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ORIGINAL RESEARCH

From Forest to Flesh: Rubber, Chitinase Biology, Colonial Power and the Accidental Engineering of Allergy

Brianna Watson, MD[1], Mrinalini Deverapalli, MBBS[1], Zarina Meer, OD[2], Tyler Thompson, MD[1], Lima Ross, MD[3], Christian Nolan, MD[1], Madison Blackwell, MD[1], Jaisha Wilson, MD[1], Mekdem Bisrat, MD[1], Miriam Michael, MD , FACP[1,4]

[1] Internal Medicine, Howard University, [2] Ophthalmology, Howard University, [3] internal Medicine, Howard University, [4] Internal Medicine, University of Maryland School of Medicine

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Background

Natural rubber latex allergy is traditionally described as a late twentieth-century occupational disease that emerged prominently among healthcare workers during expanded glove use in the HIV/AIDS era. Although this explanation accounts for the timing of clinical recognition, it incompletely addresses the deeper biologic and historical forces that shaped latex allergenicity, cross-reactive food syndromes, and the continuing relevance of latex allergy in modern medicine.

Objective

To synthesize historical, immunologic, occupational, and environmental literature in order to reframe latex allergy as a model of how large-scale societal and ecological changes can generate new patterns of allergic disease.

Research and Context

This article draws on published literature spanning environmental history, allergy and immunology, occupational medicine, infectious disease policy, and plant biology. Natural rubber latex, derived from Hevea brasiliensis, contains pathogenesis-related proteins such as hevein and class I chitinases that function in plant defense. Through colonial extraction economies, industrial manufacturing, and global medical supply chains, these proteins entered widespread human contact. The implementation of Universal Precautions during the HIV/AIDS epidemic markedly increased repetitive latex exposure in healthcare settings, facilitating sensitization and clinically apparent allergy at scale. Existing literature also demonstrates structural homology between latex allergens and proteins in foods including banana, avocado, kiwi, and chestnut, helping explain latex–fruit syndrome through IgE-mediated cross-reactivity. Broader parallels with shellfish, arthropods, fungi, and other chitin-associated organisms suggest recurring immune recognition of conserved environmental molecules across biological kingdoms. Emerging work on climate stress and agricultural biotechnology further raises the possibility that environmental and food-related allergen exposures may continue to evolve.

Implications and Conclusions

Latex allergy remains clinically relevant in perioperative medicine, emergency care, dentistry, obstetrics, and other settings where latex exposure persists. Recognition of latex–fruit syndrome can improve allergy histories, procedural risk assessment, and avoidance counseling. More broadly, latex allergy illustrates how occupational exposures, food cross-reactivity, public health policy, and ecological change intersect to shape immune-mediated disease. As environmental pressures and engineered food systems continue to change biologic exposures, latex allergy offers a useful framework for anticipating future patterns of sensitization and prevention.

Introduction

Allergic disease is often treated as a byproduct of modern excess—a world that is too industrial, too hygienic, and too artificial.[1,2] Yet allergy is not simply a consequence of contemporary life; it is part of the historical record. Allergic syndromes emerge when immune systems encounter the same molecular signals repeatedly, at scale, and under conditions that render avoidance impossible. For most of human history, such encounters were sporadic and localized.[2] In the modern era, they have become systemic. Allergy, in this sense, is not a modern aberration but a biological response to the reorganization of life by empire, industry, medicine, and policy.[3,4]

Natural rubber latex allergy offers a particularly clear lens through which to examine this transformation. Latex was neither novel nor rare when it became a clinically significant source of allergy.[1] Derived from *Hevea brasiliensis*, it circulated globally for decades as an industrial material before becoming embedded in routine medical practice.[2,3] What changed was not the substance itself, but the conditions of exposure. As latex moved from forest to factory and from factory to hospital, it crossed a threshold from intermittent contact to continuous intimacy. Only then did the immune consequences of this ancient plant defense system become visible in humans.[2]

Latex allergy is therefore best understood not as a medical accident, but as a historical event. Its emergence reflects the convergence of colonial extraction, industrial scalability, infection-control policy, and patterns of sustained exposure that transformed an ancient plant defense system into a modern clinical allergen. By tracing latex from Amazonian forests through imperial economies, into hospitals reshaped by the HIV/AIDS epidemic, and onward into the molecular logic of allergy and agricultural biotechnology, this article argues that allergic disease arises where biological conservation collides with modern systems of scale.[5] Allergy, in this view, becomes an archive written in IgE (immunoglobulin E, the antibody class associated with allergic responses) of how deeply human history has penetrated the defensive biology of the natural world.[6,7]

Discussion

Rubber, Empire, and the Political Economy of Extraction

Before latex became a medical material, it was already a political commodity. Natural latex refers to the milky sap of *Hevea brasiliensis*, whereas rubber denotes the processed industrial material derived from that sap.[1,2] The global availability of rubber cannot be understood without tracing how *H brasiliensis* was drawn into imperial systems of extraction, trade, and violence during the nineteenth century.[2,3] Following Charles Goodyear's vulcanization breakthrough in 1839, rubber became indispensable to industrial modernity, enabling telegraph cables, mechanized factories, bicycle tires, automobile manufacturing, insulation, and military logistics.[2,4] Demand surged rapidly, and the Amazon basin became the primary source of natural latex.[2,3]

This demand was sustained through coercive labor regimes involving debt peonage, forced Indigenous labor, and organized brutality.[2] Rubber was harvested from forests using structures of colonial power that imposed on landscapes and populations.[2,4,5] During the Amazon rubber boom, Indigenous communities across Brazil, Peru, and surrounding regions were displaced, enslaved, or killed in systems designed to maximize latex extraction for European and North American markets.[2,4,5] Atrocities in the Putumayo region became one of the most documented examples of violence linked to rubber commerce.[3] The biological features that made *H brasiliensis* so profitable, particularly its ability to be tapped repeatedly without killing the tree, enabled prolonged exploitation.[1,2]

The eventual collapse of Brazilian rubber dominance was driven not only by economics but also by ecology.[2,3] In 1876, thousands of *H brasiliensis* seeds were exported to Britain and used to establish plantations in Asia, especially British Malaya and Ceylon.[2,3,6] There, the absence of South American leaf blight (*Microcyclus ulei*), a fungal pathogen endemic to the Amazon, allowed dense plantation cultivation that was not feasible in South America.[2,6,7] As environmental historian Warren Dean described, disease ecology shaped the geography of empire as much as finance or trade.[3,6] By the early twentieth century, Southeast Asia had overtaken Brazil as the dominant global producer of natural rubber.[2,3]

By the time latex entered hospitals as a standardized medical commodity, its origins in imperial violence, ecological contingency, and industrial restructuring had largely been obscured by distance, processing, and institutional abstraction. Yet the underlying biology of latex itself remained unchanged.

Latex as Defense: Chitinases and the Molecular Architecture of Plant Immunity

Latex is not an inert industrial material. In living plants, latex is a biologically active defensive secretion released after tissue injury to protect against fungi, insects, and herbivores.[8,9] In *H brasiliensis*, latex contains multiple pathogenesis-related proteins, including hevein, prohevein, and class I chitinases, which function as part of the plant's innate immune system.[8-10] These proteins are not incidental contaminants of latex, but core protective molecules evolved to limit environmental attack.[8,9]

One of the principal targets of these defenses is chitin, a β-1,4-linked polymer of N-acetylglucosamine found in fungal cell walls, arthropod exoskeletons, crustacean shells, and some algae.[11,12] Because chitin is absent from vertebrate tissues, it serves as a conserved marker of potential environmental threat.[12] Plants respond by producing chitinases, enzymes that bind and degrade chitin-containing structures, thereby limiting fungal invasion and pest damage.[9,11]

Class I chitinases contain an N-terminal hevein-like chitin-binding domain stabilized by multiple disulfide bonds. These bonds help preserve protein conformation under heat, mechanical stress, and environmental degradation.[8] Such structural stability may also preserve antigenic epitopes that remain recognizable after processing or repeated exposure.[8,10] In practical terms, these proteins can persist in forms capable of interacting with the human immune system.[8]

Latex from *H brasiliensis* contains substantial concentrations of these defense proteins. Major latex allergens include Hev b 6.01 and Hev b 6.02 (hevein and prohevein) as well as Hev b 11, a class I chitinase.[8,13] Repetitive cutaneous, mucosal, or airborne exposure, particularly in healthcare and occupational settings, can lead susceptible individuals to develop IgE sensitization.[13,14] Once sensitized, subsequent exposure may trigger mast cell activation, urticaria, asthma, or anaphylaxis.[1,13] Thus, proteins that evolved to defend plants against fungi and pests can, under conditions of repeated human exposure, become clinically significant allergens.[1,8]

Hospitals, HIV/AIDS, and the Scaling of Exposure

For much of the twentieth century, latex circulated quietly through medicine. Surgical gloves, introduced by William Halsted in 1889, were used selectively and inconsistently.[15] This changed abruptly in the late 1980s, when the HIV/AIDS epidemic forced a major reconceptualization of clinical risk.[16,17] In 1987, the Centers for Disease Control and Prevention introduced Universal Precautions, recommending routine glove use for patient encounters involving potential contact with blood or bodily fluids.[16]

This policy was lifesaving, but it also transformed patterns of latex exposure.[1, 14] Healthcare workers who had previously worn gloves intermittently now donned multiple pairs each day. Powdered latex gloves released allergenic proteins into the hospital air, increasing both skin and respiratory exposure.[14] Patients with chronic illness were also affected. Children with spina bifida, for example, often underwent repeated surgeries and catheter-based procedures from early life, resulting in intense cumulative latex exposure and some of the highest reported sensitization rates in the pre–latex-safe era.[1,18]

Latex allergy became clinically prominent during this period not because latex itself changed, but because exposure crossed a population-level sensitization threshold.[1,14] Epidemiologic studies in the early 1990s reported markedly elevated rates of latex-specific IgE and symptomatic allergy among healthcare workers, with prevalence estimates in some cohorts ranging from approximately 5% to 17%, particularly among individuals with preexisting atopy.[14]

This period illustrates a broader principle of allergic disease: when repeated exposure to biologically active and structurally conserved proteins becomes widespread, sustained, and difficult to avoid, previously uncommon sensitization can emerge as a major clinical problem.[1,8]

Latex–Fruit Syndrome and Molecular Cross-Reactivity

As latex sensitization increased during the late twentieth century, clinicians began reporting a recurring and clinically significant phenomenon: many patients allergic to latex also experienced immediate hypersensitivity reactions to foods such as banana, avocado, chestnut, kiwi, and, less commonly, other plant-derived foods.[13,19,20] Depending on the cohort studied and diagnostic criteria used, food cross-reactivity has been reported in a substantial proportion of latex-allergic individuals, establishing what became known as latex–fruit syndrome.[13,19] These observations suggested a broader principle of immune recognition: IgE antibodies do not recognize exposure setting or clinical context; they recognize protein structure.[8,13]

Class I chitinases in several implicated fruits contain hevein-like domains that closely resemble those present in major latex allergens. Although primary amino acid sequences may differ, conserved disulfide bonding helps preserve three-dimensional epitopes capable of binding preexisting latex-specific IgE.[10,13] This structural homology helps explain why sensitization acquired through occupational or medical latex exposure may later manifest as allergic reactions to foods encountered outside the healthcare setting.[13,19]

Latex–fruit syndrome therefore blurred traditional boundaries between occupational and food allergy and helped establish the concept of panallergens: structurally conserved proteins capable of producing cross-

reactivity across species and routes of exposure.[4,13] It also demonstrated that the clinical consequences of sensitization may extend far beyond the environment in which sensitization first occurred.[13,19]

Chitin Across Kingdoms: Shellfish, Arthropods, and Immune Pattern Recognition

Chitin is not confined to plants. It is widely distributed across fungi, insects, mites, crustaceans, and some algae.[11,12] Human immune systems evolved to recognize chitin-associated molecular patterns as potential indicators of parasitic, fungal, or environmental threat.[12,21] The persistence of mammalian chitinases and chitinase-like proteins, despite the absence of endogenous chitin in vertebrate tissues, underscores the biologic importance of these recognition pathways in host defense.[11,21]

This broader context helps explain why allergic diseases often cluster across seemingly unrelated exposures. Although shellfish allergy is primarily mediated by tropomyosin rather than the same proteins implicated in latex–fruit syndrome,[4,13] epidemiologic overlap among shellfish allergy, dust mite sensitization, atopy, and latex allergy suggests convergence through shared type 2 immune pathways.[11,13,21] Chitin and chitin-associated proteins may enhance epithelial activation, innate immune signaling, and downstream IgE responses in susceptible individuals.[12,21]

For clinicians, the implication is not that latex allergy and shellfish allergy are interchangeable, but that sensitized patients may carry broader allergic predispositions shaped by recurring responses to conserved environmental molecules.[21] More broadly, latex allergy can be understood as one manifestation of a wider immunologic pattern in which repeated exposure to structurally conserved biological materials contributes to clustered allergic disease across occupational, dietary, and environmental settings.[8,11,13]

Climate Change and the Intensification of Plant Defense

Climate change is altering the environmental conditions under which plants maintain immune defense.[6,7] Rising temperatures, changing rainfall patterns, increased humidity in some regions, drought stress in others, and expanding fungal and pest ranges expose crops and wild plants to new and intensified biologic stressors.[7,22] These pressures are particularly relevant for food-producing species and other agriculturally important plants that rely on inducible defense systems to respond to infection and environmental injury.[22,23]

In many plant species, these stress responses include upregulation of pathogenesis-related proteins such as chitinases, glucanases, and other antimicrobial defense molecules.[8,9,22] Experimental models have shown that combined heat stress and pathogen exposure can increase chitinase expression

several-fold compared with baseline conditions.[22] As a result, environmental change may alter not only crop yield and disease susceptibility but also the protein composition of foods entering the human diet.

The clinical implications remain incompletely defined, but this pathway is biologically plausible and increasingly relevant.[7,8] Foods historically considered low risk may, under some conditions, express higher concentrations of defense-related proteins with allergenic potential, particularly in sensitized or atopic individuals.[8,9] Because many plant stress responses are dynamic, reductions in environmental burden, improved crop management, selective breeding, and controlled agricultural conditions may help mitigate excessive defense-protein expression.[7]

Viewed through this lens, latex allergy may represent an early and well-characterized example of a broader phenomenon: environmental disruption can reshape biologic exposures in ways that ultimately influence patterns of human allergic disease.[1,8,13]

Biotechnology and the Deliberate Amplification of Defense

Faced with mounting threats to food security, agricultural biotechnology has increasingly turned to plant defense proteins as tools of resilience.[9,23,24] Chitinase genes and related defense pathways have been introduced or upregulated in crops to improve resistance to fungal disease, particularly in rice and other globally important staples vulnerable to major yield losses. These approaches reflect a practical challenge in modern agriculture: improving crop survival while maintaining nutritional quality and biologic safety.[23,24]

A landmark cautionary example emerged in 1996 with the Brazil nut soybean experiment. Investigators introduced a methionine-rich Brazil nut protein into soybean to improve amino acid content, because soybeans are relatively limited in methionine.[25] However, serum from Brazil nut–allergic individuals demonstrated IgE binding to the modified soybean, and the project was halted. The key lesson was that allergenicity depends on protein epitope structure rather than the species in which the protein is expressed. Transferring a protein into a new crop does not necessarily eliminate its ability to trigger allergic reactions.[25]

By contrast, chitinase-enhanced crops more commonly involve overexpression of endogenous plant defense proteins or transfer of proteins with known antimicrobial functions rather than introduction of classic food allergens, and widespread allergic consequences have not been documented.[9,23] Nevertheless, biologic caution remains warranted. Increased concentration, altered distribution, or cumulative exposure to structurally conserved

proteins could theoretically influence sensitization risk in susceptible populations, particularly those with atopy or prior cross-reactive allergies.[8,9,13]

This issue connects directly to the history of latex allergy. In hospitals, repeated exposure transformed naturally occurring plant defense proteins into a clinically important allergen.[14] In agriculture, similar proteins may be intentionally amplified for beneficial purposes. The broader lesson is not that biotechnology is inherently hazardous, but that changes in the scale, route, and intensity of exposure to biologically active proteins should remain part of long-term safety surveillance and allergy risk assessment.[23,25]

Conclusion

Latex allergy is best understood not as an isolated occupational disorder, but as the product of historical, environmental, and biologic systems that transformed a naturally occurring plant defense secretion into a clinically significant human allergen. The rise of latex sensitization reflected the convergence of colonial rubber extraction, industrial manufacturing, infection-control policy, and repeated high-intensity exposure to structurally conserved proteins capable of provoking IgE-mediated disease. What appeared in the late twentieth century as a new medical problem was, in many respects, the delayed clinical expression of much older relationships among ecology, labor, commerce, and immunity.

The same biologic principles help explain why latex sensitization extended beyond hospitals into food allergy and cross-reactive syndromes. Conserved protein structures, recurrent environmental motifs such as chitin-associated defense pathways, and cumulative exposure across occupational and dietary settings reveal that allergic disease often follows patterns of recognition shaped by evolution rather than by modern categories such as workplace, food, or environment. Climate change may further intensify these dynamics by altering plant stress responses, while biotechnology must continue to consider how beneficial manipulation of defense proteins may influence allergen exposure.

These insights carry practical implications for medicine and public health. Continued reduction of unnecessary latex exposure, careful perioperative screening, recognition of latex–fruit syndrome, and protection of high-risk occupational groups remain important preventive priorities. Future research should examine how environmental change, food systems, and repeated exposure to conserved biologic proteins contribute to emerging allergy patterns and whether earlier intervention can reduce sensitization risk.

Ultimately, latex allergy is more than a specific diagnosis. It is a case study in how modern systems redistribute exposure and, in doing so, reshape human immunity.

Relevance

This review is relevant to clinicians, allergists, surgeons, anesthesiologists, occupational health specialists, and researchers seeking to understand how new patterns of allergic disease emerge. Latex allergy remains an important cause of preventable perioperative reactions, workplace sensitization, and cross-reactive food allergy, but it also offers a broader model for how repeated, large-scale exposure to biologically conserved proteins can convert ordinary environmental contact into clinically significant immune disease. This framework may help clinicians identify higher-risk patients, take more thoughtful exposure and cross-reactivity histories, and anticipate emerging allergens linked to changing medical practice, consumer products, agriculture, and environmental stress. It also raises important research questions regarding whether patients with one structurally related allergy are predisposed to others, how climate and food systems may alter allergen expression, and which prevention strategies can reduce sensitization before disease develops. In this sense, latex allergy is not only a specific diagnosis but also a window into how the movement of materials from forest to flesh can shape modern medicine.

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