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Leukocyte Subset Dynamics in the Tumor Microenvironment: Dual Roles, Single-Cell Insights, and Implications for Cancer Immunotherapy in Nigeria

Okluchi Hartumu

Department of Hematology and Molecular Oncology, Faculty of Basic Medical Sciences, University of Ibadan, Ibadan, Nigeria

Abstract

The tumor microenvironment (TME) constitutes a highly complex and dynamic ecosystem wherein diverse leukocyte subsets engage in intricate cross-talk that ultimately dictates the balance between tumor progression and suppression. This review provides a comprehensive analysis of the dualistic functions of key immune cell populations-including natural killer (NK) cells, effector T cells, regulatory T cells (Tregs), myeloid-derived suppressor cells (MDSCs), and tumor-associated macrophages (TAMs)-within this milieu. Particular emphasis is placed on the Nigerian context, where unique genetic backgrounds, such as the high prevalence of sickle cell trait, and environmental factors, including endemic infections, significantly shape immune phenotypes and cancer pathogenesis. We explore how cutting-edge single-cell technologies are revolutionizing our understanding of the spatial architecture, functional plasticity, and cellular heterogeneity of these leukocytes in patient cohorts, revealing mechanisms of both immune surveillance and tumor-promoting immunosuppression. The paradoxical roles of these cells elucidate the variable clinical outcomes observed with immunotherapies and underscore the critical need for precision medicine strategies that are tailored to individual immune landscapes. Finally, we discuss emerging therapeutic modalities aimed at selectively depleting pro-tumor subsets like Tregs or MDSCs while activating anti-tumor effectors such as NK cells, which hold substantial promise for reshaping the TME and improving oncology outcomes both in Nigeria and across the globe.

Keywords

Tumor Microenvironment, Leukocyte Subsets, Cancer Immunotherapy, Single-Cell Analysis, Immune Suppression, Nk Cells, Regulatory T Cells

1. Introduction

The tumor microenvironment (TME) comprises proliferating malignant cells surrounded by a heterogeneous collection of immune cells, cancer-associated fibroblasts, vascular lymphatic endothelial cells, and extracellular matrix elements. These components establish various microstructures within tumors through direct cell contact and paracrine or autocrine communication. Among these constituents, leukocyte subsets play particularly pivotal and often contradictory roles-either restraining tumor growth through cytotoxic activities or facilitating cancer progression by establishing an immunosuppressive niche.

The composition, spatial architecture, and functional states of immune cells within the TME significantly influence tumor immune evasion, progression, and responses to immunotherapy. Despite advances in understanding leukocyte biology, the dual nature of these cells continues to pose challenges for cancer therapy. This paradox is especially relevant in Nigeria, where unique genetic factors such as the high prevalence of sickle cell anemia modify hematological parameters and immune responses. For instance, young Nigerian sickle cell anemia patients demonstrate altered leukocyte profiles, with white blood cell counts ranging from 5.9-12.1×10^9/L, reflecting a chronic inflammatory state that might influence anti-tumor immunity.

Single-cell RNA sequencing technologies have dramatically enhanced our capacity to decipher the composition and functional states of immune and non-immune cells within the TME. However, the spatial distribution characteristics of these cells, their intercellular interactions, and their impact on maintaining cell states and anti-tumor or pro-tumor functions remain incompletely understood, particularly in African populations.

This review explores the current understanding of how different leukocyte subsets either suppress or promote cancer within the TME, with particular attention to insights gained from single-cell studies and their implications for Nigerian cancer research. We will examine the mechanisms underlying these opposing functions and discuss emerging therapeutic strategies aimed at reprogramming the TME toward effective anti-tumor immunity.

2. Anti-Tumor Leukocyte Subsets in the TME

2.1 Natural Killer (NK) Cells

Natural killer (NK) cells serve as the immune system's critical first-line defenders against nascent tumors and virally infected cells, capable of distinguishing and rapidly eliminating malignant targets without the need for prior sensitization or MHC-restricted antigen presentation. As pivotal components of the innate immune system, NK cells orchestrate anti-tumor responses through two primary mechanisms: direct cell-mediated cytotoxicity and the secretion of potent immunomodulatory cytokines. Upon recognition of susceptible target cells, NK cells release perforin and granzymes from their cytoplasmic granules, forming pores in the target cell membrane and initiating caspase-dependent apoptosis, respectively. Concurrently, they produce substantial quantities of interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-α), which collectively exert pleiotropic effects including inhibition of tumor cell proliferation, suppression of angiogenesis, and activation of macrophages and other immune effector cells, thereby creating an inflammatory microenvironment hostile to cancer progression and metastasis [1].

The activation threshold and functional capacity of NK cells are governed by a sophisticated balance of signals integrated from an array of germline-encoded activating and inhibitory surface receptors. Key activating receptors-including NKG2D, DNAM-1, and the natural cytotoxicity receptors (NCRs) such as NKp30, NKp44, and NKp46-recognize stress-induced ligands (e.g., MICA/B, ULBP family) and adhesion molecules that are frequently upregulated on transformed or infected cells. Conversely, inhibitory receptors, predominantly the killer cell immunoglobulin-like receptors (KIRs) and the CD94/NKG2A heterodimer, engage with MHC class I molecules, which are often downregulated on malignant cells as an immune evasion strategy. This "missing-self" recognition, wherein the loss of inhibitory signals dominates over activating cues, allows NK cells to selectively target and eliminate cells that have undergone pathological transformation while sparing healthy "self" tissues. Furthermore, NK cells express the low-affinity Fc receptor CD16 (FcγRIIIa), which enables them to mediate antibody-dependent cellular cytotoxicity (ADCC) upon engagement with antibody-opsonized target cells, a mechanism of particular therapeutic relevance in monoclonal antibody-based cancer immunotherapies.

In the Nigerian context, the functional integrity and clinical significance of NK cells are of paramount importance, especially in controlling viral-associated malignancies that constitute a substantial public health burden. High-risk human papillomavirus (HPV)-associated cervical cancer, for instance, represents a leading cause of cancer-related morbidity and mortality among Nigerian women. Research has consistently demonstrated that functional impairment or numerical deficiency of NK cells in the peripheral blood and tumor microenvironment is strongly correlated with increased risk of progression from persistent HPV infection to invasive cervical carcinoma. Similarly, Epstein-Barr virus (EBV)-associated lymphomas and nasopharyngeal carcinoma, which exhibit considerable prevalence in Nigeria, have been linked to NK cell dysfunction. Detailed immunological studies examining NK cell phenotypes and effector functions in Nigerian patient cohorts have revealed that chronic exposures to these oncogenic viruses, along with endemic infections such as malaria and hepatitis, can profoundly shape the NK cell repertoire, driving it toward an exhausted or anergic phenotype characterized by reduced cytotoxic capacity, impaired cytokine production, and altered receptor expression profiles. This virally induced NK cell exhaustion not only facilitates viral persistence but also compromises immune surveillance against transformed cells, thereby accelerating oncogenesis.

The advent of single-cell technologies has further illuminated the remarkable heterogeneity within the NK cell compartment in both healthy and diseased states. Single-cell RNA sequencing (scRNA-seq) studies have identified multiple distinct NK cell subsets with unique transcriptional signatures and functional specializations, including CD56brightCD16- cytokine-producers and CD56dimCD16+ cytotoxic effectors, as well as tissue-resident populations adapted to specific microenvironments. In the context of Nigerian patients, ongoing research aims to delineate how regional factors-such as genetic polymorphisms in NK cell receptors, endemic pathogen exposures, nutritional status, and environmental influences-collectively mold the NK cell landscape and contribute to the distinctive epidemiology and clinical behavior of cancers in this population. A deeper understanding of these population-specific determinants of NK cell biology is crucial for developing targeted immunotherapeutic interventions that can effectively harness NK cell-mediated anti-tumor immunity within the Nigerian and broader African contexts [2].

Table 1. Anti-Tumor Leukocyte Subsets and Their Mechanisms of Action

Leukocyte Subset	Key Anti-Tumor Mechanisms	Activating Receptors/Cytokines	Targets in Cancer Therapy
Natural Killer Cells	Direct cytotoxicity via perforin/granzyme, IFN-γ production, ADCC	NKG2D, DNAM-1, NCRs, NKp46	NKG2A, TIGIT, KIRs
CD8+ T Cells	Antigen-specific tumor cell killing, granzyme/perforin release, IFN-γ production	TCR, CD28, IL-2R	PD-1, CTLA-4, LAG-3
CD4+ Th1 Cells	IFN-γ production, CD8+ T cell help, macrophage activation	TCR, CD28, IL-12R, IFN-γR	OX-40, GITR, ICOS
γδ T Cells	MHC-unrestricted tumor cell killing, dendritic cell cross-priming	γδ TCR, NKG2D, DNAM-1	Bispecific antibodies, CAR-γδ T
M1 Macrophages	Phagocytosis, ROS/RNS production, pro-inflammatory cytokine secretion	TLRs, IFN-γR, GM-CSFR	CD47-SIRPα axis, CSF-1R

Table 1: This table provides an overview of how five key types of immune cells kill tumors, as well as the key molecules targeted by recent immunotherapies (such as checkpoint inhibitors, NK cell therapy, and CAR-T). It helps in understanding the roles of different cells and therapeutic targets in tumor immunology.

Engineering NK cells for enhanced anti-tumor function represents an emerging frontier in cancer immunotherapy. Chinese researchers have developed innovative approaches using NK cell engagers (NKCEs) that specifically direct NK cell cytotoxicity toward tumor cells. Additionally, pH-selective T cell engagers (TCEs) that activate only in the acidic tumor microenvironment show promise for reducing off-target toxicity while maintaining potent anti-tumor effects. These advancements hold particular relevance for Nigeria, where access to complex cellular therapies remains limited but could potentially be implemented as off-the-shelf treatments.

2.2 Effector T Lymphocytes

CD8+ cytotoxic T lymphocytes (CTLs) represent the most potent antigen-specific anti-tumor effectors of the adaptive immune system. Upon recognizing tumor-associated antigens presented by MHC class I molecules, CTLs execute direct killing through perforin and granzyme release and Fas/FasL interactions. The efficacy of CTLs correlates with improved survival across multiple cancer types, and their presence within the TME serves as a favorable prognostic indicator.

CD4+ T helper cells, particularly the Th1 subset, contribute to anti-tumor immunity through multiple mechanisms, including IFN-γ production that enhances antigen presentation and activates other immune cells, provision of help for CTL priming and maintenance, and direct cytotoxicity in certain contexts. The critical role of T helper cells is evidenced by the superior anti-tumor efficacy observed with combinations of CD4+ and CD8+ T cells compared to either subset alone [3].

In Nigerian patients, the functional capacity of effector T cells can be influenced by various factors, including nutritional status, co-infections, and genetic background. For instance, studies have documented that HIV infection, which remains prevalent in Nigeria, profoundly impairs T cell function and increases cancer risk. Additionally, research suggests that the high frequency of certain HLA alleles in African populations might confer distinctive patterns of tumor antigen recognition by T cells.

3. Pro-Tumor Leukocyte Subsets in the TME

3.1 Regulatory T Cells (Tregs)

Regulatory T cells, characterized by expression of the transcription factor Foxp3, play indispensable roles in maintaining immune homeostasis and preventing autoimmunity. However, within the TME, Tregs exert potent immunosuppressive effects that hinder effective anti-tumor immunity. Tregs utilize multiple mechanisms to suppress immune responses, including cell contact-dependent inhibition via CTLA-4-mediated downregulation of CD80/CD86 on dendritic cells, LAG-3 interaction with MHC class II molecules, and direct cytolysis via granzyme and perforin [20]. Additionally, Tregs employ cell contact-independent strategies such as secretion of inhibitory cytokines (IL-10, IL-35, TGF-β), competition for IL-2 through high-affinity CD25 expression, and generation of immunosuppressive adenosine via CD39/CD73 ectoenzymes [4].

Recent single-cell studies conducted in Nigerian research settings have revealed specialized spatial organization of Tregs within the TME. A groundbreaking investigation by Mao et al. discovered that Tregs specifically accumulate around lymphatic vessels in the tumor stroma, forming distinct niches with mature dendritic cells enriched in immunoregulatory molecules (mregDCs). This Treg-mregDC-lymphatic niche serves to maintain Tregs in an activated state and limits antigen trafficking to draining lymph nodes, thereby restraining the initiation of anti-tumor immune responses. The clinical significance of these findings is underscored by analysis of TCGA data showing that patients with low activated Treg signatures and high mregDC signatures experience significantly improved survival.

Table 2. Pro-Tumor Leukocyte Subsets and Their Mechanisms of Action

Leukocyte Subset	Key Pro-Tumor Mechanisms	Immunosuppressive Factors	Therapeutic Targeting Approaches
Regulatory T Cells	Inhibitory cytokine secretion, metabolic disruption, CTLA-4-mediated DC modulation	IL-10, TGF-β, IL-35, adenosine	Anti-CTLA-4, anti-CCR4, CD25-targeted toxins
Myeloid-Derived Suppressor Cells	Arginase/iNOS-mediated T cell suppression, cysteine sequestration, Treg induction	ARG1, iNOS, ROS, TGF-β	PDE5 inhibitors, ATRA, COX-2 inhibitors
M2 Macrophages	Angiogenesis promotion, tissue remodeling, T cell suppression	IL-10, TGF-β, PGE2, arginase-1	CSF-1R inhibitors, CD40 agonists
Regulatory B Cells	IL-10 production, T cell inhibition, Treg conversion	IL-10, TGF-β, PD-L1	Bruton's tyrosine kinase inhibitors
Neutrophil Extracellular Traps	Metastasis promotion, T cell apoptosis, coagulation induction	MMP9, histones, cathepsin G	DNase I, PAD4 inhibitors

Table 2: This information in the table is that various leukocyte subsets (Tregs, MDSCs, M2 macrophages, Bregs, NETs) support tumor growth in TME, exerting their effects through immunosuppression, metabolic regulation, tissue remodeling, and metastasis promotion.

Therapeutic strategies targeting these cells mainly include:

- Depleting or blocking their inhibitory functions (e.g., anti-CTLA-4, COX-2 inhibitors)
- Inducing their differentiation (e.g., ATRA)
- Inhibiting key pathways (e.g., BTK inhibitors, CSF-1R inhibitors)
- Disrupting their pro-tumorigenic structures (e.g., DNase I to remove NETs)

3.2 Myeloid-Derived Suppressor Cells (MDSCs)

MDSCs represent a heterogeneous population of immature myeloid cells that expand during cancer and exert potent immunosuppressive functions. These cells are broadly categorized into polymorphonuclear (PMN-MDSCs) and monocytic (M-MDSCs) subsets, both of which inhibit anti-tumor immunity through diverse mechanisms. MDSCs express high levels of arginase-1 and inducible nitric oxide synthase (iNOS), which collaboratively deplete essential amino acids and generate reactive nitrogen species that disrupt T cell signaling and function. Additionally, MDSCs produce elevated levels of reactive oxygen species, sequester cysteine essential for T cell activation, and promote Treg expansion through TGF-β and other soluble factors.

In Nigerian cancer patients, factors such as chronic inflammation and certain endemic infections might influence MDSC accumulation and function. For instance, studies have shown that parasitic infections like malaria can induce myeloid cell expansions with immunosuppressive properties that potentially worsen cancer outcomes. Additionally, the altered hematological parameters observed in conditions prevalent in Nigeria, such as sickle cell disease, might modify MDSC dynamics within the TME [5].

3.3 Tumor-Associated Macrophages (TAMs)

Tumor-associated macrophages (TAMs) represent one of the most abundant immune cell populations within the tumor microenvironment, yet their functions are typically co-opted by tumors to promote cancer progression [6].These cells predominantly exhibit an M2-like phenotype that facilitates tumor growth through multiple mechanisms.

Key pro-tumorigenic mechanisms of TAMs include:

- **Angiogenesis**: Secretion of VEGF, PDGF, and other factors to promote tumor vascularization
- **Immunosuppression**: Expression of PD-L1 and production of IL-10, TGF-β to suppress T cell function
- **Metastasis facilitation**: Production of MMP-2, MMP-9 and other metalloproteinases to degrade extracellular matrix
- **Metabolic reprogramming**: Upregulation of arginase-1 to deplete arginine essential for T cell function

Therapeutic strategies targeting TAMs:

- Inhibition of CSF-1R signaling to block macrophage recruitment
- Reprogramming M2-like TAMs toward M1-like anti-tumor phenotype using CD40 agonists
- Disrupting the CD47-SIRPα "don't eat me" signal to enhance macrophage phagocytosis

In the Nigerian context, chronic infections such as malaria, helminth infections, and tuberculosis may precondition macrophage functional states toward M2-like polarization, potentially influencing the efficacy of TAM-targeted

therapies. Understanding these region-specific immune characteristics is therefore crucial for developing effective cancer immunotherapy strategies [7].

4. Factors Influencing Leukocyte Function in the TME

4.1 Genetic Background and Ethnic Considerations

Genetic variations among ethnic populations can significantly influence immune cell function and therapeutic responses. Nigerian and broader African populations harbor unique genetic polymorphisms in immune-related genes that might impact anti-tumor immunity. For instance, variations in FCGR genes that influence antibody-dependent cellular cytotoxicity (ADCC) could modify responses to therapeutic monoclonal antibodies. Similarly, polymorphisms in cytokine genes or their receptors might alter the balance between pro-inflammatory and anti-inflammatory responses in the TME [8].

The high prevalence of sickle cell trait and disease in Nigeria represents a particularly relevant genetic factor that modifies leukocyte characteristics. Research has established that sickle cell anemia significantly alters hematological parameters, with white blood cell counts in steady-state patients ranging from 5.9-12.1×10⁹/L, reflecting a chronic inflammatory state. Additionally, the co-inheritance of α-thalassemia, present in approximately 41% of Nigerian sickle cell anemia patients, further modifies laboratory parameters and clinical manifestations. [9]. These hematological alterations likely influence the composition and function of leukocyte subsets within the TME, potentially creating unique immune contexts that differ from those in non-African populations.

4.2 Regional and Environmental Influences

In Nigeria, as in many developing regions, environmental factors significantly shape immune function and cancer biology. The high burden of infectious diseases leads to chronic immune activation that can either enhance or suppress anti-tumor immunity depending on context. For instance, chronic helminth infections typically induce Th2-polarized immune responses and Treg expansion that might potentially dampen anti-tumor immunity. Similarly, the high prevalence of aflatoxin exposure contributes to hepatocellular carcinoma development while simultaneously modifying the liver immune microenvironment.

Nutritional factors also play crucial roles in shaping immune responses. Micronutrient deficiencies prevalent in Nigeria, such as zinc, selenium, and vitamin D, can impair various immune functions including NK cell activity and T cell responses. Additionally, the urban-rural divide in dietary patterns might create distinct immune contexts that influence cancer progression and treatment responses [10].

5. Single-Cell Insights and Clinical Translation

5.1 Single-Cell Technologies in Leukocyte Analysis

The advent of single-cell technologies has revolutionized our understanding of leukocyte heterogeneity and plasticity within the TME. Single-cell RNA sequencing (scRNA-seq) enables comprehensive profiling of immune cell transcriptomes at unprecedented resolution, revealing previously unappreciated diversity within classical leukocyte subsets. For instance, scRNA-seq studies have identified multiple distinct states of T cell exhaustion in the TME, each with different functional capacities and potential for reinvigoration through immunotherapy.

Spatial transcriptomics techniques now allow researchers to map the precise anatomical locations of different leukocyte subsets and analyze how their positioning influences function. As mentioned previously, the discovery of Treg-mregDC-lymphatic niches in colorectal cancer highlights the importance of spatial organization in immune regulation. Similar analyses in Nigerian patient populations might reveal unique architectural features of the TME influenced by genetic and environmental factors specific to the region [11].

Mass cytometry (CyTOF) extends flow cytometry capabilities by enabling simultaneous measurement of over 40 parameters on single cells, providing deep immunophenotyping of leukocyte populations in the TME. Application of these technologies in Nigerian cancer patients could identify distinctive immune signatures with prognostic and therapeutic implications.

5.2 Therapeutic Strategies Targeting Leukocyte Subsets

Insights from single-cell studies have facilitated the development of novel therapeutic strategies that precisely target specific leukocyte subsets in the TME. Immune checkpoint inhibitors, including antibodies against PD-1, PD-L1, and CTLA-4, function primarily by reinvigorating exhausted T cells [12]. The efficacy of these therapies across multiple cancer types underscores the critical role of suppressed T cells in permitting tumor progression.

Emerging approaches focus on targeting other leukocyte subsets. For instance, Treg-depleting strategies using anti-CTLA-4 antibodies (ipilimumab) or anti-CCR4 antibodies (mogamulizumab) aim to reduce immunosuppression in the TME [13]. Similarly, therapies targeting MDSCs, such as phosphodiesterase-5 inhibitors or all-trans retinoic acid, seek to alleviate myeloid cell-mediated suppression.

NK cell-directed therapies represent another promising avenue. Natural killer cells are increasingly recognized as important mediators of anti-tumor immunity, particularly in hematological malignancies. As noted earlier, Nigerian researchers have contributed to developing NK cell engagers and pH-selective T cell engagers that enhance tumor-specific killing while minimizing off-target toxicity [14].

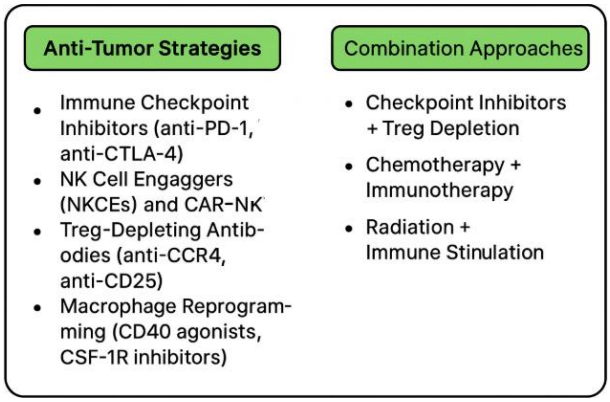

Figure 1. Therapeutic Strategies Targeting Leukocyte Subsets in the TME

Figure 1: This diagram outlines the main therapeutic strategies targeting different leukocyte subsets in the tumor microenvironment (TME), categorized into two main groups:

1. Anti-Tumor Strategies

Directly enhancing the immune response or suppressing immunosuppressive cells, including:

- Immune checkpoint inhibitors (e.g., anti-PD-1, anti-CTLA-4)
- NK cell-related therapies (NKCEs, CAR-NK)
- Treg depletion antibodies (anti-CCR4, anti-CD25)
- Reprogramming therapies targeting MDSCs and macrophages (e.g., CD40 agonists, CSF-1R inhibitors)

Main goal: To enhance the anti-tumor immune response and reduce immunosuppression.

2. Combination Approaches

Enhancing anti-cancer effects through the synergistic use of multiple therapies, including:

- Checkpoint inhibitors + Treg depletion
- Chemotherapy + Immunotherapy
- Radiotherapy + Immunostimulation

Main goal: To overcome the limitations of single-therapy approaches and comprehensively activate the immune system.

5.3 Clinical Trials and Outcomes in Nigerian Context

While Nigeria's clinical trial infrastructure remains developing, participation in international trials and initiation of local studies are increasingly generating data relevant to the Nigerian population. Preliminary analyses suggest that immune-related adverse events and treatment responses to immunotherapies might exhibit ethnic and regional variations, potentially influenced by genetic background, microbiome differences, and distinct TME compositions [15].

A critical consideration for implementing immunotherapies in Nigeria is the cost and infrastructure requirements. Cheaper, more accessible alternatives such as cancer vaccines or repurposed drugs with immunomodulatory properties might offer more immediate benefits. Additionally, leveraging Nigeria's rich biodiversity for drug discovery represents an promising approach-several indigenous medicinal plants contain compounds with demonstrated immunomodulatory properties [16].

6. Conclusion

Leukocyte subsets within the tumor microenvironment (TME) demonstrate remarkable functional plasticity, playing paradoxical roles as both potent defenders against cancer progression and unexpected facilitators of tumor immune evasion. The ultimate impact of these immune populations-whether protective or pathogenic-depends on complex multi-layered interactions between different cell types, their functional states, spatial organization within the tumor niche, and the broader host context. In Nigerian patients, this immunological balance is particularly influenced by unique genetic backgrounds including sickle cell trait and specific HLA polymorphisms, diverse environmental exposures, and high prevalence of endemic infections such as malaria and tuberculosis, all of which collectively create distinctive immune landscapes that significantly influence cancer biology, disease progression, and treatment responses. The recent application of single-cell technologies, particularly single-cell RNA sequencing and spatial transcriptomics,

has dramatically enhanced our understanding of leukocyte heterogeneity and functional dynamics within the TME, revealing previously unappreciated cellular states, transitional phenotypes, and critical spatial relationships that define immune activity in both physiological and pathological conditions. These revolutionary insights have already catalyzed the development of novel immunotherapeutic strategies that precisely target specific leukocyte populations, such as Treg-depleting antibodies, MDSC-targeting agents, and NK cell engagers. However, despite these advances, significant knowledge gaps remain, particularly regarding the unique characteristics of the TME in African populations, where genetic diversity, environmental factors, and socioeconomic determinants intersect to create distinct therapeutic challenges and opportunities that demand focused investigation to achieve equitable cancer care outcomes globally.

Future research directions should include:

1. Comprehensive characterization of the TME in Nigerian cancer patients using single-cell technologies

2. Investigation of how common genetic variants in African populations influence leukocyte function and therapeutic responses

3. Development of cost-effective immunotherapies accessible to resource-limited settings

4. Exploration of Nigeria's biodiversity for novel immunomodulatory compounds

5. Clinical trials specifically designed to address the unique needs and characteristics of African cancer patients

Addressing these priorities will require strengthened collaboration between Nigerian research institutions and international partners, increased investment in research infrastructure, and cultivation of local expertise in cancer immunology and immunotherapy. By pursuing these directions, we can work toward realizing the promise of precision immuno-oncology for Nigerian and global populations alike.

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