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Tumor Immunotherapy Progress: Arsenic Trioxide and Bee Venom from a Traditional Chinese Medicine Perspective

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Abstract

Tumor immunotherapy, particularly immune checkpoint inhibitors (ICIs), has become a cornerstone of comprehensive cancer treatment. Nevertheless, primary and acquired resistance remain persistent challenges in solid tumors, largely driven by an immunosuppressive tumor microenvironment (TME) and insufficient tumor immunogenicity. The traditional Chinese medicine (TCM) philosophy of Fuzheng Quxie—strengthening the body's vital qi while eliminating pathogenic factors—offers a compelling conceptual foundation for modulating the TME and converting immunologically "cold" tumors into "hot" ones. From the perspective of cellular stress and immune activation, this review systematically examines the immunomodulatory mechanisms and recent therapeutic advances of two representative TCM-derived agents. We highlight how arsenic trioxide (ATO), beyond its classical success in acute promyelocytic leukemia (APL), triggers a "viral mimicry" effect by pharmacologically rescuing mutant p53, thereby potentiating systemic antitumor immunity via the cGAS-STING pathway. We also examine how melittin, the principal active component of bee venom, induces immunogenic cell death (ICD) and serves as a potent adjuvant in biomimetic nanovaccines for dendritic cell cross-presentation. Furthermore, emerging clinical evidence suggests that specific TCM formulae may extend this paradigm, improving response rates and mitigating immune-related toxicities when combined with ICIs. Looking forward, the integration of single-cell multi-omics, advanced nanodelivery systems, and biomarker-driven clinical trials is expected to transform TCM-assisted immunotherapy from empirical practice into evidence-based precision medicine.

Keywords

tumor immunotherapy, traditional Chinese medicine (TCM), arsenic trioxide (ATO), melittin, immunogenic cell death (ICD), cGAS-STING pathway, tumor microenvironment (TME), nanodelivery, Fuzheng Quxie

1. Introduction

Cancer immunotherapy, particularly immune checkpoint inhibitors (ICIs) and adoptive cell therapies, has reshaped the clinical management of multiple solid tumors [1, 2]. Yet durable responses remain confined to a minority of patients, as most solid tumors exhibit either primary or acquired resistance. A central driver of this resistance is the immunologically "cold" tumor microenvironment (TME)—one characterized by sparse T cell infiltration, abundant immunosuppressive cells, and low tumor immunogenicity. Converting such "cold"

tumors into "hot," immune-infiltrated lesions therefore stands as a defining challenge in contemporary immuno-oncology.

Within this landscape, the philosophy of traditional Chinese medicine (TCM) provides an underexplored conceptual resource. The classic TCM principle of *Fuzheng Quxie*—strengthening normal *qi* while eliminating pathogenic factors—can be reinterpreted in modern immunological terms as a coordinated program of immune activation and immune normalization. A particularly intriguing branch of this tradition is the strategy of "fighting poison with poison" (*Yi Du Gong Du*), which employs inherently toxic natural substances to attack malignant lesions. From a contemporary perspective, the value of such agents may extend well beyond direct cytotoxicity: by inflicting controlled cellular stress, they can induce immunogenic cell death (ICD), releasing danger signals that reprogram the TME.

This review focuses on two paradigmatic toxic TCM-derived agents—arsenic trioxide (ATO) and bee venom (primarily its active peptide melittin)—to illustrate how the *Fuzheng Quxie* framework is being realized at the molecular level. We first establish an immunological interpretation of *Fuzheng Quxie* as a theoretical basis. We then systematically examine the mechanisms by which ATO and melittin induce ICD, activate innate immune signaling pathways, and potentiate antitumor immunity. Finally, we assess the available clinical evidence and outline key challenges that must be addressed to advance this field from empirical practice toward mechanism-guided precision immunotherapy.

2. 2. Theoretical Basis: An Immunological Reinterpretation of *Fuzheng Quxie*

In classical TCM doctrine, disease is understood as an imbalance between *Zheng Qi* (the body's vital defensive energy) and *Xie Qi* (pathogenic factors). The therapeutic principle of *Fuzheng Quxie* accordingly aims to reinforce the former while eliminating the latter. Within the context of tumor immunology, this dual mandate finds a striking parallel: *Quxie* aligns with the induction of tumor cell death and the release of tumor antigens, whereas *Fuzheng* corresponds to the activation of antigen-presenting cells, the priming of effector T cells, and the restoration of an immune-permissive microenvironment.

Crucially, certain forms of cell death are inherently immunogenic. ICD is defined by the spatiotemporally coordinated emission of damage-associated molecular patterns (DAMPs)—including surface-exposed calreticulin (CRT), secreted ATP, and released high-mobility group box 1 (HMGB1)—that collectively recruit dendritic cells, enhance antigen cross-presentation, and provide the costimulatory signals necessary for T cell activation [13]. ICD thus serves as a molecular bridge: it is simultaneously an act of Quxie (eliminating tumor cells) and a trigger for *Fuzheng* (awakening innate and adaptive immunity).

This framework holds particular relevance for toxic TCM agents employed under the "fighting poison with poison" paradigm. Unlike conventional chemotherapeutics that often induce immunologically silent apoptosis, ATO and melittin impose a multimodal cellular stress—encompassing oxidative injury, mitochondrial damage, and, in the case of ATO, transcriptional reactivation of endogenous retroelements—that converges on the ICD pathway. The resulting immunological cascade not only destroys the targeted tumor cells but also reconditions the TME, creating a favorable environment for T cell infiltration and restoring sensitivity to ICIs. The following sections dissect the specific molecular circuits through which ATO and melittin execute this *Fuzheng Quxie* program.

3. Literature Review

3.1 Clinical Progress and Limitations of Immune Checkpoint Inhibitors

Since the approval of the first CTLA-4 antibody in 2011, ICIs targeting the PD-1/PD-L1 axis have achieved clinical breakthroughs across more than 20 malignancies. Despite these advances, the majority of patients still face primary or acquired resistance. The biological basis for ICI failure is multifactorial: low tumor mutational burden (TMB), defects in antigen presentation (e.g., β2-microglobulin mutations), activation of oncogenic pathways such as Wnt/β-catenin that exclude T cells, and the establishment of an immunosuppressive TME replete with regulatory T cells (Tregs), myeloid-derived suppressor cells (MDSCs), and M2-polarized tumor-associated macrophages (TAMs). Together, these cellular and molecular barriers dampen both the priming and the effector phases of the antitumor immune response. Current combination strategies partially address these

hurdles but often exacerbate systemic toxicity, underscoring the urgent need for tumor-selective immunostimulatory modalities.

Against this backdrop, toxic TCM-derived agents capable of inducing ICD offer distinctive translational value. While conventional chemotherapeutics such as oxaliplatin can also trigger ICD, their narrow therapeutic windows limit broader application. By contrast, ATO and melittin engage unique immune-activating mechanisms: ATO primarily mediates p53-dependent DNA damage responses and mitochondrial stress, whereas melittin activates innate immune sensors through direct membrane disruption. Empowered by modern systems biology, the immunological dimensions of these traditional medicines are being progressively deciphered, opening new avenues for overcoming ICI resistance.

3.2 Arsenic Trioxide: From Standard APL Therapy to Immune Remodeler

Arsenic trioxide (ATO) originates from the traditional TCM mineral medicine Pi Shuang. In the 1990s, researchers in China pioneered the "Shanghai Protocol," which combined ATO with all-trans retinoic acid (ATRA) for the treatment of acute promyelocytic leukemia (APL), achieving a remarkable cure rate exceeding 90% [14]. Although ATO was initially thought to act primarily by degrading the PML-RARα oncoprotein and inducing apoptosis, recent systematic investigations reveal that its therapeutic value extends deeply into immune regulation and TME remodeling.

The central mechanism underlying ATO-induced immune remodeling is the pharmacological rescue of mutant p53. Trivalent arsenic ions released by ATO covalently bind to specific mutant p53 conformations, restoring wild-type-like folding and transcriptional activity [3], although rescue efficacy varies considerably across different p53 mutations [4]. In susceptible cancer models, ATO treatment derepresses endogenous retroviruses (ERVs) and drives the transcription of endogenous double-stranded RNA (dsRNA), thereby establishing a "viral mimicry" state [5]. These nucleic acid species are sensed by intracellular pattern recognition receptors, activating the cGAS-STING pathway and triggering a type I interferon (IFN-I) response that, in turn, promotes $CD8^+$ T cell recruitment [5]. It should be noted that the dependency of this antitumor immunity on $CD8^+$ T cells and IFN-γ signaling has thus far been demonstrated in specific transplantable and genetically engineered models; its generalizability to the heterogeneous landscape of human solid tumors remains an active area of investigation. Recent work further suggests that physical interventions, such as hypothermia, may synergize with ATO to rescue temperature-sensitive p53 mutants, expanding the scope of this pharmacological strategy [21].

Regarding multi-dimensional immune cell regulation, ATO affects a broad spectrum of targets. Single-cell RNA sequencing (scRNA-seq) has revealed significant expansion of specific $CD8^+$ T cell subsets and activated natural killer (NK) cells in the bone marrow of APL patients following ATO treatment [6]. Notably, ATO exerts a dual regulatory effect on NK cells: it may delay their early reconstitution while simultaneously enhancing their cytotoxic activity at certain concentrations. In hepatocellular carcinoma (HCC) models, ATO treatment has been associated with mitochondrial damage, mtDNA leakage, activation of the cGAS-STING pathway, enhanced $CD8^+$ T cell infiltration, and PD-L1 upregulation [17, 18]. Evidence of macrophage reprogramming toward an M1-like phenotype was also observed [18]. However, the extent to which this reflects a direct effect of ATO on macrophages versus an indirect consequence of IFN-I signaling from tumor cells remains unclear.

Capitalizing on ATO's highly immunogenic nature, cutting-edge nanotechnology is accelerating its translation into novel vaccine platforms. ATO-treated tumor cells used as whole-cell vaccines can elicit robust immunological memory responses [19]. Moreover, PLGA nanoparticle-based ATO delivery and tumor antigen microparticle-based nanovaccines co-loaded with ATO and CpG have both been shown to efficiently induce ICD, thereby facilitating the conversion of "cold" tumors into "hot" ones [20].

In summary, ATO possesses dual functionality: direct cytotoxicity and profound TME remodeling. It not only triggers viral mimicry and innate immune sensing through mutant p53 reactivation, but also more broadly potentiates adaptive antitumor immunity by inducing ICD, modulating NK cells, and activating the cGAS-STING axis. Its immunomodulatory effects are therefore not confined to tumors harboring p53 mutations, underscoring the generality and translational potential of this approach.

3.3 Melittin: From Cytotoxic Agent to in Situ Nanovaccine Adjuvant

Melittin, the principal active component of bee venom, is a 26-amino acid amphipathic α-helical peptide. Although its canonical mechanism involves intercalating into lipid bilayers to form pores and cause membrane lysis, recent studies have uncovered its deeper immunomodulatory potential.

Melittin can induce ICD through multiple converging pathways. Its membrane-disruptive property triggers the release of ATP—a potent "find-me" signal that robustly recruits dendritic cells (DCs). The ensuing mitochondrial damage exposes calreticulin (CRT), an "eat-me" signal that facilitates phagocytosis of antigens. Furthermore, bee venom inhibits the PI3K/Akt/mTOR survival pathway. It sensitizes tumor cells to extrinsic apoptosis mediated by effector lymphocytes by upregulating death receptors, such as Fas and TNFR2 [10], thereby significantly potentiating NK cell cytotoxicity against tumors.

However, the clinical translation of free melittin faces formidable obstacles: its indiscriminate membrane-lytic activity causes severe hemolysis and systemic toxicity; its peptide nature makes it susceptible to rapid enzymatic degradation and poor tumor accumulation; and its heterologous origin can provoke anti-drug antibodies or even anaphylaxis. Consequently, unmodified melittin has proven exceptionally difficult to develop as a direct therapeutic agent.

To overcome these barriers, a range of nanoscale delivery platforms has been explored. Liposomal encapsulation and polymer-based nanoparticles can shield melittin from enzymatic degradation and reduce nonspecific hemolysis, while tumor-targeting ligands, such as RGD peptides or hyaluronic acid, enhance tumor accumulation. pH-responsive and matrix metalloproteinase (MMP)-sensitive nanocarriers exploit TME cues to achieve local, on-demand melittin release. More recently, biomimetic strategies—including coating nanoparticles with tumor cell membranes or loading melittin into exosome-like vesicles—have shown promise in extending circulation time and enhancing tumor homing.

A particularly instructive example is the multicomponent biomimetic nanovaccine platform nAg-MRDE/Mn, which integrates melittin, tumor neoantigens, and manganese ions (a STING agonist) [9]. In preclinical models, intratumoral injection of this platform achieved a tripartite synergy—ICD induction, enhanced DC cross-presentation, and STING-mediated IFN-I amplification—resulting in robust $CD8^+$ T cell responses and durable immunological memory [9]. This and related work demonstrate that melittin, once considered too toxic for systemic administration, can be repositioned as a potent in situ vaccination adjuvant when delivered through rationally engineered nanomaterials.

3.4 From Monomers to Formulae: Clinical Validation of the "Toxicity-Driven Immune Activation" Paradigm

Notably, the mechanistic logic established by ATO and melittin—whereby controlled cellular stress enhances tumor immunogenicity and remodels the TME—finds indirect clinical corroboration in studies of multi-herb TCM formulae combined with ICIs. A meta-analysis of 41 randomized controlled trials (RCTs) demonstrated that specific TCM-based adjuvant therapies significantly improved objective response rate (RR = 1.34), disease control rate (RR = 1.15), and overall survival (WMD = 1.46 months) compared with ICI monotherapy, while concurrently reducing the incidence of immune-related adverse events (RR = 0.82) [7]. These benefits, however, are not conferred by generic herbal interventions; they depend on regimens tailored to disease-specific pathogenesis.

For instance, in advanced HCC, the Shenqi Jiedu (SQJD) decoction, when combined with PD-1 inhibitors, enhanced intratumoral $CD8^+$ T cell infiltration, an effect mechanistically linked to gut microbiota remodeling and the enrichment of beneficial probiotics via the gut–liver axis [8]. Similarly, the Jianpi Huayu (JPHD) decoction was shown to impede M2-like macrophage polarization, thereby removing an immunosuppressive barrier to effector T cell trafficking into the tumor core [8]. Although the active constituents responsible for these effects remain largely unidentified, the functional outcomes—enhanced T cell infiltration, macrophage reprogramming, and reduced immune-related toxicity—closely parallel the immunological endpoints achieved by ATO and melittin in preclinical models. This convergence suggests that a subset of classical Fu-Zheng formulae may operate, at least in part, through a toxicity-driven or stress-induced immune activation mechanism analogous to that of ATO and melittin.

These pooled clinical results are encouraging, but they are subject to the substantial heterogeneity of the included formulae and trials, and their interpretation is constrained by the absence of consistent patient stratification or biomarker-guided enrollment across studies. These data should therefore be regarded as hypothesis-generating rather than definitive. Future clinical studies must prioritize standardized, quality-controlled formulations and incorporate biomarker-driven patient stratification—for example, by PD-L1 expression, TMB, or gut microbiome signatures—to rigorously validate TCM's contribution to immune sensitization.

4. Discussion and Synthesis

The existing literature signals a fundamental shift in how TCM-derived toxic agents are perceived. As demonstrated above, ATO and melittin converge on ICD induction and innate immune activation, directly addressing the core unmet need in the ICI era: converting immunologically “cold” tumors into “hot” ones. Although ICIs show remarkable efficacy in tumors with pre-existing T cell infiltration, they frequently fall short against poorly immunogenic solid tumors. ATO remodels tumor immunogenicity at the transcriptional level by pharmacologically rescuing p53 and triggering viral mimicry and STING activation. Melittin, by contrast, creates a pro-inflammatory microenvironment through physical membrane disruption and the massive release of DAMPs, conditions highly conducive to the recruitment of antigen-presenting cells. Whether deployed as monotherapies or in combination regimens, both agents establish essential immunological prerequisites for subsequent ICI intervention, forming an innovative sequential strategy akin to the “prime-and-boost” approach in vaccine development.

The integration of cutting-edge nanotechnology represents a transformative step toward precision TCM immunotherapy. As exemplified by the nAg-MRDE/Mn nanovaccine platform and related work on ginseng-derived nanovesicles [12], TCM-derived molecules are evolving from crude extracts into core functional modules within engineered immune materials. Nanosystems confer several key advantages on traditional toxic drugs: optimized pharmacokinetics and reduced systemic toxicity through controlled release; synergistic co-delivery of multiple therapeutic modalities within a single carrier; tumor-specific triggering via TME-responsive elements such as low pH, reactive oxygen species, and specific enzymes; and protection of labile peptides from degradation. This convergence of materials science, immune engineering, and TCM pharmacology has been increasingly recognized as a transformative strategy for tumor immunotherapy [11], holding the promise of overcoming the long-standing bottlenecks of TCM—namely, low bioavailability and poorly defined mechanisms—while preserving its intrinsic advantage of multi-target regulation.

Despite these advances, the further development of this field must address three major challenges.

First, the systemic elucidation of mechanisms remains incomplete. For ATO, the relative contribution of mutant p53 rescue to immune activation in vivo versus p53-independent pathways—such as direct mitochondrial damage and ROS-mediated ICD—has yet to be quantitatively dissected. For melittin, the relative importance of membrane pore formation, mitochondrial DNA release, and death receptor upregulation to the overall antitumor immune response is poorly defined. It may shift with peptide concentration and the local biochemical milieu of the TME. Addressing these questions with single-cell multi-omics and spatial transcriptomics will be critical for guiding rational combination strategies.

Second, significant translational barriers persist in the development of nanomedicine. ATO-loaded nanocarriers must reconcile the high aqueous solubility and rapid renal clearance of free arsenic ions with the requirement for sustained, tumor-localized release. For melittin-based formulations, key challenges extend beyond pharmacokinetics to include the peptide's intrinsic immunogenicity, which can trigger anti-drug antibodies or hypersensitivity upon repeated administration. Furthermore, scaling up the production of complex biomimetic nanovaccines while maintaining batch-to-batch consistency represents a non-trivial engineering challenge that demands early interdisciplinary collaboration.

Third, high-quality clinical evidence remains scarce. As the case studies of ATO and melittin illustrate, future progress depends on identifying the specific active constituents within complex TCM formulae and mapping their immunological mechanisms at single-cell resolution. Prospectively designed, mechanism-driven, and biomarker-guided randomized controlled trials will be essential to definitively validate TCM's contribution to immune remodeling in cancer therapy.

5. Conclusion

This review has shown that TCM-derived toxic agents, exemplified by ATO and melittin, are being redefined—from traditional cytotoxic drugs into potent immunological stressors capable of inducing ICD, activating innate sensing pathways, and converting immunologically "cold" tumors into "hot" lesions responsive to ICIs. The ancient wisdom of "fighting poison with poison," when interpreted through the *Fuzheng Quxie* framework, thus acquires a concrete molecular foundation. Nanodelivery technologies have further accelerated this translation by improving targeting, enabling multimodal synergy, and mitigating toxicity. Looking ahead, integrating single-cell multi-omics, AI-driven target discovery, and biomarker-guided clinical trials will be essential to transform this emerging paradigm from empirical practice into evidence-based precision immunotherapy.

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