

Targeted Allergy and Auto-immune Disease Treatment

Allergen desensitization treatment to reduce or eliminate inflammatory reactions to allergens or auto-immune diseases.

Severe allergic disease affects millions of people globally, causing significant morbidity and mortality. Currently, the most effective therapy for these disorders is allergen desensitization, which involves prolonged exposure to small doses of allergens over several years. This process is expensive and time consuming for patients, while also carrying significant risk of adverse effects ranging from injection site reactions to anaphylactic shock and death. VCU researchers have developed a treatment that circumvents the need for allergen desensitization by reprogramming immune responses to allergens, allowing for safe and rapid allergen desensitization.

The technology

Allergic inflammation responsible for anaphylaxis is caused by over exuberant IgE antibody responses to allergens. This novel therapy developed at VCU impairs IgE production, resulting in reduced inflammation in response to allergens. This therapy, which may be used by patients at home in a convenient oral, inhaled, or topical formulation. Further, this new treatment does not have broad immunosuppressive effects, mitigating the risk of severe, potentially life-threatening infections that are seen with existing immunomodulatory medications. This new treatment provides a safe, fast, and effective treatment for suppressing severe allergic reactions and has potential broader uses for autoimmune diseases with promising results already shown in animal studies (Figure 1).

Benefits

- » Fast and effective
- » Broad usage
- » Targeted therapy

Applications

- » Allergies
- » Auto-immune diseases

Patent status:

Patent pending: U.S. and foreign rights are available.

License status:

This technology is available for licensing to industry for further development and commercialization.

Category:

Biomedical

VCU Tech #:

22-091

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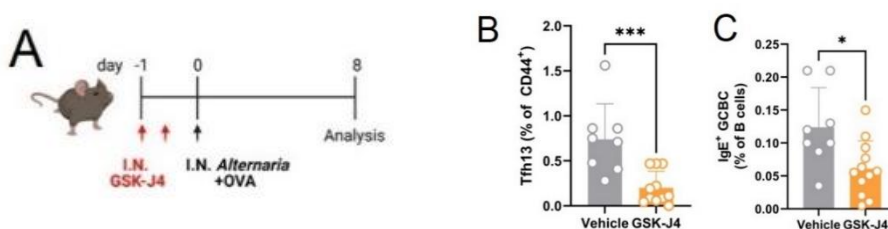


Figure 1. (A) Schematic diagram of experimental design. Mice received 50 µg of GSK-J4 via intranasal inhalation 24 hours and 6 hours prior to exposure to the allergen *Alternaria alternata*. (B) Day 8 analysis revealed reduced T follicular helper 13 (Tfh13) cell number and (C) IgE+ germinal center B cell (GCBC) numbers.