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Evaluating Race in Air Pollution and Health Research: Race, $PM_{2.5}$ Air Pollution Exposure, and Mortality as a Case Study

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Abstract

Purpose of Review Racial inequities in air pollution exposure have been documented. There is also interest in documenting the modifying role of race in the link between air pollution and health. However, the empirical literature in this area has yielded mixed results with potentially unclear policy implications. We critically evaluate recent empirical papers on the interactive association between race and air pollution exposure on adult mortality in the USA as a case study of the race, pollution, and health literature. Specifically, we evaluate these studies for the conceptualization and discussion of race and the use of race variables that may contribute to the ambiguous results and policy implications both in this specific literature and in the broader literature.

Recent Findings We evaluate ten empirical studies from 2016 to 2022 on the modifying role of race in the association between short- and long-term $PM_{2.5}$ exposure and specific types of adult mortality (all cause, non-accidental, and heart or cardiovascular diseases) in the USA. In addition to comparing and contrasting the empirical results, we focus our review on the conceptualization, measurement, modeling, and discussion of race and the race variables. Overall, the results indicate no consistent role of race in the association between $PM_{2.5}$ exposure and mortality. Moreover, conceptualization and discussion of race was often brief and incomplete, even when the empirical results were unexpected or counterintuitive.

Summary To build on recent discussions in the epidemiology and environmental epidemiology literature more specifically, we provide a detailed discussion of the meaning of race, the race variables, and the cultural and structural racism that some argue are proxied by race variables. We use theoretical scholarship from the humanities and social sciences along with empirical work from the environmental literature to provide recommendations for future research that can provide an evidence base to inform both social and environmental policy.

Keywords Environmental inequalities · Racial inequalities · Air pollution · Structural racism

Introduction

The evidence on racial inequalities in the health toll related to air pollution exposure is growing, with recent reports indicating the importance of both inequities in exposure and inequities in the health impact of exposures [1, 2]. Clarifying the linkages among race, air pollution, and health is critical for policy intervention. The EPA recently evaluated the health and welfare thresholds for $PM_{2.5}$ exposure, including the role of sociodemographic and health factors that may modify these associations [2]. After reviewing the literature, the authors stated that, "… the evidence is adequate to conclude that race and ethnicity modify $PM_{2.5}$-related risk and that nonwhites, particularly blacks, are at increased risk for $PM_{2.5}$-related health effects, in part due to disparities in exposure" [2]. Notably, there are two components to this

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EPA statement, that there are inequities in air pollution exposures and inequities in the health impact of air pollution exposures. The latter component is the focus of this review and commentary, as we critical evaluate the recent empirical literature on the role of race as an effect modifier in the association between air pollution and health. Our objective is to address a major gap in the environmental epidemiology literature in the detailed conceptualization of race and the meaning of the race variable. We will draw from the experts on race in the humanist and social science scholarship, moving beyond black box, simplistic conceptualizations of "race as a social construct." We will use specific examples from the extant empirical literature, providing specific interpretations of the results and recommendations for empirical tests that may provide evidence upon which we can develop policy.

To facilitate a deeper discussion, we focus on empirical papers on the association between short- and long-term $PM_{2.5}$ and mortality in adults published from 2016 to 2022 as this specific literature has a solid empirical foundation to evaluate, including the recent EPA review [2]. Furthermore, $PM_{2.5}$ has been causally linked to numerous health outcomes and may play a major role in health inequities. By narrowing our focus to this association, we can provide more detailed discussions on the meaning of race. While we focus on this $PM_{2.5}$–mortality association for this case study, the points we discuss will apply to any examination of the role of race in air pollution and health studies, including race and exposure studies. We are not the first to discuss the meaning of race in epidemiology more broadly or in environmental epidemiology specifically. Therefore, to deepen the discussion and address gaps, will build on and extend recent commentaries on the assessment of racial inequities in health that include the importance of inequities in exposures [3•] and the causal meaning of race in environmental epidemiology studies [4•, 5].

We agree with the need to develop environmental pollutant exposure standards to protect our vulnerable citizens. However, it is not that Black Americans as a racial group, for example, are inherently or uniformly more vulnerable than White Americans. It is the landscape of unequal social, economic, and political conditions that renders social groups differentially vulnerable to the health impact of environmental hazards, including air pollution [6]. This distinction is not academic or trivial, but has direct research and policy implications. Rather than continuing to state that race is a social construct while repeatedly documenting racial patterns in the association between air pollution and health without theoretical justification, it is time to build the evidence on the features of the landscape that render different social groups differentially vulnerable to the health impacts of air pollution. While we set our pollutant standards, we also need to target policies to address the conditions that render Black and other non-White groups more vulnerable to the health effects of these pollutants [6, 7].

On Studying Race: from Humanities to Environmental Epidemiology

What Does Race Mean? To evaluate the meaning of race in effect modification studies of $PM_{2.5}$ and mortality is to evaluate the meaning of race in the general epidemiology literature. This general question contains two subparts: (a) What is race? and (b) What does the race variable capture? Modern notions of race in the USA were developed by colonists to reconcile the hypocrisy of a new country founded upon a notion of freedom and equality for all men and reality that some humans were kept as property [8, 9]. Race continues to serve as a categorization schema developed to differentially surveille and control the social, economic, and political circumstances of different social groups [8, 10, 11]. This conceptualization of race (and ethnicity) moves beyond those widely used in epidemiology, and draws from humanist scholarship to highlight the historical origins, intentionality, and the often insidious purpose of racial categorizations. While the US Office of Management and Budget (OMB) currently recognizes five racial groups and one ethnicity in a static manner, the humanist and social science literatures underscore the dynamic nature of race and ethnicity in the concept of racialization. This is a process by which crude, perceptible features of different social groups are identified and stigmatized through power inequities. Sociologists have documented the ways in which Arab Americans and Muslim Americans (groups often conflated) have been racialized through clothing, language, and religion since 9/11 [12, 13]. Similarly, different groups of Latino Americans, defined as an "ethnicity" by the OMB, have been racialized through legalized status, language, occupational class, and skin tone at different points in history [14, 15, 16]. While we focus on the US context here, the process of racialization occurs in other countries and on a global scale as well [17, 18, 19, 20]. For example, information on race, per se, is not collected by the French government (similar to many European governments). However, immigrant status and religious affiliation have been racialized to reflect the underlying French version of cultural racism [21].

Once groups are racialized, differential rights and restrictions follow through formal policies and informal social mores. Implied in this conceptualization of race, borne from decades of scholarship in the humanities and social sciences, is that the social meaning of each racial (and ethnic) category and the relation of these categories to each other, to exposures, and to health will vary over place and time. Perhaps ironically, the notion of five races and one ethnicity, often taught as a static absolute in biomedicine

and public health, serves as a barrier to the deeply nuanced understanding that race is socially constructed—that racialized categories are continually altered and adapted to meet the social and political mores of the time and place.

What Does the Race Variable Proxy? What Is Structural Racism? If the concept of race, through the lens of racialization, is developed and maintained as a tool of sociopolitical surveillance and control, then it may be understood that race stands in as a crude proxy for exposure to features of structural racism. Examples of race-based inequities in surveillance and control discussed in the literature include police presence and violence [22, 23], school resource officers, foster care system interaction [24], and Immigration and Naturalization Service interaction. To understand structural racism, we need to understand the general link between a society's culture and its structure and the meaning of cultural racism. A society's culture is its underlying collective ideologies and value systems, its social and behavioral norms, and its lens through which knowledge is defined and interpreted. The structure of a society, then, is the interrelated network of formal and informal institutions that reflect its culture. The institutions that we have in the USA and the ways in which they operate reflect our values, our norms, our priorities; in other words, our overarching American culture.

Cultural racism can be considered the ideologies and value systems, social and behavioral norms, and overall epistemology that is based on the often-implicit understanding that some racialized groups are superior to others [25•]. Cultural racism is a particularly insidious form of racism as it is a part of our shared social subconscious. We may not be fully aware of or be able to explicate the nuances of our American racial hierarchy but, as with any other aspect of our culture, it becomes part of the lens through which we view and understand people's lives. Also called the "White racial frame" [26], colorblind racism [27], or the racial contract [28••], cultural racism shapes our answers both at the societal and individual level to the questions: "Who is fully American?" and "Whose life matters?" This American epistemology shaped by cultural racism operating in the background means that our society is structured to privilege racialized White Americans in ways that, on the surface, seem neutral and rational without explicit reference to race [26, 29••, 30]. Furthermore, cultural racism includes a temporal amnesia that delinks historical processes, that often were explicitly racial, from current institutional policies and practices that seem non-racial. With this amnesia that filters our knowledge implicitly through whiteness, our contemporary institutional policies and practices appear neutral and rational [28••, 31•].

Structural racism can be considered the ubiquitous, real-world application or actualization of cultural racism. Structural racism is the "interrelated network of a society's institutions, with their policies and practices that favor racial groups over others and operate without the need on the part of their actors to intend harm or hold dislike of certain racial groups" [31•, 32]. In other words, institutional policies and practices reflect the underlying ideas of who "deserves" to live a long and healthy life and whose health will be sacrificed. Of critical importance is the understanding that, while structural racism may be ubiquitous, its features are both temporally and spatially local, changing to adapt to local sociopolitical norms, in what philosopher Achilles Mbembe would call "a more civilized way of killing" [31•, 33•]. The interrelated nature of the institutions that comprise our structure is a key feature in that the bonds that connect them allow for institutions to operate together [33•]. Scholars have documented that neighborhoods characterized by the segregation of Black residents are burdened by, for example, hypersurveillance by the criminal justice system in the form of police violence and mass incarceration as well as disproportionate exposures to environmental pollution [34]. Evidence suggests that these social and environmental exposures together may maintain racial inequities in health [34]. Furthermore, these institutions may operate together, as suggested by evidence that prisoners are used as cheap labor for hazardous waste disposal without the same workplace protections as provided in other settings [35, 36].

Structural racism reflects the racialized hierarchy that stems from cultural racism which means that racism is relational in the sense that some racialized groups are sacrificed for the comfort of others [37, 38, 39••, 40]. For example, in a recent analysis, researchers examined the racial inequity in $PM_{2.5}$ emissions related to the consumption of goods and services [38]. Not only did Black and Hispanic Americans experience greater $PM_{2.5}$ exposure, as has been reported by others but, importantly, these exposures were linked to the greater consumption by White Americans [38]. In other words, the $PM_{2.5}$ emissions linked to the consumption of White Americans is linked to the greater $PM_{2.5}$ emission burden of Black and Hispanic Americans. It may be that Black and Hispanic residents benefit economically from proximity to polluters through employment, an argument that appears rational and to address economic inequities. However, evidence suggests that this is not the case. In another recent analysis, researchers examined the notion that neighborhoods with high proportions of Black residents would reap economic benefit from proximity to polluters but reported that the jobs of these polluting companies went to residents of other communities, leaving those Black communities burdened with the pollution and without the economic benefit [41].

Race in Air Pollution Effect Modification Studies

With this substantive foundation, we turn to review the empirical literature on $PM_{2.5}$ exposure and adult mortality that test effect modification by the interaction between race and air pollution variables. We narrow the study to the USA because race categories and meanings vary across sociopolitical contexts. We exclude studies with samples exclusively with persons with diseases (e.g., cancer registries, dialysis patients) which would require discussion on inequities in access to health care and treatment within the healthcare system. We exclude studies that examine ecological models (e.g., county average exposure and county mortality rates). The resulting studies modeled mortality due to different conditions; we included only those causes that were the focus of more than one study to facilitate qualitative comparisons. Thus, we include ten studies that model all-cause mortality, mortality due to non-accidental causes, and mortality due to heart or cardiovascular diseases (some studies examine mortality due to multiple causes; see Table 1):

- Four studies that model long-term $PM2_{.5}$ exposure and all-cause mortality: three based on data from Medicare enrollees [42–44] and one based on data from the National Health Interview Survey (NHIS), an annual cross-sectional sample of non-institutionalized of adults in the USA [45]; NHIS annual data can be combined across years;
- Three studies that modeled long-term $PM_{2.5}$ exposure and mortality from non-accidental causes, one each based on data from Medicare enrollees [46], the NHIS [47], and the US Veterans Administration [48];
- Three studies that modeled long-term $PM_{2.5}$ exposure and mortality from heart or cardiovascular diseases: one was based on data from Medicare enrollees [46], one from NHIS [47], and one from the National Institutes of Health (NIH)-American Association of Retired Persons (AARP) Diet and Health Study, a sample of AARP members in six states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and two cities (Atlanta, GA, and Detroit, MI) [49];
- Two studies that modeled short-term $PM_{2.5}$ exposure and all-cause mortality: one using data from Medicare enrollees [50] and the other using individual-level data from the North Carolina mortality files [51].

Overall, the modifying role of race on the association between $PM_{2.5}$ exposure (either long-term or short-term) and mortality was inconsistent without many clear patterns. Depending on the dataset used, White adults might exhibit a stronger [44], weaker [42, 43], or same association as other adults [45, 47] (see Table 2). For example, regarding the association between long-term $PM_{2.5}$ exposure and all-cause mortality, White Medicare enrollees showed a weaker positive association compared to enrollees in any other racial category while Black enrollees showed the strongest positive association [42]. When modeling only $PM_{2.5}$ exposures < 12 μg/m^3, however, White enrollees showed a stronger positive association compared to Black enrollees [44]. There were no racial differences in the $PM_{2.5}$–mortality association in the 1986–2014 NHIS sample with all racial groups showing a positive association [45]. Notably, the inconsistencies in the racial patterns persisted even though studies employed similar approaches to the inclusion of covariates, including sociodemographic and health behavior information, as well as a number of contextual variables (see Table 1).

As another example of these inconsistencies, we look to the association between long-term $PM_{2.5}$ exposure and cardiovascular mortality. White Medicare enrollees showed a weaker positive association compared to Black enrollees while Asian and Hispanic enrollees showed an *inverse* association [46]. On the other hand, White, but not Black, participants in the NIH-AARP study showed a positive association, although the interaction term was not statistically significant suggesting that there was no racial difference in the pollution–mortality association [49]. A similar pattern was reported among NHIS participants in that White but not others showed a positive association, but the interaction term was not statistically significant [47].

The pattern of results did vary when racial comparisons are made in different parts of the USA. For example, in a national Medicare study, Black enrollees showed a stronger positive long-term $PM_{2.5}$–all-cause mortality association compared to White enrollees, while Asian and Hispanic enrollees showed an *inverse* association [42]. In a separate study that only included only enrollees in seven southern states, Black enrollees still showed a stronger positive association than White enrollees, but a *weaker* positive association compared to a composite category that included Asian, Hispanic, and Native American enrollees [43]. Indeed, this composite category of Asian, Hispanic, and Native American enrollees from the southern USA exhibited a markedly stronger positive association between air pollution and mortality compared to either White or Black enrollees [52]. In a second example, in a different national Medicare study modeling short-term $PM_{2.5}$ exposure and all-cause mortality, Black but not Hispanic, Native American, or White enrollees showed a positive association, while Asian enrollees showed an inverse association [50]. On the other hand, based on mortality data from North Carolina, White and Black residents showed the same positive association while

Table 1 Dataset and model covariate description

Dataset characteristics	Covariates, individual-level	Covariates, area-level	Covariates, other
[49] *AARP Diet and Health Study*, 1995–2014 • Age: 55.00–57.89 yrs, 25%[a]; 57.90–62.78 yrs, 25%; 62.79–66.72 yrs, 25%; 66.72 + yrs, 25% • Women: 40% • Race: A/NA, 1.6%; B, 3.9%; H, 1.9%; W, 91.2%	• Age, sex • Education • Marital status • 3 Health[b]	• 2 Tract-level sociodemographic	
[42] *Medicare enrollees*, 2000–2012 • Age, mean: 70.1 yrs • Women: 56% • Race: A, 1.8%; B, 8.7%; H, 1.9%; NA, 0.3%; W, 85.4%	• Age, sex • Medicaid eligibility	• 8 ZC-level sociodemographic • 2 CO-level health • 3 Hospital-level service • 2 ZC-level meteorological • Region	• Ozone
[43] *Medicare enrollees*, 2000–2013 • Includes only enrollees from Alabama, Florida, Georgia, Mississippi, North Carolina, South Carolina, and Tennessee • Age: 65–74 yrs, 53.7%; 75–84 yrs, 53.1%; > 84 yrs, 11.7% • Women: 59% • Race: B, 13.1%; W, 84.3%, other, 2.6%	• Age, sex • Medicaid eligibility • 2 Health measures	• 4 ZC-level sociodemographic • 2 CO-level health • 1 ZC-level meteorological • State	
[44] *Medicare enrollees*, 2000–2016 • Only enrollees whose $PM_{2.5}$ exposure levels were < 12 μg/m^3 • Age: 54–74 yrs, 66.8%; 75–85 yrs, 23.9%; > 84 yrs, 9.3% • Women: 53.7% • Race: B, 7.1%; W, 84.8%; other, 8.1%	• Age, sex • Medicaid eligibility	• 8 ZC-level sociodemographic • 2 CO-level health • 7 Hospital-level service and health • 1 ZC-level meteorological • Census division	• Distance to nearest hospital
[46] *Medicare enrollees*, 2000–2008 • Characteristics reported for some decedent categories only • Age, range: 65–120 yrs • Women, decedents: 55.6% • Race, decedents: W, 87.3%; other, 12.7%	• Age, sex	• 1 ZC-level sociodemographic • ZC • Urban/rural	• Ozone
[50] *Medicare enrollees*, 2000–2012 • Age at death: ≤ 69 yrs, 10.38%; 70–74 yrs, 13.37%; 75–84 yrs, 38.48%; ≥ 85 yrs, 37.78% • Women: 55.27% • Race: A, 1.03%; B, 8.87%; H, 1.51%; NA, 0.31%; W, 87.34%	• Age, sex • Medicaid eligibility	• 1 ZC-level sociodemographic • 1 ZC-level meteorological	• Ozone
[45] *National Health Interview Survey*, 1986–2015 • Age mean, range: 43.9 yrs; 18–84 yrs • Women: 52.9% • Race: B, 13.00%; H, 15.37%; W 66.77%; other, 4.86%	• Age, sex • Income • Education • Marital status • 2 Health	• Census region • Urban/rural	• Year
[47] *National Health Interview Survey*, 1997–2011 • Age: 25–35 yrs, 21.3%; 36–45 yrs, 23.5%; 46–55 yrs, 21.6%; 56–65 yrs, 15.1%; 66–75 yrs, 10.1%; > 75 yrs, 8.5% • Women: 52.2% • Race: B, 12.9%; H, 18.4%; W, 64.3%; other, 4.4%	• Age, sex • Income • Education • Marital status	• 1 CO-level sociodemographic • Urban/rural • Climate region	

Table 1 (continued)

Dataset characteristics	Covariates, individual-level	Covariates, area-level	Covariates, other
[51] *North Carolina mortality files*, 2002–2013 • Age at death: < 65 yrs, 24.8%; ≥ 65 yrs, 75.2% • Women: 52.3% • Race: A, 0.4%; B, 20.4%; H, 0.7%; NA, 0.8%; W, 77.7%	• Age, sex • Education • Marital status	• 1 Tract-level sociodemographic • 2 CO-level meteorological • Urban/rural	• Distance to water bodies • Average vegetation
[48] *US Veterans Health Administration*, 2006–2016 • Age, median (IQR): 64.1 yrs (55.7–75.5 yrs) • Women: 6.2% • Race: B, 14.8%; W, 82.0%, other, 3.2%	• Age, sex • 1 Health	• 3 CO-level sociodemographic • 3 CO-level health	

AARP American Association of Retired Persons, *CO* county, *IQR* interquartile range, *NR* not reported, *yrs* years, *ZC* zip code, *A* Asian, *B* Black, *H* Hispanic, *NA* Native American

[a]Percentages are the percent of participants in the age, sex/gender, or race categories listed; they may not sum to 100 due to rounding imprecision

[b]Denotes the number of this type of measure (e.g., 2 individual-level health measures)

Asian, Hispanic, and "other" residents showed no association [51]. Finally, in a third example, a national Medicare study showed that White enrollees showed a weaker positive association between long-term $PM_{2.5}$ exposure and cardiovascular mortality compared to Black enrollees [46]. On the other hand, using the NIH-AARP sample in six states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania), White participants showed a positive association while Black participants showed no association; the confidence intervals around the interaction term suggest that there were no racial differences in this air pollution–mortality association [49]. These disparate results, even among Medicare enrollees, suggest that race has different social meanings in different places.

While there were few consistencies among the empirical results, there were clear patterns in the discussion and treatment of race across studies. First, detailed justification for the study of race and the justification and clear description of the categories used was rarely provided. Many studies provided no justification for the use of race as an effect modifier [45, 48–50] while others simply stated that certain populations might be more vulnerable or susceptible to the mortality impact of air pollution [42–44, 46, 47, 51]. When an explicit justification was provided, it was hypothesized that there might be racial patterns in the pollution–mortality association due to racial patterns in individual-level factors such as comorbidities, access to health care, socioeconomic status, and other unspecified risk factors [43, 47]. No study provided justification for the categorization schema used. In most studies, the authors listed categories that integrated race and Hispanic ethnicity [42, 45–51]. However, some were unclear whether "White" included both Hispanic and non-Hispanic White adults, for example, or which groups were included in an "other" category, or why all non-White adults or all non-Black adults were combined into a single category [43, 44, 48, 50].

Second, discussion of the results of racial patterns were generally unclear and incomplete. When results fit with expected patterns (e.g., Black or other non-White groups showed stronger pollution–mortality associations), then these results were mentioned in the discussion section, generally without any discussion of potential reasons [42]. However, when the results were unexpected (e.g., White adults showed a stronger association, Black adults did not show the strongest association), few provided discussion on the potential reasons [43–45, 49–51]. When unexpected results were discussed, it was not in terms of the social meaning of the racial categories, but in terms of potential missing individual-level covariates [46, 47]. These patterns suggest that the research questions are not based on frameworks that conceptualize race as dynamic with social meaning derived from cultural and structural racism. Without strong frameworks built from scholarship in the humanities and social sciences, apparent paradoxes arise that may elude simple explanations.

Conclusions and Recommendations for Future Research

Considerable effort and resources are spent to improve the accuracy and precision of our measures and modeling of air pollution and health and to train cadres of scholars develop expertise in these areas, but the same cannot be said for the study of race. If we hope to develop an evidence base to inform policy to address exposure and health

Table 2 Summary of results

Study	Outcome(s)	Results by race					
		Asian	Black	Hispanic	Native American	White	Other
Long-term $PM_{2.5}$ exposure and all-cause mortality							
[42]	HR: 11,908,888 deaths 60,925,443 persons	1.096 (1.075, 1.117) $p=0.002$[a]	1.208 (1.199, 1.217) $p<0.001$[a]	1.116 (1.100, 1.133) $p<0.001$[a]	1.100 (1.060, 1.140) $p=0.067$[a]	1.063 (1.060, 1.065) Ref	
[43]	HR: 4,700,000 deaths[c] 13,100,000 persons		~1.025[b] Sig diff[d]			~1.018[b] Ref	~1.061[b,g] Sig diff[d]
[44]	RD: 10,365,012 deaths 40,422,099 persons		0.058% (0.044, 0.073) Sig diff[d]			0.076% (0.073, 0.079) Ref	NR[g]
[45]	HR: 267,204 deaths 1,599,329 persons		1.15 (1.05, 1.27) Not sig diff[e]	1.20 (1.11, 1.30) Not sig diff[e]		1.11 (1.07, 1.15) Ref	1.10[g] (0.94, 1.28) Not sig diff[e]
Long-term $PM_{2.5}$ exposure and non-accidental mortality							
[46]	RR: 15,324,059 deaths 52,954,845 persons	~0.9[b] Sig NR[f]	~1.2[b] Sig diff[d]	~0.8[b] Sig NR[f]		~1.1[b] Ref	
[46]	RR: 15,324,059 deaths 52,954,845 persons					1.064 (1.058, 1.071) Ref	1.041[h] (1.034, 1.049) Sig diff[d]
[47]	HR: 65,936 deaths 657,238 persons		1.11 (0.97, 1.28) Not sig diff[e]	0.97 (0.88, 1.06) Not sig diff[e]		1.05 (1.00, 1.11) Ref	0.89[g] (0.70, 1.13) Not sig diff[e]
[48]	MR: 1,570,798 deaths 4,522,160 persons		55.2 (50.5, 60.6) Ref	48.9 (44.9, 53.4) Sig diff[d]			51.5[i] (46.4, 50.4) Sig diff[d]
Long-term $PM_{2.5}$ exposure and CVD mortality							
[49]	HR: 135,289 deaths 565,477 persons		0.98 (0.73, 1.32) $p=0.63$[a]			1.15 (1.09, 1.21) Ref	
[46]	RR: 6,371,173 deaths 52,954,845 persons	~0.8[b] Sig diff[d]	~1.2[b] Sig diff[d]	~0.8[b] Sig NR[f]		~1.1[b] Ref	
[46]	RR: 6,371,173 deaths 52,954,845 persons					1.109 (1.099, 1.119) Ref	1.054[h] (1.042, 1.066) Sig NR[f]
[47]	HR: 21,152 deaths 657,238 persons		1.08 (0.89, 1.32) Not sig diff[e]	1.07 (0.91, 1.25) Not sig diff[e]		1.21 (1.11, 1.32) Ref	0.81 (0.57, 1.15) Sig NR[f]
Short-term $PM_{2.5}$ exposure and all-cause mortality							
[50]	RR: 22,433,862 deaths					1.01 (0.91, 1.12) Ref	1.27[h] 1.01, 1.53) p=0.07[a]
[51]	RelR: 775,338 deaths	0.21% (−0.56, 0.99) $p=0.047$[a]	1.51% (1.19, 1.63) $p=0.003$[a]	1.16% (0.42, 1.91) $p=0.692$[a]	1.78 (−0.40, 4.01) $p=0.494$[a]	1.01 (0.91, 1.12) Ref	

CVD cardiovascular disease, *HR* hazard ratio, *HS* high school, *NR* not reported, *Not sig diff* no statistically significant difference, *RD* risk difference, *Ref* referent group, *RelR* relative risk, *RR* risk ratio, *Sig diff* statistically significant difference

[a]Reports of statistical significance for race × pollution interaction terms are taken from the source manuscript

[b]When "~" is used, it is because the coefficients were taken from a figure and the precise results were not listed elsewhere in the paper

[c]Precision reported in table is taken from the source paper

[d]Statistically significant difference is reported in the text of the source paper without coefficients

[e]No statistically significant difference is reported in the text of the source paper without coefficients

[f]Statistically significant difference exists for some comparisons but is not clearly reported in the source paper

[g] "Other" is inferred by the authors to include Asian, Hispanic, and Native American, as it is not clearly listed in the source paper

[h]"Other" is inferred by the authors to include Asian, Black, Hispanic, and Native American, as it is not clearly listed in the source paper

[i] "Other" is inferred by the authors to include Asian, White, and Native American, as it is not clearly listed in the source paper

inequities, we need to treat the study of race with the same rigor and intention as we would any of our exposures and health outcomes. We strongly recommend that, at the most fundamental level, we base our empirical tests on strong research frameworks in which the concepts of race and cultural and structural racism are drawn from the humanities and those social sciences drawn from humanist traditions. These disciplines have been studying the complexity of race and cultural and structural racism for many decades and can provide important direction for our measures and models in environmental epidemiology. Public health, and epidemiology in particular, is in its infancy with regard to the theory-informed study of race, meaning that we must look outside of our own discipline to develop these frameworks and empirical models.

Building our empirical models on these interdisciplinary frameworks will also guide our use of covariates in both general studies of race and environmental health and specific studies that examine the modifying role of race in the association between air pollution and health. When an interaction between race and air pollution is modeled, confounders on the effect modification scale are generally not modeled. However, research indicates that race and these potential confounders (e.g., income, education, housing values) may interact to impact health. For example, the robust association between education and morbidity and mortality is stronger for White compared to Black Americans [53], suggesting that White Americans have been more able to translate increases in education into better health compared to Black Americans. These frameworks will also help guide the inclusion of area-level covariates as confounders rather than mediators in the association between air pollution and health.

Use Racial Categories with Purpose When using frameworks based on a rigorous understanding of race and the race variables we generally have available, we can use these variables in innovative ways to better reflect theory. For example, examining social and spatial variation within racial categories may provide clues to the social features that may confer vulnerability to the health impact of air pollution. For example, race has different meaning in relation to health across place that is not simply related to markers of socioeconomic status (SES) [54–56]. Evidence suggests that risk of mortality in poor urban areas is markedly higher for Black residents compared to their counterparts in poor rural areas [57]. It may be that looking at place within race categories will provide insight into the factors that make residents vulnerable to the health impact of air pollution. Indeed, in urban areas in North Carolina, the association between $PM_{2.5}$ exposure and all-cause mortality is strongest among those living in neighborhoods with the lowest median income [51]. Further research may capitalize on the large geographic coverage of many datasets to examine race and place together.

If Race Is Meant to Proxy Structural Racism, Then Model Features of Structural Racism Rather Than or in Addition to Race Models that examine the sociospatial features of our social structure that give race its meaning will provide a stronger evidence base for policy intervention than the continued documentation of race patterns. Even within the narrow topic of $PM_{2.5}$ exposure and adult mortality, the race patterns are inconsistent and defy meaningful interpretation, particularly for policy intervention.

Segregation can be considered a feature of structural racism, a tool to systematically and unequally invest and disinvest in people of different racial groups. Residential segregation is linked to unequal social, economic, and civic investments. Therefore, it may be that for some of the population, segregation, as a feature of structural racism, rather than racial group category, is an important link between levels of air pollution exposure as well as social vulnerability to the health impact of air pollution. In epidemiology studies where segregation is linked to study participants, segregation is broadly conceptualized in two ways, as global (e.g., city, county) and local (e.g., neighborhood). For the former, the research question may be: "Does living in a city in which different races live separate from each other relate to the health of study participants, independent of their race or the characteristics of their own neighborhood?" It may be that cities that perpetuate segregation also do not invest in safety net policies and programs that affect the entire city or that the unequally invest in resources that would maintain social inequities in the city. Using a very crude proxy of city segregation, researchers reported that Medicare enrollees, regardless of race, who live in cities with a greater proportion of Black residents exhibited a stronger association between $PM_{2.5}$ and mortality compared to enrollees who lived in cities with a lower proportion of Black residents [58]. This modifying role of cities is independent of the race of the enrollee or the median household income of their zip code. It may be that cities with high proportions of Black residents experience widespread social, economic, and political disinvestment [59]. Furthermore, outmigration from cities into surrounding communities by White residents may take along with it important social, economic, and political resources.

With regard to neighborhood segregation, the research question may be: "Does living in a neighborhood characterized by a clustering of one racial group from others relate to health?" Segregated Black neighborhoods experience marked disinvestment compared to other neighborhoods, regardless of the socioeconomic status of the residents [60,

61]. Researchers recently examined the modifying roles of two proxies of neighborhood segregation (i.e., racial composition) and a measure of one type of neighborhood segregation (i.e., clustering of racial groups) on the association between short-term $PM_{2.5}$ exposure and mortality in Massachusetts [62]. They reported that the association between $PM_{2.5}$ and mortality was not different between Black and White residents. However, residents living in neighborhoods characterized by a higher proportion of Black neighbors exhibited a stronger $PM_{2.5}$–mortality association compared to those living in other neighborhoods. On the other hand, residents living in neighborhoods characterized by a higher proportion of White neighbors exhibited a weaker $PM_{2.5}$–mortality association compared to those living in other neighborhoods, although the standard error was relatively large for the interaction term.

More research on the modifying role racial residential segregation is recommended, particularly that which is built on a solid theoretical framework of how segregation might modify air pollution–health associations and that which then matches the measure of segregation appropriate to that framework. When examining city-level segregation, it is recommended that researchers articulate the ways in which segregation impacts vulnerability to the health impacts of air pollution and then select both the appropriate measures (e.g., clustering versus isolation) and modeling approach (e.g., health of total population, health of different racial groups, health inequality among racial groups). When examining neighborhood-level segregation, spatial (e.g., Getis–Ord) rather than aspatial (e.g., dissimilarity index) or proxies (e.g., proportion Black) are recommended. Spatial (but not aspatial) measures of segregation take the location of different local areas (e.g., neighborhoods) within the larger area (e.g., city) into account. This means that when the racial composition of a neighborhood changes within the city, the spatial measure of segregation will also change. Spatial measures may better capture the theoretical notions that residents of the same racial group are each clustered together (e.g., Black residents live primarily with other Black residents) and isolated away from other residents of other racial groups (e.g., Black residents live apart from other residents) into different parts of the city. This clustering and isolating by race can then facilitate unequal investment and disinvestment by place—but ultimately by race.

While not yet adapted to environmental epidemiology studies, spatial spillover studies are an innovative way to examine the role of cultural and structural racism on population health. Spillover in this sense is the notion that circumstances that are thought to directly affect some people may have indirect impacts on the larger community. For example, mass imprisonment, as a marker of inequities at multiple points in the criminal justice system, has been shown to be related to the health of the broader Black community, even among those who have not had contact with the criminal justice system themselves [63, 64]. Others have shown that workplace raids by the Immigration and Naturalization Service (INS) was linked to the health of the surrounding Latinx communities, including those residents with documented status [65]. Similar results have been documented with respect to Muslim and Arab American communities after 9/11 [14, 66]. Collectively, these studies suggest that features of cultural and structural racism can impact the broader racialized community. These features, rather than race category, may make racialized groups more vulnerable to the health impacts of air pollution. Built on a strong theoretically informed framework, using these approaches to race, pollution, and health studies have the potential to inform policy intervention.

Leading experts are calling for a movement away from continued documentation of these inequities toward solutions [67]. We propose that this includes a movement away from documenting race patterns to modeling the features of American society thought to confer vulnerability to racialized populations based on theoretically informed interdisciplinary research frameworks. In this review and critical commentary, we examined the recent empirical literature on the modifying role of race in the association between $PM_{2.5}$ exposure and mortality as a case study for the literature on race, air pollution, and health. Collectively, this literature exemplifies the challenges with the study of race without a theoretical foundation built from the humanities. The results are mixed, with some studies reporting stronger, weaker, or similar $PM_{2.5}$–mortality associations for White compared to Black adults. Some studies report inverse $PM_{2.5}$–mortality associations for Asian and Hispanic adults while others report positive associations.

The empirical literature on the modifying role of race, as discussed in this review, does not have clear policy implications. Not only is the literature unclear as to the role of race, but it is unclear as to the intervention point. Building an empirical literature that models the modifying role of features of cultural and structural racism on air pollution and health has clearer policy implications. Research on the interactive and/or joint effects of both social and air pollution exposures can complement the research on inequities in air pollution exposures. This combined evidence base can guide social policies that yield inequities in vulnerability to the health impact of air pollution as well as policies that set pollution threshold standards. As we continue to build an evidence base that can inform both social and environmental policies, using theory-informed, interdisciplinary frameworks that explicate the meaning and role of race, and cultural and structural racism, will help us better clarify where and how to intervene.

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Declarations

Conflict of Interest The authors declare no competing interests.

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