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Racial Differences in Allergic Sensitization: Recent Findings and Future Directions

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Abstract

Racial disparities are present in many facets of health and disease. Allergy and asthma are no exceptions. Secondary results from cross-sectional and cohort studies have provided information on the scope of racial disparities in allergic sensitization in the United States. African American/Black individuals tend to be sensitized more frequently than White individuals. Little is known about rates in other race groups. Genetics are unlikely to be the sole or major cause of the observed differences. Home dust allergen and endotoxin levels cannot explain the differences. Studies that have been designed to specifically address the sources of these racial disparities are needed. A “Multilevel Framework” that considers the roles of the individual, family and community presents an excellent approach to guide design of future studies of the causes of these disparities. Understanding the causes of the disparities could lead to interventions that would improve the health of all individuals.

Keywords

racial disparities; racial differences; atopy; sensitization; allergies; asthma

Introduction

While racial disparities have been well documented with respect to the outcomes of asthma prevalence and severity, far less is known about racial differences in allergen sensitization. Sensitization to allergens may affect the risk of asthma incidence and exacerbation. Liu et al. reported that in the NHANES III data, those with a doctor diagnosis of asthma had greater odds of having food allergy.^[1] In a study of asthmatic children in southern California, controller medication and β -agonist use were associated with the number of positive skin prick tests.^[2] It has been shown that inner city children with moderate to severe asthma who are sensitive and exposed to cockroach allergen experience more asthma symptom days, more caretaker interrupted sleep and more school days missed than children who were neither sensitized nor exposed.^[3] In the United States, cockroach allergen sensitization and exposure has been associated with increased asthma morbidity and this may specially affect

African Americans who are disproportionately exposed to cockroach allergen compared to White individuals.^[4]

Thus, it may be inferred that racial differences in allergic sensitization may contribute to the racial disparities in asthma incidence and outcomes. Further, understanding the causes of racial differences in sensitization could provide novel insights into understanding the etiology of allergies and asthma and could lead to the improvement of health for all individuals at risk of these diseases. The objectives of this paper are to briefly summarize recent evidence about racial differences in allergic sensitization, identify deficiencies in the literature and discuss potential next steps for investigations into this important area of research. There are surprisingly few publications focused on racial disparities. There are, however, more results on racial disparities that are nested within larger studies and presented as secondary findings.

What We Know

Defining Race

Race has been defined in many ways in recent history, not only in the groups used to classify individuals but also in the method for that classification (self-report versus assigned by another party). The 2010 Census included 15 race groups for people to self-identify including: White; Black, African Am., or Negro; American Indian or Alaska Native; Asian Indian; Chinese; Filipino; Japanese; Korean; Vietnamese; Native Hawaiian; Guamanian or Chamorro; Samoan; Other Pacific Islander; Other Asian; and Some Other Race.^[5] There was also an ethnicity question on whether the person reports themselves as Hispanic or Latino.

The role of race in medical research has been debated and the meaning of race in analyses has been discussed.^[6,7] Some have argued it is a proxy for social and economic factors and others cite genetic differences between race groups while many recognize all these factors are influential. This paper will not focus on the debate over the meaning of race, but rather on the literature that has included race in studies of allergic sensitization. By identifying group membership of any kind that is associated with differential risk for sensitization, much knowledge can be gained. Understanding how groups with high risk differ from other groups with lower risk is an important step that can lead to understanding of etiology. Essentially, much is learned through understanding the sources of differences. This summary has been written under the assumption that race is a way to group people by suspected risk similarity. The terms African American and Black and White and Caucasian are used throughout the paper and reflect the actual terminology from the publication.

Results of Large National Studies

The National Health and Nutrition Examination Surveys (NHANES) have provided several recent cross-sectional estimates of prevalence rates of sensitization in the United States. Some key study-specific characteristics (sample size, participant's age, allergens studied, skin prick test or allergen-specific IgE measured) are summarized in Table 1 for the NHANES analyses discussed here. Based on data from NHANES III (n=10,508), non-

Hispanic black individuals were more likely than non-Hispanic white individuals to have had at least one positive skin test result among the United States population ages 6-59 years (62% (SE=1.26) versus 51.3% (SE=1.17); odds ratio=OR=1.6, 95% confidence interval=CI=1.4, 1.9).^[8] The odds ratios were similar for females (OR=1.8, 95%CI 1.4, 2.2) and males (OR=1.5, 95%CI 1.2, 1.8). The 10 allergens included in the study are listed in Table 1. The potential sources of the differences in rates by race were not investigated as this was beyond the scope of the study. Race was based on participant report with some classifications based on interviewer report.

Fessler et al. examined the association between serum cholesterol levels and atopy in the 6854 NHANES 2005-2006 participants who were at least 6 years of age.^[9] Atopy was defined as having at least one detectable allergen-specific IgE (≥ 0.35 kU/l) in a panel of 19 allergens (listed in Table 1). Non-Hispanic Black individuals (57.9%, SE=1.6) had the highest rate of atopy compared to Non-Hispanic White individuals (41.5%, SE=1.3).

Analyses of food allergen sensitization in children in NHANES 2005-2006 by Branum and Lukacs included children ages 1-17 years.^[10] The food allergens included were peanut, milk and egg and also shrimp in those at least 6 years of age. Higher percentages of Non-Hispanic black children were sensitized to peanut (twice as likely, $p<0.01$), milk (twice as likely, $p<0.0001$) and shellfish (4 times as likely; $p<0.0001$) compared to Non-Hispanic white individuals. No differences in rates of egg sensitization were reported.

Liu et al. included 8203 NHANES 2005-2006 participants (children and adults) in their analyses of food allergy in relationship to asthma.^[1] They defined sensitization as having a food-specific IgE level ≥ 0.35 kU/l. Non-Hispanic black individuals had the highest rates of sensitization to food allergens and Non-Hispanic white individuals had the lowest rates (milk: 7.6% (SE=0.9) versus 5.2% (SE=0.6); egg: 4.0% (SE=0.5) versus 3.9% (SE=0.4); peanut: 13.4% (SE=0.9) versus 5.9% (SE=0.5); shrimp: 12.8% (SE=1.2) versus 3.8% (SE=0.3)). Non-Hispanic black individuals also had higher rates of multiple food sensitization compared to Non-Hispanic white individuals (7.9% (SE=0.8) versus 3.9% (SE=0.4)).

Another analysis of food sensitization of milk, egg and peanut in the NHANES 2005-2006 population was conducted for individuals ages 1-21 years ($n=3550$) to investigate the role of nativity.^[11] Among US-born children and adolescents, African American individuals (OR=2.43; 95%CI 1.12, 2.67) were more likely to be sensitized to food than white individuals.

While these studies are very large in sample size, they are cross-sectional studies and subject to the limitations of this study design. These studies have allowed the scope of racial disparities to be assessed in well-represented subgroups (i.e., African American versus White individuals). However, cross-sectional studies do not permit sources of the disparity to be investigated since there is no way to establish that most individual factors preceded sensitization. Further, subgroup analyses by residence location (urban, suburban or rural) or adjustment for social and economic variables were not included in these large studies. However, despite their limitations, large, cross-sectional national studies have contributed

much information about the scope of the racial disparity in sensitization between African American/Black and White individuals.

Other Studies of Children

Some key study-specific characteristics (sample size, participant ages, source of participants, allergens studied, skin prick test or allergen-specific IgE measured) are summarized in Table 2 for the non-NHANES analyses. In a cross-sectional analysis of 569 middle-class children in suburban Detroit, African American children were more likely to be sensitized than white children to ragweed (23.0% versus 11.8%, $p=0.02$) and bluegrass (23.0% versus 12.3%, $p=0.02$) as determined by sIgE ≥ 0.35 IU/mL. Bluegrass sensitization was also higher among African Americans when determined by skin prick tests (17.7% versus 9.5%, $p=0.03$).^[12] The allergens studied are in Table 2. This letter was interesting as it compares children of similar socioeconomic status living in the same suburban city. Few studies have examined racial disparities in allergic diseases in children living in similar residential areas.

In the Wayne County Health Environment Allergy and Asthma Longitudinal Study (WHEALS) birth cohort of 466 Black and 223 White children living in urban and suburban Detroit, African American children were more likely to have had at least 1 allergen-specific IgE ≥ 0.35 IU/mL among 10 common allergens tested when compared to White children (54% versus 42.9%, $p=0.02$) by the age of 2 years.^[13] (Table 2) The differences persisted even after adjusting for a host of socioeconomic and environmental factors including household income, number of older siblings, parental history of allergy, or housedust endotoxin and allergen levels (dog, cat, and cockroach). In a subset of 512 children who also had interview information about wheezing, the 358 Black children were statistically significantly more likely to be sensitized than 154 White children to the following allergens: cockroach (7.1% versus 2%, $p=0.02$), *Dermatophagoides farinae* (10.1% versus 1.3%, $p=0.001$), egg (28.5% versus 16.3%, $p=0.004$), peanut (14.3% versus 7.3%, $p=0.028$) and milk (35.4% versus 23.5%, $p=0.008$).^[14] In the same analyses, black children had more allergens to which they were sensitized compared to white children ($p=0.02$). For example, 9.2% of black and 3.5% of white children were sensitized to four or more allergens. In WHEALS, race was reported by the child's parent or guardian.

In a cohort of 1104 children recruited 24-72 hours after delivery at the Boston Medical Center for the Boston Birth Cohort (BBC), black children ($n=672$) were more likely to have had food sensitization (allergen-specific IgE ≥ 0.35 kUA/L for egg white, cow's milk, peanut, soy, shrimp, walnut, wheat and cod) at age 2 years compared with white children ($n=65$) even after adjusting for various confounders including maternal atopy, c-section, firstborn status and breastfeeding (aOR=2.34, 95% CI 1.24, 4.44).^[15] The black children were also more likely to have been sensitized to at least three foods compared with white children (aOR=3.76; 95% CI 1.09, 12.97). In this study, race was based on self-report. The analyses did not take important socioeconomic variables such as income and maternal education into consideration.

In their cohort of 791 asthmatic children ages 4-18 years who sought care in the Hartford, Connecticut, area, African American children were less likely to be sensitized to dog (aOR=0.5; 95%CI 0.2, 1.0) but more likely to be sensitized to mixed tree pollen (aOR=2.3;

95%CI 1.3, 3.9), mixed grass pollen (aOR=2.7; 95%CI 1.6, 4.8), ragweed (aOR=2.1; 95%CI 1.2, 3.8) and mugwort/sage (aOR=3.1; 95%CI 1.6, 6.0) than white children.^[16] While no statistically significant difference was found for cockroach or dust mite, the study may have been underpowered to detect these differences. The allergens that were included in skin prick testing are in Table 2. Race was based on parental report.

In the Cincinnati area, a cohort of 275 children without a personal or family history of allergic disease or symptoms was skin prick tested with a panel of 11 allergens.^[17] (Table 2) The study population was 48% Caucasian and 46% African-American. Forty-eight percent of the African-American children and 30% of the Caucasian children had at least one positive skin prick test. The African-American children were more likely to be sensitized to any aeroallergen even after adjusting for age and household income (aOR=2.14, 95%CI 1.23, 3.84). Race was based on parental and child report.

Other Studies of Adults

The Boston Epidemiology of Home Allergens and Asthma Study is a cross-sectional study of 882 pregnant women (169 were Black and 577 were White). Black women were approximately 2.5 times more likely than white women to be sensitized to 3 allergens after adjusting for income (aOR=2.76, 95%CI 1.51, 5.05) as defined by allergen-specific IgE 0.35IU/mL to dust mite, dog, cat, cockroach, *Alternaria* species, *Aspergillus* species, ryegrass and ragweed.^[18] Self-reported race was used.

Additional recent work has highlighted racial differences in allergic sensitization among mothers participating in the WHEALS birth cohort study.^[19] These analyses were also cross-sectional. After controlling for numerous socioeconomic variables and variables including house dust endotoxin and allergen levels (dog, cat, cockroach), Black women (n=563) were more likely than White women (n=219) to have been sensitized (allergen-specific IgE 0.35 IU/mL) to at least one allergen (aOR=3.35, 95%CI 1.96, 5.73). The allergens are listed in Table 2. Black women were also more likely to have been specifically sensitized to dog (23.2% versus 14.6%), *Alternaria* (26.8% versus 11.9%), cockroach (19.5% versus 5.5%), short ragweed (38.3% versus 20.6%) and Timothy grass (31.4% versus 17.4%) than White women (all p<0.05). Race was self-reported by the women.

Ancestry Informative Marker Studies

More recently, race as defined genetically by ancestral informative markers (AIMs) has become a novel area of study with an attempt to categorize and quantify race by geographical origin. However, varied methodologies for assigning racial admixture have been used in these studies. It is not clear if there are any effects on the study results due to these different methodologies.

In the WHEALS cohort study, AIMs were used to estimate biogeographic ancestry through the analysis of 493 single nucleotide polymorphisms (SNPs).^[20] Admixture was estimated for each individual. The analyses were performed on 601 WHEALS mothers who identified themselves as white (n=216, 35.9%) or African American (n=385, 64.1%). Self-identified African American women were more likely to be sensitized to at least one allergen (allergen-specific IgE 0.35 IU/mL) than white women (aOR=2.19, 95%CI 1.22, 3.93).

However, genetic ancestry was not statistically significantly associated with allergic sensitization (aOR=1.34, 95%CI 0.73, 2.43) after adjusting for location of residence (urban versus suburban). These data suggest that genetics may not explain the observed racial disparity in allergic sensitization.

In their study of food sensitization in children who were approximately two years of age, the Boston Birth Cohort genotyped 150 AIMS.^[15] African Ancestry was associated with food sensitization (aOR=1.07, 95%CI 1.02, 1.14 for a 10% increase in African ancestry). There was no adjustment for location of residence, income, maternal education or other socioeconomic factors.

Summary of Study Results

Although a small number of studies indicate that Black/African American children and adults tend to experience more sensitization to both food and aeroallergens than White individuals and that racial disparities emerge as early as age 2 years, there is relatively little evidence to assess whether disparities exist between African Americans or Whites and other racial groups such as Asian and Native American Individuals. With so few longitudinal studies, risk (probability of incidence) cannot be quantified for specific racial groups. The roles that genetics and socioeconomic and other factors play in the observed differences are not well understood, but the limited extant data do not indicate a clear and overwhelming role for genetics in the observed racial disparities

What do we need to know and how do we get there?

Few studies of allergic sensitization have set out to do what investigators have attempted with asthma – specifically study the effect of race on incidence. Most of the results discussed in this review have been secondary results of analyses focused on other research questions. Because of this, there are many outstanding questions. To answer these questions, new studies will be needed. The most immediate questions and discussion of the study design elements follow.

Are there racial differences in the risk of sensitization?

There is evidence that African American/Black individuals are more likely than White individuals to be sensitized and to be sensitized to more allergens and the studies supporting this premise have been summarized here. However, there is little information on other racial groups in the United States such as Asian Indian, Japanese, Korean or American Indian individuals. While these groups represent smaller percentages of the United States population, they still represent large segments and warrant study.

Are there racial differences in specific allergens to which the individuals are sensitized?

In the reviewed papers, African American/Black individuals tended to be more frequently sensitized to food and aeroallergens compared with White individuals. However, in some of the studies, African American/Black children did not have higher rates of sensitization to some of the allergens that have been investigated for their role in asthma incidence and

exacerbation (i.e., cockroach and dust mites). Again, there is a paucity of data on individuals who are neither African American/Black nor White.

What are the sources of these observed racial disparities?

By identifying the sources of these differences, great insight into the etiology of sensitization will be obtained and allow improvement in health for all individuals. For example, if a particular exposure is more common in African American children than White children and biological plausibility exists, the exposure becomes an important candidate for study of disease development. The hygiene hypothesis, which has been supported mainly by evidence from Europe, may one day be able to explain, at least in part, the differential rates of sensitization as methods to accurately assess general and specific environmental microbial exposures develop and gain widespread use in epidemiological studies.

Other candidates for a potentially causal relationship to sensitization include factors such as genetics, socioeconomic factors (income, education) and the environment, which can encompass both physical (e.g., allergens, particulate matter, etc.) and societal components (e.g., neighborhood poverty and violence). The case for genetics would be a difficult one to make as the epidemic of allergy has emerged over the last couple of decades – far too quickly for genetics to be the sole or even main explanation. Further, the analyses of Yang et al. argues against genetics as self-reported race was more strongly associated with sensitization than ancestry.^[20] Some argue that environmental exposures are the most probable cause – although in some studies like WHEALS, differences persist even after adjusting for key environmental factors such as allergen and endotoxin levels in home dust. Although admittedly incomplete, existing data is consistent with the notion that combinations of factors including exposure and host-susceptibility such as gene-environment interactions and epigenetic changes that occur in the context of time-related “windows of opportunity” are important mechanisms. Wright and Subramanian present an interesting “Multilevel Framework” for epidemiologic research on asthma disparities.^[21] They propose that there are multiple levels of causation that lay between neighborhood contexts and asthma in the individual. They state that: “The etiology of health problems is increasingly recognized as a result of the complex interplay of influences operating at several levels, including the individual, the family and the community.” This approach could prove very successful in studies of causes of racial disparities in allergic sensitization.

How should future studies be designed?

While there are many promising leads, well-designed studies are needed to examine potential causal factors in a way that produces the strongest evidence. Designing data collection to support the analyses of causes through a “Multilevel Framework” would likely be the best approach at this time based on extant evidence. Further, longitudinal studies are needed to allow assessment of exposures that precede onset of sensitization. Given that racial disparities were present as early as age 2 years, studies should include the prenatal and very early life intervals with environmental measures also performed during these potentially critical periods. This includes elements of both the physical (allergens, pollutants, microbiome) and social environment (poverty, crime, racism). Individual data on health conditions, nutrition and behavioral factors are also essential.

Sample size calculations must take into consideration the need to analyze subgroups jointly (simultaneously) defined by race, socioeconomic factors (income, education, family size) and residential location (urban versus suburban versus rural). The need for subgroup analyses will increase the sample size.

There must also be careful selection of the study population. Careful thought should be given to the residential location of the participants so as to minimize residual confounding. African American individuals and individuals with low socioeconomic status have traditionally been more difficult to recruit and retain in scientific studies. However, thoughtful and strategic recruitment and retention plans will be needed and could employ research techniques such as community-based participatory research to achieve them.

Race must be defined uniformly across studies and within studies. Census definitions would be a strong option as they often change concomitantly as the concept of race evolves in the United States, but the categories are not the same as those on a United States Department of Health and Human Services Public Health Grant Application (PHS 398) enrollment table. Outcomes would ideally be made comparable across studies. In the papers reviewed, sensitization was defined by levels of allergen-specific IgE above a pre-specified cutpoint and skin prick results. Different allergen panels (in quantity and type) were used across the studies. For each study region, the use of a standardized panel combining region-specific and ubiquitous allergens would be helpful in teasing apart differences in sensitization that may be related to geographically-driven exposure versus potential differences in allergen-specific susceptibility related to race or inherent biological factors.

Conclusion

The papers reviewed here demonstrate that African American/Black individuals tend to be sensitized more frequently than White individuals but remarkably few studies address this issue and little is known about sensitization rates in other race groups. The sources of this disparity are not known but are unlikely to be solely genetic. Studies have not been conducted with the specific goal of understanding causes of the racial disparities. This lack of knowledge provides an opportunity for carefully designed studies that assess a multilevel model of causation to provide insight into the causes of disparities at the individual, family and community levels. Once these causes are identified and the etiology of sensitization is better understood, interventions can be designed to improve the health of individuals of all races.

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Table 1

Summaries of recent United States' National Health and Nutrition Examination Survey (NHANES) analyses with results on racial disparities and allergic sensitization.

Study name	Authors	Sample size	Participant ages	Allergens studied	Test
NHANES III	Arbes et al.	10,508	6-59 years	Dust mite, German cockroach, cat, perennial rye, short ragweed, Bermuda grass, Russian thistle, white oak, <i>Alternaria alternata</i> and peanut	Skin prick test
NHANES 2005-2006	Fessler et al.	6854	6 years	<i>Dermatophagoides farinae</i> , <i>Dermatophagoides pteronyssinus</i> , cat, dog, cockroach, <i>Alternaria</i> species, peanut, egg, milk, ragweed, ryegrass, Bermuda grass, white oak, birch, shrimp, <i>Aspergillus</i> species, thistle, mouse and rat	Allergen-specific IgE
NHANES 2005-2006	Branum and Lukacs	Not provided	1-17 years	Peanut, egg, milk and shrimp	Allergen-specific IgE
NHANES 2005-2006	Liu et al.	8203	Children and adults	Milk, egg, peanut and shrimp	Allergen-specific IgE
NHANES 2005-2006	Keet et al.	3550	1-21 years	Milk, egg and peanut	Allergen-specific IgE

Table 2

Summaries of other analyses in the United States with results on racial disparities and allergic sensitization.

Study name	Authors	Sample size	Study population	Participant ages	Allergens studied	Test
Southfield Childhood Allergy Study (SCAS)	Joseph CLM, et al.	569	Middle class, suburban Detroit, Michigan residents	6-8 years	<i>Dermatophagoides farinae</i> , <i>Dermatophagoides pteronyssinus</i> , cat, dog, <i>Alternaria</i> , short ragweed, bluegrass and cockroach	Skin prick test and allergen-specific IgE
WHEALS	Wegienka et al. (CEA) Wegienka et al. (Allergy Asthma Proc)	466 African American 223 White	Urban and suburban Detroit, Michigan residents	2-3 years	<i>Dermatophagoides farinae</i> , Timothy grass, cat, dog, <i>Alternaria</i> , ragweed, egg, peanut, milk and cockroach (<i>Blatella germanica</i>)	Allergen-specific IgE
Boston Birth Cohort	Kumar et al.	672 black 65 white	Born at Boston Medical Center	2 years	Egg white, cow's milk, peanut, soy, shrimp, walnut, wheat and cod	Allergen-specific IgE
Easy Breathing	Celedon et al.	791	Asthmatic children in urban and suburban Hartford, Connecticut	4-18 years	cockroach (<i>Blatella germanica</i>), house dust mite (<i>Dermatophagoides pteronyssinus</i>), cat dander, dog dander, mold mix, mixed tree pollen, mixed grass pollen, ragweed, weed mix, and mugwort/sage	Skin prick test
None provided	Stevenson et al.	275	No family or personal history of allergic diseases		2 mold mixes, grass mix, ragweed (giant and short), 2 tree pollen mixes, weed mix, dust mite mix (<i>der f</i> and <i>der p</i>), cat (fel d 1), dog (hair and dander) and cockroach mix (<i>Blatella germanica</i> and <i>Periplaneta americana</i>)	Skin prick test
Boston Epidemiology of Home Allergens and Asthma	Lintonjua et al.	577 white 169 black	Women who delivered at Brigham and Women's Hospital		dust mite, dog, cat, cockroach, <i>Alternaria</i> species, <i>Aspergillus</i> species, ryegrass and ragweed	Allergen-specific IgE
WHEALS	Wegienka et al. JACI	563 Black women 219 White women	Pregnant women in urban and suburban Detroit	21-49 Years	<i>Dermatophagoides farinae</i> , Timothy grass, cat, dog, <i>Alternaria</i> , ragweed, egg and cockroach (<i>Blatella germanica</i>)	Allergen-specific IgE