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# Autologous Cytokine Fragments for Targeted Modulation of the Immune Response: Two Clinical Case Reports

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## Abstract

Cytokines play a central role in immune regulation, normally maintaining serum levels below the threshold for systemic activation. In pathological conditions, however, they may be excessively or insufficiently expressed, contributing to immune imbalance. This study explored the effects of microdoses of autologous cytokines fractionated into α and β subunits, hypothesizing that α subunits exert inhibitory modulation while β subunits stimulate endogenous activity. Two patients with altered cytokine profiles were included: one with interleukin-2 (IL-2) overexpression and one with reduced interleukin-6 (IL-6). Peripheral blood was processed to isolate cytokines, using plasma separation, magnetic microspheres, and differential centrifugation. The IL-2 α fraction was administered intramuscularly in the first case, aiming to downregulate expression, while the IL-6 β fraction was given in the second case to stimulate production. Cytokine levels were assessed before treatment, after treatment, and at six months. Both patients showed clinical improvement. IL-2 progressively decreased in the first case, while IL-6 increased and IL-2 decreased in the second, with values returning to physiological ranges. No adverse effects were observed. These findings suggest that administration of autologous cytokine fragments, separated into functional subunits, may offer a selective and well-tolerated strategy for modulating immune responses. Larger studies are needed to confirm these preliminary results and better define clinical applications.

***Keywords:*** immunomodulation; cytokine modulation; biological therapy; autologous therapy; low-dose immunotherapy

## Abbreviations

interleukin (IL), antigen (Ag), antibody (Ab), natural killer cells (NK), high-dose (HD), C-reactive protein (CRP).

## Introduction

The immune system is a complex and interconnected network of organs, tissues, cells, and molecules, with plasma playing a central role. Plasma contains numerous biologically active substances: protein hormones, immunoglobulins, signaling and transport molecules, coagulation factors, and fibrinogen. Approximately 90% of the elements responsible for tissue response and regeneration are contained in plasma, making it a reservoir of reparative and stimulatory molecules.

At the core of immune processes lies the interaction between a reactive element—such as an antigen (Ag)—and its specific counterpart capable of neutralizing the reactions such as an antibody (Ab). This interaction is defined by specificity, i.e., the ability of a given antibody to recognize a particular antigenic determinant, or the capacity of a polyclonal population of antibodies to selectively recognize a single antigen. Functionally, antibodies possess two domains: Fab regions that bind to the antigen, and Fc regions that interact with components of the innate and adaptive immune system, such as NK cells, phagocytes, and complement. Fc domains are fundamental for the in vivo efficacy of passive immunization strategies. Monoclonal antibodies, widely used in clinical practice, exploit these properties, including epitope specificity, neutralization, and Fc-mediated effector functions (Abraham, 2020).

The immunopathology of allergies is based on recognition of allergens by the immune system as if they were pathogenic threats. This triggers overproduction of cytokines such as IL-4, IL-5, and IL-13, accompanied by mast cell and eosinophil activation, leading to histamine release and inflammatory mediator cascades that manifest as clinical symptoms (Galli et al., 2008). A validated therapeutic approach to allergic diseases is allergen-specific immunotherapy, based on repeated administration of gradually increasing doses of causative allergen to induce tolerance (Anagnostou, 2023). This principle, first introduced by Leonard Noon in 1911, represents the foundation of active allergen immunization: controlled stimulation of the immune system can reduce rather than amplify the allergic response (Noon, 1911; Akdis, 2015).

Historically, the antibody-antigen interaction has been the cornerstone for vaccine development and therapeutic monoclonal antibody design. Immunity—specific or nonspecific—against an antigen can be induced either through vaccine administration (active immunization) or via exogenous antibody administration (passive immunization). Both approaches are supported by experimental and clinical evidence demonstrating their ability to induce immune protection and memory (Caserta et al., 2021; Sallusto et al., 2010).

In allergology, evidence suggests that administration of microdoses of antigens (via sublingual or intradermal routes) may induce faster desensitization with greater tolerability compared to conventional high-dose regimens, and with fewer systemic adverse effects (Smith et al., 2004). Moreover, low-dose IL-2 protocols have shown efficacy in modulating allergic responses through expansion of regulatory T cells, introducing new perspectives for low-intensity immunomodulation (Rosenzwajg et al., 2016; Klatsmann et al., 2024).

In recent years, the rationale for low-dose therapy has expanded beyond allergic disease. Microdoses of bioactive molecules, including antigens, hormones, antibodies, and cytokines, are being studied for their ability to interact with the immune system in a non-aggressive way, supporting endogenous regulation and tolerance. These approaches are based on nonlinear pharmacodynamics, where the clinical effect depends not on the absolute dose but on the ability of the molecule to activate receptor-mediated signals at very low thresholds (Floris et al., 2020; Jacques and Floris, 2022; Saxton et al., 2023).

The common goal of these strategies is not suppression, but rebalancing the immune response, acting physiologically and preserving the self-regulatory properties of the immune system. An alternative therapeutic paradigm is thus emerging controlled administration of sub-immunogenic doses of immunoactive molecules as a possible treatment for inflammatory diseases, minimizing adverse effects while restoring tolerance.

Passive immunization provides a logical basis for the therapeutic use of cytokines (Radonjic-Hoesli et al., 2022). Indeed, it is possible to stimulate or modulate immune activity by using a subject's own plasma, which acts via cytokine release (Peci et al., 2021).

Cytokines are crucial signaling molecules of the immune system, regulating activation, recruitment, and suppression of immune cells. They can initiate self-regulatory, reparative, and regenerative tissue processes. Their release is tightly regulated: in homeostasis, serum levels remain below threshold to avoid unnecessary immune responses. In pathological conditions—chronic inflammation, allergies, cancer, autoimmune diseases—cytokine dysregulation drives dysfunctional immune activity (Jones & Jenkins, 2018; Boyman & Sprent, 2012; Elenkov et al., 2005).

Allergic diseases such as rhinitis and asthma are marked by Th1/Th2 imbalance, with increased IL-4, IL-5, IL-6, mast cell activation, and mucosal inflammation (Rosenzwajg et al., 2024). Genetic variants, such as IL-6 promoter polymorphism rs1800795, have been linked to higher risk of rhinitis and asthma, suggesting genetic regulation of cytokine expression (Yang et al., 2022). In this context, Treg expansion through low-dose IL-2 has proven effective in pollen allergies, reducing nasal symptoms and improving respiratory function (Rosenzwajg et al., 2024). In food allergy models, low-dose IL-2 prevented allergic reactions by modulating Th1/Th2 balance (Bonnet et al., 2016; Smith, 2018). Clinical trials confirm that low-dose IL-2 increases Tregs and improves symptoms with favorable safety (Rosenzwajg et al., 2016; Klatsmann & Rosenzwajg, 2015).

Interleukin-2 (IL-2) plays a central role in T-cell and NK-cell proliferation and differentiation. Historically, high-dose IL-2 (HDIL-2) was used against cancers such as melanoma and renal carcinoma, showing durable responses but with severe toxicity (Im et al., 2024; Zhou et al., 2023). Conversely, low-dose IL-2 has demonstrated Treg expansion and reduction of autoimmune activity, with improved safety (Hartemann et al., 2013; Kosmaczewska, 2014; Saadoun et al., 2011). Trials in SLE, RA, and other autoimmune diseases confirm its tolerability and efficacy without excess infection risk (He et al., 2016; Humrich et al., 2015). Separating IL-2 into α and β subunits introduces a novel strategy to selectively modulate immune responses (Paul, 2012).

Similarly, IL-6 is a key mediator of inflammation, acting through JAK/STAT3 signaling via the β subunit (gp130). It has both protective and pathogenic roles, including promoting tumor proliferation (Putoczki et al., 2013; Heimberger et al., 2023).

Use of autologous cytokine fragments represents a non-conventional therapeutic approach: patient blood serves as the source, and cytokines are purified and fractionated into α and β subunits for personalized therapy. This strategy offers advantages in immunogenicity, precision, and adaptation compared to recombinant cytokines or monoclonal antibodies (Rutgers et al., 2010).

In this study, we describe two clinical cases: (1) a patient with IL-2 overexpression treated with the α subunit to test a downregulatory effect; and (2) a patient with IL-6 deficiency in post-chemotherapy immunosuppression, treated with the β subunit to stimulate immune recovery. The aim was to evaluate whether microdoses of autologous cytokine fragments could modulate cytokine levels and restore immune balance.

## Materials and Methods

### *Study Design*

The study consisted of clinical and laboratory observation of two patients treated with autologous cytokine fragments, fractionated into their α and β subunits and obtained from peripheral venous blood. Both patients were selected on the basis of a documented alteration in their cytokine profile, confirmed by serum quantification using ELISA.

### *Case Report A*

Subject A: male, 42 years old, employed, with a degree in economics and business. He presented with persistent morning joint pain, reported for several years, which worsened after SARS-CoV-2 infection in 2021. Following the acute infection, new symptoms emerged, including episodes of mental confusion and lexical difficulties (temporary word loss), affecting his work and social life.

Medical history included pneumonia at age 24, two hospitalizations for nephrolithiasis, and appendectomy. No chronic medications. He has two healthy children. Family history revealed rheumatoid arthritis in his mother, suggesting an inflammatory predisposition. Rheumatological evaluation excluded autoantibody positivity, though slight CRP elevation and sub-threshold lymphocytopenia were

present. Cytokine testing was therefore performed.

Baseline screening (January 3rd, 2022) revealed inflammatory cytokine alterations. Autologous cytokine therapy was initiated on March 12th, 2022, personalized according to the altered profile (IL-2 excess). Follow-up assessments were performed at 3 months (June 10th, 2022) and 6 months (September 2nd, 2022).

***Case Report B***

Subject B: female, 55 years old, employed, postmenopausal. She presented after a diagnosis of ductal carcinoma of the left breast, treated with total mastectomy. No prior comorbidities except for previous progestin therapy for hormonal irregularities. Preoperatively, she underwent 12 cycles of radiotherapy, followed by prophylactic chemotherapy with Paclitaxel and Cisplatin.

Family history was unremarkable. She was not on chronic medications apart from oncological therapy. Given her clinical background and immune-modulating treatments, cytokine assessment was performed to evaluate systemic inflammation.

Baseline screening (01.03.2019) revealed significant cytokine alterations, especially IL-2 and IL-6. The patient began treatment on 12.03.2019, with follow-up at 3 months (05.06.2019) and 6 months (30.09.2019).

***Blood Sampling and Cytokine Extraction***

Extraction of autologous cytokines followed a multi-phase procedure. After venous blood collection, plasma was separated by decantation. For each cytokine, the following steps were performed: suspension in solution, addition of magnetic microspheres specific to the cytokine, magnetic separation, removal of microspheres, disruption of molecular chains via centrifugation, separation of α helices and β sheets, dilution, stabilization, and storage of the preparation.

***Cytokine Extraction: Phase 1***

The skin was disinfected with cotton soaked in antiseptic solution. Venipuncture was performed with a sterile 10 mL syringe and a 20G/0.9 mm needle, collecting 10 cc of blood. Smaller-gauge needles were avoided to prevent cytokine damage.

The sample was left to decant vertically for 5-48 hours at room temperature (15-25 °C), or in a temperature-controlled device for the same period. Decantation was chosen instead of centrifugation, since the latter would irreversibly damage cytokines, compromising the final product.

After decantation, the supernatant plasma (~5 cc) was collected with a micropipette under sterile conditions. Cytokines of interest are in the intermediate layer between plasma and serum. Collecting 1-2 mL of serum ensured retrieval of cytokines without interfering with the activity of magnetic beads. Without this step, extraction of the intermediate cytokine-rich fraction would be incomplete.

***Cytokine Extraction: Phase 2***

Plasma obtained by decantation was suspended in 0.9% NaCl solution containing magnetic microspheres coated with specific ligands for the target cytokine.

For separation, a secondary magnet from the kit was placed against the container wall. The magnet attracted the cytokines bound to the microspheres. The supernatant was removed with a micropipette, isolating the pellet containing cytokine-bound microspheres.

The bound cytokines were then eluted in 0.9% NaCl solution to release them from the microspheres. A magnet was again applied to attract the beads, leaving cytokines free in solution. The cytokine-containing supernatant was collected and centrifuged at 400g for 5 minutes to break cytokine molecular chains.

After centrifugation, the sample rested for 20-30 minutes, followed by differential centrifugation at 4500g for 10 minutes to separate α helices and β sheets. β sheets deposited at the bottom, while α helices remained suspended in a thin layer at the surface.

The final sample was diluted 1:1000 in 0.9% NaCl solution for therapeutic use. It was stabilized for 24 hours at -25 °C and stored at 0-5 °C until administration, with a maximum shelf life of 6 months.

This protocol was repeated for each cytokine of interest. Separation of α and β chains enabled selective use: α subunits for suppression/modulation, β subunits for stimulation.

The choice of chains for administration was based on quantitative analysis of each patient's cytokine profile, determined in the laboratory.

***Administration and Follow-up***

Autologous cytokine therapy, strictly individualized and tailored to each patient, was administered intramuscularly or subcutaneously at peripheral sites, with frequency and dosage established according to baseline cytokine profile and therapeutic goal (inhibition or stimulation). Preparations contained isolated molecular fragments of autologous cytokines, obtained through α or β subunit separation from peripheral plasma, as described in section 2.2.

Clinical and laboratory follow-up were performed at 3 months (T1) and 6 months (T2) after treatment initiation to monitor immunological and cytokine parameters.

Patient monitoring included clinical evaluation of symptoms and serum quantification of major interleukins, with particular focus on the target cytokines in each case.

All cytokine measurements were performed using high-sensitivity chemiluminescent immunoassay. Data was analyzed longitudinally and compared to baseline values (T0), using the same certified laboratory for all time points.

No severe adverse events or systemic reactions were recorded. Data were analyzed in parallel to evaluate correlations between cytokine trends and subjective clinical outcomes.

## Results and Discussion

***Case Report A: findings***

At baseline screening (T0), a panel of 16 cytokines was analyzed. At subsequent assessments (T1 and T2), only IL-2—found to be altered at baseline—was measured. Table 1 summarizes cytokine values at the three points.

At T0, IL-2 levels were markedly elevated (67 U/mL), indicating abnormal immune activation. After 3 months of therapy with the IL-2 α subunit, IL-2 decreased substantially to 42 U/mL, maintaining a downward trend. At the 6-month follow-up, levels normalized to 3 U/mL, well within the physiological range, confirming the success of the targeted inhibitory approach (Figure 1).

| *Interleukin (IL)* | *T0* | *T1* | *T2* | *Normal Range* |
|---|---|---|---|---|
| IL-1α | 130 pg/mL | — | — | 39-250 pg/mL |
| IL-1β | 520 pg/mL | — | — | 30-1400 pg/mL |
| IL-2 | 67 U/mL | 42 U/mL | 3 U/mL | 1-30 U/mL |
| IL-3 | 90 pg/mL | — | — | 78-500 pg/mL |
| IL-4 | 70 U/mL | — | — | 6-100 U/mL |
| IL-5 | 180 pg/mL | — | — | 130-750 pg/mL |
| IL-6 (Elisa) | 90 pg/mL | — | — | 100-44 pg/mL* |
| IL-7 | 500 pg/mL | — | — | 150-1000 pg/mL |
| IL-8 | 240 pg/mL | — | — | 7-750 pg/mL |
| IL-10 | 420 pg/mL | — | — | 11-1335 pg/mL |
| IL-12 | 910 pg/mL | — | — | 15-1300 pg/mL |
| IL-13 (Elisa) | 870 pg/mL | — | — | 312-2000 pg/mL |
| IL-15 (Elisa) | 1120 pg/mL | — | — | 39-2500 pg/mL |
| IL-16 (Elisa) | 850 pg/mL | — | — | 34-1500 pg/mL |
| IL-17 (Elisa) | 340 pg/mL | — | — | 156-1000 pg/mL |
| IL-18 (Elisa) | 210 pg/mL | — | — | 140-1000 pg/mL |

***Table 1:*** Interleukin values at baseline (T0), 3 months (T1), and 6 months (T2) in Patient A.

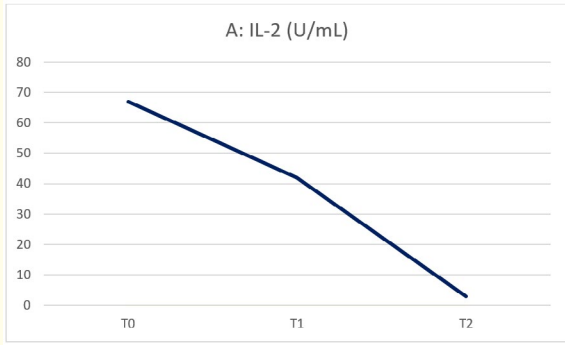


***Figure 1:*** Progressive decline of IL-2 values from baseline (T0) to 3 months (T1) and 6 months (T2) in Patient A.

***Case Report B: findings***

At baseline screening (T0), a panel of 13 cytokines was analyzed. At subsequent assessments (T1 and T2), only IL-2 and IL-6—found to be altered at baseline—were measured. Table 2 summarizes cytokine values at the three points.

At T0, IL-2 was elevated (44 U/mL), clearly above the physiological range (1-30 U/mL), indicating significant baseline immune activation. After 3 months of therapy, IL-2 decreased to 32 U/mL, still above normal but trending downward. At the 6-month follow-up, levels normalized (4 U/mL), confirming the effectiveness of the targeted inhibitory strategy (Figure 2).

| *Interleukin (IL)* | *T0* | *T1* | *T2* | *Normal Range* |
|---|---|---|---|---|
| IL-1α | 212 pg/mL | — | — | 39-250 pg/mL |
| IL-1β | 45 pg/mL | — | — | 30-1400 pg/mL |
| IL-2 | 44 U/mL | 32 U/mL | 4 U/mL | 1-30 U/mL |
| IL-3 | 200 pg/mL | — | — | 78-500 pg/mL |
| IL-4 | 40 U/mL | — | — | 6-100 U/mL |
| IL-5 | 190 pg/mL | — | — | 130-750 pg/mL |
| IL-6 (ELISA) | 10 pg/mL | 42 pg/mL | 89 pg/mL | 100-36 pg/mL |
| IL-7 | 610 pg/mL | — | — | 150-1000 pg/mL |
| IL-8 | 110 pg/mL | — | — | 7-750 pg/mL |
| IL-10 | 140 pg/mL | — | — | 110-1335 pg/Ml |
| IL-12 | 400 pg/mL | — | — | 200-1300 pg/mL |
| IL-13 (Elisa) | 1220 pg/mL | — | — | 312-2000 pg/mL |
| IL-15 (Elisa) | 1430 pg/mL | — | — | 39-2500 pg/mL |

***Table 2:*** Interleukin values at baseline (T0), 3 months (T1), and 6 months (T2) in Patient B.

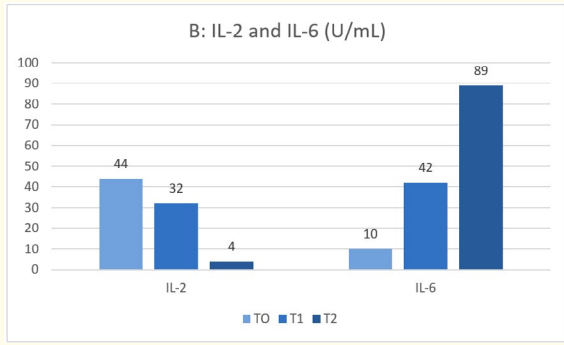


***Figure 2:*** Trends of IL-2 and IL-6 across baseline (T0), 3 months (T1), and 6 months (T2) in Patient B.

Regarding IL-6, baseline levels were markedly reduced, consistent with immunosuppression following prior pharmacological treatments. Therapy with β fragments of autologous cytokines, aimed at selective stimulation of the IL-6 pathway, produced results consistent with the working hypothesis.

IL-6 showed a gradual and steady increase: from 10 pg/mL at T0, to 42 pg/mL at T1 (suggesting an initial stimulatory effect), and to 89 pg/mL at T2, within the physiological range, sufficient to sustain a functional immune response without triggering pathological inflammation (Figure 2).

## Conclusion

Personalized administration of autologous cytokine fragments, selectively separated into α and β subunits, represents an innovative and biologically consistent strategy for targeted modulation of the cytokine axis in patients with inflammatory dysregulation. The two clinical cases presented here provide preliminary evidence of the possibility to direct the immune response in opposite ways—inhibition or stimulation—depending on the clinical condition and baseline cytokine profile.

In the first case, a patient with neuroinflammation and chronic joint pain presented with elevated serum IL-2 levels (67 U/mL at T0) in the absence of specific autoantibodies. Administration of autologous microdoses containing the α subunit of IL-2, designed to competitively block the overexpressed cytokine, resulted in a progressive decline in plasma levels, reaching normalization at 6 months (3 U/mL), in parallel with subjective clinical remission.

In the second case, an oncological patient undergoing chemotherapy and radiotherapy exhibited functional immunosuppression, with initially low IL-6 levels (10 U/mL) and elevated IL-2 levels (56 U/mL) at the time of screening. The treatment was implemented using autologous α and β cytokine fragments, aimed at selectively stimulating the IL-6 pathway while reducing IL-2 levels.

The IL-6 trajectory confirmed a progressive and controlled increase, from 10 U/mL (T0) to 42 U/mL (T1), reaching 89 U/mL (T2), always within the physiological range. Simultaneously, a marked reduction in IL-2 levels was observed, decreasing from 44 U/mL to 32 U/mL (T1), and ultimately to 4 U/mL (T2). This pattern suggests a secondary regulatory effect, mediated by a systemic rebalancing of the cytokine profile in response to the treatment.

Both cases highlight that autologous cytokine therapy derived from plasma offers:

- Selective and adaptive modulation, without the systemic adverse effects commonly associated with recombinant cytokines or monoclonal antibodies.
- Clinical responses proportional to the individual cytokine profile, consistent with the principles of personalized medicine.
- Fine-tuned dose-effect control, mediated by endogenous regulatory mechanisms.
- Restoration of immune homeostasis, through rebalancing of the cytokine network.

Although these observations are limited to two single-patient experiences, the results support the hypothesis that microdoses of fractionated autologous cytokines may represent a safe and effective alternative for immune modulation, paving the way for broader clinical protocols in immunology. Further studies on larger cohorts will be necessary to consolidate these findings and strengthen the scientific evidence.

***Conflict of interest***

The authors declare no conflicts of interest.

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