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Dual roles of cancer cell-expressed immunoglobulins in cancer immunology

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While the expression of both immunoglobulins and T cell receptors on cancer cells have been well-established for decades, the potential roles and mechanisms of action behind these cancerous antigen receptors have not been fully elucidated. A monoclonal antibody designated as RP215, which reacts specifically with the carbohydrate-associated epitope located on the heavy chain region of cancerous immunoglobulins and T cell receptors and not on normal immune cells, was used as a unique probe to study the roles of antigen receptors in the immunology of cancer cells. Through extensive cell-based biological and immunological studies, both anti-antigen receptors and RP215 were found to demonstrate similar actions on the gene regulations involved in the growth/proliferation of cancer cells, as well as on toll-like receptors involved in innate immunity. In addition, RP215-specific cancerous immunoglobulins are believed to capture or neutralize circulating antigen/antibodies which may be harmful to cancer cells within the human body. In contrast to normal B and T cells and their respective receptors in the conventional immune system, cancer cells co-express both immunoglobulins and T cell receptors and immune protection is exercised by unique mechanisms. For example, these cancer cell-expressed antigen receptors display a lack of class switching, limited hyper-mutation, aberrant glycosylations and a strong influence on the toll-like receptors of cancer cells. Therefore, it is hypothesized that both normal and cancerous immune systems may co-exist and operate simultaneously within the human body. The balance of these two immune factors for respective surveillance and protection may be relevant to the outcome of cancer immunotherapy in humans. A potential therapeutic strategy is being developed by using RP215 as a drug candidate to target cancer cells based on these observations.

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An update on monoclonal antibody-based therapy for asthma

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Current therapies for asthma are aimed at controlling disease symptoms and for the majority of asthmatics inhaled corticosteroid anti-inflammatory therapy is effective. However, this approach requires life-time therapy while a subset of patients remains symptomatic despite optimal treatment creating a clear unmet medical need. It is recognised that airway inflammation is key to asthma pathogenesis. Biopharmaceutical approaches have identified new therapies that target key cells and mediators that drive the inflammatory responses in the asthmatic lung. Such an approach may provide disease-modifying treatments. Significant areas of drug development include humanised monoclonal antibodies (mAb) for asthma therapy including those targeting the Th2 cytokines IL-4, IL-5 and IL-13. However, early clinical trials with these biologics in patients with asthma were for the most part disappointing even though they were highly effective in animal models of asthma. It is now generally accepted that asthma is a complex, heterogeneous mixture of syndromes that can be sub-divided into several phenotypes on the basis of clinical, physiological and inflammatory markers that in turn can result in variable responses to treatment. This in turn led to a rapidly evolving realisation that significant clinical effects with anti-cytokine-based therapies are observed in carefully selected patient populations that take asthma phenotypes into account. The development of discriminatory biomarkers and genetic profiling may aid identification of such patients with asthma. This talk will summarise the current evidence demonstrating the effectiveness of targeting Th2 cytokines in patients with poorly controlled asthma.

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