International Journal of Biology and Pharmacy Research Updates

Journal homepage: https://orionjournals.com/ijbpru/

(REVIEW ARTICLE)

Allergies, acute infections and humoral skin creams

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International Journal of Biology and Pharmacy Research Updates, 2022, 02(01), 014–016

Publication history: Received on 21 August 2022; revised on 26 September 2022; accepted on 28 September 2022

Article DOI: https://doi.org/10.53430/ijbpru.2022.2.1.0033

Abstract

Allergies can engender a healthy immune response to harmful viruses. The humoral immune system (i.e., antibody-mediated immunity) creates and regulates endogenous proteins that support a fully integrated immune response to acute infections. A robust and active humoral immune system may inhibit the development of cytokine/bradykinin storms and affect angiotensin-converting enzyme 2 (ACE2) expression. Humoral skin cream therapy is immune-stimulating and may affect the global burden of morbidity and mortality from acute infections.

Keywords: ACE2; Allergies; Bradykinin; Cytokines; COVID-19; Humoral skin cream

1. Introduction

Allergies are associated with the humoral immune system and antibody-mediated immunity (i.e., immunoglobulin-E or IgE). An allergy is when your body reacts to something harmless. Common allergens include pollen, foods such as peanuts, milk, and eggs; insect stings, such as bee and wasp stings; and natural rubber latex (hevea brasiliensis.) Minor symptoms of an allergic reaction can include a runny nose, tenderness around your eyes, coughing, wheezing, itchy skin, or a raised rash [1].

The COVID-19 pandemic caused by severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) affects people differently based on their immune system. Infections can be asymptomatic (no symptoms) or cause a wide range of symptoms like body aches, a cough, diarrhea, difficulty breathing, fatigue, fever, loss of smell and taste, and nausea [2].

In the United States of America, mortality from SARS-CoV-2 infection based on an observed case-fatality ratio is 1.1 percent [3]. Elezkurtaj et al. [4] show that the causes of death were directly related to COVID-19 in most decedents, while they appear not to be an immediate result of preexisting health conditions and comorbidities.

Wilczynski et al. [5] show that cytokine/bradykinin storms are often the fatal consequences of SARS-CoV-2 infection (e.g., heart, kidney, and lung failure). Cytokine/bradykinin storm theories offer intriguing explanations for the diversity of symptoms and organ systems affected after SARS-CoV2 infection. Odeh [6] discusses that the intricacies of these two hypotheses have a crucial overlap that can lead to an understanding of the pathology of the SARS–CoV-2 and how it relates to the virus in vulnerable patients.

Dochniak [7] shows that hyper-allergenic skin cream therapy may inhibit cytokine storm development during acute infections. Forced humoral immunity may also mediate the renin-aldosterone system to decrease the severity of COVID-19; limiting angiotensin-converting enzyme 2 (ACE2) expression abates the point of entry for the virus into the host cell.

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2. Discussion

Can allergies affect the severity of symptoms during a COVID-19 infection? Oprea et al, [8] show that increased mortality occurred in IgE deficient patients compared to those with normal and higher IgE levels. Furthermore, higher eosinophil counts have better outcomes during COVID-19 infection. In continuation, Seibold et al. [9] show that food allergies are associated with a lower risk of SARS-CoV-2.

How do allergies affect the pathogenesis of acute infections? Research shows that allergies affect the expression of endogenous proteins (e.g., signaling molecules, enzymes, and receptors) to decrease the severity of COVID-19.

Zarnowski et al. [10] show that atopic comorbidities have no unfavorable impact on the severity and course of COVID-19. A decreased ACE2 expression in atopic manifestations may reduce the viral entrance of SARS-CoV-2 and thus lowers susceptibility for COVID-19 infection or disease severity in individuals with an atopic background.

Jackson et al. [11] show that respiratory allergy and controlled allergen exposures are each associated with significant reductions in ACE2 expression. ACE2 expression was lowest in those with increased allergic sensitization and asthma. Given that ACE2 serves as the receptor for SARS-CoV-2, our findings suggest a potential mechanism for reduced COVID-19 severity in patients with respiratory allergies. However, additional factors beyond ACE2 expression likely modulate the response to COVID-19 in individuals with allergies; elucidation of these factors may provide valuable insights into COVID-19 disease pathogenesis.

Gebremeskel et al. [12] show that mast cells and eosinophils express ACE2 receptors on their cell membrane. SARS-CoV-2 can use the ACE2 on mast cells and eosinophils to cause degranulation, releasing inflammatory cytokines. Wu et al. [13] show that SARS-CoV-2-triggered mast cells causes rapid degranulation and induces alveolar epithelial inflammation and lung injury.

Humoral immunity provides IgE-primed mast cells and eosinophils; it is speculated herein that increased steric factors associated with IgE-primed mast cells and eosinophils decrease SARS-CoV-2 cellular entry.

Globally, the continuous and rapid spread of infectious diseases makes it impossible to create herd immunity through vaccinations. Gammon [14] wrote, “Even if the developed world gets its citizens vaccinated in a year, virus mutations and economic instability will roil unvaccinated countries for years – and end up costing everyone.”

Humoral skin creams are an immune-stimulating therapy that may be used to decrease the global burden of morbidity and mortality from acute infections. Dochniak [15] shows examples of humoral skin creams in the United States application number 20210015912 A1 (2019) titled, “Topical hyper-allergenic composition and method of treating using the same.”

3. Conclusion

There remains a need for an effective and economical medicament (i.e., skin cream) that reduces morbidity and mortality from acute infections. Research continues to explore if forced humoral immunity has an immuno-therapeutic effect on cytokine/bradykinin storm development and ACE2 expression.

Compliance with ethical standards

Acknowledgments

The author wishes to thank Christopher Glancey, Cherie Benson, and David Bartlett for their continued support.

Disclosure of conflict of interest

Michael J. Dochniak is the co-founder of Alleam-it Corporation, Minnesota, and USA.

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