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Immunodominance as Complex Patterned Interplay of Antigen Quantitation Phenomena in Pressure Immunity

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Abstract

The basis of biology of immunodominance of antigens is defining term in the evolution of a specific antigenicity as carried forward by complex dimensions of an immune response to tumor epitopes. In such terms, immune pressure dynamics contextualize the evolving process of epitope redistribution as anergic states of non-reactivity or as terms of reference in defining such immunodominance of given antigenicity of individual tumor cells. It is further to such considerations that quantitative antigen presentation both correlates with evolution of antigen binding to the Major Histocompatibility Complex (MHC) structured molecules and with system profiles of redistribution inherent to antigen presentation on the surface membrane of both antigen presenting cells and also tumor cells.

Introduction

The immunodominance of given specific antigens is linked to the development of potentially robust immune response to tumors in general or to the emergence of profiles of reactivity of T cells in general. Neoadjuvant PD-1 immune checkpoint blockade reverses functional immunodominance amongst tumor antigen-specific T cells [1]. The complexities of immune response to neoplasms if further increased by antigen presentation phenomena inherently activate CD4+ and CD8+ T lymphocytes. Immunization with *in vitro* selected cancer variants that lack the immunodominant antigen can disrupt the immunodominance and avoid escape of cancers from host immunity [2]. In such terms, the evolving redistribution of antigen epitopes is a potential series of mechanisms that dominants in various ways in regard to particular quantitative degrees of expression levels of specific antigen epitopes. Somatic mutations can induce cancer-specific neoepitopes there recognized by autologous T cells as foreign and constitute ideal cancer vaccine targets [3]. Hence, it may be relevant to consider the variability of heterogeneity of epitope antigenicity as provided not only by antigens among various different types of tumor but also heterogeneity of such antigen expression within the same neoplastic lesion.

Demarcation Profiles of Antigenicity

In conceptual terms, demarcation profiles between tumors or tumor types are system parameters with the biologic significance of the immune response to mitotically active infiltrating lesions. A significant fraction of non-synonymous cancer mutations is immunogenic and that most of the immunogenic mutanome is recognized by CD4+ T cells; mutational MHC class II epitopes drive therapeutic immune response to cancer [4]. In such terms, the explicit dimensions of increments in the immune response might arise within systems of immunodominance of given expressed epitopes. It is not known whether the human immune system often mounts a T cell response against motions expressed by epithelial cancers [5]. In the sense of evolutionary pressure phenomena the redistribution of antigenic profiles may allow for heterogeneous differentiation of various multiple clones of malignant tumor cells.

Immunodominance

Immunodominance is hence a redistribution of epitopes within actively growing neoplasms within the system biology attempts of immunosurveillance attempts in creating reactivity to given tumor cell clones within a given neoplasm and neoplasm type. Immunohistochemistry often appears to highlight the variability of epitope expression as projected by antigenicity profiles of a tumor. In a restrictive sense, the evolutionary emergence of particular epitope expression is beyond homeostatic control and is thus a biologically inherent attribute of reactivity as dimensionalized by T cell subtypes.

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Antigenicity

Poor degrees of antigenicity or non-expression of antigens are simple parametric emergence phenomena in the inherent activation mechanics of T lymphocytes. Immunodominance constitutes the breadth of antitumor T CD8 responses and is as such considered an impediment to effective vaccination against cancer [6]. The simple juxtaposition of given antigen moieties is enhanced redistribution of epitope specificity as dictated by systems of predominantly quantitative degrees of expressivity and of binding affinity of the given epitope. Mucin-associated sialyl-Tn antigens bind to receptors on dendritic cells, macrophages and natural killer cells that result in overall immunosuppression by receptor masking or inhibit of cytolytic ability [7]. Major histocompatibility complex binding is a system profile determinant within the biologic expressivity inherent to epitope presentation to T lymphocytes. Robust antitumor immune responses are induced in pediatric acute lymphoblastic leukemia despite their low mutation burden and stress the importance of immunodominance in modulating cellular immune responses [8].

Expressivity of Antigen

Prominence of immunodominance is further contextual expressivity within the identity profile of the cell type origin of a specific tumor lesion and tumor histologic type. The evolving dimensions of tumor cell growth are determinants in terms of antigen loss phenomena on a clonal basis and on an individual tumor cell.

Potency of the specificity of antigen expression is secondary consideration of the activation phenomena of lymphocytes as projected within system biology of the molecular identity profiles as given expression of the immune response. Development of strategies that aim to enhance the total Tumor-associated antigens-specific CD8+ T cell response may be obtained by therapeutic boosting and/or specificity diversification [9]. The nature of molecular expression and of immune response bespeak of a whole series of complex interplay within the variability of anergic phenomena of various multiple complexes of antigenicity. Treatment with interferon gamma, which up regulates the transcription of MHC class I is involved in antigen presentation and most of tutor lines are able to respond normally [10].

Antigen Identity

Identity of particular antigens in antigenicity studies allows for a possible reappraisal of system reactivities within the given dimensions of expressed antigen expression by tumor cell heterogeneity phenomena. To date, the alterations in the expression of MHC molecules play a critical role in tutor development due to defective antigen presentation to T-lymphocytes and regulation of natural killer cell function [11]. The redistribution of presented antigen epitopes is further complex consideration as terms of quantitative degrees of expression on the tumor cell plasma membrane.

Dynamics of inclusion of immunodominance are permissive redistribution as recharacterized indices for further potential increases of epitope expression to T lymphocytes. Recombinant tumor-specific proteins are currently among the most promising candidate anticancer vaccines, and monitoring of cancer vaccine trials should systemically include the assessment of HLA association with responsiveness [12].

Complex nature dynamics of the dominance of antigen presentation is hence a consideration of vital significance in turnover processing of particularly antigen presenting cells in general terms. Distribution preference dynamics are prominent mechanisms within the profile specificity of indices of activation of T lymphocytes. MHC class I expression is up regulating during progression and therapy of HPV16-associated tutors and this may have important implications in the development of immunotherapeutic strategies [13]. Realization of system profiles of antigenicity are hence a complex integrative interplay of molecular dominance as determined by the evolutionary exposure of epitopes within the further development of sequence specificity of T cell receptor.

T Cell Specific Receptor

Lymphocyte specific antigen receptor is hence a process of selectivity in terms of antigenicity phenomena as expressed within the profile dynamics of redistribution of given antigen moieties as expressed on the lymphocyte/antigen presenting cells on the cell plasma membrane. Immunodominance redefines the quantitative affinity of receptivity of T lymphocytes as terms of pressure dynamics of redistributed membrane epitopes.

With regard to such immune defined pressure dynamics, the fluidity of the cell membrane may encompass the T lymphocyte specific receptor in terms that include the realization of binding affinity of the groove molecular structure of the MHC bearing cells. The status of the immune system in tumor-bearing animal is capable of defining the MHC profile of the tumor cells; MHC class I-negative metastatic colonies are generated in immunocompetent animals, and MHC class I positive colonies in T cell deficient individuals [14]. The production of incremental expressivity of such binding is itself dictated as gain/loss dimensions of surface antigen as expressed by tumor cells that in turn enhances the properties of biologic activation of the immune cells and of the antigen presenting cells. Tutor neoantigen heterogeneity thresholds provide a time window for combination immunotherapy where checkpoint blockade immunotherapy can become more effective [15]. Specificity issues allow for permissive interplay as possessed by and further enhanced by the antigen moiety interactivity with the antigen presenting cells. Such conditions provide general and specific attributes to immunodominant epitopes in general.

Concluding Remarks

Bypass phenomena of direct and indirect cross-reacting antigen presentation are integral to systems of potentiality that are significantly dependent on pathways of molecular interplay as defining receptivity and epitope presentation. It is with regard to system profiles of immunity that the reactivity of T lymphocytes both dimensionalizes the attributes of antigen dominance and also of potent interactivity of the MHC molecule as dictated by antigen/receptor binding. The close semblance theme with regard to profile shifts in epitope expression allow for a significant degree of interplay that is itself self-potentiating.

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