BIO Integration

Armored and Cold Tumors: A Refractory Subset of Solid Cancer

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Abstract

Solid tumors are characterized by extensive extracellular matrix (ECM) remodeling prominently featuring massive collagen deposition. This dense collagen network does not act as inert scaffolding but actively orchestrates critical aspects of tumor progression and therapy resistance, thereby shaping the fate of cancer cells. Collagen influences cellular behavior through multiple mechanisms, including providing structural rigidity, modulating mechanotransduction signaling pathways, creating physical barriers to immune cell infiltration and drug penetration, and serving as a reservoir for signaling molecules. Here, we discuss recent findings regarding the critical roles of collagen in tumors and potential therapies for armored and cold tumors, a refractory subset demonstrating high collagen deposition and low immune infiltration.

Keywords

Armored and cold tumors, collagen, immune exclusion, tumor microenvironment.

Intratumoral collagen supports tumor progression

Collagen, the structural component in the extracellular matrix (ECM), provides structural integrity and support to various cell types in tumor tissues. Collagen also modulates cellular activities through signal transduction via interaction with cell surface receptors in specific cell types, including integrins, the discoidin domain receptors, osteoclast-associated receptor, glycoprotein VI, G6b-B, leukocyte-associated immunoglobulin-like receptors, and the mannose family receptor [1]. Intratumoral collagen, characterized by high density and stiffness, has diverse roles in malignant progression in most solid cancer types [1, 2], such as promoting tumor proliferation, metastasis, and resistance to therapeutic agents while hindering tumor progression.

Intratumoral collagen, primarily type I collagen, serves as a dual physical and biochemical barrier against anti-tumor immunity. It forms as a result of synergistic multifactorial effects. First, hyperactivation of tumor cells and recruited cancer-associated fibroblasts (CAFs) is the core mechanism driving aberrant collagen synthesis and deposition, wherein activated CAFs secrete excessive collagen and other ECM components under stimuli such as transforming growth factor-β (TGF-β) [3]. Second, imbalanced ECM remodeling exacerbates collagen accumulation. In this process, dysregulation of matrix metalloproteinases and their inhibitors impairs collagen degradation, thus promoting excessive cross-linking and thickening of deposited collagen fibers, and the formation of a dense, rigid fibrotic network [4]. Finally, this collagen-enriched microenvironment establishes immunosuppression through multiple mechanisms, including physical blockade and biochemical inhibition, primarily by (i) physically impeding contact between immune cells and tumor cells [5], (ii) restricting the cytotoxic functions of immune cells via mechanical stress [6], and (iii) releasing bioactive fragments that modulate immune cell activity [7]. Our previous research has systematically summarized the critical role of targeting collagen in sensitizing immunotherapy [8].

Definition of armored and cold tumors

Despite the close association between the ECM and immune cells in the tumor microenvironment (TME), the contribution

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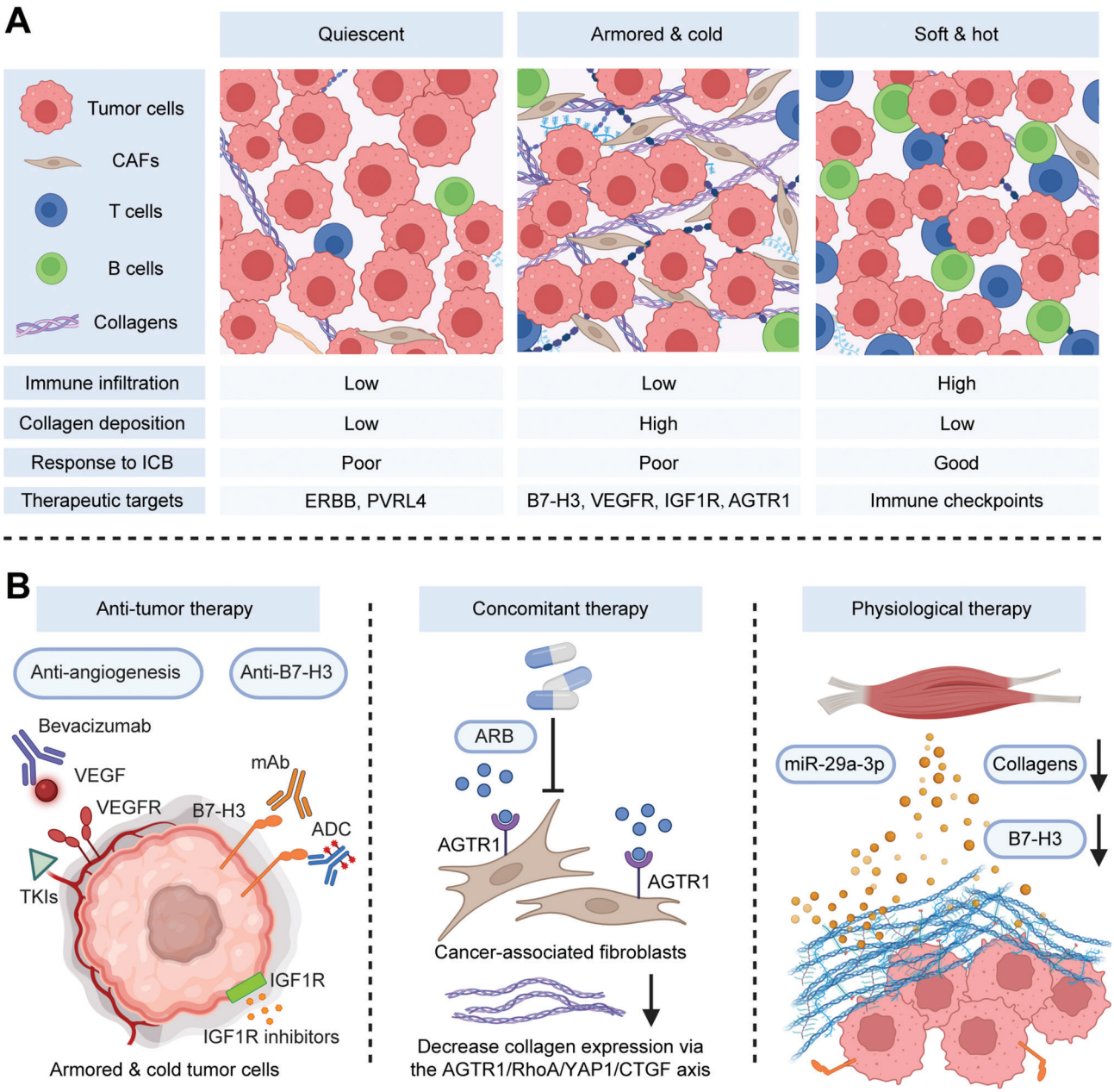

Figure 1 Schematic description of the molecular features of armored and cold tumors and corresponding treatments. (A) A framework was developed to stratify tumors according to collagen deposition and immune activity. Armored and cold tumors are accompanied by low immune infiltration and high collagen deposition, and show poor prognosis and resistance to ICB therapy. These tumors highly express B7-H3, angiogenesis markers, IGF1R, and AGTR1 (the target of ARBs). (B) Possible therapeutic strategies from three perspectives (anti-tumor therapy, concomitant therapy, and physiological therapy) are shown. Created with BioRender.com. Abbreviations: CAF, cancer associated fibroblast; TME, tumor microenvironment; ICB, immune checkpoint blockade; VEGF/VEGFA, vascular endothelial growth factor/A; VEGFR, VEGF receptor; AGTR1, angiotensin II receptor type 1; ARB, angiotensin receptor blocker; IGF1R, insulin like growth factor 1 receptor; mAb, monoclonal antibody; ADC, antibody–drug conjugate; TKIs, tyrosine kinase inhibitors; CAR T, chimeric antigen receptor T cell; NK, natural killer; RhoA, Ras homolog family member A; YAP1, Yes associated protein 1; CTGF, connective tissue growth factor.

of the interaction between ECM and immune cells to patient stratification and immune checkpoint blockade (ICB) response prediction remains poorly understood. In our previous research [9], analysis of transcriptomic collagen activity and immune signatures in large-scale public cohorts led to the identification of three distinct immuno-collagenic subtypes predictive of ICB responses. Validation was performed using paraffin embedded cancer tissue microarrays obtained from the National Engineering Center for Biochip (Outdo Biotech, Shanghai), comprising 1,012 cases across 10 solid cancer types. The tumors were categorized into three subtypes: “soft and hot” (low collagen deposition and high immune infiltration), “armored and cold” (high collagen deposition and low immune infiltration), and “quiescent” (low collagen deposition and immune infiltration) (**Figure 1A**) [9]. In addition, Hamidi et al. have established a novel stratification incorporating ECM, immune infiltration, and tumor cell heterogeneity to predict clinical benefits from programmed death ligand 1 (PD-L1) blockade in urothelial carcinoma [10]. That study provided both an independent validation of, and an important complement to, our own research, thereby underscoring the critical importance

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of elucidating tumor cell heterogeneity in quiescent tumors [10]. In summary, although armored and cold tumors and quiescent tumors are "cold" tumors, as previously defined [11], armored and cold tumors demonstrate unique collagen-rich microenvironments that enable specific therapeutic strategies [12].

Candidate therapies for armored and cold tumors

Anti-tumor therapy

To determine potential anti-tumor targets for armored and cold tumors, we conducted a pan-cancer analysis to assess the expression of various drug targets. Targets such as fibroblast growth factor receptor, platelet-derived growth factor receptor, immune inhibition-associated targets [such as B7 homolog 3 protein (B7-H3)], and angiogenesis-related targets [such as vascular endothelial growth factor A (VEGFA) and VEGF receptor (VEGFR)] exhibited high expression in armored and cold tumors [9]. B7-H3 has been associated with immuno-cold features and collagen accumulation in triple-negative breast cancer [13] and melanoma [14]. Interestingly, anti-B7-H3 therapy has been validated to be a feasible and robust candidate immunotherapy against advanced prostate cancer [15]. Therefore, anti-B7-H3 therapy might provide a valuable supplemental therapeutic strategy for armored and cold tumors. B7-H3-related clinical trials in solid cancer are summarized in **Table 1**. We believe that subgroup analyses based on the three distinct immuno-collagenic subtypes in these clinical trials are warranted to identify whether patients with armored and cold tumors might achieve enhanced benefits.

Furthermore, collagen deposition markedly influences tumor angiogenesis. Our prior systematic analysis across multiple cancer types has revealed a robust association between immuno-collagenic subtypes and angiogenic activity, and demonstrated that armored and cold tumors exhibit the highest angiogenic levels. Critically, collagen inhibition through diverse approaches effectively suppresses tumor angiogenesis in vivo [16]. In addition, armored and cold tumors have been found to display superior responsiveness to anti-angiogenic therapy in advanced lung adenocarcinoma cohorts [16]. Therefore, anti-angiogenic therapy is another promising strategy for targeting armored and cold tumors. Furthermore, insulin like growth factor 1 receptor (IGF1R) has emerged as an additional therapeutic target for armored and cold tumors. The IGF1R inhibitor picropodophyllin has been observed to enhance immunotherapy efficacy in preclinical models [17].

Concomitant therapy

Patients with cancer undergoing anti-tumor therapy are frequently prescribed multiple medications for pre-existing comorbidities or adverse effects from the anti-tumor therapy, and polypharmacy is common [18]. Considering the high prevalence of concomitant medications, we screened potential concomitant medications according to the established immuno-collagenic subtypes, and found that angiotensin II receptor 1 (AGTR1), the target of angiotensin receptor blockers (ARBs), has high expression in armored and cold tumors. ARBs inhibit type I collagen expression in CAFs via negatively regulating the RhoA/YAP axis, thus shaping an inflamed TME [19]. Therefore, the combination of ARBs with existing anti-tumor therapies might be effective for armored and cold tumors. Notably, a phase 2 clinical trial has reported that a combination of losartan followed by chemoradiotherapy has led to downstaging of locally advanced pancreatic ductal adenocarcinoma, a highly fibrotic solid tumor, and is associated with an R0 resection rate of 61% [20]. Therefore, more clinical trials of ARB combinations in settings not limited to pancreatic cancer, or to chemotherapy and radiotherapy, are urgently needed in armored and cold tumors.

Physiological therapy

Physiological therapies are aimed at enhancing and restoring the normal physiological functions of the human body. This spectrum of treatment includes primarily rehabilitation training and biofeedback therapy, among other therapies. Physical activity is widely understood to be healthful among both the scientific community and the public. Exercise has been found to increase T and natural killer (NK) cell infiltration, thereby controlling tumor growth [21]. However, how exercise promotes anti-tumor immunity and immunotherapy remains elusive. On the basis of the established immuno-collagenic subtypes, we have reported that exercise promotes muscle-derived extracellular vesicle-associated miR-29a-3p in ECM inhibition by directly targeting COL1A1, thereby enabling immune cell infiltration and immunotherapy. Clinically, miR-29a-3p correlates with decreased ECM and increased T cell infiltration in various cancer types [22]. In addition, B7-H3, a therapeutic target highly expressed in armored and cold tumors, is targeted by exercise-stimulated miR-29a-3p [23]. Although exercise exerts broad antitumor effects, based on the molecular features of the immuno collagenic subtypes and exercise induced molecular profiles, patients with armored and cold tumors are expected to achieve greater relative benefits than those with soft and hot or quiescent tumors—specifically in terms of extracellular matrix reduction, increased intratumoral T/NK cell infiltration, and potentiation of immunotherapy responses.

Shortcomings and future research directions

The studies described herein first defined the concept of armored and cold tumors in the scientific community and suggested possible therapeutic strategies from three perspectives: anti-tumor therapy, concomitant therapy, and physiological

Table 1 B7-H3-Related Clinical Trials in Solid Cancers

NCT Number	Cancer types	Interventions
NCT01918930	Melanoma	MGA271
NCT02381314	Melanoma and non-small cell lung cancer	Enoblituzumab
NCT02628535	Advanced solid tumors	MGD009
NCT02982941	Neuroblastoma, rhabdomyosarcoma, osteosarcoma, and others	Enoblituzumab
NCT03198052	Lung cancer	CAR-T cell
NCT03406949	Advanced solid tumors	MGD009
NCT03729596	Solid tumors	MGC018
NCT04022213	Desmoplastic small round cell tumors, and peritoneal cancer	Omburtamab I^{131}
NCT04077866	Brain and nervous system tumors	CAR-T cell
NCT04129320	Head and neck cancer	Enoblituzumab
NCT04145622	Advanced solid tumors	DS7300
NCT04185038	Brain and nervous system tumors	CAR-T cell
NCT04385173	Brain and nervous system tumors	CAR-T cell
NCT04432649	Solid tumors	CAR-T cell
NCT04433221	Sarcoma, osteoid sarcoma, and Ewing sarcoma	CAR-T cell
NCT04483778	Solid tumors	CAR-T cell
NCT04544592	B-cell acute lymphoblastic leukemia/non-Hodgkin lymphoma	CAR-T cell
NCT04630769	Ovarian cancer	Enoblituzumab
NCT04634825	Head and neck cancer	Enoblituzumab
NCT04637503	Neuroblastoma	CAR-T cell
NCT04670068	Epithelial ovarian cancer	CAR-T cell
NCT04691713	Solid tumors	CAR-T cell
NCT04692948	Acute myeloid leukemia	CAR-T cell
NCT04743661	Brain and nervous system tumors	Omburtamab I^{131}
NCT04842812	Solid tumors	CAR-T cell
NCT04864821	Osteosarcoma, neuroblastoma, gastric cancer, and lung cancer	CAR-T cell
NCT04897321	Solid tumors	CAR-T cell
NCT05063357	Brain and nervous system tumors	Omburtamab I^{131}
NCT05064306	Brain and nervous system tumors	Omburtamab I^{131}
NCT05143151	Advanced pancreatic cancer	CAR-T cell
NCT05190185	Malignant melanoma, lung cancer, and colorectal cancer	CAR-T cell
NCT05211557	Ovarian cancer	CAR-T cell
NCT05241392	Brain and nervous system tumors	CAR-T cell
NCT05276609	Advanced solid tumors	HS-20093
NCT05280470	Extensive-stage small-cell lung cancer	DS7300
NCT05293496	Advanced solid tumors	MGC018
NCT05323201	Hepatocellular carcinoma	CAR-T cell
NCT05341492	EGFR/B7H3-positive advanced lung cancer and breast cancer	CAR-T cell
NCT05366179	Brain and nervous system tumors	CAR-T cell
NCT05405621	Advanced solid tumors	BAT8009
NCT05474378	Brain and nervous system tumors	CAR-T cell
NCT05515185	Advanced solid tumors	CAR-T cell
NCT05562024	B7-H3-positive relapsed/refractory neuroblastoma	CAR-T cell
NCT05722171	Relapsed/refractory acute myeloid leukemia	CAR-T cell
NCT05731219	Relapsed/refractory acute myeloid leukemia	CAR-T cell
NCT05752877	Brain and nervous system tumors	CAR-T cell
NCT05768880	Brain and nervous system tumors	CAR-T cell
NCT05835687	Brain and nervous system tumors	CAR-T cell
NCT05914116	Advanced solid tumors	DB-1311
NCT05991583	Advanced malignant tumors	IBB0979
NCT06018363	Brain and nervous system tumors	CAR-T cell
NCT06052423	Extensive-stage small-cell lung cancer	HS-20093
NCT06112704	Advanced solid tumors	HS-20093
NCT06158139	Pancreas cancer and relapse/resistant cancer	CAR-T cell
NCT06203210	Small cell lung cancer	DS7300
NCT06221553	Brain and nervous system tumors	CAR-T cell
NCT06305299	Ovarian cancer	CAR-T cell
NCT06347068	Triple-negative breast cancer	CAR-T cell

Table 1 Continued

NCT Number	Cancer types	Interventions
NCT06362252	Extensive-stage small-cell lung cancer	DS7300
NCT06372236	B7-H3-positive relapsed/advanced malignant solid tumors	CAR-T cell
NCT06422520	Advanced solid tumors	BGB-C354
NCT06482905	Brain and nervous system tumors	CAR-T cell

therapy (**Figure 1B**). However, further in-depth research should be conducted. First, in addition to concomitant medications, pre-existing comorbidities in patients with cancer warrant investigation. Attention should be paid to common pre-existing comorbidities, including type 2 diabetes, hypertension, abnormal lipid metabolism, and other diseases. Moreover, potential effects on the armored and cold transformation of the TME and the underlying molecular mechanisms should be investigated. Given the plasticity of the TME, non-invasive diagnostic technologies, such as deep learning with B-mode ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), and visualization of the collagen in the TME, are urgently needed. Another promising research direction is using physical stimulation, such as ionizing radiation, light, electricity, magnetic fields, or ultrasound, to potentially decrease collagen production or induce collagen denaturation, thereby boosting anti-tumor immune responses [24]. Clinical trials guided by the characteristics of armored and cold tumors stand to benefit both cancer patients and oncologists. In addition, the development of novel biomaterials for collagen degradation is an important therapeutic method for further investigation.

Concluding remarks

Cancer is a complex systemic disease involving constant interactions among cancer cells, the ECM, and other cell types present in the TME. Collagen in the ECM plays an essential role during cancer progression, and consequently is a promising therapeutic target for cancer management. Although collagen has traditionally been considered a structural scaffold, it elicits a myriad of biophysical, biochemical, and cell biological alterations that extensively affect tumor metabolism, growth, and immunity. Leveraging the biological association between collagen deposition and immune activity in the TME has enabled the identification of armored and cold tumors, a refractory subset of solid cancer, as well as the proposal of potential therapeutic strategies. With advances and interdisciplinary integration in cell biology, oncology, material science, and nanotechnology, current translational priorities include (i) developing collagen-based imaging biomarkers to non-invasively stratify tumors and (ii) designing combination regimens that disrupt collagen barriers while enhancing immunotherapy penetration in armored and cold tumors. Such strategies have promise in accelerating clinical translation and improving patient quality of life.

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Conflict of interest

Jie Mei is an Editorial Board Member of *BIO Integration*. He was not involved in the peer-review or handling of the manuscript. The other authors have no other competing interests to disclose.

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