WHITE PAPER

The Tumor Microenvironment: A Complex Ecosystem

- Understand how the tumor microenvironment (TME) drives cancer progression, immune evasion and therapeutic resistance
- Explore emerging strategies that target the TME to improve cancer therapy outcomes
- Discover how proteomic technologies provide complementary insights across molecular, cellular and spatial levels
- Learn how characterizing the TME can reveal prognostic biomarkers and mechanisms of treatment response or failure

Introduction

Cancer is a heterogeneous disease, driven not only by the genetic instability and diversity of tumor cells but also by the complexity of the tumor microenvironment (TME)[1]. The TME is a dynamic and multifaceted ecosystem composed of stromal cells, immune infiltrates, extracellular matrix (ECM) components and signaling molecules, all of which interact with tumor cells to influence disease progression[2]. It plays a critical role in shaping tumor behavior by enabling proliferation, promoting resistance to apoptosis, facilitating immune evasion and driving metastasis.

This white paper explores how the TME contributes to cancer development and therapeutic resistance. It also highlights how 4 advanced proteomic technologies provide unique and complementary insights – ranging from high-throughput immunoprofiling to spatial protein mapping – to support a more integrated understanding of this clinically relevant and biologically complex environment (Table 1).

Table 1. Proteomic technologies at Standard BioTools

Technology	Analytical Focus	Sample Type	Description
SomaScan™ Assay	Protein profiling	Biological fluid*	Quantifies up to 11,000 unique human proteins with approximately 5% coefficient of variation (CV).
KREX™ microarrays	Antibody profiling	Biological fluid*	Profiles 1,800-plus antibodies with a false discovery rate (FDR) below 1%.
CyTOF™ technology	Single-cell proteomics	Cells	Measures 50-plus surface and intracellular proteins per cell, the highest multiplexing capability on the market.
Hyperion™ Imaging System [Imaging Mass Cytometry™ (IMC™) technology]	Spatial proteomics	Tissue	Analyzes 40-plus protein markers in any tissue with high spatial resolution and no autofluorescence interference.

* Pre-validated biological fluids include plasma and serum. For information on additional compatible sample types, please **contact us**.

Understanding the TME

The TME is now recognized as a central driver of cancer progression and therapeutic resistance, contributing to a sharp rise in research publications over the past 2 decades (Figure 1). Far from being a passive bystander, the TME evolves alongside the tumor, influencing nearly every stage of its development through both direct and indirect mechanisms (Figure 2)[1, 2].

Composed of non-malignant cells, soluble factors, signaling molecules and structural components, the TME can both support and restrain tumor growth, which is often through immunomodulation (Table 2)[2, 3]. Additional factors such as hypoxia, acidity and nutrient depletion create a metabolically hostile environment that further impairs immune cell function and promotes tumor survival[1].

The ECM is a particularly influential component of the TME[2]. It can serve as a physical barrier that blocks immune cell infiltration and impedes the delivery of therapeutic agents, reducing treatment efficacy. Its biochemical makeup and mechanical properties also facilitate tumor cell migration and invasion, promoting metastatic spread. Additionally, the ECM functions as a reservoir for signaling molecules like TGF-β and VEGF, which are released during remodeling events to further shape both tumor and microenvironmental behavior.

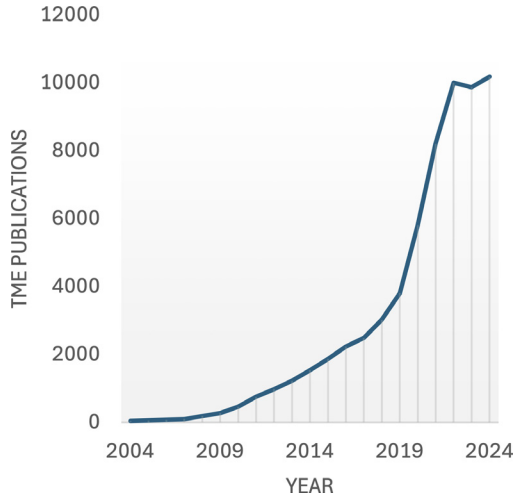

Figure 1. Growth in TME-related publications (2004–2024). The number of scientific publications referencing the TME has increased substantially over the past 2 decades, reflecting rising interest and recognition of its critical role in cancer biology. Source: NCBI PubMed

Even immune cells typically considered antitumor, such as CD8+ T cells, can lose their effectiveness within the TME2. Prolonged exposure to tumor antigens and immunosuppressive signals often drives these cells into an exhausted or dysfunctional state, compromising their cytotoxic activity and enabling immune evasion.

Taken together, the TME is highly dynamic and interactive. Understanding its cellular composition, structural organization and molecular signaling is essential for developing more effective, personalized cancer therapies.

Targeting the TME for cancer treatment

Insights into the TME are fueling the development of more effective cancer therapies, from early-stage discovery and preclinical models to treatments approved by the United States Food and Drug Administration (FDA) (Tables 3, 4) [3–5]. While tumor cells are genetically unstable and capable of rapidly evolving drug resistance, most cells within the TME are more genetically stable[1]. This relative stability makes them less prone to resistance and more amenable to durable therapeutic targeting.

By disrupting key cellular interactions, modulating immune responses or remodeling the ECM, researchers can reprogram the TME to enhance treatment response and limit disease progression. As a result, combination therapies that address both the tumor and its surrounding microenvironment are becoming a foundational strategy in modern oncology.

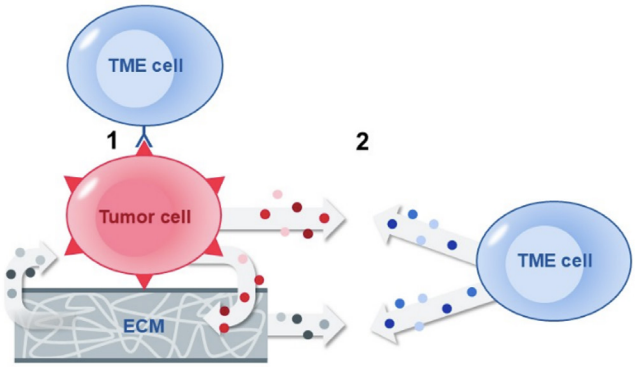

Figure 2. Mechanisms of tumor-TME interactions. Tumor cells and components of the TME, including cells and the ECM, communicate through (1) direct cell-to-cell contact and (2) indirect paracrine signaling. These dynamic interactions regulate key processes such as angiogenesis, cancer stem cell maintenance, ECM remodeling, immune evasion, inflammation, metastasis and response to therapy.

One of the most successful examples is the use of immune checkpoint inhibitors (ICIs). Many tumors overexpress PD-L1, an immune checkpoint protein that binds to the PD-1 receptor on T cells, suppressing immune surveillance[3, 6]. Blocking this interaction with anti-PD-1 or anti-PD-L1 antibodies has produced durable responses in cancers such as melanoma, non-small-cell lung cancer and renal cell carcinoma, leading to multiple FDA approvals (Table 3). Although designed to activate T cells, these therapies are inherently TME-targeting, as they modulate immune activity within the tumor's local environment. However, only about one-third of patients respond favorably to ICIs, underscoring the heterogeneity of the TME and the need for additional or combinatorial therapeutic strategies[6].

In response, researchers are now exploring ways to target other components of the TME, including cancer-associated fibroblasts (CAFs) and tumor-associated macrophages (TAMs)[1, 7]. These efforts reflect a growing consensus: Cancer therapy must consider the tumor ecosystem, not just the tumor itself.

TME insights enabled by advanced proteomics

Understanding the TME requires analyzing its complexity and heterogeneity across multiple biological levels: molecular, cellular and spatial. No single approach can fully capture the diverse interactions between tumor cells and their microenvironment. To address this challenge, recent advances in proteomic technologies now enable researchers to quantify proteins and antibodies in high-plex formats, perform immunoprofiling with greater resolution and examine protein expression within intact tissue

Table 2. Functional roles of major TME components in cancer progression

Component Type	Specific Cell or Molecule	Primary Tumor Effect	Mechanism(s) of Action
Immune cells	T cells	Context-dependent	CD8+ T cells: antitumor (kill cancer cells); regulatory T cells: protumor (immune suppression)
	B cells	Context-dependent	Can present antigens (antitumor) or secrete immunosuppressive cytokines (protumor)
	Tumor-associated macrophages (TAMs)	Context-dependent	M1 phenotype: antitumor (pro-inflammatory); M2 phenotype: protumor (immunosuppressive, pro-angiogenic)
	Natural killer (NK) cells	Antitumor	Direct killing of tumor cells
	Tumor-associated neutrophils (TANs)	Context-dependent	N1: antitumor (cytotoxic, immune-activating); N2: protumor (promote angiogenesis, suppress immunity)
	Dendritic cells (DCs)	Antitumor	Antigen presentation to T cells, activate adaptive immune response
	Myeloid-derived suppressor cells (MDSCs)	Protumor	Suppress T cell activation
Stromal cells	Cancer-associated fibroblasts (CAFs)	Protumor	Remodel ECM; secrete cytokines; and stimulate angiogenesis, tumor formation and metastasis
	Mesenchymal stem cells (MSCs)	Protumor	Immune suppression, differentiate into other pro-tumorigenic stromal components, stimulate angiogenesis
	Pericytes	Protumor	Form and stabilize vasculature, secrete pro-inflammatory cytokines and angiogenic growth factors
Secreted molecules	Cytokines and chemokines	Context-dependent	Can be either pro- or anti-inflammatory, affecting immune response and inflammation
	Growth factors	Protumor	Stimulate angiogenesis, proliferation
	Extracellular vesicles	Protumor	Critical role in tumor-TME crosstalk
ECM	Collagen, fibronectin, laminin	Protumor	Provide structural support, enable migration, store and secrete signaling molecules

architecture. This section demonstrates how 4 proteomic approaches are generating unique insights into the TME, each ultimately contributing to a more integrated systems-level understanding (Tables 1, 5).

Protein profiling defines prognostic signatures

Peritoneal fluid (PF), a key component of the TME in **ovarian cancer**, contains soluble factors that influence tumor progression, chemoresistance and immune evasion[8]. In high-grade serous ovarian carcinoma (HGSC), the most common and lethal subtype, fewer than 20% of patients remain relapse-free for more than 5 years, and no validated prognostic biomarkers currently exist.

To address this gap, Finkernagel et al. used the SomaScan 1.3K Assay to profile PF proteins from 70 HGSC patients[8]. Compared with matched plasma samples, 356 proteins were significantly upregulated in PF, including 45 elevated more than 5-fold. While some of these proteins were already known to be associated with ovarian cancer, the analysis also revealed novel candidates.

Hierarchical clustering identified 2 distinct TME protein expression profiles: one enriched for metastasis-related proteins and associated with poor relapse-free survival (RFS), the other enriched for immune regulatory proteins and linked to longer RFS. Many of the differentially expressed proteins correlated with extracellular vesicle (EV) markers, suggesting a role for EVs in shaping the metastatic niche.

A 9-protein signature derived from the data accurately distinguished long-term (100%) and short-term (85.3%) survivors, underscoring the prognostic potential of high-plex protein profiling in the TME and its value for future biomarker development.

Protein profiling maps immune suppression

In **acute myeloid leukemia (AML)**, disease originates and progresses within the bone marrow, making it essential to understand the local TME. To investigate this niche, Çelik et al. used the SomaScan 1.3K Assay to profile bone marrow plasma from AML patients and matched healthy controls[9]. The analysis revealed 168

Table 3. Anti-PD-1/L1 antibody therapies approved by the FDA

Drug Name	Company	Cancer Types	First Year Approved
Atezolizumab (Tecentriq)	Roche	Bladder, breast, liver, lung, multiple solid cancers, sarcoma, skin	2016
Avelumab (Bavencio)	EMD Serono	Bladder, kidney, skin	2017
Cemiplimab (Libtayo)	Regeneron	Lung, skin	2018
Cosibelimab (Unloxcyt)	Checkpoint Therapeutics	Skin	2024
Dostarlimab (Jemperli)	GlaxoSmithKline	Endometrial	2021
Durvalumab (Imfinzi)	AstraZeneca	Biliary tract, bladder, liver, lung	2017
Nivolumab (Opdivo)	Bristol Myers Squibb	Bladder, colorectal, esophageal, gastric, head and neck, Hodgkin lymphoma, kidney, liver, lung, multiple solid cancers, skin	2014
Pembrolizumab (Keytruda)	Merck	Biliary tract, bladder, breast, cervical, colorectal, endometrial, esophageal, gastric, head and neck, Hodgkin lymphoma, kidney, liver, lung, non-Hodgkin lymphoma, skin	2014
Tislelizumab (Tevimbra)	BeiGene	Esophageal	2024
Toripalimab-tpzi (Loqtorzi)	Coherus Biosciences	Head and neck	2023

differentially expressed proteins, with 102 uniquely altered in the bone marrow, highlighting the distinct molecular profile of the leukemic TME.

Functional enrichment identified a dense cytokine and chemokine signaling network, dominated by IL-8, as a defining feature of the leukemic TME. Downregulated proteins were associated with neutrophil degranulation and platelet function, reflecting known immune impairments in AML.

The study also identified significantly elevated levels of 2 MPIF-1 isoforms (CKβ8 and CKβ8-1), myelosuppressive chemokines not previously associated with myeloid malignancies. Additional findings, including reduced osteonectin and increased CCL3, indicated disrupted bone remodeling and enhanced immune evasion. Overall, the results establish the value of high-plex proteomic profiling in uncovering immune and stromal signaling dynamics within the hematologic TME, providing a foundation for novel biomarker discovery and therapeutic targeting.

Antibody profiling reveals a tolerogenic yet inflamed immune landscape

Autoantibodies (AAbs), or antibodies that target self-molecules, can emerge well before clinical signs of cancer, offering an early window into immune dysregulation within the TME. In a recent study, Maimela et al. used KREX technology to profile AAbs in both serum and tumor tissue lysates from early-stage **pancreatic ductal adenocarcinoma** (PDAC) patients (n=30)[10].

The analysis revealed significantly elevated IgA AAbs in tumor tissue compared with adjacent normal tissue, with a dominant IgA2 subclass profile – suggesting a pro-inflammatory local immune response. In contrast, IgG subclass profiling showed a predominance of IgG4, a non-activating tolerogenic isotype, while the immune-activating subclasses IgG1 and IgG3 were underrepresented. This skewed isotype distribution points to impaired antibody-mediated immune clearance and an immunosuppressive yet inflamed TME.

Importantly, a 12-antigen IgA signature demonstrated high diagnostic accuracy [area under the curve (AUC) = 0.968; sensitivity = 1.00; specificity = 0.833 at 1% FDR], demonstrating the utility of AAb profiling for both early biomarker discovery and monitoring immune regulation in solid tumors.

Table 4. Examples of non-PD-1/L1 therapies approved by the FDA

Target/ Pathway	Therapy Name	Drug Type	Cancer Type(s)	Mechanism of Action
CTLA-4	Ipilimumab (Yervoy)	Antibody	Colorectal, esophageal, kidney, mesothelioma, liver, lung, skin	Blocks CTLA-4, promoting T cell activation
	Tremelimumab-actl (Imjudo)	Antibody	Liver, lung	
VEGF	Bevacizumab (Avastin)	Antibody	Breast, cervical, colorectal, fallopian tube, glioblastoma, kidney, liver, lung, ovarian, primary peritoneal	Inhibits VEGF, preventing angiogenesis
	Ramucirumab (Cyramza)	Antibody	Colorectal, gastric, liver, lung	Targets VEGFR-2, inhibiting angiogenesis
CSF1R	Pexidartinib (Turalio)	Small molecule	Tenosynovial giant cell tumor	Depletes TAMs by inhibiting the tyrosine kinase receptor CSF1R
LAG3	Relatlimab + nivolumab (Opdualag)	Antibody (2)	Skin	Blocks T cell inhibition by binding to LAG-3 and PD-1
BCR	Ibrutinib (Imbruvica)	Small molecule	CLL, SLL, Waldenström's macroglobulinemia	Inhibits B cell survival pathways
	Acalabrutinib (Calquence)	Small molecule	CLL, MCL, SLL	

CLL = chronic lymphocytic leukemia; MCL = mantle cell lymphoma; SLL = small lymphocytic lymphoma

Single-cell proteomics reveals immune remodeling in the TME

Multiple myeloma arises from malignant plasma cells within the bone marrow. Investigating the cellular heterogeneity of bone marrow in multiple myeloma patients is therefore essential to understanding how the TME shapes immune dynamics in this cancer. CyTOF analysis revealed a progressive decline in the CD4+/CD8+ T cell ratio from Stage 1 to Stage 3 disease, reflecting a shift in T cell composition as the TME evolves[11]. Additionally, a marker of naive CD8+ T cells (CD45RA) was significantly downregulated in Stage 3 patients, suggesting increased activation of these cells in advanced disease. Together, these phenotypic changes reveal how the TME influences T cell behavior and may serve as useful indicators of disease activity and progression in multiple myeloma.

Single-cell drug screening links CAF-induced plasticity to therapy resistance

CAFs are the most abundant stromal cell type in the TME and are known to influence therapeutic outcomes, yet their patient-specific effects remain poorly defined. To explore this, Ramos Zapatero et al. co-cultured **colorectal cancer** patient-derived organoids (PDOs) with CAFs to simulate direct stromal-tumor interactions[12]. PDOs serve as personalized models that closely replicate individual treatment responses.

Using CyTOF technology, the researchers profiled over 2,500 co-cultures treated in triplicate with either vehicle control or combinations of standard chemotherapies (5-fluorouracil, SN-38, oxaliplatin) and immunotherapy (cetuximab). The study found that CAFs consistently induced a slow-cycling, chemoresistant “revival stem cell” state in PDOs. Drug-induced apoptosis was rare and highly variable across patients, reflecting unique signaling profiles. The data show how CAF-driven plasticity actively remodels the TME to promote resistance and highlight the importance of incorporating stromal context into high-throughput drug screens to inform personalized treatment strategies.

Single-cell analysis discovers a novel cell subset driving immunotherapy resistance

CD8+ T cell dysfunction is a defining feature of the TME. To explore its origins, Sanmamed et al. analyzed paired tumoral and non-tumoral lung tissue samples from patients with resectable non-small-cell **lung cancer** (NSCLC) (n=25–35) using CyTOF and the Hyperion Imaging System[13].

Their analysis identified a novel subset of hyperactivated yet dysfunctional CD8+ T cells, termed “burned-out” effector (Ebo) cells, that are terminally differentiated, highly proliferative and prone to apoptosis. Expression of LAG-3, a key marker of T cell exhaustion, was exclusively increased. Ebo cells were selectively enriched within the TME and were nearly absent from adjacent non-tumor lung tissue and peripheral blood mononuclear cells (PBMC).

Ebo cell abundance was significantly associated with tumor progression ($p = 0.006$), reduced overall survival ($p < 0.05$) and lack of durable clinical benefit from anti-PD-1 therapy ($p = 0.004$). These findings suggest that anti-PD-1 treatment may act not by restoring T cell function but by interrupting an apoptotic death program in this exhausted T cell population.

Taken together, this study identifies a previously unrecognized mechanism of PD-1 resistance driven by the accumulation of dysfunctional, overactivated CD8+ T cells. The presence of Ebo cells may serve as a predictive biomarker for ICI failure, and therapeutic strategies aimed at preventing or reversing T cell burnout may be critical for improving outcomes in NSCLC.

On-demand webinar: Drug screening using PDOs

Explore how CAFs drive therapy resistance in colorectal cancer. Watch the on-demand webinar featuring Chris Tape, PhD (University College London Cancer Institute), corresponding author of the featured Ramos Zapatero et al. study[12].

Spatial proteomics reveals stromal drug sequestration in the ECM

Cisplatin is a platinum-containing chemotherapy used to treat a wide range of **solid tumors**. However, 50% of patients eventually develop resistance to treatment[14]. To investigate its biodistribution, Chang et al. employed the Hyperion Imaging System in pancreatic cancer patient-derived xenografts[15]. The analysis revealed that cisplatin accumulated extensively in the tumor stroma, binding preferentially to collagen fibers within the ECM rather than to malignant epithelial cells.

This spatial insight underscores the ECM's role as a drug reservoir, potentially mediating delayed drug release while limiting immediate therapeutic efficacy. Notably, similar collagen-bound cisplatin deposits were also detected in normal tissues, suggesting that such tissue-resident reservoirs could contribute to off-target toxicity and long-term side effects.

By mapping drug localization at cellular resolution, this study exemplifies how spatial proteomics can reveal critical interactions between therapeutic agents and stromal components of the TME. These insights are essential for understanding resistance and improving drug delivery strategies.

Spatial proteomics uncovers immune aggregates that predict long-term survival

ICIs only benefit only 20–30% of patients with **oropharyngeal squamous cell carcinoma** (OPSCC), underscoring the need to better understand how the TME influences treatment response. To investigate this, Abdulrahman et al. used the Hyperion Imaging System to analyze tissue samples from immune-responsive (IR+) and non-responsive (IR–) OPSCC patients[16].

In IR+ patients, the TME featured highly organized intratumoral immune microaggregates composed of dendritic cells (DCs), CD8+ T cells, CD4+ T cells and tumor cells. Chemokine secretion by T cells appeared to further recruit both T cells and DCs into the TME, suggesting a positive feedback loop that reinforced immune cell infiltration. These immune microaggregates were strongly associated with long-term survival (>10 years), underscoring the importance of spatial immune organization in sustaining effective antitumor responses. T cell-DC interactions, in particular, are known to be critical for successful anti-PD-1 therapy.

These tumor-specific immune aggregates may serve as a novel biomarker for OPSCC and for predicting ICI responsiveness. Their structured architecture also suggests that

Table 5. Featured studies exploring the TME using Standard BioTools™ platforms

Biological Level	Technology	Sample Type	Cancer Type	Key Insight	Representative Study
Molecular	SomaScan	Peritoneal fluid	Ovarian	Prognostic signature	Finkernagel (2019)
	SomaScan	Bone marrow plasma	AML	Immune suppression signaling	Çelik (2020)
	KREX	Serum/tissue lysate	Pancreatic	Immune tolerance	Maimela (2024)
Cellular	CyTOF	Cells	Multiple myeloma	TME role in T cell behavior, disease activity signature	Yao (2022)
	CyTOF	Cells	Colorectal	CAF role in TME remodeling and treatment resistance	Ramos Zapatero (2023)
Cellular + spatial	CyTOF + Hyperion Imaging System	Cells/tissue	Lung	Mechanism of ICI resistance	Sanmamed (2021)
Spatial	Hyperion Imaging System	Tissue	Pancreatic	ECM and normal tissue as a drug reservoir	Chang (2016)
	Hyperion Imaging System	Tissue	Oropharyngeal	Immune aggregates as a diagnostic and predictive biomarker	Abdulrahman (2022)

tumor-specific T cells play a central role in local immune control, potentially through DC-mediated antigen presentation that enhances effector T cell activation and amplifies the antitumor immune response.

Conclusion

The heterogeneity of the TME requires a comprehensive multidimensional research strategy. As illustrated by the studies herein, selecting the right proteomic platform for the right biological question enables powerful insights. From profiling soluble factors to mapping cellular heterogeneity and spatial architecture, these tools offer the depth and resolution needed to understand – and ultimately disrupt – the tumor-supportive microenvironment to improve patient outcomes.

Standard BioTools Omics Services give researchers immediate access to the technologies featured in this white paper for rapid study startup, including the SomaScan Platform, KREX microarrays, CyTOF and the Hyperion Imaging System. From study design to data analysis, our end-to-end support accelerates discovery, reduces technical burden and helps generate actionable insights into the TME. As the cancer research landscape evolves, our services are here to empower investigators to translate complex data into meaningful outcomes.

References

1. Wang, Y. et al. “The solid tumor microenvironment and related targeting strategies: a concise review.” *Frontiers in Immunology* 16 (2025): 1563858.
2. De Visser, K.E. and Joyce, J.A. “The evolving tumor microenvironment: from cancer initiation to metastatic outgrowth.” *Cancer Cell* 41 (2023): 374–403.
3. Glaviano, A. et al. “Harnessing the tumor microenvironment: targeted cancer therapies through modulation of epithelial-mesenchymal transition.” *Journal of Hematology & Oncology* 18 (2025): 6.
4. Drugs@FDA: FDA-Approved Drugs. U.S. Food and Drug Administration (2025): accessdata.fda.gov/scripts/cder/daf/index.cfm.
5. Timeline of Anti-PD-1/L1 Antibody Approvals by the FDA. Cancer Research Institute, Inc. (2025): public.tableau.com/app/profile/cancer.research.institute/viz/PDxapprovaltimeline/PD-1approvallandscape.
6. Khosravi, G-R. et al. “Immunologic tumor microenvironment modulators for turning cold tumors hot.” *Cancer Communications* 44 (2024): 521–553.
7. Xiao, Y. and Yu, D. “Tumor microenvironment as a therapeutic target in cancer.” *Pharmacology & Therapeutics* 221 (2021): 107753.
8. Finkernagel, F. et al. “Dual-platform affinity proteomics identifies links between the recurrence of ovarian carcinoma and proteins released into the tumor microenvironment.” *Theranostics* 9 (2019): 6,601–6,617.
9. Çelik, H. et al. “Highly multiplexed proteomic assessment of human bone marrow in acute myeloid leukemia.” *Blood Advances* 4 (2020): 367–379.
10. Maimela, P.W.M. et al. “Humoral immunoprofiling identifies novel biomarkers and an immune suppressive autoantibody phenotype at the site of disease in pancreatic ductal adenocarcinoma.” *Frontiers in Oncology* 14 (2024): 1330419.
11. Yao, L. et al. “Comprehensive characterization of the multiple myeloma immune microenvironment using integrated scRNA-seq, CyTOF, and CITE-seq analysis.” *Cancer Research Communications* 2 (2022): 1,255–1,265.
12. Ramos Zapatero, M. et al. “Trellis tree-based analysis reveals stromal regulation of patient-derived organoid drug responses.” *Cell* 186 (2023): 5,606–5,619.e24.
13. Sanmamed, M.F. et al. “A burned-out CD8+ T-cell subset expands in the tumor microenvironment and curbs cancer immunotherapy.” *Cancer Discovery* 11 (2021): 1,700–1,715.
14. Lugones, Y. et al. “Cisplatin resistance: genetic and epigenetic factors involved.” *Biomolecules* 12 (2022): 1365.
15. Chang, Q. et al. “Biodistribution of cisplatin revealed by Imaging Mass Cytometry identifies extensive collagen binding in tumor and normal tissues.” *Scientific Reports* 6 (2016): 36641.
16. Abdulrahman, Z. et al. “Tumor-specific T cells support chemokine-driven spatial organization of intratumoral immune microaggregates needed for long survival.” *Journal for ImmunoTherapy of Cancer* 10 (2022): e004346.

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