



## CLINICAL PHARMACOLOGICAL APPROACH TO THE RATIONAL USE OF IMMUNOCORRECTIVE DRUGS

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**Abstract:** This article provides a clinical pharmacological framework for the rational use of immunocorrective drugs — agents that modulate the immune response to restore homeostasis. Rather than categorizing these agents solely by their chemical structure or general effect, the discussion focuses on their functional classification based on immune system targets, mechanisms of dysregulation, and clinical scenarios. Emphasis is placed on tailoring immunocorrection to immune profile types, avoiding non-selective immune modulation, and integrating pharmacodynamic, pharmacokinetic, and immunogenetic data into therapeutic decisions.

**Keywords:** Immunocorrection, immunomodulators, clinical pharmacology, immune homeostasis, T-cell regulation, cytokine therapy, immune stratification.

### INTRODUCTION

The immune system maintains internal equilibrium not only by defending against pathogens but also by regulating tissue repair, surveillance against malignancy, and the resolution of inflammation. Dysregulation in this system can manifest as immunodeficiency, autoimmunity, chronic inflammation, or immune hyperresponsiveness. Immunocorrective drugs aim not merely to suppress or stimulate the immune response but to normalize its function according to pathological need.



Unlike traditional pharmacological interventions that target isolated molecular pathways, immunocorrection requires a systems-level understanding of immune architecture, redundancy, and plasticity. Rational use of these agents demands more than matching a drug to a diagnosis; it requires interpreting dynamic immune markers, recognizing immunopathological phenotypes, and accounting for pharmacogenomic variability. This article presents a clinical pharmacological perspective on when and how to use immunocorrective agents effectively and responsibly.

## **MATERIALS AND METHODS**

Immunocorrective drugs are often broadly labeled as "immunostimulants" or "immunosuppressants," but this binary classification obscures the complexity of immune modulation. A rational approach begins with immune stratification — assessing whether the dysfunction lies in the innate immune response (e.g., macrophage deficiency, dendritic cell dysfunction), adaptive immunity (T- or B-cell imbalance), or in the regulatory feedback loops (e.g., Treg suppression, cytokine overexpression).

Clinical scenarios where immunocorrection is relevant include:

- Post-infectious immune exhaustion (e.g., post-COVID-19 immunosuppression)
- Primary or secondary immunodeficiencies (e.g., iatrogenic after chemotherapy)
- Chronic inflammation with immune escape (e.g., viral hepatitis)
- Autoimmune flare regulation (e.g., systemic lupus with paradoxical immunodeficiency)
- Rational use implies choosing agents that restore specific deficits rather than globally activating or suppressing immunity.

## **RESULTS AND DISCUSSION**

Immunocorrection, while conceptually restorative, is not without risks. Overstimulation may lead to autoimmune flares, allergic sensitization, or immune exhaustion. In oncology patients, excessive stimulation may activate dormant neoplastic



processes. On the other hand, prolonged use of immune “boosters” in the absence of indication can suppress natural feedback loops, blunting endogenous immunity.

Rational clinical pharmacology entails using immunocorrective therapy only when benefit clearly outweighs risks, and when there is a biologically plausible target. It also implies time-bound therapy: these are not lifetime drugs, but interventions designed to reorient the immune system over a defined window [1].

One of the most forward-looking areas in immunocorrective pharmacology is the exploration of epigenetic regulation as a therapeutic target. The immune system's functionality is determined not only by its genetic architecture but also by epigenetic modifications—such as DNA methylation, histone acetylation, and microRNA expression—which govern immune cell differentiation and activation. Dysregulation at this level is increasingly recognized in various chronic immune disorders, including autoimmune diseases, allergies, and immunodeficiency syndromes with no identifiable genetic basis.

Modern immunocorrective strategies now include experimental and early-clinical epigenetic modulators, such as histone deacetylase (HDAC) inhibitors or DNA methyltransferase inhibitors, aimed at reprogramming dysfunctional immune responses. These agents offer a more nuanced immune recalibration, allowing for the restoration of immune homeostasis without broadly suppressing or overstimulating the entire system. This precision becomes particularly relevant in disorders with immune polarity shifts, such as lupus or mixed connective tissue diseases, where blanket immune stimulation may exacerbate pathology.

Beyond synthetic molecules, nutritional immunopharmacology focuses on the therapeutic application of vitamins, trace elements, and other bioactive compounds with immunoregulatory properties. Substances like vitamin D, zinc, selenium, omega-3 fatty acids, and glutamine have demonstrated clear immunomodulatory effects in clinical and



experimental settings. Their role includes enhancing antigen presentation, supporting T-cell maturation, and modulating cytokine release patterns [2].

Such agents are especially relevant in chronic low-grade immune dysfunctions, such as in elderly populations, patients with chronic fatigue syndrome, or those recovering from viral illnesses like COVID-19. However, their use as pharmacological tools demands precise dose monitoring, as therapeutic windows are often narrow and bioavailability can vary significantly depending on the form of administration, the nutritional state of the patient, and concurrent therapies. Thus, while these agents are often viewed as "natural" or safe, clinical oversight remains essential to avoid toxicity or ineffective under-dosing.

The role of the gut microbiota in systemic immune regulation has opened entirely new dimensions for immunocorrective therapy. It is now established that dysbiosis—a disrupted microbial balance—contributes to various immune-related conditions, including atopic dermatitis, inflammatory bowel disease, and even certain autoimmune disorders. This has led to the emergence of microbiota-based immunocorrection, which includes not only probiotics, but also postbiotics (bacterial metabolites like short-chain fatty acids) and prebiotics (selective substrates for beneficial bacteria).

Notably, compounds like butyrate and propionate—metabolites produced by microbial fermentation—can enhance regulatory T-cell (Treg) activity, reduce systemic inflammation, and restore mucosal barrier function. Such effects are especially valuable in conditions like post-viral immune fatigue, non-specific immunosuppression, and recurrent respiratory infections. However, the clinical use of microbiota-targeted therapies must be individualized, as each patient's microbiome is unique, and generalized regimens may not produce consistent or safe results.

Because immune modulation affects multiple physiological systems and can lead to non-linear and delayed responses, continuous clinical monitoring and pharmacovigilance are indispensable components of rational immunocorrective therapy. Immune-related



adverse effects may not be immediately apparent; for instance, overstimulation of the immune system may trigger autoimmune flares, allergic sensitization, or even lymphoproliferative syndromes in vulnerable populations [3].

Thus, rational use implies setting clear therapeutic endpoints, using validated immune biomarkers (e.g., CD4/CD8 ratio, immunoglobulin levels, pro- and anti-inflammatory cytokines), and limiting treatment duration to avoid dependency or immune suppression. Clinical pharmacologists and prescribers must approach immunocorrection as a dynamic process, not a static prescription—requiring periodic reassessment and adaptive dosing.

Additionally, attention must be given to drug–immune system interactions, especially in patients with complex medication regimens. For example, certain immunomodulators may potentiate or diminish the effects of vaccines, anticoagulants, or antineoplastic agents. Hence, immunocorrective pharmacotherapy must be integrated into a broader therapeutic ecosystem, guided by multidisciplinary input.

Despite their growing use, many immunocorrective agents lack robust validation through large-scale randomized controlled trials (RCTs). This is particularly true for low-molecular immunomodulators, bacterial lysates, and non-conventional agents such as peptide-based immunoregulatory molecules. Their use is often guided by real-world evidence, post-marketing surveillance, or expert consensus, rather than strict RCT-based hierarchies. While such evidence may support their effectiveness in certain patient groups, it also limits the strength of formal recommendations in clinical guidelines [4].

This gap highlights the need for rigorous pharmacological research, including mechanism-specific trial design, biomarker-stratified patient enrollment, and long-term safety assessments. Until then, clinicians must balance therapeutic optimism with scientific caution, ensuring that the drive to enhance immune performance does not outpace empirical validation.

## CONCLUSION



Immunocorrective pharmacology must move beyond empirical immunostimulation and toward targeted immune homeostasis restoration. This demands a shift in clinical mindset — from reactive to profile-driven prescribing, from general immune enhancement to selective functional repair. By anchoring therapy in measurable immune deficits, integrating pharmacokinetic awareness, and monitoring immune trajectories, clinicians can use these agents more safely and effectively. The future lies in personalized immunocorrection, where immune diagnostics and drug action are finely aligned to restore balance without collateral immune disruption.

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