

Immunology in lung cancer

Editorial

Human body have evolved active defenses that compose the immune system in response to harmful challenges which include cancer cells, bacteria, virus, unicellular and multicellular pathogens. Several previous experiments have demonstrated that the growth of a syngeneic tumor in a mouse could be prevented by prior immunization with that same tumor. These studies have demonstrated T cells to play a major role in antitumor immune response. Several animal model studies revealed human T cells to be capable of specifically lysing antilogous tumor cells *in vitro*. T cells specifically secrete cytokines, such as interleukin-2 (IL-2), interferon- γ (IFN- γ), tumor necrosis factor- α (TNF- α) and granulocyte-macrophage colony stimulating factor, and proliferate in response to stimulation with antilogous tumor cells. *In vitro*, antitumor T-cells are able to be grown to large number and adoptively transferred to treat even substantial tumor burdens in mice and humans. Finally, tumor antigens recognized by antilogous human T cells have been identified by a variety of independent techniques. Collectively, these findings provide nearly interconvertible evidence that a T-cell immune response is able to occur against an antilogous tumor. Several previous experimental and clinical approaches have been developed to use recombinant cytokines, either singly or in combination, to augment against lung cancer. Several cytokines, such as IFN- α , β and γ , IL-1, IL-2, IL-4, IL-5 and IL-12; GM-CSF and TNF have been evaluated in cancer immunotherapy. The most notable obstacle of cytokine therapy is the complexity of the cytokine network itself. Three IFNs have demonstrated to increase class I MHC expression on tumor cells, whereas IFN- γ has demonstrated to increase class II MHC expression on macrophages. IFNs inhibit normal and malignantly transformed cells *in vitro*. Additionally, IFN- γ directly or indirectly increases the activity of macrophages, T cells, and NK cells, all of which play a major role in the immune response to tumor cells. TNF- α and TNF- β exhibit direct antitumor activity by tumor hemorrhage and regression, whereas TNF- α inhibit tumor angiogenesis by damaging the vascular endothelial cells.

Previous studies in human lung cancer focused on boosting immune response against putative tumor antigens with adjuvant, such as Bacille Calmette-Guerin (BCG), *Corynebacterium parvum* and levamisole. Each adjuvant was known to stimulate either humoral and/or cellular immune response and all these clinical trials were classified as “active non-specific immunotherapy”. BCG was the most commonly tested adjuvant and was administered intratumoral, intrapleural, intradermal or by aerosol routes. *Corynebacterium parvum* and levamisole have failed to alter survival in lung cancer. Active specific immunotherapy involves immune stimulation with tumor vaccine containing irradiated antilogous or allogeneic

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tumor cells obtained from tumor specimens. These tumor vaccines have been administered intralesional, intradermal, intralymphatic or subcutaneous, and were occasionally combined with adjuvant products, such as BCG, lytic virus, or Freund’s complete adjuvant. For lung cancer, despite several previous studies, no clear benefit has been demonstrated. Part of the failure in tumor vaccine immunotherapy has been due to the previous impossibility of matching immunogens to patients. The recent identification of TRAs and their encoding genes should newly lighten on active specific immunotherapy in lung cancer patients.

In conclusions, when considering the poor prognosis of patients with inoperable lung cancer, new therapeutic strategies need to be developed. Regarding a better understanding of the immune mechanisms involved in lung cancer could provide efficacious alternative therapy, either alone or adjuvant therapy.

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Conflict of interest

The author declares no conflict of interest.