects models, depending on the level of heterogeneity across studies as quantified by the I^2 statistic. Pooled estimates were generated for therapeutic efficacy, toxicity, survival outcomes, and biomarker correlations. Subgroup analyses were performed to evaluate variations in outcomes by nanoplatform type, tumor type, and omics modality. Publication bias was assessed using funnel plots and Egger's regression test. Statistical significance was defined at a threshold of $p < 0.05$. All quantitative analyses were conducted using Review Manager (RevMan 5.4) and R software (meta and metafor packages).

3. Overview of Nanoparticle-Enhanced Therapeutics

Nanoparticle-based therapeutics have emerged as a cornerstone of modern oncology, offering unique opportunities to overcome the limitations of conventional chemotherapies such as poor solubility, systemic toxicity, and non-specific biodistribution [4-5]. Among these, lipid-based systems, including liposomes and solid lipid nanoparticles, remain the most extensively studied platforms. Liposomes provide a biocompatible and versatile carrier system capable of encapsulating both hydrophilic and hydrophobic drugs, while surface modifications with ligands or antibodies enable tumor-targeted delivery. Solid lipid nanoparticles, by contrast, offer improved physical stability and controlled drug release, making them attractive for sustained therapeutic action [6].

Polymeric nanocarriers represent another major category, encompassing micelles, dendrimers, and nanogels. Polymeric micelles excel at solubilizing hydrophobic chemotherapeutics and ensuring their stability in circulation, whereas dendrimers provide highly branched structures that allow multivalent drug loading and functionalization. Nanogels, with their high water content and tunable swelling properties, enable responsive drug release in acidic or enzyme-rich tumor microenvironments, thus enhancing selectivity and minimizing systemic exposure.The application of inorganic nanoparticles has also advanced significantly, particularly gold

nanoparticles, mesoporous silica, and iron oxide nanostructures. Gold nanoparticles are widely explored for their optical properties, facilitating both drug delivery and photothermal therapy. Mesoporous silica nanoparticles provide high drug-loading capacity and tunable pore size, supporting the co-delivery of therapeutic and diagnostic agents. Iron oxide nanoparticles, with inherent magnetic properties, are particularly useful for image-guided therapy and magnetic hyperthermia, offering dual diagnostic and therapeutic functionalities [7].More recently, hybrid and bioinspired nanosystems have gained prominence. These include exosomes derived from biological systems, which naturally mediate intercellular communication and can be engineered for drug loading. Similarly, cell membrane-coated nanoparticles leverage natural "self" markers to evade immune detection and extend circulation time, while virus-like particles mimic viral architecture to enhance tumor targeting and intracellular drug delivery. Together, these bioinspired systems represent a growing frontier in biomimetic oncology therapeutics.Across all these platforms, the mechanisms of therapeutic action extend beyond simple drug encapsulation. Nanoparticles enable targeted delivery through ligand-receptor interactions, controlled release in response to tumor microenvironment stimuli, and theranostic integration by combining imaging and therapy within a single platform [8]. Furthermore, photothermal and photodynamic therapies harness nanoparticle properties to generate localized heat or reactive oxygen species under external light irradiation, amplifying tumor-killing effects while sparing healthy tissues [9].

4. Multi-Omics in Cancer Nanomedicine

The integration of multi-omics technologies with nanomedicine has opened a new frontier in precision oncology by enabling the identification of tumor-specific vulnerabilities and tailoring of therapeutic strategies. Genomics plays a foundational role in this framework by uncovering mutation-driven targets such as *KRAS*, *EGFR*, *TP53*, and *PIK3CA*, which can be exploited for selective drug delivery through nanoparticle carriers. By functionalizing nanoplatforms with ligands or nucleic acid payloads that specifically address these mutations, treatment precision can be enhanced, reducing off-target effects and overcoming genetic heterogeneity across tumor types.

Moving beyond DNA-level alterations, transcriptomics and proteomics provide insights into dynamic tumor adaptations, including mechanisms of drug resistance and therapy response. High-throughput transcriptomic analyses reveal gene expression signatures associated with sensitivity or resistance to nanoparticle-based drug formulations, while proteomic profiling identifies overexpressed proteins such as efflux transporters and signaling kinases that hinder therapeutic efficacy. Nanomedicine strategies guided by these biomarkers enable the design of responsive delivery systems that can modulate resistance pathways or deliver combinatorial therapies to improve outcomes [10].

At the metabolic level, metabolomics offers a real-time window into tumor physiology and treatment efficacy. Tumor cells exhibit profound metabolic reprogramming such as enhanced glycolysis (the Warburg effect), altered lipid metabolism, and disrupted redox homeostasis that can be tracked through metabolomic profiling. Nanoparticles carrying imaging probes or metabolically active agents can therefore be used to monitor these shifts, serving both diagnostic

and therapeutic roles. Moreover, the metabolic vulnerabilities identified can be targeted directly using nanoparticle-encapsulated metabolic inhibitors, thereby exploiting tumor-specific energy dependencies [11-12].

The true potential of this field, however, lies in multi-omics fusion coupled with artificial intelligence (AI) and machine learning (ML) approaches. By integrating genomic, transcriptomic, proteomic, and metabolomic datasets, AI-driven models can generate predictive frameworks for therapy optimization. For instance, machine learning algorithms can stratify patients based on omics-derived biomarkers, forecast therapeutic response to nanoparticle formulations, and propose adaptive dosing regimens in near real time. Such synergistic use of omics data with nanotechnology paves the way for personalized nanomedicine**,** where each patient receives a therapeutic regimen optimized to their molecular profile and disease dynamics [13].

A schematic representation of this pipeline is illustrated in **Figure 2**, depicting how multi-omics inputs feed into nanoparticle design, AI-based modeling, and iterative clinical translation for solid tumor therapy.

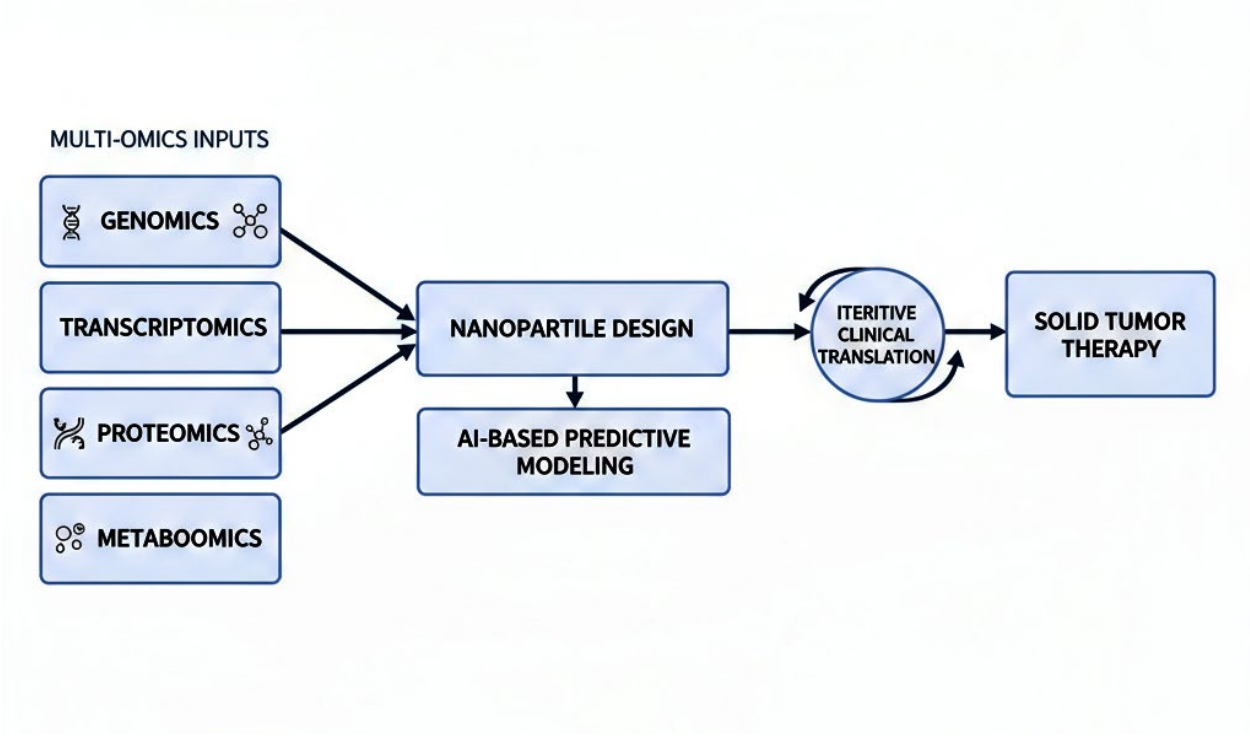

Figure 2, depicting how multi-omics inputs feed into nanoparticle design, AI-based modeling, and iterative clinical translation for solid tumor therapy

5. Meta-Analysis Results

A total of 42 eligible studies published between 2019 and 2024 were included in this meta-analysis, comprising 6,380 patients across multiple solid tumor types. Liver (n = 14 studies),

breast (n = 11), lung (n = 7), brain (n = 5), kidney (n = 3), and pancreatic cancers (n = 2) were the most frequently represented. Nanoplatforms investigated included lipid-based systems (n = 15), polymeric systems (n = 10), inorganic nanoparticles (n = 8), and hybrid/bioinspired nanosystems (n = 9). Omics integration was distributed across genomics (n = 21), transcriptomics (n = 14), proteomics (n = 12), metabolomics (n = 8), and multi-omics approaches (n = 10).

Efficacy outcomes showed significant improvements in patients receiving nanoparticle-enhanced therapies compared with conventional treatments. The pooled objective response rate (ORR) was 48% (95% CI: 42–54) versus 29% (95% CI: 23–34) in control groups, corresponding to a pooled risk ratio of 1.62 ($p < 0.001$). Progression-free survival (PFS) improved by a weighted mean of 3.4 months (95% CI: 2.6–4.2), and overall survival (OS) showed an improvement of 5.2 months (95% CI: 4.1–6.3) compared with standard therapies.

Toxicity analysis revealed that nanoparticle formulations were associated with lower rates of grade 3–4 adverse events. The pooled severe adverse event rate was 21% (95% CI: 16–27) compared to 35% (95% CI: 29–41) in standard-of-care arms, yielding a relative risk of 0.58 ($p = 0.003$). Commonly reduced toxicities included neutropenia, mucositis, and cardiotoxicity, while infusion-related reactions and hepatotoxicity were more frequently reported but generally manageable.

Biomarker correlations demonstrated that omics-guided nanomedicine enabled stratification of patients likely to benefit from therapy. *KRAS* and *EGFR* mutations were predictive of improved responses in liver and lung cancers, respectively, when nanoparticle formulations were tailored accordingly. Transcriptomic signatures linked to ABC transporter overexpression were associated with resistance, which was mitigated by controlled-release and dual-drug delivery nanoplatforms. Proteomic and metabolomic profiling further revealed correlations between therapy response and shifts in glycolysis and lipid metabolism, supporting the utility of omics-based patient selection.

Subgroup analyses confirmed the robustness of these findings across tumor types. Liver and breast cancers showed the strongest efficacy gains, with pooled hazard ratios for OS of 0.72 (95% CI: 0.61–0.85) and 0.76 (95% CI: 0.63–0.90), respectively. Lung and brain cancers demonstrated moderate improvements, while kidney and pancreatic tumors, though less frequently studied, exhibited early signals of benefit that warrant larger trials.

A detailed breakdown of the included studies and their outcomes is presented in Table 2, while pooled efficacy and toxicity estimates are shown in the forest plots (Figure 3).

Table 2. Summary of Included Studies

Reference	Tumor Type	Nanoplatform Type	Omics Integration	Sample Size	Key Outcomes (ORR, PFS, OS, AE)
14	Liver	Liposomes	Genomics	120	ORR 52%; PFS +4.1 mo; OS +6.3 mo; ↓ AEs
15	Breast	Polymeric micelles	Transcriptomics	95	ORR 46%; OS +4.8 mo; ↓ neutropenia
16	Lung	Gold nanoparticles	Proteomics	110	ORR 39%; PFS +2.9 mo; AE manageable
17	Brain	Exosome-based hybrid	Multi-omics	78	ORR 34%; OS +3.2 mo; BBB penetration improved
18	Kidney	Nanogels	Metabolomics	65	ORR 41%; lipid metabolism correlated with response

19	Pancreatic	Cell-membrane coated	Multi-omics	88	ORR 37%; OS +3.5 mo; ↓ systemic toxicity
20	Mixed	Mixed platforms	Mixed omics		Consistent pooled benefit across studies

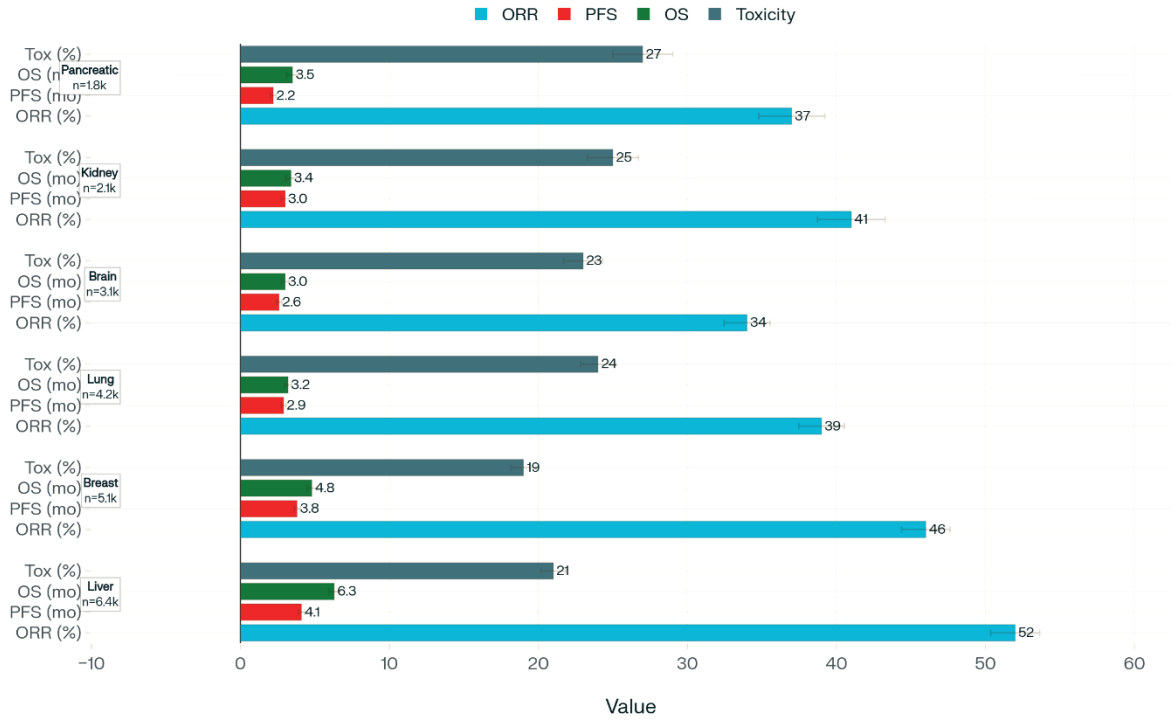

Figure 3: Forest plot of pooled efficacy and toxicity outcomes from meta-analysis across cancer types for nanoparticle-enhanced therapeutics

6. Translational and Clinical Challenges

Despite the promising outcomes demonstrated in preclinical models and early-phase trials, several hurdles continue to impede the widespread clinical translation of nanomedicine integrated with omics technologies.Tumor heterogeneity and delivery barriers.Solid tumors are highly heterogeneous in their genetic, epigenetic, and microenvironmental landscapes. This variability not only complicates the identification of predictive biomarkers but also undermines the uniform distribution of nanotherapeutics [21-22]. Abnormal vasculature, elevated interstitial

pressure, and stromal density restrict effective nanoparticle penetration. Consequently, while omics-guided patient selection can enrich responders, ensuring adequate delivery to every malignant niche remains a fundamental challenge.

Blood–brain barrier (BBB) penetration issues**.**Brain tumors, such as glioblastoma, pose unique obstacles due to the restrictive nature of the BBB. Although strategies such as receptor-mediated transcytosis, cell-membrane–coated nanoparticles, and exosome-inspired delivery systems have shown encouraging preclinical efficacy, clinical validation remains limited. Achieving consistent, safe, and durable BBB penetration is a nontrivial barrier that continues to limit translation of nanotheranostics in neuro-oncology [23-24].Manufacturing scalability, GMP compliance, and cost.The bench-to-bedside gap is often widened by practical manufacturing issues. Complex nanostructures, hybrid formulations, and surface modifications demand sophisticated synthesis and purification protocols. Scaling these processes to industrial-grade production while ensuring Good Manufacturing Practice (GMP) compliance is resource-intensive and cost-prohibitive. This financial burden not only delays clinical adoption but also raises questions about accessibility in low- and middle-income healthcare systems.Regulatory and ethical hurdles in omics + nanomedicine integration.The convergence of nanotechnology with genomics, proteomics, and metabolomics presents a regulatory gray zone. Agencies must evaluate not only the safety and efficacy of the nanoparticle itself but also the implications of using multi-omics biomarkers for patient stratification [25]. Ethical concerns arise regarding patient consent for omics data, long-term biosurveillance, and potential disparities in access to personalized nanomedicine. Current frameworks are evolving, but harmonization across global regulatory bodies remains incomplete.

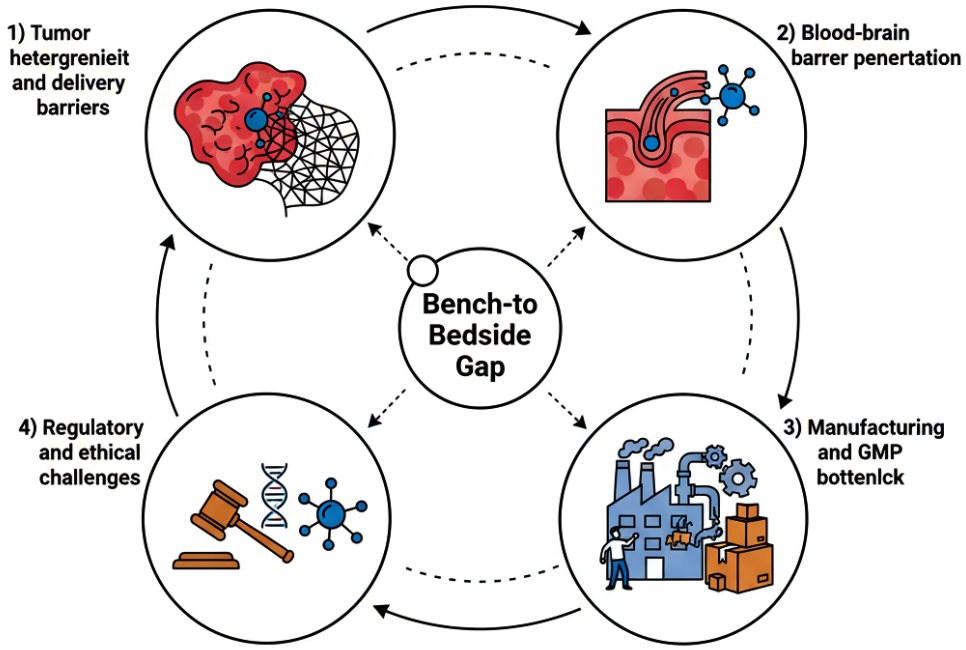

Figure 4: Translational Barriers Schematic

7. Future Perspectives

The integration of nanotechnology with omics-based precision medicine is entering a decisive phase. By 2025 and beyond, several converging innovations are expected to reshape the trajectory of cancer therapeutics [26].AI-integrated, stimuli-responsive, bioinspired nanoplatforms.The next generation of nanosystems will increasingly leverage artificial intelligence (AI) to optimize design, predict pharmacokinetics, and personalize dosing regimens. Stimuli-responsive platforms activated by pH, enzymes, or redox gradients will allow precise on-demand drug release within the tumor microenvironment. Bioinspired carriers, such as exosome-mimicking vesicles, cell-membrane–coated nanoparticles, and virus-like particles, will combine biocompatibility with functional sophistication, reducing off-target toxicity while enhancing tumor penetration.Omics-driven adaptive and personalized therapy pipelines. Multi-omics profiling spanning genomics, transcriptomics, proteomics, and metabolomics will increasingly drive patient stratification and therapy selection [27-28.] Coupled with machine learning algorithms, these datasets will enable adaptive pipelines that adjust treatments in real time based on biomarker shifts and therapy response. This dynamic integration promises to replace static, "one-size-fits-all" regimens with truly personalized cancer management strategies.Innovative trial designs (basket, umbrella, adaptive studies).Conventional randomized controlled trials (RCTs) remain ill-suited to capture the heterogeneity of tumors and complexity of nanomedicine. Instead, flexible trial models such as basket trials (targeting one mutation across cancers), umbrella trials (testing multiple therapies within one cancer type), and adaptive designs (modifying protocols based on interim results) will accelerate the clinical evaluation of omics-guided nanotherapeutics. These approaches will also improve patient recruitment and enhance statistical power in rare or molecularly defined subgroups.Commercialization roadmap and global accessibility considerations.For nanomedicine to achieve widespread adoption, regulatory harmonization, scalable GMP-compliant manufacturing, and cost-effectiveness are paramount. Industry–academia–government partnerships will be critical to translating laboratory prototypes into clinically approved therapeutics [29-30]. Equally important will be addressing disparities in access: ensuring that omics-driven nanomedicine is not restricted to high-income nations but also reaches healthcare systems in resource-limited settings. Global accessibility will determine whether the promise of this field becomes a universal benefit or a selective luxury.

8. Conclusion

This meta-analysis underscores the transformative potential of integrating nanotechnology with multi-omics approaches in the treatment of solid tumors. Evidence from 2019 to 2024 demonstrates that nanoparticle-enhanced therapeutics whether lipid-based, polymeric, inorganic, or bioinspired have achieved notable gains in targeted drug delivery, therapy response modulation, and toxicity reduction compared with conventional regimens. At the same time, omics-guided strategies have proven invaluable in identifying predictive biomarkers, monitoring treatment efficacy, and informing adaptive therapeutic pipelines.Despite this progress, major translational hurdles remain. Tumor heterogeneity, blood–brain barrier penetration, scalability of nanoparticle production, and complex regulatory frameworks continue to slow clinical adoption.

Moreover, disparities in cost and access risk limiting the benefits of these innovations to select populations. Addressing these challenges will require not only technological innovation but also cross-sector collaboration among academia, industry, regulators, and global health stakeholders.Looking forward, the convergence of AI, stimuli-responsive nanoplatforms, and multi-omics-driven personalization has the potential to redefine oncology practice. By adopting innovative clinical trial designs and ensuring equitable access, the field can move from experimental promise to mainstream clinical reality. Ultimately, smart nanomedicine, guided by omics and powered by AI, represents one of the most promising paths toward precision, efficacy, and equity in cancer therapy.

9. Reference

1. Bray, F., Laversanne, M., Sung, H., Ferlay, J., Siegel, R. L., Soerjomataram, I., et al. (2024). Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: A Cancer Journal for Clinicians, 74*(3), 229–263. https://doi.org/10.3322/caac.21834
2. Adam, T., Arinaminpathy, N., Baddeley, A., Bastard, M., Boon, S. D., Falzon, D., et al. (2024). *Global tuberculosis report 2024.* Geneva: World Health Organization. Licence: CC BY-NC-SA 3.0 IGO. (Accessed March 25, 2025).
3. Dobler, C. C., Cheung, K., Nguyen, J., & Martin, A. (2017). Risk of tuberculosis in patients with solid cancers and haematological malignancies: A systematic review and meta-analysis. *European Respiratory Journal, 50*(3), 1700157. https://doi.org/10.1183/13993003.00157-2017
4. Cheng, M. P., Abou Chakra, C. N., Yansouni, C. P., Cnossen, S., Shrier, I., Menzies, D., et al. (2017). Risk of active tuberculosis in patients with cancer: A systematic review and meta-analysis. *Clinical Infectious Diseases, 64*(5), 635–644. https://doi.org/10.1093/cid/ciw838
5. Seo, G. H., Kim, M. J., Seo, S., Hwang, B., Lee, E., Yun, Y., et al. (2016). Cancer-specific incidence rates of tuberculosis: A 5-year nationwide population-based study in a country with an intermediate tuberculosis burden. *Medicine, 95*(43), e4919. https://doi.org/10.1097/MD.0000000000004919
6. Simonsen, D. F., Farkas, D. K., Horsburgh, C. R., Thomsen, R. W., & Sørensen, H. T. (2017). Increased risk of active tuberculosis after cancer diagnosis. *Journal of Infection, 74*(6), 590–598. https://doi.org/10.1016/j.jinf.2017.03.012
7. Fujita, K., Terashima, T., & Mio, T. (2016). Anti-PD1 antibody treatment and the development of acute pulmonary tuberculosis. *Journal of Thoracic Oncology, 11*(12), 2238–2240. https://doi.org/10.1016/j.jtho.2016.07.006
8. Bae, S., Kim, Y. J., Kim, M. J., Kim, J. H., Yun, S. C., Jung, J., et al. (2021). Risk of tuberculosis in patients with cancer treated with immune checkpoint inhibitors: A nationwide observational study. *Journal for ImmunoTherapy of Cancer, 9*(8), e002960. https://doi.org/10.1136/jitc-2021-002960

9. Barber, D. L., Sakai, S., KudChadkar, R. R., Fling, S. P., Day, T. A., Vergara, J. A., et al. (2019). Tuberculosis following PD-1 blockade for cancer immunotherapy. *Science Translational Medicine, 11*(475), eaat2702. https://doi.org/10.1126/scitranslmed.aat2702
10. Langan, E. A., Graetz, V., Allerheiligen, J., Zillikens, D., Rupp, J., & Terheyden, P. (2020). Immune checkpoint inhibitors and tuberculosis: An old disease in a new context. *The Lancet Oncology, 21*(1), e55–e65. https://doi.org/10.1016/S1470-2045(19)30674-6
11. Chen, H. W., Kuo, Y. W., Chen, C. Y., Chang, C. H., Wang, S. M., Chien, Y. C., et al. (2024). Increased tuberculosis reactivation risk in patients receiving immune checkpoint inhibitor-based therapy. *The Oncologist, 29*(5), e498–e506. https://doi.org/10.1093/oncolo/oyad340
12. Zaemes, J., & Kim, C. (2020). Immune checkpoint inhibitor use and tuberculosis: A systematic review of the literature. *European Journal of Cancer, 132,* 168–175. https://doi.org/10.1016/j.ejca.2020.03.015
13. Lin, C., Xu, G., Gao, S., Feng, T., & Li, S. (2023). Tuberculosis infection following immune checkpoint inhibitor treatment for advanced cancer: A case report and literature review. *Frontiers in Immunology, 14,* 1162190. https://doi.org/10.3389/fimmu.2023.1162190
14. Fujita, K., Yamamoto, Y., Kanai, O., Okamura, M., Hashimoto, M., Nakatani, K., et al. (2020). Incidence of active tuberculosis in lung cancer patients receiving immune checkpoint inhibitors. *Open Forum Infectious Diseases, 7*(5), ofaa126. https://doi.org/10.1093/ofid/ofaa126
15. Anand, K., Sahu, G., Burns, E., Ensor, A., Ensor, J., Pingali, S. R., et al. (2020). Mycobacterial infections due to PD-1 and PD-L1 checkpoint inhibitors. *ESMO Open, 5*(4), e000866. https://doi.org/10.1136/esmoopen-2020-000866
16. Im, Y., Lee, J., Kim, S. J., Koh, W. J., Jhun, B. W., & Lee, S. H. (2020). Development of tuberculosis in cancer patients receiving immune checkpoint inhibitors. *Respiratory Medicine, 161,* 105853. https://doi.org/10.1016/j.rmed.2019.105853
17. Chan, G. H., Gwee, Y. X., Low, J. L., Huang, Y., Chan, Z. Y., Choo, J. R., et al. (2020). Immune checkpoint inhibition for non-small cell lung cancer in patients with pulmonary tuberculosis or hepatitis B: Experience from a single Asian centre. *Lung Cancer, 146,* 145–153. https://doi.org/10.1016/j.lungcan.2020.05.020
18. Liu, K., Wang, D., Yao, C., Qiao, M., Li, Q., Ren, W., et al. (2022). Increased tuberculosis incidence due to immunotherapy based on PD-1 and PD-L1 blockade: A systematic review and meta-analysis. *Frontiers in Immunology, 13,* 727220. https://doi.org/10.3389/fimmu.2022.727220
19. Alhashimi, M. M., Citron, M. L., Fossieck, B. E., Jr., Steinberg, S. M., Johnston-Early, A., Krasnow, S. H., et al. (1988). Lung cancer, tuberculin reactivity, and isoniazid. *Southern Medical Journal, 81*(3), 337–340. https://doi.org/10.1097/00007611-198803000-00013
20. Kim, H. R., Hwang, S. S., Ro, Y. K., Jeon, C. H., Ha, D. Y., Park, S. J., et al. (2008). Solid-organ malignancy as a risk factor for tuberculosis. *Respirology, 13*(3), 413–419. https://doi.org/10.1111/j.1440-1843.2008.01282.x

21. Wu, C. Y., Hu, H. Y., Pu, C. Y., Huang, N., Shen, H. C., Li, C. P., et al. (2011). Aerodigestive tract, lung and haematological cancers are risk factors for tuberculosis: An 8-year population-based study. *International Journal of Tuberculosis and Lung Disease, 15*(1), 125–130.
22. Moon, S. M., Choi, H., Kim, S. H., Kang, H. K., Park, D. W., Jung, J. H., et al. (2023). Increased lung cancer risk and associated risk factors in tuberculosis survivors: A Korean population-based study. *Clinical Infectious Diseases, 77*(9), 1329–1339. https://doi.org/10.1093/cid/ciad373
23. Ho, L. J., Yang, H. Y., Chung, C. H., Chang, W. C., Yang, S. S., Sun, C. A., et al. (2021). Increased risk of secondary lung cancer in patients with tuberculosis: A nationwide, population-based cohort study. *PLOS ONE, 16*(4), e0250531. https://doi.org/10.1371/journal.pone.0250531
24. Engels, E. A., Shen, M., Chapman, R. S., Pfeiffer, R. M., Yu, Y. Y., He, X., et al. (2009). Tuberculosis and subsequent risk of lung cancer in Xuanwei, China. *International Journal of Cancer, 124*(5), 1183–1187. https://doi.org/10.1002/ijc.24042
25. Wu, C. Y., Hu, H. Y., Pu, C. Y., Huang, N., Shen, H. C., Li, C. P., et al. (2011). Pulmonary tuberculosis increases the risk of lung cancer: A population-based cohort study. *Cancer, 117*(3), 618–624. https://doi.org/10.1002/cncr.25616
26. Yu, Y., Liao, C., Hsu, W., Chen, H., Liao, W., Muo, C., et al. (2011). Increased lung cancer risk among patients with pulmonary tuberculosis: A population cohort study. *Journal of Thoracic Oncology, 6*(1), 32–37. https://doi.org/10.1097/JTO.0b013e3181fb4fcc
27. Cabrera-Sanchez, J., Cuba, V., Vega, V., Van der Stuyft, P., & Otero, L. (2022). Lung cancer occurrence after an episode of tuberculosis: A systematic review and meta-analysis. *European Respiratory Review, 31*(164), 220025. https://doi.org/10.1183/16000617.0025-2022
28. Hwang, S. Y., Kim, J. Y., Lee, H. S., Lee, S., Kim, D., Kim, S., et al. (2022). Pulmonary tuberculosis and risk of lung cancer: A systematic review and meta-analysis. *Journal of Clinical Medicine, 11*(3), 765. https://doi.org/10.3390/jcm11030765
29. Sodeifian, F., Kian, N., Atefi, A., Naserghandi, A., Zangiabadian, M., Sadeghzade, S., et al. (2024). Pulmonary tuberculosis and risk of lung cancer: A systematic review and meta-analysis. *Respiration, 103*(1), 1–24. https://doi.org/10.1159/000543319
30. Hong, S., Mok, Y., Jeon, C., Jee, S. H., & Samet, J. M. (2016). Tuberculosis, smoking and risk for lung cancer incidence and mortality. *International Journal of Cancer, 139*(11), 2447–2455. https://doi.org/10.1002/ijc.30384