

IMMUNOMODULATION POTENTIAL OF CHIMERIC ELASTASE FROM *SCHISTOSOMA MANSONI* ON HUMAN PERIPHERAL BLOOD CELLS FROM ALLERGIC AND NON-ALLERGIC INDIVIDUALS

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The hygiene hypothesis suggests that reduced exposure to infectious agents in early childhood may impair immune development, increasing allergy risks, particularly in high-income countries. Helminth infections, like allergies, induce Th2 responses, with CD4+ T cells producing IL-4 and IL-5. However, helminths also promote IL-10 production and activate regulatory T cells (Tregs), helping to control inflammation. Thus, allergen-specific immunotherapy based on parasite molecules offers promising alternatives to corticosteroids, which are palliative and often cause side effects. Among *Schistosoma mansoni* proteins, elastase (SmCE) plays a key role in immune evasion during cercarial invasion, inducing both Th1 and Treg responses, which may help rebalance Th2-dominant allergic responses. This study aimed to evaluate the immunoregulatory potential of a chimeric protein (Q2) based on *S. mansoni* elastase. Q2 was expressed in *Escherichia coli* strains through IPTG induction. Protein extracts were solubilized in Tris-HCl buffer and purified by affinity chromatography. Identity and purity were confirmed by Western blot using a 6×His tag. Peripheral blood mononuclear cells (PBMCs) from healthy and allergic donors were isolated by Ficoll-Hypaque separation and stimulated with Q2 for 48 and 72 hours. Unstimulated cells served as controls. Polymyxin B was used to neutralize residual endotoxins. After incubation, cell viability was assessed, and cytokines (IL-1 β , IL-4, IL-5, IFN- γ , IL-10, IL-13, TNF- α) were measured in the culture supernatant. We observed that Q2 stimulation increased TNF- α production without altering baseline IL-10 levels, suggesting a Th1 response without suppressing Treg activity. These findings highlight the potential of Q2 as an immunotherapeutic agent, but further studies are needed to confirm these preliminary results and ensure its safety and efficacy.

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