

CME Review

Socioeconomic determinants of food allergy burden



A clinical introduction

Christopher Warren, PhD^{*}; Tami Bartell, MPH[†]; Sai R. Nimmagadda, MD^{‡,§};
Lucy A. Bilaver, PhD^{||}; Jennifer Koplin, PhD^{¶,¶}; Ruchi S. Gupta, MD, MPH^{**,*†}

^{*} Department of Preventive Medicine and Center for Food Allergy and Asthma Research, Northwestern University Feinberg School of Medicine, Chicago, Illinois

[†] Patrick M. Magoon Institute for Healthy Communities, Ann & Robert H. Lurie Children's Hospital of Chicago, Chicago, Illinois

[‡] Center for Food Allergy and Asthma Research, Northwestern University Feinberg School of Medicine, Chicago, Illinois

[§] Division of Allergy and Immunology, Ann & Robert H. Lurie Children's Hospital of Chicago, Chicago, Illinois

^{||} Department of Pediatrics and Center for Food Allergy and Asthma Research, Northwestern University Feinberg School of Medicine, Chicago, Illinois

[¶] Murdoch Children's Research Institute Melbourne, Melbourne, Victoria, Australia

[¶] Department of Paediatrics and School of Population and Global Health, University of Melbourne, Melbourne, Victoria, Australia

^{**} Center for Food Allergy & Asthma Research and Institute for Public Health and Medicine, Northwestern Feinberg School of Medicine, Chicago, Illinois

^{*†} Ann & Robert H. Lurie Children's Hospital of Chicago, Chicago, Illinois

Key Messages

- US epidemiologic data suggest that rates of food allergy and food-allergic sensitization are disproportionately high among historically marginalized groups—particularly, non-Hispanic Black Americans—likely owing to systematic racism and other discriminatory social processes.
- US epidemiologic data indicate that food allergy management tends to be suboptimal among patients with lower socioeconomic status.
- When conducting clinical trials for food allergen immunotherapies and prevention interventions, care should be taken to recruit and retain participants who reflect the full socioeconomic and demographic diversity of the affected population.
- When implementing clinical food allergy risk assessment algorithms and advancing food allergy prevention efforts (eg, early introduction of allergenic solids), providers should work to ensure equitable implementation across socioeconomic strata.
- When incorporating emerging technologies such as telehealth visits into the clinical workflow, providers should be attentive to ways in which their implementation might exacerbate socioeconomic disparities in health care delivery and outcomes.
- Efforts at the clinical level to support equitable care for children and adults with FA should be supported by advocacy at the individual, community, and society levels to address social and structural factors that lead to health inequities.

ARTICLE INFO

Article history:

Received for publication May 3, 2022.

Received in revised form June 30, 2022.

Accepted for publication July 25, 2022.

ABSTRACT

Objective: This review characterizes what is currently known about how prevalence, severity, distribution, and management of food allergy (FA) differ across socioeconomic strata and provides guidance for practicing clinicians about how to improve equity in research participation, health care delivery, and patient outcomes through a deeper understanding of the socioeconomic determinants of FA.

Data Sources: Epidemiologic and biomedical literature published before April 2022.

Reprints: Christopher Warren, PhD, 750 N Lake Shore Drive, 6th Floor, Chicago, IL 60611. E-mail: Christopher.Warren@northwestern.edu.

Disclosures: Dr Warren reports receiving research support from the National Institutes of Health (NIH), Food Allergy Research & Education (FARE), and the Sunshine Charitable Foundation; being currently employed by Northwestern University; and being an Assistant Professor of Preventive Medicine at Northwestern University Feinberg School of Medicine. Dr Nimmagadda reports having previous speaking engagements for AstraZeneca and GlaxoSmithKline. Dr Bilaver reports receiving research support from the NIH (R21 ID # 1AI159562, AI135705, R01 ID # AI130348, U01 ID # AI138907), FARE, Thermo Fisher Scientific, and Genentech; being currently employed by Northwestern University; and being an Associate Professor of Pediatrics at Northwestern University Feinberg School of Medicine. Dr Gupta reports receiving research support from the NIH (R21 ID # AI135705, R01 ID # AI130348, U01 ID # AI138907), FARE, Melchiorre Family Foundation, Sunshine Charitable Foundation, The Walder Foundation, UnitedHealth Group, Thermo Fisher Scientific, and Genentech; and serving as a medical consultant and advisor for Genentech, Novartis, Aimmune LLC, Allergenics LLC, and FARE. Dr Gupta reports having ownership interest in Yobee Care, Inc; being currently employed by Ann & Robert H. Lurie Children's Hospital of Chicago; and being a Professor of Pediatrics & Medicine at Northwestern University Feinberg School of Medicine.

Funding: The authors have no funding sources to report.

<https://doi.org/10.1016/j.anaai.2022.07.021>

1081-1206/© 2022 American College of Allergy, Asthma & Immunology. Published by Elsevier Inc. All rights reserved.

Instructions

Credit can now be obtained, free for a limited time, by reading the review article and completing all activity components. Please note the instructions listed below:

- Review the target audience, learning objectives and all disclosures.
- Complete the pre-test.
- Read the article and reflect on all content as to how it may be applicable to your practice.
- Complete the post-test/evaluation and claim credit earned. At this time, physicians will have earned up to 1.0 AMA PRA Category 1 Credit™. Minimum passing score on the post-test is 70%.

Overall Purpose

Participants will be able to demonstrate increased knowledge of the clinical treatment of allergy/asthma/immunology and how new information can be applied to their own practices.

Learning Objectives

At the conclusion of this activity, participants should be able to:

- Describe socioeconomic status and common methods of assessment in research studies.
- Discuss the role of socioeconomic status as a determinant of food allergy management and outcomes.

Release Date: October 1, 2022

Expiration Date: September 30, 2024

Target Audience

Physicians involved in providing patient care in the field of allergy/asthma/immunology.

Accreditation

The American College of Allergy, Asthma & Immunology (ACAAI) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

Designation

The American College of Allergy, Asthma & Immunology (ACAAI) designates this journal-based CME activity for a maximum of 1.0 AMA PRA Category 1 Credit™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Disclosure Statement

As required by the Accreditation Council for Continuing Medical Education (ACCME) and in accordance with the American College of Allergy, Asthma and Immunology (ACAAI) policy, all individuals in a position to control or influence the content of an activity must disclose all financial relationships with any ineligible company that have occurred within the past **24 months**. The ACCME defines a “ineligible company” as companies whose primary business is producing, marketing, re-selling, or distributing health care goods or services, used by or on patients. Examples of such organizations include:

- Bio-medical startups that have begun a governmental regulatory approval process
- Compounding pharmacies that manufacture proprietary compounds
- Device manufacturers or distributors
- Diagnostic labs that sell proprietary products
- Growers, distributors, manufacturers or sellers of medical foods and dietary supplements
- Manufacturers of health-related wearable products
- Pharmaceutical companies or distributors
- Pharmacy benefit managers
- Reagent manufacturers or sellers

The ACCME does not consider providers of clinical service directly to patients to be commercial interests. For more information, visit www.accme.org. All identified relevant relationships must be mitigated and the educational content thoroughly vetted for fair balance, scientific objectivity, and appropriateness of patient care recommendations. It is required that disclosure of or absence of relevant financial relationships be provided to the learners prior to the start of the activity.

Learners must also be informed when off-label, experimental/investigational uses of drugs or devices are discussed in an educational activity or included in related materials.

Disclosure in no way implies that the information presented is biased or of lesser quality. It is incumbent upon course participants to be aware of these factors in interpreting the program contents and evaluating recommendations. Moreover, expressed views do not necessarily reflect the opinions of the ACAAI. **All identified relevant financial relationships have been mitigated.**

Planner

- Anna Nowak Wegrzyn, MD, Advisor: Aimmune, Aquestive, DBV Technologies, Novartis, Regeneron; Advisor/Speaker: Nestle; Speaker: Thermo Fisher Scientific; Speaker/Researcher: Nutricia.

Authors:

- Christopher Michael Warren, PhD, Consultant: Alladapt Immunotherapeutics.
- Tami Bartell, MPH, has no relevant financial relationships with ineligible companies to disclose.
- Sai Nimmagadda, MD, has no relevant financial relationships with ineligible companies to disclose.
- Lucy Bilaver, PhD, has no relevant financial relationships with ineligible companies to disclose.
- Jennifer Koplin, PhD, has no relevant financial relationships with ineligible companies to disclose.
- Ruchi S. Gupta, MD, MPH, Researcher/Consultant: Genentech; Consultant: Novartis.

Recognition of Commercial Support: This activity has not received external commercial support.

Copyright Statement: © 2015–2022 ACAAI. All rights reserved.

CME Inquiries: Contact the American College of Allergy, Asthma & Immunology at education@acaaai.org or 847-427-1200.

Results: Socioeconomic status (SES) is a complex concept that encompasses not only economic resources (eg, income, wealth) but also a person's social, economic, and political power and standing, each of which can affect health. However, in many studies of individuals and families with FA, assessment of SES has been limited and often a respondent's membership within a racial and ethnic group is used as a proxy for low SES. As a whole, findings from US population-based studies indicate a consistent trend: those who self-identify as non-Hispanic Black, and to a lesser extent other subpopulations who identify as being of non-White race and ethnicity, experience a greater burden of food-allergic sensitization and disease including higher rates of emergency health care utilization and food-induced anaphylactic fatality as compared with those identifying as non-Hispanic White.

Conclusion: Reports of FA management and outcomes highlight inequities among specific low SES populations in the United States. Clinicians can and should act to reduce inequities by engaging more diverse populations in clinical research, equitably implementing FA risk screening and prevention, thoughtfully using emerging technologies to ameliorate disparities based on SES in health care delivery and outcomes, and advocating for social change.

© 2022 American College of Allergy, Asthma & Immunology. Published by Elsevier Inc. All rights reserved.

Introduction

In recent years, immunoglobulin E (IgE)-mediated food allergy (FA) has become a major public health concern¹ affecting millions of children and adults around the world. In the United States, an estimated 8% of children and 10% of adults have at least 1 FA, imposing a substantial population-level burden of disease.^{2,3} However, emerging data suggest that this burden is heterogeneous and differs considerably across age,⁴ sex,⁵ geographic,⁶ racial,⁷ ethnic,⁸ and socioeconomic⁹ strata. The specific goal of this review is to characterize the burden of FA in North America across various socioeconomically disadvantaged populations—with a focus on providing context for the practicing clinicians who care for them.

Socioeconomic Status and Health Outcomes

Socioeconomic status (SES) is understood to be a key predictor of a person's physical and mental health and educational achievement.¹⁰ At the individual level, SES as a measure of absolute poverty has been associated with health and educational achievement, and low SES has been identified as a risk factor for economic and social deprivation at the community and societal levels.¹¹ Socioeconomic stratification has been found to correlate with risk of disease, disability, and death across major racial and ethnic groups in the United States and arguably lies at the root of these differences.^{12,13}

These differences in health and well-being are observed throughout the life course,¹⁴ as SES has been found to affect nearly every aspect of life in the United States. This begins with birth outcomes and childhood household- and community-level factors, which have a broad influence on educational opportunities, work and career options, outlets for leisure, availability of neighborhood-level resources, including green space and healthy food, absence of adverse environmental exposures, and access to adequate and quality medical care. As a result, considerable research has identified the presence of historically pervasive and prolonged health disparities that are caused and compounded by SES.^{15–17}

If we are to address these deep inequities in our society, greater efforts to better understand how SES affects population patterns leading to health disparities are needed. Although we know that an individual's SES can affect their health outcomes and health care access,^{18–20} knowing more about the specific ways that health and disease are patterned across SES strata can inform how best to frame future research and interventions,²¹ starting with who should be included in research studies.

Understanding Socioeconomic Status

SES has been defined and described as a complex concept that encompasses not only economic resources, including income, wealth, and home ownership, but also a person's social connectedness and economic and political power and standing.^{22,23} Each of these

components can influence a person's health at all levels of the socioecological model (eg, individual, household, neighborhood, society) by different pathways.

Because the impacts of SES extend across the life course and generations, its effects on quality of life and the lived experiences of individual patients can be profound. Poverty reflects these interwoven physical and psychosocial variables and their collective impact on individuals and communities.¹⁰ In the US context, poverty—or the state of having insufficient material resources to maintain physical and mental well-being and protection from the elements—is often estimated using thresholds of household income that vary based on family size and region.^{24–26} Although we know something about the complex connection between poverty, SES, and health, because researchers do not have access to data related to poverty or SES for the populations they observe and study, largely because these constructs are multifactorial and many relevant variables are not assessed or well understood, race, ethnicity, income, occupation, and educational attainment are frequently used as proxies for SES. Table 1 contains a list of currently recommended measures for assessment of these constructs. However, when these data are available, because there is no standardized method for measuring SES, researchers use a variety of approaches to define SES in their studies making comparisons difficult.²² New definitions, reconceptualizations, and measurement tools for SES and poverty are needed as understanding about these concepts continues to evolve, as has language to describe different racial and ethnic groups (ie, Black, Indigenous, and People of Color [BIPOC]).

Among those with FA, economic and social deprivation require further clinical consideration, particularly because FA can contribute to further social isolation and deprivation, which could increase both its economic and psychosocial burden.²⁹ The connection between and impact of economic resources and food consumption is vital and encompasses issues of food security, availability, quality (including access to allergenic-free food), and the experience of hunger and its connections to climate change.^{30,31} As economic circumstances become more precarious, individuals increasingly experience additional adverse environmental exposures in their homes, neighborhoods, occupations, and relationships that increase the burden of FA and result in the cumulative embodiment of deprivation.³²

However, in most previous studies of individuals and families with FA, assessment and analyses of SES have been limited. Historically, US data reporting health differences across groups with FA have been stratified only by racial or ethnic groups, and much less frequently by other factors associated with SES (eg, household income, educational attainment).²⁷ For instance, data are mostly lacking regarding how each of these factors acts as independent predictors of adverse health outcomes among children and adults with FA.

It is for the above-mentioned reasons that much of the rest of this review focuses on associations between race and ethnicity on FA outcomes, as these other factors constitute important and widely reported indicators of SES in the US context. However, we do acknowledge the need to collect, track, and analyze factors related to

Table 1
Recommended Practices for Assessing Socioeconomic Status, Race, and Ethnicity in Health Research

Indicator	Recommended survey/Assessment methodology ^a	Source of recommended survey questions
Educational attainment ^b	- Assess in single years completed up to 5+ years of college - Assess whether individual obtained a high school diploma or GED; including associates, bachelor's, and postsecondary degree attainment	US Census Bureau American Community Survey
Annual household income ^b	- Assess individual income and household income - If feasible, income information should include measurement of total income, earned or unearned, from specific sources (eg, wages or salaries, dividends interest, social security, unemployment insurance, disability income)	US Centers for Disease Control and Prevention Behavioral Risk Factor Surveillance System
Occupation ^b	- Assess by 2 standardized questions, one for occupation and another for industry - Consider asking participant to briefly summarize their work tasks and employer	US Census Bureau American Community Survey
Family size and relationships	Assess information on the number of individuals residing in the respondent's household and household composition during the past year; these data are crucial for derivation of poverty status using federal- and state-level income cutoffs	US Census Bureau American Community Survey
Race and ethnicity ²⁷	- Assess participant race and ethnicity in a manner that permits harmonizing data with census data and other federal data sets that follow the Federal 1997 OMB standard - Permit selection of multiple categories of the following (at a minimum): White; Hispanic/Latino/Latinx; Black/African American; Asian; American Indian/Alaskan Native/Indigenous; Middle Eastern or North African; Native Hawaiian or Other Pacific Islander; Other - Allow participants to write-in specific racial and ethnic groups	US Census Bureau Combined Race and Ethnicity Question with checkboxes

Abbreviations: GED, general educational development; SES, socioeconomic status.

^aThese recommendations are published by the National Center of Vital and Health Statistics, within the United States Department of Health and Human Services.²⁸

^bThese SES indicators are often referred to as the “Big 3.”

systematic socioeconomic disadvantage. Until then, attempts to understand the impact of SES on health outcomes through data on race and ethnicity will be necessary though insufficient.

Although uncomfortable for many to acknowledge, it is a historical fact that the United States was founded as a settler colonial state and an active participant in the trans-Atlantic slave trade, which brought approximately 10 to 12 million enslaved Africans to the Americas. In their quest to dominate indigenous American peoples and maintain political power, European-American elites imposed a racial hierarchy that discriminated against “non-White” residents in many ways.³³ As a result of this well-documented history of systemic racism and other related social dynamics (eg, discrimination, differential ability to accumulate wealth and transfer it intergenerationally, social stratification, and geographic segregation resulting from exclusionary zoning policies, ie, red lining), race and ethnicity have come to be important predictors of disease outcomes in the United States,³⁴ including atopic conditions, such as FA. However, it is crucial to acknowledge that race is not rooted in biology but rather is a nonbiological categorization system based on a combination of personal identity and phenotypical indicators.

When considering associations between race and health outcomes, it is crucial that race be understood as a social construct—as indicated in Figure 1—because many, if not all racial differences in health outcomes are likely caused by structural and socioenvironmental determinants, including differential treatment by institutions and individual actors within the health care system itself.^{35–37} Race must not be misconstrued as having an essential biological or genetic basis, because it is socially constructed in a specific way based on national context. A corollary of this is that analyses of specific racial and ethnic health disparities may be somewhat limited in their international applicability.³⁸ However, in the United States, historical patterns of racism, discrimination, and migration have rendered race (and ethnicity) an important

indicator of health outcomes, including SES, which is why race (and ethnicity) are highlighted throughout this review.

Ethnicity, a separate social construct apart from race, refers to social groups with shared cultural origin.³⁹ The US Census Bureau⁴⁰ distinguishes Hispanic and Latino ethnicity from race and defines ethnicity as the heritage, nationality, lineage, or country of birth of the person or the person's parents or ancestors before they arrived in the United States. Consequently, US residents identifying as Hispanic and Latinx may identify as any race, leading to the most often used racial and ethnic categories of non-Hispanic White, non-Hispanic Black, and non-Hispanic Asian. However, the term “Latinx” has emerged recently as a gender-neutral term for persons of Latin American ethnicity.⁴¹

As clinicians, it is important to be knowledgeable about this history and about a patient's self-identified race and ethnicity and to acknowledge how these constructs have been used as proxies for SES and may influence (consciously or unconsciously) the provision of clinical care, conduct of research, and ultimately, patient outcomes. Of course, this is not a substitute for the role that clinicians can and should undertake as advocates for their patients at the individual, community, and society levels—not only to ensure that they get the information and care they need but also to advance the necessary upstream social and structural changes that can help eradicate health disparities and lead to improvements in health and well-being for children and adults with FA, irrespective of race, ethnicity, or SES. Certainly, changes that clinicians can make at the individual patient level cannot fully address the broader systemic inequities in environmental exposures, which are stratified across SES. In contrast, systematic efforts to identify and address the adverse structural and social determinants of health that tend to cluster in communities with lower SES are needed if we are to ever address these underlying reasons for why under-resourced communities lack resources and experience differential FA outcomes.

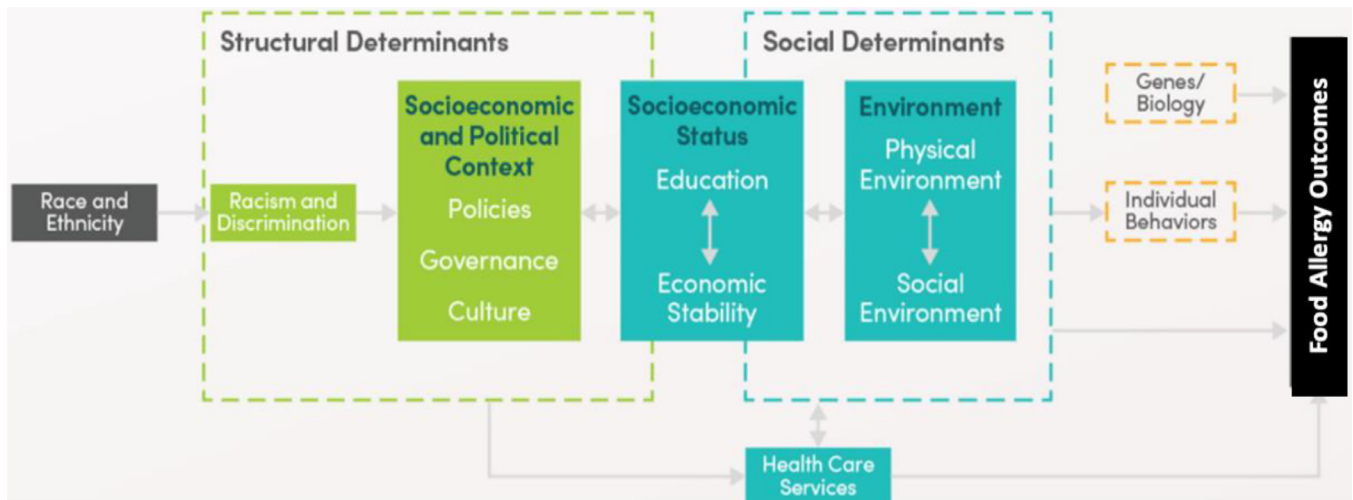


Figure 1. Conceptual model of how an individual's race and ethnicity may influence their food allergy outcomes, incorporating structural and social determinants of health. Adapted from: <https://www.aafa.org/asthma-disparities-burden-on-minorities.aspx>.

Racial, Ethnic, and Socioeconomic Differences in Food Allergy Prevalence

In the past 20+ years, US population-based studies indicate a consistent trend: those who self-identify as non-Hispanic Black, and to a lesser extent other subpopulations who identify as being of non-White race and ethnicity, experience a greater burden of food-allergic disease compared with those who identify as White. This includes elevated FA prevalence rates and more frequent utilization of health care resources for FA.⁴² The disproportionate burden of FA on non-Hispanic Black and other populations, who on average experience lower SES in the US context, is consistent with epidemiologic data from other related atopic disease states (eg, asthma,^{43,44} eczema,^{45–48} and environmental allergies).⁴⁹ A summary of this epidemiologic evidence follows.

The first national population-based effort to estimate FA prevalence and temporal trends was undertaken by Sicherer et al^{50–53} in 1997, 2002, and 2008 using random-digit-dial surveys. Although the first of these 3 surveys did not assess respondent race and ethnicity, the second survey estimated the prevalence of seafood allergy to be much higher among non-Hispanic Black respondents, relative to their non-Hispanic White counterparts (3.7% vs 1.9%, respectively). In contrast, the relative frequency of shellfish allergies being reported as physician diagnosed was comparable (40% vs 38%)—suggesting that the differences in prevalence may not be primarily driven by systematic racial and ethnic differences in health care access.

More recent prevalence estimates from follow-on surveys identified similar disparities and indicated a rising burden of FA among non-Hispanic Black Americans. For instance, these studies estimated that peanut and tree nut allergy prevalence increased from 0.7% to 1.9% among non-Hispanic Black Americans from 2002 to 2008, compared with an observed increase of 1.2% to 1.4% among non-Hispanic White Americans.⁵³ Again, comparable rates of physician diagnosis among reported peanut and tree nut allergy cases were identified. It is important to note, however, that these surveys did not estimate the prevalence of other major FAs, including those to hen's egg, cow's milk, wheat, or soy.

In a 2011 survey,⁵⁴ which used a national sampling frame to interview parents and caregivers of approximately 40,000 US children, the odds of having FA were approximately double among non-Hispanic Black children relative to non-Hispanic White children, adjusting for sex, age, household income, and census geography. In addition, irrespective of race and ethnicity, children identified as having FA by families reporting an annual household income of less than \$50,000 were considerably less likely to receive a physician diagnosis, compared with their higher earning peers.⁵⁴ This highlights a potential disparity

in access to specialist allergy care, which is discussed subsequently. It should be noted that it is possible that some of the observed increases in FA prevalence may be driven by improved recognition and knowledge of FA among these groups. In any event, the potential for differential awareness and recognition of FA and differences in access to specialist allergy care pose problems for population-based surveys of FA prevalence in different racial, ethnic, and SES groups. If these factors are not considered, estimates may not reflect the true underlying distribution of FA by race, ethnicity, and SES.

A meta-regression⁵⁵ of previously published FA prevalence estimates, including both federal data and the aforementioned national surveys,^{50–53} concluded that pediatric FA increased in prevalence by an estimated 1.2 percentage points per decade in the previous 2 decades. However, it increased by 2.1 percentage points per decade among non-Hispanic Black Americans. More recent data from national population-based surveys reported that current FA prevalence is now higher among Black Americans compared with White Americans irrespective of the specific case definition used (eg, self-reported FA vs convincingly IgE-mediated food-induced reaction symptom-report, vs physician-diagnosed FA).⁴²

Recent epidemiologic data also report that US pediatric and adult populations who self-identify as Hispanic or Latinx^{2,3} experience higher clinically diagnosed FA prevalence than their non-Hispanic White peers. In a multisite cohort, observed rates of pediatric shellfish and finned fish allergy were substantially higher in Hispanic (and non-Hispanic Black) children compared with their non-Hispanic White peers.^{56,57} Findings for elevated seafood allergy risk among individuals of non-White race were also recently corroborated by data from the National Institutes of Health-supported FORWARD cohort,⁵⁸ which reported that non-Hispanic Black study participants had significantly higher adjusted odds of allergist diagnosed allergy to finned fish (odds ratio [OR], 2.54; $P < .01$) and shellfish (OR, 3.10; $P < .001$), which included statistical adjustment for sex, age, household income, and recruitment site. In this cohort, children with shellfish allergy also had significantly elevated adjusted odds of asthma (OR, 1.94; $P = .02$).

Together, findings for high rates of seafood allergy among non-Hispanic Black and Latinx children and adults in these national and regional datasets have led to mechanistic hypotheses that have the potential to inform clinical care and future preventive interventions. For example, it has been hypothesized that sensitization to shellfish may occur through inhalation of tropomyosin,^{59–61} a protein that shares a common peptide chain and structure with other inhaled allergens (eg, house dust mites, cockroach) and shellfish. Shrimp tropomyosin (Pen a

1) and cockroach (Per a 7) have similar amino acid sequences,⁶² and elevated levels of household exposure to cockroach allergen have been linked to higher shrimp- and cockroach-specific IgE.^{63,64} Data from Black participants from predominantly low-resourced households in the National Cooperative Inner City Asthma Consortium⁶³ indicate that greater exposure to cockroach allergen—such as that which is more likely to occur in older, less well-maintained rental housing disproportionately inhabited by urban residents in lower SES communities⁶⁵—can provoke cross-sensitization for shellfish allergy. It is plausible that similar processes may also drive the observed racial differences in finned fish allergy prevalence, given that tropomyosins are present in most often consumed finned fish,^{66,67} and IgE binding to finned fish tropomyosins was recently found in patients who have allergy to finned fish.⁶⁸ Although further research is needed to elucidate the specific mechanism(s), evidence suggests that efforts to better understand and mitigate household exposures to common aeroallergens (eg, cockroach, house dust mite) may improve clinical FA outcomes.

Allergic Sensitization

With respect to food-allergic sensitization, data from the Project Viva cohort⁶⁹ found that non-Hispanic Black adolescents had higher rates of sensitization to peanut, cow's milk, hen's egg, soy, and wheat—all 5 common food allergens evaluated in this study—although these rates were only elevated for peanut and wheat after adjustment for maternal education, maternal smoking during pregnancy, and infant exposure to environmental tobacco smoke, household income, and census tract median income. Similar racial and ethnic differences were observed with respect to estimated FA prevalence based on parental symptom report. In this study, maternal education and food sensitization were negatively associated, although relationships were less straightforward when household income was evaluated. For instance, income was only associated with elevated odds of soy sensitization, and maternal educational attainment was associated with lower odds of cow's milk and wheat sensitization.⁶⁹

The Detroit-area Wayne County Health, Environment, Allergy, and Asthma Longitudinal Study (WHEALS)⁷⁰ found similar patterns in their large birth cohort of non-Hispanic Black and White children. At the year 3 follow-up, this study of environmental influences on pediatric allergy found that sensitization to cow's milk, hen's egg, or peanut was more prevalent among non-Hispanic Black children compared with non-Hispanic White children, even after adjusting for income, Medicaid insurance status, and maternal education.⁴⁹ However, physician-assessed cow's milk, hen's egg, or peanut allergy did not differ in prevalence by race or ethnicity. Previous work in this cohort at the year 2 follow-up found that atopic dermatitis, wheeze, and sensitization to a larger panel of 10 environmental and food allergens were each more common among non-Hispanic Black infants compared with non-Hispanic White infants.⁷¹ At the year 10 follow-up, allergic non-Hispanic Black children still had higher rates of sensitization, even after adjusting for socioeconomic variables (eg, income, maternal education, and difficulty making housing payments) and a variety of lifestyle variables (eg, exposure to pets, siblings, and antibiotic use). These findings from the WHEALS study are consistent with data published from the National Health and Nutrition Examination Survey III,⁷² which found that among US children aged 6 to 19 years, rates of sensitization to peanut, hen's egg, cow's milk, and shrimp were higher among non-Hispanic Black vs White children, with the most pronounced differences observed for shrimp (21% vs 7%) and cow's milk (13% vs 6%).

The above-mentioned estimates of racial and ethnic differences in food-allergic sensitization, with and without evidence for clinical allergy, raise questions about the extent to which the current set of 95% positive predictive values (PPVs) used to guide clinical decision-making are generalizable to patients from different racial and ethnic populations. These cutoffs⁷³ serve as useful decision points for the practicing allergist as

they consider whether to proceed with a confirmatory oral food challenge (OFC) or further testing (eg, component resolved diagnostics, basophil activation testing, mast cell activation testing, and epitope mapping). However, recent data⁷⁴ suggest that a given patient's total IgE level influences the 95% PPV of specific IgE (sIgE) and that a large proportion of children with elevated total IgE and moderate-to-severe atopic dermatitis passed an OFC despite having sIgE values above the 95% PPV.

Other recent data from the multisite Food Allergy Management and Outcomes Related to White and African American Racial Differences (FORWARD) cohort of children with physician-diagnosed FA suggest that non-Hispanic Black children have elevated total IgE relative to non-Hispanic White children.⁷⁵ Furthermore, a previous review⁷⁶ highlighted how non-Hispanic Black race is associated with higher levels of allergen-specific IgE with decreased clinical relevance—specifically how 95% cutoffs defined in the United Kingdom⁷⁷ for peanut sIgE and Ara h 2 sIgE provided lower PPVs in South African⁷⁸ peanut-sensitized patients. Altogether, these data indicate that further, concerted efforts may be warranted to evaluate the diagnostic performance of currently used laboratory cutoffs in samples that reflect the full diversity of populations affected by FA.

Physical Health Outcomes: Anaphylaxis and Fatality

The most severe FA outcome is fatal food-induced anaphylaxis. Data from the US National Mortality Database⁷⁹ recorded from 1999 to 2010 suggest that in the United States, non-Hispanic Black populations are at greatest risk of fatal food-induced anaphylaxis relative to their White peers. Not only that, but annual rates of fatal food-induced anaphylaxis increased from 0.06 deaths per million (1999–2001) to 0.21 deaths per million (2008–2010) in non-Hispanic Black males, but did not increase among non-Hispanic White, Hispanic, or non-Hispanic Black females during the same period.⁷⁹ The extent to which these patterns have persisted in the last decade remains unclear, as do the underlying causes of these observed differences in mortality by race and ethnicity, although similar trends⁸⁰ have been observed with respect to asthma mortality.

With respect to healthcare utilization, recent data^{2,3} from a population-based survey of more than 50,000 households found that non-Hispanic Black individuals with FA are more likely to report the following: (1) more than or equal to 1 FA-related emergency department (ED) visit within the last 12 months; (2) a “severe” food-allergic reaction history (indicated by multiple organ system involvement); and (3) use of an epinephrine autoinjector (EAI) to treat a food-allergic reaction, despite very similar rates of reporting a current epinephrine prescription. Furthermore, data⁵⁷ from chart reviews of 817 patients with FA across the midwestern United States provide further evidence that allergic disease may have a disproportionate impact on non-Hispanic Black children, with rates of food-induced anaphylaxis and FA-related ED visits more than 2 times higher among non-Hispanic Black patients relative to non-Hispanic White patients, even though these populations had similar age distributions.

That said, it is noteworthy that the lack of universal health care in the United States and patient-level heterogeneity in health care insurance coverage makes ED utilization an imperfect indicator of FA severity. However, it is essential that emergency health care providers are educated and equipped to either administer epinephrine directly or facilitate the administration of carried or undesignated epinephrine to individuals experiencing anaphylaxis, given that it remains the only first-line treatment for anaphylaxis and has an excellent safety profile. Moreover, evidence^{81,82} suggests that the pathophysiology of food-induced anaphylaxis can differ from that of other types of anaphylaxis (eg, medication, sting, venom)—specifically, fatal food-induced anaphylaxis outcomes are nearly always caused by respiratory collapse—highlighting the importance of timely administration of epinephrine over administration of antihistamine and other adjunct therapies (eg, corticosteroids, inhaled beta-agonists). Given that non-White, non-

Table 2
Recommended Validated Clinical Screening Measures for Identification of Patients and Families at Risk of Food Insecurity

Measure ⁹⁴⁻⁹⁷	Specific instructions	Question	Response items	Coding
Hunger Vital Sign		“Within the past 12 months we worried whether our food would run out before we got money to buy more.” “Within the past 12 months the food we bought just didn’t last and we didn’t have money to get more.”	☐ Often true ☐ Sometimes true ☐ Never true ☐ Often true ☐ Sometimes true ☐ Never true	The Hunger Vital Sign identifies households as being at risk for food insecurity if they answer that either or both of the following 2 statements is “often true” or “sometimes true” (vs “never true”):
US Household Food Security Survey Six-Item Short-Form	“I’m going to read you several statements that people have made about their food situation. For these statements, please tell me whether the statement was <u>often</u> true, <u>sometimes</u> true, or <u>never</u> true for (you/your household) in the last 12 months.”	“The food that I/we bought just didn’t last, and I/we didn’t have money to get more.” Was that often, sometimes, or never true for (you or your household) in the last 12 months? “(I/we) couldn’t afford to eat balanced meals.” Was that often, sometimes, or never true for (you/your household) in the last 12 months?” “In the last 12 months, did you or others in your household ever cut the size of your meals or skip meals because there wasn’t enough money for food?” “How often did this happen?” “In the last 12 months, did you ever eat less than you felt you should because there wasn’t enough money for food?” “In the last 12 months, were you every hungry but didn’t eat because there wasn’t enough money for food?”	☐ Often true ☐ Sometimes true ☐ Never true ☐ Do not know ☐ Often true ☐ Sometimes true ☐ Never true ☐ Do not know ☐ Yes ☐ No (skip next question) ☐ Do not know (skip next question) ☐ Almost every month ☐ Some months but not every month ☐ Only 1 or 2 mo ☐ Do not know ☐ Yes ☐ No ☐ Do not know ☐ Yes ☐ No ☐ Do not know	Responses of “often” or “sometimes” on questions HH3 and HH4, and “yes” on AD1, AD2, and AD3 are coded as affirmative (yes). Responses of “almost every month” and “some months but not every month” on AD1a are coded as affirmative (yes). The sum of affirmative responses to the six questions in the module is the household’s raw score on the scale. Food security status is assigned as follows: Raw score 0–1—high or marginal food security Raw score 2–4—low food security Raw score 5–6—very low food security

English-speaking patients are disproportionately represented among those receiving FA care in the ED,⁵⁷ it is especially important that ED providers are equipped to provide appropriate, comprehensible guidance regarding the importance of allergen avoidance, recognition of allergic reactions, and appropriate utilization of EALs, which should also be prescribed on discharge. Although limited to English and Spanish language speakers, some excellent educational materials—including anaphylaxis action plan templates—do exist.⁸³

Racial and Ethnic Differences in Food Allergy-Related Health Care Utilization

Medicaid is the single largest health insurer of children in the United States. A recent study⁸⁴ evaluated guideline-informed care for a FA diagnosis among children aged 0 to 19 enrolled in the Medicaid program for more than or equal to 1 year. Using national Medicaid claims data, the authors found that the estimated rates of allergy specialist follow-up were lower for Asian, Black, and Hispanic children compared with non-Hispanic White children after receiving a FA diagnosis by a primary care provider. Among those groups, only non-Hispanic Black children were less likely to follow-up with an allergist after an ED visit for FA. The rates of filling prescriptions for EALs were also evaluated. Among Medicaid-enrolled children, non-Hispanic Black children were 28% more likely to have an epinephrine prescription filled after diagnosis by a pediatrician or family medicine physician, whereas Hispanic children were less likely to fill an epinephrine prescription after an ED visit for FA, as compared with non-Hispanic White children.⁸⁴

Racial and Ethnic Differences in Food Allergy-Related Access to Care

In the same study of Medicaid claims data,⁸⁴ the results revealed that living in a US county with a high proportion of individuals living

in poverty (>20%) was consistently associated with a lower likelihood of visiting an ED for FA, attending an allergy follow-up visit, and filling an epinephrine prescription after receiving a FA diagnosis from a pediatrician or family medicine provider, compared with children living in US counties with less poverty. Food allergy-related ED visits were also more common in US counties with higher poverty rates.⁸⁴

In a multisite cohort study of N = 817 patients recruited across Chicago, Illinois, and Cincinnati, Ohio,⁵⁷ it was determined that, relative to non-Hispanic Black patients, non-Hispanic White patients received follow-up allergy care for considerably longer (4.3 years vs 3.2 years), adjusting for age, sex, and insurance status. At the same time, patients covered by Medicaid (a proxy for lower income) reported a shorter duration of follow-up care (2.3 years vs 3.2 years) than patients with private insurance, even after adjusting for race. This is potentially worrisome because shorter duration and earlier detachment from pediatric follow-up allergy care are likely to negatively affect FA management and outcomes. Follow-up with allergists during childhood is important because children can outgrow allergies, and without follow-up, many may unnecessarily avoid foods in adulthood owing to missed opportunities for confirmatory food challenges. At the same time, sensitization may worsen with prolonged avoidance. To ensure routine carriage and EAL access for individuals with allergy during an anaphylactic emergency, epinephrine prescriptions must be renewed; but this most often also occurs at follow-up allergy visits. Taken together, the above-mentioned data underline the need to implement strategies that improve access to outpatient specialist care for all children with FA, especially those from families who experience lower SES with correspondingly reduced access to care. Furthermore, these data call for the development and implementation of tailored programs designed to educate families from diverse cultural and socioeconomic backgrounds.

On a related note, the Center for Disease Control’s National Health Interview Survey (NHIS)⁸⁵ found substantial racial and ethnic disparities in health care access among respondents with FA, even after

statistical adjustment for differences in household income and education. Overall, NHIS respondents with FA were more likely than those without FA to report difficulties accessing health care and food. Furthermore, parents and caregivers of non-Hispanic Black and Hispanic children with FA were more likely to report difficulty affording necessary health care and medications and low food security when compared with parents and caregivers of non-Hispanic White children with FA. Although these associations were somewhat attenuated after adjusting for household income and parental educational attainment, non-Hispanic Black survey respondents with FA still had elevated odds of reporting low food security and problems affording medical care and prescriptions.⁸⁵ As in many studies, the treatment of household income and parental education as confounders of associations between race and ethnicity and health outcomes, rather than exposures of interest themselves, make it especially difficult to disentangle the impact of each individual factor. In addition, the corresponding lack of reporting these parameter estimates in the published reports further prevents a deeper understanding of the potential causal role of these unique social and structural determinants of health that are so tightly entangled with SES.

Access to food, and to allergenic-free food, is a critical issue for children and families with FA. Food insecurity refers to the inability to acquire sufficient food for household members owing to low-income or lack of access. More than 38 million US households are food insecure,⁸⁶ and this problem has grown substantially as an economic consequence of coronavirus disease 2019.⁸⁷ Using federal and local nutrition programs is one way to fill the nutritional gap; however, these programs may not be able to address the specialized diets of individuals with FA.⁸⁸ Allergen-free foods that can be used to supplement the diets of many individuals with FA are often expensive and inaccessible to patients relying on supplemental nutrition assistance (SNAP). Before the coronavirus disease 2019 pandemic, only 4 of 60,000 food pantries specifically served the needs of food insecure patients with FA. During the pandemic, the number in operation dropped to 2—Food Equality Initiative (FEI)⁸⁹ and the Rachel Way Food Pantry—although additional organizations have recently emerged to help address these needs (eg, Securing Safe Foods,⁹⁰ FODiversity,⁹¹ and FEI's affiliate—Free From Marketplace).⁹²

Given its adverse impact on disease prevention and management, it is increasingly incumbent on practicing allergists to recognize and address issues of food insecurity among their patients, although screening for food insecurity remains uncommon during most clinical encounters. To wit, a recent survey⁹³ of members of the Academy of Allergy, Asthma, and Clinical Immunology found that just 24.5% of respondents indicated that they screen patients for food insecurity, though most (60%) expressed interest in being able to screen if appropriate resources were available. Table 2 lists validated screening measures for identifying food insecurity.

Ensuring Access to Emerging Food Allergy Immunotherapies Irrespective of Socioeconomic Status

Oral immunotherapy (OIT) is an emerging treatment modality offered by a limited number of allergists. Recent national survey data⁹⁸ revealed that 72% of patients with FA and their caregivers did not know what OIT was before the survey. Respondents with an annual household income more than \$100,000 had greater odds of OIT awareness compared with lower income, non-college-educated respondents. Both Food and Drug Administration (FDA)-approved products and off-label peanut flour have been used for peanut OIT. Analysts have estimated the cost of *Palforzia*—the only currently FDA-approved food allergen immunotherapy—to be \$4200/y/patient; this is the cost of the standardized peanut flour alone and does not consider costs of office visits. Using off-label peanut flour reduces the average cost to \$2500/y/patient to reach maintenance dosing, not including the additional \$730/y cost

for the optional peanut capsules that enable ingestion. However, none of these estimates include additional expenses, such as optional OFCs, travel, time off work, and unanticipated ED visits. Moreover, because these costs only cover desensitization to a single allergen, those with multiple FAs would incur even more costs. Although efforts are currently underway to advance treatments (eg, monoclonal antibodies, multifoed OIT, vaccines) that simultaneously target multiple food allergens, many investigational therapies remain allergen specific (eg, sublingual and epicutaneous immunotherapies).⁹⁹ However, to date, no curative food allergen therapy currently exists and all approaches typically necessitate ongoing treatment. Therefore, unless advances are made in promoting equitable access to more cost-effective food allergen immunotherapies, the disparate burden of FA across socioeconomic strata is likely to continue, if not become further exacerbated.

Engaging Socioeconomically Diverse Populations in Clinical Trials

Clinical trials are not only the primary means through which novel immunotherapeutic approaches are evaluated but are also an essential means through which patients can access emerging therapies at minimal cost. Unfortunately, as summarized in a recent review,¹⁰⁰ researchers often are unsuccessful in enrolling and retaining clinical trial participants who reflect the underlying heterogeneity of the affected population. For instance, in the year 2020, the FDA granted approval to 53 new drugs, whose clinical trials enrolled approximately 32,000 patients. Among the trial participants, three-quarters were non-Hispanic White, whereas 11% were Hispanic and 8% were non-Hispanic Black. This reflects substantially less racial and ethnic diversity than found in the general population not to mention the racial and ethnic distribution of people with peanut allergy. To wit, the phase III trial of *Palforzia* included just 9 non-Hispanic Black participants among the intent-to-treat population of 551 participants and did not report in their landmark publication¹⁰¹ whether or not patients self-reported Hispanic or Latinx ethnicity. The public health implications for such underrepresentation of historically marginalized populations in clinical research are considerable and have been recently reviewed.¹⁰⁰

Advancing Equitable Food Allergy Screening and Targeted Prevention Efforts

As data accumulate, it has become clear that an impaired skin barrier, as is characteristic of eczema and atopic dermatitis, is a crucial risk factor for subsequent development of FA.¹⁰² Data also indicate clear racial and ethnic differences in the relative prevalence and severity of eczema, with the highest rates observed in non-Hispanic Black children.^{103,104} For instance, non-Hispanic Black children may experience more itchiness and inflammation that requires higher doses of medications to get relief. Non-Hispanic Black and Hispanic children tend to develop more severe cases of atopic dermatitis compared with non-Hispanic White children and are also more likely than non-Hispanic White children to miss school owing to eczema. Although debate continues regarding the extent to which prompt, aggressive treatment of eczema can reduce the risk of subsequent FA, guidelines^{105,106} clearly state that eczematous infants should be considered a high clinical priority for encouraging introduction of allergenic solids. However, because of its unique presentation in more pigmented skin—and other social and structural determinants of health that can lead to delayed specialist evaluation and affect access to quality care—eczema is susceptible to misdiagnosis among patients of color. Moreover, given the general lack of skin color diversity among physicians relative to the populations whose skin they are evaluating, it is important that these clinicians appreciate the diverse presentation of eczema in patients with different degrees of skin pigmentation. One publicly available resource for physicians seeking to gain experience in differentiating eczema from other skin diseases on diverse skin types is *Eczema In Skin of Color*¹⁰⁷; the

website also has information to help patients recognize and manage eczema.

As efforts to promote timely, frequent introduction of allergenic solids continue, it will be important to monitor uptake of early introduction guidelines across key racial, ethnic, and SES strata to ensure equitable implementation and to identify where the need for additional targeted outreach and education efforts may be warranted.

Leveraging Technology to Ameliorate—Not Exacerbate—Health Disparities

Another important topic for ensuring that health care delivery does not further entrench the aforementioned socioeconomic disparities in FA burden pertains to telemedicine. Although telemedicine was originally designed to be a powerful tool for increasing access to care, given the well-acknowledged differences in technological literacy by SES and variable access to high-quality electronic devices, cameras, and broadband internet service that can facilitate an effective telemedical encounter, it is important for clinicians to be cognizant of how the quality of care delivered through this modality may not be equitable across SES strata. For example, with poor lighting or camera equipment, it may be extremely difficult to adequately assess a patient's skin by telemedicine. It is also likely that care delivery may be more effective when high-quality video is possible, as opposed to choppy video or audio only, the latter of which may be more likely among patients of lower SES because of a variety of contextual factors.

Conclusion

In conclusion, the practicing clinician must be attentive to the role that SES plays as a key determinant of FA—and health outcomes more generally. More broadly, efforts to advance FA prevention and treatment should consider the role of upstream social and structural determinants of health and the ways in which they are likely to influence the distribution of FA burden at the population level. More specifically, it is essential that clinicians dedicated to caring for people with FA work to ensure the provision of equitable clinical care, so that advancements in FA disease management and prevention reach across the socioeconomic gradient.

References

- Warren CM, Jiang J, Gupta RS. Epidemiology and burden of food allergy. *Curr Allergy Asthma Rep*. 2020;20(2):1–9.
- Gupta RS, Warren CM, Smith BM, Blumenstock JA, Jiang J, Davis MM, et al. The public health impact of parent-reported childhood food allergies in the United States. *Pediatrics*. 2018;142(6):e20181235.
- Gupta RS, Warren CM, Smith BM, Jiang J, Blumenstock JA, Davis MM, et al. Prevalence and severity of food allergies among US adults. *JAMA Netw Open*. 2019;2(1):e185630.
- Sicherer SH, Warren CM, Dant C, Gupta RS, Nadeau KC. Food allergy from infancy through adulthood. *J Allergy Clin Immunol*. 2020;8(6):1854–1864.
- Pali-Schöll I, Jensen-Jarolim E. Gender aspects in food allergy. *Curr Opin Allergy Clin Immunol*. 2019;19(3):249–255.
- Gupta RS, Springston EE, Smith B, Warrier MR, Pongracic J, Holl JL. Geographic variability of childhood food allergy in the United States. *Clin Pediatr*. 2012;51(9):856–861.
- Kumar R, Tsai HJ, Hong X, Liu X, Wang G, Pearson C, et al. Race, ancestry, and development of food-allergen sensitization in early childhood. *Pediatrics*. 2011;128(4):e821–e829.
- Krishna MT, Warren CM, Jiang J, Gupta RS. *Ethnicity-based disparities in immune-mediated diseases—time for action!* Mayo Clin Proceedings. 96. Amsterdam: Elsevier; 2021:2523–2527.
- Saegert SC, Adler NE, Bullock HE, Cauce AM, Liu WM, Wyche KF. *American Psychological Association Task Force on Socioeconomic Status*. Report of the APA Task Force on Socioeconomic Status; 2007.
- Anderson NB, Bulatao RA, Cohen B, National Research Council (US) Panel on Race, Race/Ethnicity, Socioeconomic Status, and Health. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*. Washington, DC: National Academies Press (US); 2004.
- Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, et al. Socioeconomic status and health: the challenge of the gradient. *Am Psychol*. 1994;49(1):15–24.
- Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav*. 1995:80–94.
- Williams DR, Collins C. US socioeconomic and racial differences in health: patterns and explanations. *Annu Rev Sociol*. 1995;21(1):349–386.
- Chen E, Miller GE. Socioeconomic status and health: mediating and moderating factors. *Annu Rev Clin Psychol*. 2013;9:723–749.
- Fiscella K, Williams DR. Health disparities based on socioeconomic inequities: implications for urban health care. *Acad Med*. 2004;79(12):1139–1147.
- Kennedy BM, Paeratakul S, Ryan DH, Bray GA. Socioeconomic status and health disparity in the United States. *J Hum Behav Soc Environ*. 2007;15(2–3):13–23.
- Williams DR, Mohammed SA. Racism and health I: pathways and scientific evidence. *Am Behav Sci*. 2013;57(8):1152–1173.
- Williams DR, Mohammed SA. Racism and health II: a needed research agenda for effective interventions. *Am Behav Sci*. 2013;57(8):1200–1226.
- Williams DR, Mohammed SA, Leavell J, Collins C. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci*. 2010;1186(1):69–101.
- Braveman PA, Cubbin C, Egerter S, Williams DR, Pamuk E. Socioeconomic disparities in health in the United States: what the patterns tell us. *Am J Public Health*. 2010;100(suppl 1):S186–S196.
- Braveman PA, Cubbin C, Egerter S, Chideya S, Marchi KS, Metzler M, et al. Socioeconomic status in health research: one size does not fit all. *JAMA*. 2005;294(22):2879–2888.
- Noël RA. *Race, Economics, and Social Status*. Washington, DC: U.S. Bureau of Labor Statistics; 2018.
- Spicker P. *Definitions of poverty: twelve clusters of meaning*. Pover Int Glossary. 2007;229:84277–84824.
- Hagenaars AJ. The definition and measurement of poverty. *Economic Inequality and Poverty*. Routledge; 2017:134–156.
- Williams DR, Priest N, Anderson NB. Understanding associations among race, socioeconomic status, and health: patterns and prospects. *Health Psychol*. 2016;35(4):407–411.
- National Committee on Vital and Health Statistics. *About NCVHS*. April 2, 2022.. Available at: <https://ncvhs.hhs.gov>. Accessed.
- Jones NA, Bentley M. *Overview of 2015 National Content Test Analysis Report on Race & Ethnicity*. US Census Bureau; 2017.
- Krieger N. Methods for the scientific study of discrimination and health: an ecological approach. *Am J Public Health*. 2012;102(5):936–944.
- Agyeman J, McEntee J. Moving the field of food justice forward through the lens of urban political ecology. *Geogr Compass*. 2014;8(3):211.
- Thornton RL, Glover CM, Cené CW, Glik DC, Henderson JA, Williams DR. Evaluating strategies for reducing health disparities by addressing the social determinants of health. *Health Aff*. 2016;35(8):1416–1423.
- Krieger N. Embodiment: a conceptual glossary for epidemiology. *J Epidemiol Community Health*. 2005;59(5):350–355.
- Zinn H. *A People's History of the United States*. Routledge; 2015:1492–present.
- McIntosh K, Moss E, Nunn R, Shambaugh J. *Examining the Black-White Wealth Gap*. Washington, DC: Brookings Institutes; 2020.
- Egede LE. Race, ethnicity, culture, and disparities in health care. *J Gen Intern Med*. 2006;21(6):667.
- Washington HA. *Medical Apartheid: the Dark History of Medical Experimentation on Black Americans From Colonial Times to the Present*. Doubleday Books; 2006.
- Nelson A. Unequal treatment: confronting racial and ethnic disparities in health care. *J Natl Med Assoc*. 2002;94(8):666.
- Pickert RE, Saperstein A, Penner AM. Placing racial classification in context. *Socius*. 2019;5: 2378023119851016.
- Ford ME, Kelly PA. Conceptualizing and categorizing race and ethnicity in health services research. *Health Serv Res*. 2005;40(5 Pt 2):1658–1675.
- US Census Bureau. *Hispanic origin*. Available at: <https://www.census.gov/topics/population/hispanic-origin.html>. Accessed April 2, 2022.
- Noe-Bustamante L, Mora L, Lopez MH. *About One-in-Four U.S. Hispanics Have Heard of Latinx, but Just 3% Use It*. Washington, DC: Pew Research Center; 2020.
- Warren CM, Brewer AG, Grobman B, Jiang J, Gupta RS. Racial/ethnic differences in food allergy. *Immunol Allergy Clin North Am*. 2021;41(2):189–203.
- Akinbami LJ, Simon AE, Rossen LM. Changing trends in asthma prevalence among children. *Pediatrics*. 2016;137(1):1–7.
- Canino G, McQuaid EL, Rand CS. Addressing asthma health disparities: a multi-level challenge. *J Allergy Clin Immunol*. 2009;123(6):1209–1217.
- Kim Y, Blomberg M, Rifas-Shiman SL, Camargo Jr CA, Gold DR, Thyssen JP, et al. Racial/ethnic differences in incidence and persistence of childhood atopic dermatitis. *J Invest Dermatol*. 2019;139(4):827–834.
- Brunner PM, Guttman-Yassky E. Racial differences in atopic dermatitis. *Ann Allergy Asthma Immunol*. 2019;122(5):449–455.
- Silverberg JL. Racial and ethnic disparities in atopic dermatitis. *Curr Dermatol Rep*. 2015;4(1):44–48.
- Shaw TE, Currie GP, Koudelka CW, Simpson EL. Eczema prevalence in the United States: data from the 2003 National Survey of Children's Health. *J Invest Dermatol*. 2011;131(1):67–73.
- Wegienka G, Havstad S, Joseph CL, Zoratti E, Ownby D, Woodcroft K, et al. Racial disparities in allergic outcomes in African Americans emerge as early as age 2 years. *Clin Exp Allergy*. 2012;42(6):909–917.
- Sicherer SH, Muñoz-Furlong A, Burks AW, Sampson HA. Prevalence of peanut and tree nut allergy in the US determined by a random digit dial telephone survey. *J Allergy Clin Immunol*. 1999;103(4):559–562.

50. Sicherer SH, Muñoz-Furlong A, Godbold JH, Sampson HA. US prevalence of self-reported peanut, tree nut, and sesame allergy: 11-year follow-up. *J Allergy Clin Immunol*. 2010;125(6):1322–1326.
51. Sicherer SH, Muñoz-Furlong A, Sampson HA. Prevalence of seafood allergy in the United States determined by a random telephone survey. *J Allergy Clin Immunol*. 2004;114(1):159–165.
52. Sicherer SH, Muñoz-Furlong A, Sampson HA. Prevalence of peanut and tree nut allergy in the United States determined by means of a random digit dial telephone survey: a 5-year follow-up study. *J Allergy Clin Immunol*. 2003;112(6):1203–1207.
53. Gupta RS, Springston EE, Warrier MR, Smith B, Kumar R, Pongracic J, et al. The prevalence, severity, and distribution of childhood food allergy in the United States. *Pediatrics*. 2011;128(1):e9–e17.
54. Keet CA, Savage JH, Seopaul S, Peng RD, Wood RA, Matsui EC. Temporal trends and racial/ethnic disparity in self-reported pediatric food allergy in the United States. *Ann Allergy Asthma Immunol*. 2014;112(3):222–229.
55. Robinson A, Sharma H, Coleman A, Abdikarim I, Catlin P, Chura A, et al. Food allergy resolution and subsequent introduction among black and white children in the forward study. *Ann Allergy Asthma Immunol*. 2019;123(5):S10.
56. Mahdavinia M, Fox SR, Smith BM, James C, Palmisano EL, Mohammed A, et al. Racial differences in food allergy phenotype and health care utilization among US children. *J Allergy Clin Immunol*. 2017;5(2):352–357.
57. Mahdavinia M, Tobin MC, Fierstein JL, Andy-Nweye AB, Bilaver LA, Fox S, et al. African American children are more likely to be allergic to shellfish and finfish: findings from FORWARD, a multisite cohort study. *J Allergy Clin Immunol*. 2021;9(7):2867–2873.
58. Wong L, Huang CH, Lee BW. Shellfish and house dust mite allergies: is the link tropomyosin? *Allergy Asthma Immunol Res*. 2016;8(2):101–106.
59. Ayuso R, Reese G, Leong-Kee S, Plante M, Lehrer SB. Molecular basis of arthropod cross-reactivity: IgE-binding cross-reactive epitopes of shrimp, house dust mite and cockroach tropomyosins. *Int Arch Allergy Immunol*. 2002;129(1):38–48.
60. Ayuso R, Lehrer SB, Reese G. Identification of continuous, allergenic regions of the major shrimp allergen Pen a 1 (tropomyosin). *Int Arch Allergy Immunol*. 2002;127(1):27–37.
61. Wang J, Calatroni A, Visness CM, Sampson HA. Correlation of specific IgE to shrimp with cockroach and dust mite exposure and sensitization in an inner-city population. *J Allergy Clin Immunol*. 2011;128(4):834–837.
62. McGowan EC, Peng R, Salo PM, Zeldin DC, Keet CA. Cockroach, dust mite, and shrimp sensitization correlations in the National Health and Nutrition Examination Survey. *Ann Allergy Asthma Immunol*. 2019;122(5):536–538. e1.
63. Rosenstreich DL, Eggleston P, Kattan M, Baker D, Slavin RG, Gergen P, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med*. 1997;336(19):1356–1363.
64. Sarpong SB, Hamilton RG, Eggleston PA, Adkinson Jr. NF. Socioeconomic status and race as risk factors for cockroach allergen exposure and sensitization in children with asthma. *J Allergy Clin Immunol*. 1996;97(6):1393–1401.
65. Gonzalez-Fernandez J, Alguacil-Guillen M, Cuellar C, Daschner A. Possible allergenic role of tropomyosin in patients with adverse reactions after fish intake. *Immunol Investig*. 2018;47(4):416–429.
66. Ruethers T, Taki AC, Nugraha R, Cao TT, Koerber M, Kamath SD, et al. Variability of allergens in commercial fish extracts for skin prick testing. *Allergy*. 2019;74(7):1352–1363.
67. Ruethers T, Taki AC, Karnaneedi S, Nie S, Kalic T, Dai D, et al. Expanding the allergen repertoire of salmon and catfish. *Allergy*. 2021;76(5):1443–1453.
68. Coulson E, Rifas-Shiman SL, Sordillo J, Bunyavanich S, Camargo CA, Platts-Mills TA, et al. Racial, ethnic, and socioeconomic differences in adolescent food allergy. *J Allergy Clin Immunol*. 2020;8(1):336–338.
69. Joseph CL, Zoratti EM, Ownby DR, Havstad S, Nicholas C, Nageotte C, et al. Exploring racial differences in IgE-mediated food allergy in the WHEALS birth cohort. *Ann Allergy Asthma Immunol*. 2016;116(3):219–224.
70. Havstad S, Johnson CC, Kim H, Levin AM, Zoratti EM, Joseph CL, et al. Atopic phenotypes identified with latent class analyses at age 2 years. *J Allergy Clin Immunol*. 2014;134(3):722–727.
71. McGowan EC, Matsui EC, Peng R, Salo PM, Zeldin DC, Keet CA. Racial/ethnic and socioeconomic differences in self-reported food allergy among food-sensitized children in National Health and Nutrition Examination Survey III. *Ann Allergy Asthma Immunol*. 2016;117(5):570–572.
72. Foong RX, Dantzer JA, Wood RA, Santos AF. Improving diagnostic accuracy in food allergy. *J Allergy Clin Immunol*. 2021;9(1):71–80.
73. Paradis N, Galarneau-Vincent R, Paradis B, Graham F, Paradis L, Paradis J, et al. Total IgE influences the cutoff values of specific IgE with 95% PPV. *J Allergy Clin Immunol*. 2019;143(2):AB162.
74. Chura A, Ding L, Mersha T, Ortiz-Rodriguez I, Andy-Nweye A, Bilaver L, et al. Racial difference in total IgE between black and white children in the food allergy management and outcomes related to white and African American racial differences (FORWARD) cohort. *Allergy*. 76. NJ: WILEY; 2021:329.
75. Santos AF, Brough HA. Making the most of in vitro tests to diagnose food allergy. *J Allergy Clin Immunol Pract*. 2017;5(2):237–248.
76. Roberts G, Lack G. Avon Longitudinal Study of Parents and Children Study Team. Diagnosing peanut allergy with skin prick and specific IgE testing. *J Allergy Clin Immunol*. 2005;115(6):1291–1296.
77. Gray CL, Levin ME, Du Toit G. Which test is best for diagnosing peanut allergy in South African children with atopic dermatitis? *S Afr Med J*. 2016;106(2):214–220.
78. Jerschow E, Lin RY, Scaperotti MM, McGinn AP. Fatal anaphylaxis in the United States, 1999–2010: temporal patterns and demographic associations. *J Allergy Clin Immunol*. 2014;134(6):1318–1328. e7.
79. Akinbami LJ, Moorman JE, Simon AE, Schoendorf KC. Trends in racial disparities for asthma outcomes among children 0 to 17 years, 2001–2010. *J Allergy Clin Immunol*. 2014;134(3):547–553.
80. Krajewski GS, Krajewski T. Evaluation and management of food allergies in the emergency department. *Emerg Clin*. 2022;40(1):57–67.
81. Davis CM, Kelso JM. Food allergy management. *Immunol Allergy Clin North Am*. 2018;38(1):53–64.
82. Food Allergy Research and Education. Living with food allergies, newly diagnosed. Available at: <https://www.foodallergy.org/living-food-allergies/information-you/newly-diagnosed>. Accessed June 20, 2022.
83. Kanaley MK, Dyer AA, Negris OR, Fierstein JL, Ciacchio CE, Gupta RS, et al. Guideline-informed care among Medicaid-enrolled children with food allergy. *Am J Manag Care*. 2020;26(12):505–512.
84. Johns CB, Savage JH. Access to health care and food in children with food allergy. *J Allergy Clin Immunol*. 2014;133(2):582–585.
85. Feeding America. Hunger in America. Available at: <https://www.feedingamerica.org/hunger-in-america/#:~:text=FACTS%20about%20hunger%20in%20America,United%20States%20are%20food%20insecure>. Accessed April 2, 2022.
86. Parekh N, Ali SH, O'Connor J, Tozan Y, Jones AM, Capasso A, et al. Food insecurity among households with children during the COVID-19 pandemic: results from a study among social media users across the United States. *Nutr J*. 2021;20(1):73.
87. Brown E, Das R, Brewer AG, Martinez E, Bilaver LA, Gupta RS. Food insecure and allergic in a pandemic: a vulnerable population. *J Allergy Clin Immunol*. 2020;8(7):2149–2151.
88. Food Equality Initiative. Food is medicine. Available at: <https://foodequalityinitiative.org/>. Accessed April 2, 2022.
89. Securing Safe Food. Food pantry initiative. Available at: <https://securingSAFEfood.org/>. Accessed April 2, 2022.
90. Facebook. FOODiversity. Available at: <https://www.facebook.com/carolinafoodiversity>. Accessed April 2, 2022.
91. Free from Market. Eat your way to better health. Available at: <https://freefrommarket.com/>. Accessed April 2, 2022.
92. Shroba J, Das R, Bilaver L, Vincent E, Brown E, Polk B, et al. Food insecurity in the food allergic population: a work group report of the AAAAI adverse reactions to foods committee. *J Allergy Clin Immunol*. 2022;10(1):81–90.
93. Economic Research Service U.S. Department of Agriculture. Available at: <https://www.ers.usda.gov/topics/food-nutrition-assistance/food-security-in-the-u-s/survey-tools/#household>. Accessed April 2, 2022.
94. Gundersen C, Engelhard EE, Crumbaugh AS, Seligman HK. Brief assessment of food insecurity accurately identifies high-risk US adults. *Public Health Nutr*. 2017;20(8):1367–1371.
95. Hager ER, Quigg AM, Black MM, Coleman SM, Heeren T, Rose-Jacobs R, et al. Development and validity of a 2-item screen to identify families at risk for food insecurity. *Pediatrics*. 2010;126(1):e26–e32.
96. Baer TE, Scherer EA, Fleegler EW, Hassan A. Food insecurity and the burden of health-related social problems in an urban youth population. *J Adolesc Health*. 2015;57(6):601–607.
97. Warren CM, Roach A, Das R, Casale TB, Vickery BP, Wasserman RL, et al. Oral immunotherapy-related awareness, attitudes, and experiences among a nationally representative sample of food allergy patients/caregivers. *J Allergy Clin Immunol*. 2021;9(11):4087–4094.
98. Hwang DW, Nagler CR, Ciacchio CE. New and emerging concepts and therapies for the treatment of food allergy. *Immunother Adv*. 2022;2(1):ltac006.
99. Warren CM, Brown E, Wang J, Matsui EC. Increasing representation of historically marginalized populations in allergy, asthma, and immunologic research studies: challenges and opportunities. *J Allergy Clin Immunol Pract*. 2022;10(4):929–935.
100. PALISADE Group of Clinical Investigators Vickery BP, Vereda A, Casale TB, Beyer K, du Toit G, et al. AR101 oral immunotherapy for peanut allergy. *N Engl J Med*. 2018;379(21):1991–2001.
101. Martin PE, Eckert JK, Koplin JJ, Lowe AJ, Gurrin LC, Dharmage SC, et al. Which infants with eczema are at risk of food allergy? Results from a population-based cohort. *Clin Exp Allergy*. 2015;45(1):255–264.
102. Torfi Y, Bitarafan N, Rajabi M. Impact of socioeconomic and environmental factors on atopic eczema and allergic rhinitis: a cross sectional study. *Excli J*. 2015;14:1040.
103. Togias A, Cooper SF, Acebal ML, Assa'ad A, Baker JR, Beck LA, et al. Addendum guidelines for the prevention of peanut allergy in the United States: report of the National Institute of Allergy and Infectious Diseases-sponsored expert panel. *World Allergy Organ J*. 2017;10(1):1–8.
104. Fleischer DM, Chan ES, Venter C, Spergel JM, Abrams EM, Stukus D, et al. A consensus approach to the primary prevention of food allergy through nutrition: guidance from the American Academy of Allergy, Asthma, and Immunology; American College of Allergy, Asthma, and Immunology; and the Canadian Society for Allergy and Clinical Immunology. *J Allergy Clin Immunol*. 2021;9(1):22–43. e4.
105. Eczema in Skin of Color. Atopic dermatitis in skin of color. Available at: <https://eczemainskinofcolor.org>. Accessed April 2, 2022.
106. Barbosa W, Zhou K, Waddell E, Myers T, Dorsey ER. Improving access to care: telemedicine across medical domains. *Annu Rev Public Health*. 2021;42:463–481.
107. Vogels EA. Digital divide persists even as Americans with lower incomes make gains in tech adoption. 2021. Available at: <https://www.pewresearch.org/fact-tank/2021/06/22/digital-divide-persists-even-as-americans-with-lower-incomes-make-gains-in-tech-adoption/>. Accessed April 2, 2022.