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Editorial

Detailed study of Immunopharmacology

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ABSTRACT

Immunopharmacology is one among the youngest areas of pharmacology. The primary generation of immune-modulating agents included molecules drawn from oncology. These drugs, like methotrexate, azathioprine and cyclophosphamide, dampened immune responses through their anti-proliferative action. The second generation, notably cyclosporine, exploited some natural agents ready to block several signal transduction pathways. Many of those agents were suffering from several severe side effects, mainly thanks to their ability to affect every fast-proliferating cell type (alkylating agents, purine and pyrimidine analogues) or to affect signal transduction in several organs and tissues, with toxic effects (e.g. cyclosporine). Moreover, many of those agents were quite non-specific in their ability to depress immune function, thus generating a profound impairment in biological responses.

Keywords: Immunopharmacology, immune-modulating agents, immune responses

INTRODUCTION

However, in recent years, advances in our knowledge about how the system works have identified several molecular targets suitable for more selective modulation of immune function. These targets are often broadly divided into surface molecules and soluble mediators. Surface molecules play a fundamental role in antigen recognition, immune reaction activation, and homing and effector functions. Soluble mediators are involved in lymphocyte proliferation and differentiation, inflammatory response and cell recruitment. Likewise, there are currently two broad categories of agents suitable for targeting these molecules: small-molecule drugs and monoclonal antibodies (MAb). While small molecule drugs offer the advantage of potential oral administration and use in an outpatient setting, their experimental designing and testing are quite complex. MAb are clearly bulky and wish to be injected or infused, but their half-life is typically longer.

In fact, they typically engage their target molecule but they are doing not act as physiological ligands, even as antagonists, preventing the binding of the physiological counterpart. Furthermore, retaining their antibody properties, these molecules can activate cell-mediated cytotoxicity or quick removal of complexes from the circulation. However, a minimum of one drug targeted against inhibitory T cell subset may very well end in immune reaction stimulation. Much more difficult to supply are immune stimulating drugs. Cloning and expression technology has made an outsized array of cytokines available in highly purified form, many of which have made their thanks to clinical trials.

The intricacies of the cytokine network have hindered a more widespread application of this approach. In fact, many cytokines have overlapping and sometimes redundant roles; this suggests that infusion of one cytokine could also be unable to elicit the sort of response that that cytokine is putatively responsible in in vitro models. Furthermore, cytokine infusion may result in massive and non-specific activation of the system, leading to a clinical effect resembling overwhelming infection or septic shock. Nevertheless, a minimum of for one cytokine (namely, Interleukin-2), some benefits in clinical trials are observed, and further refinement of administration route and schedule are yielding promising results.

A third class of specific biotechnological agents includes chimeras, during which one cytokine or one antibody is conjugated to bacterial toxins or to radioactive substances. This approach delivers an increased toxic effect (radiation or toxins) with the high selectivity produced by the antibody/antigen or cytokine/receptor interaction. In theory, it might be possible to selectively affect just one limited subset of immune cells. However, several technical hurdles have delayed the event of those agents, and then far just one of them has gained approval for clinical use.

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