International Journal of Cancer Medicine

Review Article | Vol 6 Issue S7

Cancer Immunotherapy - Allergies and Degranulation

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Received: February 28, 2023; Accepted: March 16, 2023; Published: March 23, 2023

ABSTRACT

Can the allergy cascade provide a rate-limiting step for metastatic disease? Pro-allergy therapy using humoral-IgE skin creams increase the production of IgE-primed effector cells. Effector cells are known to permeate solid tumors. Degranulation of IgE-primed effectors cells in the tumor microenvironment releases cytotoxins that may inhibit malignancy. This allegro-oncology review will discuss degranulation as cancer immunotherapy.

KEYWORDS

Allergy; Cancer; Degranulation; IgE; Immunotherapy; Skin cream

INTRODUCTION

Research indicates that the immune system can inhibit metastasis. Discovering which mediators are involved and how they impart their effect identifies new targets to prevent metastatic disease [1].

Pushing the boundaries of cancer immunotherapy into atopy (i.e., many allergies) research increases our knowledge and understanding of allegro oncology.

Sherman et al. [2] suggest a prophylaxis hypothesis wherein IgE antibodies and their associated allergy symptoms may serve a protective function: the rapid expulsion of pathogens, dangerous natural toxins, and other carcinogenic antigens before they can trigger malignant neoplasia in exposed tissues.

Dochniak discusses pro-allergy cancer immunotherapy to starve out cancerous cells through cross reactivity [3,4], immune-metabolic interference [5], cancer attrition immunotherapy [6], and diet [7,8].

Can induced allergies and degranulation inhibit cancer progression? A new frontier in allegro-oncology is to evaluate the safety and efficacy of humoral-IgE skin creams.

Humoral-IgE skin creams [9] activate the body's adaptive immune system through contact dermatitis.

Engkilde et al. [10] showed an inverse association between contact allergy and non-melanoma skin- and breast cancer. Furthermore, an inverse association was present for brain cancer in women with contact allergies.

The humoral-IgE skin cream anti-cancer mechanism of action may involve the release of cytotoxic mediators during degranulation. For example, during the allergy cascade, allergen-specific IgE antibodies bind to effector

Citation: Michael J Dochniak, Cancer Immunotherapy - Allergies and Degranulation. Int J Can Med J 6(S7): 4-6.

cells to form IgE-primed effector cells. Allergen re-exposure causes degranulation and the release of mediators into the microenvironment.

Examples of IgE induced effector cell degranulation or stimulation and their anti-cancer effect.

BASOPHILS

Marone et al. [11] teach that activated basophils release mediators such as granzyme B, TNFα, and histamine to regulate tumor growth. There is importance to circulating basophil populations, their propensity for activation and degranulation, and their prognostic value for cancer patients.

EOSINOPHILS

Cromier et al. [12] discuss tumor-associated eosinophilia in numerous human cancers, and eosinophils are part of an early inflammatory reaction at the site of tumorigenesis.
Acharya et al. [13] teach that eosinophils contain granules carrying a plethora of cytotoxic mediators, such as the cationic proteins, eosinophil cationic protein, eosinophil peroxidase, and granzyme-A that can destroy target cells or tissues. However, the mechanisms of granule protein secretion (i.e., degranulation) in eosinophils leading to tumor cytotoxicity remain to be defined.

MAST CELLS

Oldford and Marshal [14] teach that mast cells are ideal candidates for targeted tumor immunotherapy due to their abundance at the periphery of many solid tumors, proximity to blood vessels, and ability to secrete distinct profiles of mediators. The specific approach required to target mast cells in cancer therapy will depend on the type of cancer, the stage of progression, the tumor microenvironment, and potential interactions with other immunotherapies. Mast cells can produce pre-formed and de-novo synthesized mediators following activation. Activated mast cells can initiate multifaceted responses with tumor cells, stromal cells, and immune cells.

Fereydouni et al. [15,16] discuss harnessing the antitumor mediators in mast cells as a new strategy for adoptive cell transfer for cancer. For example, tumor specific IgEs are processed on mast cells and reinfused into the cancer patient.

MACROPHAGE

Pellizarri et al. [17] teach IgE re-programs alternatively activated human macrophages towards pro-inflammatory anti-tumoural states. The research shows the potential of anti-tumour IgE antibodies to mediate immune effector cell-mediated killing of cancer cells and impact immune stroma, influencing macrophage phenotype and maturation states. The findings provide new insights into the interactions between human macrophages and IgE class antibodies. The clinical development of new immunotherapy strategies; targeting these often tissue and tumor-resident cells may point to previously unappreciated cascades with the potential to activate and re-educate immune stroma.

ANTIGEN-PRESENTING CELLS

Nigo et al. [18] disclose that antigen-presenting cells such as B-lymphocytes, monocytes, Langerhans cells, and dendritic cells in tumor infiltrate express CD23 and FcεRI. The researchers write that one of the mechanisms of immune escape by tumor cells is the defective differentiation and maturation of antigen-presenting cells, with the consequent reduction of adaptive immune responses against tumor antigens. IgE bound to the surface of dendritic cells via FcεRI interaction may increase the efficacy of antigen uptake and presentation by a 100–1,000 fold, leading to an efficient activation of T cells that results in an antitumor adaptive immune response.

CONCLUSION

An allergy cascade using humoral-IgE skin creams may be an effective cancer immunotherapy. Cytotoxic degranulation is a formidable frontier in allegro-oncology,

although, research efforts continue to explore safe and effective approaches to activate natural immunity for cancer inhibition.

CONFLICT OF INTEREST

The author is a co-founder of Alleamit, Inc., Minnesota, USA.

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