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Allergy-assisted passive immunotherapy: Harnessing IgE-mediated responses for cancer treatment

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Abstract

Allergy-assisted passive immunotherapy proposes an anti-tumor strategy by transferring allergen-specific immunoglobulin E (IgE) antibodies from atopic donors to cancer patients via transfusion, followed by controlled allergen exposure to induce localized degranulation and inflammation within the tumor microenvironment. This review assesses the biological plausibility, potential mechanisms, and safety considerations of allergy-assisted passive immunotherapy - emphasizing its applicability to immunocompromised patients. While promising, the approach requires rigorous clinical validation.

Keywords: Immunotherapy; IgE; Allergy Cascade; Tumor Microenvironment; Passive Immunity

1. Introduction

Cancer immunotherapy enhances immune recognition and elimination of malignant cells, showing success in cancers like melanoma and lung carcinoma [1]. However, immunocompromised patients or those with genetic resistance to immune activation often respond poorly [2]. Allergy-assisted passive immunotherapy offers an alternative by exploiting the allergy cascade—typically a deleterious immune response—to target tumors. IgE antibodies, central to allergic reactions, activate effector cells (e.g., mast cells, basophils) to release inflammatory mediators upon allergen binding [3]. This review explores allergy-assisted passive immunotherapy and its potential to disrupt tumor progression through passive IgE transfer and localized allergen-induced inflammation.

2. Discussion

2.1. Mechanisms of Allergy-Assisted Passive Immunotherapy

Allergy-assisted passive immunotherapy involves two steps: #1 transfusion of allergen-specific IgE from atopic donors and #2 allergen exposure to trigger degranulation within the tumor microenvironment. IgE binds FcεRI receptors on effector cells, persisting in recipients for up to 7 weeks post-transfusion. (4) Studies confirm effector cell infiltration into solid tumors (e.g., breast and pancreatic cancers), where they release histamine, cytokines (e.g., TNF-α, IL-6), and proteases upon activation. (5) These mediators may disrupt tumor vasculature, induce apoptosis, or recruit additional immune cells. (6)

For immunocompromised patients, passive IgE transfer bypasses the need for endogenous antibody production, a limitation linked to genetic factors. (7) Localized allergen delivery (e.g., intratumoral injection) could enhance specificity, minimizing systemic reactions. (8)

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2.2. Safety Considerations

The primary risk of allergy-assisted passive immunotherapy is anaphylaxis, a systemic IgE-mediated reaction with a mortality rate of 0.5–1% in severe cases. (9) Cancer patients, often frail, may tolerate such events poorly. Epinephrine auto-injectors mitigate acute symptoms, but their efficacy in this context remains untested. Multiple transfusions could amplify IgE levels, necessitating careful dosing and monitoring. Short-term sensitization (no immunological memory) reduces chronic inflammation risks. (10)

2.3. Challenges

Allergy-assisted passive immunotherapy leverages a well-characterized immune pathway for an unconventional purpose. Approximately 25% of the U.S. population has allergies, providing a viable donor pool. (11) Advances in allergen-specific IgE production (e.g., U.S. Patent 11,911,463) could standardize supply, though efficacy requires validation beyond patent claims. If applicable, preclinical studies may demonstrate IgE-mediated tumor reduction via mast cell activation to support plausibility. (12)

However, challenges persist. The tumor microenvironment complexity—hypoxia, immunosuppression—may dampen inflammatory effects. (13) Optimal allergens, delivery methods, and patient selection criteria are undefined. The right-to-try framework could facilitate early trials after addressing informed consent from terminal patients. (14)

3. Conclusion

Allergy-assisted passive immunotherapy represents a hypothesis in cancer immunotherapy, merging allergy and oncology principles. Its reliance on passive IgE transfer offers hope for immunocompromised patients, but efficacy, safety, and feasibility hinge on future research. Controlled clinical trials are essential to elucidate mechanisms, optimize protocols, and assess therapeutic potential.

Compliance with ethical standards

Acknowledgments

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Author Profile

Michael John Dochniak has authored several books on Alzheimer's disease, Artificial Intelligence, Autism Spectrum Disorders, and Climate Change through Cambridge Scholars Publishing and Nova Science Publishers.

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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