Commentary

Confronting Racism in Environmental Health Sciences: Moving the Science Forward for Eliminating Racial Inequities

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BACKGROUND: The twin pandemics of COVID-19 and systemic racism during 2020 have forced a conversation across many segments of our society, including the environmental health sciences (EHS) research community. We have seen the proliferation of statements of solidarity with the Black Lives Matter movement and commitments to fight racism and health inequities from academia, nonprofit organizations, governmental agencies, and private corporations. Actions must now arise from these promises. As public health and EHS scientists, we must examine the systems that produce and perpetuate inequities in exposure to environmental pollutants and associated health effects.

OBJECTIVES: We outline five recommendations the EHS research community can implement to confront racism and move our science forward for eliminating racial inequities in environmental health.

DISCUSSION: Race is best considered a political label that promotes inequality. Thus, we should be wary of equating race with biology. Further, EHS researchers should seriously consider racism as a plausible explanation of racial disparities in health and consider structural racism as a factor in environmental health risk/impact assessments, as well as multiple explanations for racial differences in environmental exposures and health outcomes. Last, the EHS research community should develop metrics to measure racism and a set of guidelines on the use and interpretation of race and ethnicity within the environmental sciences. Numerous guidelines exist in other disciplines that can serve as models. By taking action on each of these recommendations, we can make significant progress toward eliminating racial disparities. https://doi.org/10.1289/EHP8186

Introduction

Statements of solidarity with the Black Lives Matter movement and against anti-Asian hate and commitments to fight racism and health inequities have proliferated since the summer of 2020. These statements and commitments have included calls from academia, nonprofit organizations, governmental agencies, and private corporations. We have been forced to reckon with the twin pandemics of COVID-19 and systemic racism. The environmental health sciences (EHS) community has joined this movement by pledging to enhance diversity within its ranks (McCarthy 2020), launching new initiatives on environmental health equity, and atoning for its own racist history and past relationship with the eugenics movement (Brune 2020). Significantly, these changes are voiced not only by individuals, but also by professional societies (Casey et al. 2020) and governmental agencies (Lenox 2020).

Of course, actions must arise from these promises. As public health and environmental health scientists, we must contribute by taking a longer, harder look at environmental racism —the systems that produce and perpetuate inequities in exposure to environmental pollutants and associated health effects. The disciplines that contribute to EHS—including epidemiology, biostatistics, exposure science, and toxicology—were developed in the context of racist laws, practices, and policies. Indeed, Francis Galton, the founder of the concepts of statistical correlation, also coined the phrase eugenics and advocated for avoiding racial admixture (Markel 2018). Let us at least accept this fundamental truth: EHS

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institutions and scholarship have not avoided racism's influence. Further, EHS research has often ignored or excluded indigenous or traditional ecological knowledge that emphasizes interconnectivity between physical cultural and spiritual roles and responsibilities within a system (Arquette et al. 2002; Daniel 2019; Kelly 2020). What, then, is required of EHS so that this moment is not wasted and supports the social justice movement? As an initial step, we offer five recommendations for strengthening the science of racism and developing guidelines on the meaning and use of race and ethnicity in environmental health research.

Recommendation #1. Recognize That Race is a Social/ Political Construct, Not a Fixed Biological Trait. Investigate All Potential Causes of Racial Disparities in Environmental Health instead of Assuming They Are Due to Intrinsic Biological Differences

Race is often conceptualized as a fixed biological characteristic in research, but changes in racial classifications over time clearly demonstrate that race is a social construct that reflects political exigencies, not biological differences. For example, Asians and Pacific Islanders were historically construed as one race. However, after 1997, Pacific Islanders lobbied to become classified as a distinct race, a practice that was codified in Directive 15 (Office of Management and Budget 1997), which mandates how federal agencies collect data on race and ethnicity. These categories continue to evolve. For example, ongoing debates (Robbin 2000; Khoshneviss 2019) consider whether people from the Middle East should be considered a separate race (they are currently classified as White). Moreover, Directive 15 considers people from Spanishspeaking countries to be an ethnic group rather than a racial group. Yet, this practice confounds many who consider LatinX peoples to be a race and fail to see the distinction between race and ethnicity. For this reason, many have also argued that LatinX persons should be a racial group (Allen et al. 2011; Telles 2018).

These racial labels are not merely about identity, but reflect and reinforce structural inequalities (Roberts 2012). For example, in *United States v. Bhagat Singh Thind* (1923), the Supreme Court in 1923 intervened to classify people from the country of India as Asian even though they were considered to be Caucasian by many anthropologists at that time. The reason for this reclassification

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was discriminatory because federal policies during that period allowed Caucasians, but not Asians, to seek naturalized citizenship (NRC 2001). As another example, in 1970, Louisiana passed a statute defining someone who is Black as having 1/32 Black ancestry, which means that someone who is 97% White should be classified as Black (Marcus 1983). Assuming that any differences between Black and White individuals are genetic in this context is clearly absurd. The law was originally passed in the 1700s to "keep the [White] landowner from having to share his land with his illegitimate children" who were conceived from rape or extramarital affairs with his Black slaves (Marcus 1983). The 1970 Louisiana law establishing the race formula was repealed in 1983 (Marcus 1983). These examples illustrate how race is a social construct that fundamentally reflects wealth, power, and privilege. For these reasons, commentators in other disciplines have argued against conceptualizing race as a biological construct, for example:

- "The use of biological concepts of race in human genetic research ... is problematic at best and harmful at worst" (Yudell et al. 2016).
- "Attributing differences in biologic endpoints to race is not only imprecise, but also of no proven value in treating an individual patient" (Schwartz 2001).

We would not expect other social labels, such as political party affiliation, to represent an underlying biological trait. For example, if we found that Republicans and Democrats differed in risk for hypertension, we would not assume that these disparities resulted from differences in some cluster of alleles. Rather, we would investigate environmental and social mechanisms, such as environmental exposures, social class, culture, stress, health care access, and so forth. Yet, health differences across races are often assumed to be due to underlying biological differences. The U.S. Food and Drug Administration (FDA) approved the antihypertensive drug Bidil for Black patients, and no other races, based on an underlying assumption that biological differences exist between Blacks and Whites (Temple and Stockbridge 2007; Johnson 2019). It was believed that Blacks did not benefit as much as Whites from angiotensin converting enzyme (ACE) inhibitors and that alternative drugs were needed to provide better care. The role of genetics in explaining the difference remains an untested hypothesis. Hypertension, one of the major risk factors for congestive heart failure, is more common within the Black community (CDC 2010), and chronic social stress has been implicated as a possible contributor to the development of hypertension (Brody and Hunt 2006). Diet, exercise, and other environmental variables are also possible mediators (Brody and Hunt 2006). Later research suggested that the differential effect of ACE inhibitors by race was mostly due to higher sodium intake (Flack et al. 2000).

Assumptions about biological differences in drug effectiveness and metabolism are embedded, sometimes insidiously, within medical practice (Vyas et al. 2020) and have also appeared in toxicological research relevant to EHS. For example, higher levels of cotinine have previously been reported for Black smokers even though they report smoking fewer cigarettes compared with White smokers (Caraballo et al. 1998). Further, although Blacks usually smoke fewer cigarettes and start smoking cigarettes at an older age, they are more likely to die from smoking-related diseases than Whites (CDC 2020). Many investigators hypothesize that racial differences in the metabolism of tobacco toxins may explain differences in tobacco-related morbidity and mortality (Benowitz et al. 1999; Hukkanen et al. 2005). However, in a recent study aimed at evaluating the hypothesis that melanin (skin pigment) affects nicotine disposition kinetics in humans, researchers concluded there was no evidence of correlations between melanin and the pharmacokinetic parameters of nicotine or cotinine or tobacco dependence measures among a group of Black smokers (Liakoni et al. 2019).

Meanwhile, as well documented in records made public from numerous lawsuits, tobacco companies have targeted Black communities with mint-flavored menthol cigarettes for decades (Kaplan 2021). Mentholated cigarettes have been shown to have greater addictive potential than their nonmentholated counterparts, possibly influencing the metabolism of nicotine (Wickham 2020). The banning of menthol cigarettes has long been an elusive goal for public health regulators (Kaplan 2021). The tobacco industry has successfully delayed FDA action on menthol products (Delnevo et al. 2020). Thus, the difference between Black and White smokers appears to be due to the types of cigarettes marketed to Black communities rather than an intrinsic difference in metabolism.

Accordingly, the most dangerous interpretation of race is that of biological differences because such interpretations have been the basis for eugenics and harmful research (Braun 2006; Phelan et al. 2013; Byrd and Ray 2015). Investigators should consider multiple explanations simultaneously and do their best to rule out competing explanations. Importantly, we are not arguing that researchers should stop evaluating differences in exposures and outcomes according to race. Rather, we should first document whether racial differences exist and then investigate specific factors that might cause such differences. In short, researchers should define, measure, and investigate race as rigorously as they would any other exposure or outcome and acknowledge its limitations as a proxy measure of the underlying construct or mechanism(s) it is meant to represent (Nelson et al. 2018).

Recommendation #2. Seriously Consider Racism as a Plausible Explanation of Racial Disparities in Health

Advances in EHS have revolved around the deployment of observational, experimental, computational, and clinical approaches to more fully uncover the biological mechanisms hypothesized by the environmental public health paradigm (Sexton et al. 1992; U.S. EPA 2021). But as EHS researchers aim to discover and explain how factors—including chemical, physical, synthetic, and infectious agents, as well as social stressors—affect biological systems, it is not enough to just describe social disparities in exposures and health outcomes. We need to also investigate social mechanisms (Pellow 2000; Powell 2008) as plausible explanations of environmental exposures and illnesses. A social mechanism that is particularly relevant for the study of race disparities is racism and racial discrimination (Gee and Payne-Sturges 2004).

In the United States, racism is the social system that provides the basis of allocating power and privilege to Whites and disadvantages to racial/ethnic minorities (Jones 2000; Williams et al. 2019). Racism fundamentally alters one's circumstances across the life course, such as where one lives and their educational and occupational opportunities (Gee and Ford 2011; Gee et al. 2019). Racism can confer stress, restrict opportunities, and contribute to adverse exposures (Morello-Frosch 2002; Lewis et al. 2015). Racism has occurred throughout history but, importantly, persists today (Ford and Airhihenbuwa 2010; Lewis and Van Dyke 2018). Because racism is such a powerful driver of social circumstances, it has been considered a fundamental cause of illness by numerous scientific bodies including the Institute of Medicine (Institute of Medicine Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care 2003), the American Medical Association (AMA 2020), and the American Public Health Association (Benjamin 2020). As several meta-analyses have shown, when individuals experience racial discrimination, they are more likely to have a variety of health problems (Pascoe and Smart Richman 2009; Paradies et al. 2015).

Racial discrimination, the behavioral manifestation of racism, has been defined as having two components (NRC 2004). The first

is differential treatment based on race. This is how people often think of discrimination, that is, that racial minorities are treated poorly due to explicit racial animus. However, the second way of thinking of discrimination is based on the concept of disparate impact, which is treatment on the basis of inadequately justified factors other than race that harms a racial group. In other words, a practice can be discriminatory if it creates a racial disparity and if the practice does not have any compelling reason to exist. No explicit racial animus is necessary in the second definition. This idea was the basis for *Griggs v. Duke Power Co.* (1971), a suit levied against an employer who required employees to take aptitude tests. On the surface, such tests appeared race neutral because they were required of both Black and White employees. However, these tests had two qualities that made them discriminatory under the idea of disparate impact. The tests a) disadvantaged Black employees owing to their lower educational levels and, just as importantly, b) had no bearing on the job duties. Thus, the Supreme Court ruled that the tests were discriminatory against Black workers.

Rigorous science requires that we seriously consider racism and discrimination as plausible explanations for racial/ethnic inequities in environmental exposures and racial/ethnic differences in the impacts of such exposures (Mohai and Bryant 1992; Morello-Frosch et al. 2002; Morello-Frosch 2002; Gee and Payne-Sturges 2004). Such consideration includes studying how racism

- Accounts for the disproportionately higher rates of exposure among racial/ethnic minorities (Clark et al. 2017; Ash and Boyce 2018; Mikati et al. 2018; Tessum et al. 2019)
- Leads to preexisting health conditions (e.g., asthma) that render people more susceptible to environmental exposures (e.g., air pollution) (Guarnieri and Balmes 2014)
- Amplifies the effects of environmental exposures [e.g., effect modification between stress and lead poisoning on high blood pressure, effect modification by socioeconomic status (SES) of lead effects on intelligence quotient] (Bellinger et al. 1988; Hicken et al. 2013)
- Impairs one's ability to obtain effective medical care (Gonzales et al. 2014; Kugelmass 2016)
- Creates spillover effects that impair the well-being not simply of an individual but of their families and communities (e.g., when racism puts Black fathers in prison) (Priest et al. 2013; Lee et al. 2015; Williams et al. 2019)
- Impedes the ability to conduct rigorous scientific research (e.g., when researchers stereotype participants, when mistrust rooted in the Tuskegee Study diminishes participation in research) (Corbie-Smith et al. 2002; Ford and Airhihenbuwa 2010)
- Affects the reporting, interpretation, publication, and funding of racial disparities research (Tyer 2005; Ginther et al. 2011; Kubota 2020).

Thus, as EHS researchers, we need to start asking different questions (Payne-Sturges 2011) that can address the complex interactions between conditions, policies, and social, natural, and built environmental systems that result in unequal environmental health conditions or disproportionate impacts among (diverse) disadvantaged population groups, communities, neighborhoods, and individuals. For example, how does environmental inequality arise and why does it persist? What is the role of institutionalized racism and the economics of industrial development and production (i.e., industrial location, racialized division of labor, suburbanization, and economic restructuring)? Given the important role of toxicology in EHS, how can animal models be reformulated to be more directly relevant to the environmental context of human chemical exposures? Ultimately, what new strategies can be developed for alleviating systemic drivers of racial and socioeconomic disparities in environmentally mediated health outcomes and access to healthy environments?

In order to address these questions, we will also need to consider how best to build interdisciplinary research teams and to integrate methods and theories from systems and social sciences with EHS and corresponding training programs. The point is not to throw away the conventional research questions (e.g., what is the exposure—disease response relationship on average?), but to investigate new ones too, especially if we are concerned about disproportionate impacts and environmental health inequities.

Recommendation #3. Develop New Measures of Racism

Within EHS research, discrimination has been operationalized in two main ways. First, discrimination has been inferred when a racial disparity remains after accounting for compelling covariates (e.g., socioeconomic conditions, land use ordinances, zoning policies). The assumption that the residual disparity represents the effects of racism relies on the assumption that key confounders have been accounted for and that racism is the true driver of the disparity (Bullard 1993). Second, racial residential segregation, which represents the cumulative impact of historic and contemporary racist practices that spatially separate races, is used as a measure of discrimination. Practices that contribute to residential segregation range from outright racial hostility (e.g., lynchings) to more subtle practices, such as redlining by banks or steering by real estate agents. Segregation has been linked to a variety of environmental exposures and poor outcomes (Morello-Frosch and Jesdale 2006; Mehra et al. 2017).

However, we need to think beyond these metrics. Research in other disciplines has documented racial inequities in police shootings, mass incarceration, educational tracking, media portrayals, citation practices, hospital case-management algorithms, emergency department waiting times, National Institutes of Health funding, and innumerable other segments of life (Krieger 1999; Gee and Ford 2011; Ginther et al. 2011; Bailey et al. 2017). In addition, some measures have been developed to focus on personal experiences of discrimination (Williams et al. 1997; Krieger et al. 2005; Landrine et al. 2006). This suggests that many aspects of environmental racism are yet to be documented, conceptualized, and measured.

As the example of the lead poisoning crisis in Flint, Michigan, showed us, there are numerous points along the continuum where inequality occurs, ranging from governmental policies, to the manipulation of data, to remediation and restoration (Michigan Civil Rights Commission 2017). Racism occurs partly due to explicit racial animus, but it also manifests as willful neglect and purposeful prioritization of advantaged communities. Although it has been recognized that the Flint crisis was due to structural racism (Michigan Civil Rights Commission 2017; Hammer 2019), there are yet no good metrics for detecting such discrimination.

Therefore, we call upon researchers to develop new measures of racism and racial inequities that may be particularly relevant to EHS. This may necessitate working in transdisciplinary teams that include scholars from fields such as sociology, history, and ethnic studies. This requires working with members of racial minority communities, who often are able to see the manifestations of racial bias long before the ideas diffuse into the academic literature (Israel et al. 2005, 2010). These measures then need to be incorporated into surveys such as the National Health and Nutrition Examination Survey or must permit linkages with such surveys and other data sets so that race and racism can be rigorously studied.

Recommendation #4. Consider Structural Racism as a Factor in Environmental Health Risk/Impact Assessment

Risk assessment plays an important role in EHS because it shapes environmental health policy decisions at local and national levels. Although the majority of EHS researchers are not directly involved in risk assessment, their work (including basic research on chemical properties; estimating fate, transport models, and pollutant exposure models; toxicity testing on animal models; and epidemiologic studies of human populations) feeds directly into risk assessment. However, unfortunately, evidence shows that the system of environmental health protection based on risk assessment does not work well for the people who need it the most (Israel 1994).

Since the mid-1970s, quantitative risk assessment—a method to identify and measure the risk that a particular environmental contaminant presents at a given exposure level—has been critical to many federal environmental regulatory and policy decisions. Risk assessment consisting of a four-step process (hazard identification, dose-response assessment, exposure assessment, and risk characterization) is a reductionist approach used mainly to assess and regulate individual chemicals. Risk assessment has guided the development of ostensibly race-neutral environmental policies. The limitations of chemical-by-chemical risk assessment to address real-world exposures have been acknowledged but seldom taken seriously (Israel 1994). Risk assessment typically omits multiple chemical exposures, cumulative and synergistic effects, and consequences of co-occurring nonchemical stressors and their potential downstream convergence that can lead to the enhancement of biological effects.

Yet the human environment includes multiple risk factors in addition to a multiplicity of chemicals acting concurrently or sequentially. This fact has direct relevance to both epidemiological and basic science studies, where the focus is predominantly on single chemical exposures and usually at the mean or median level. Given that chemical exposures and other environmental risk factors can operate on the same biological substrates, they open the door for multiple interactive effects with the potential to modify the toxicity of chemicals.

Currently, risk assessments conducted by the U.S. Environmental Protection Agency generally limit consideration of susceptible populations to the elderly, children, pregnant women, and perhaps people with comorbidities, without considering the broader socialenvironmental context. Epidemiologic studies ignore interactions between chemical and nonchemical exposures (Wing 1994) or suffer from an absence of methods allowing such assessments when sample sizes are modest. Animal models that inform risk assessments are significantly removed from human relevance in their assessment of the impacts of single chemical exposure effects in the absence of any such human relevant contexts; although stress is sometimes examined, the relevance of stress paradigms typically used in animal models (e.g., restraint stress, forced swim, social isolation) needs serious reconfiguration so as to relate to the types and magnitude of stressors associated with poverty, including lower income, SES, neighborhood poverty, and social and neighborhood resource deprivation, factors that are above and beyond those experienced in higher economic strata (Evans and Kim 2013; Perry et al. 2019). Similarly, epidemiologic studies need to query stress exposures relevant to the context of the populations being studied.

More relevant paradigms might be considered for both animal models and human epidemiologic studies. One possible avenue would be through stress paradigms contextually related to inequity aversion, that is, receipt of unequal reward for the same task, a phenomenon seen even in children as well as in other mammalian species. In human studies, such stress has also been shown to modify decision making, including enhancing the tendency to choose smaller and earlier rewards over larger delayed ones (a phenomenon known as delay discounting) (Haushofer and Fehr 2014). Because animal models are the basis for much of the research used for risk assessment and because disadvantaged human populations often experience the highest chemical

exposures, it is imperative to develop animal models of social disparity and acknowledge the potential for cumulative effects.

Further, the risk assessment framework, which is predicated on the environmental public health paradigm, does not examine the upstream economic and social forces that create disparities in exposure to environmental chemicals in the first place and contribute to enhanced toxicity. Because of this, environmental policies are likely based on underestimations of true health risks.

Moreover, the interpretation of risk varies considerably by demographic group. Many studies have documented a "White male effect" whereby White men are more likely to downplay risks associated with a variety of hazards (e.g., air pollution, climate change, cigarette smoking) compared with women and people of color (Finucane et al. 2000; Marshall et al. 2006; Sansani 2018). Therefore, if White men dominate decision making about risks that disproportionately affect people of color (Marshall 2006), risk assessment can help reinforce structural racism, exacerbate racial inequalities, and perpetuate race-related differences in environmental exposures and their effects. In this way, risk assessment can serve as a tool of White supremacy, which is, at its most fundamental level, about the exercise of power to privilege Whites and disenfranchise racial minorities (Jones 2000; Bailey et al. 2017; Gee et al. 2019; Walsdorf et al. 2020). With these limitations taken together, it comes as no surprise that we continue to have race-based differences in environmental exposures and health effects.

The EHS field needs to devise new ways to assess the cumulative health toll/burden of pollution by focusing on structural causes (racialized policies, practices, and decisions) of disparities, incorporating concerns of impacted communities, and promoting policies that not only address these structural causes but also move society toward a more regenerative environment, especially among communities that are already overburdened (O'Brien 2000; Cousins et al. 2019; Sengupta 2020). Health Impact Assessment (Yuen and Payne-Sturges 2013) and system dynamics (Homer and Hirsch 2006; Hovmand 2014) are promising approaches for overcoming key limitations of risk assessment in this regard. These approaches facilitate the explicit consideration of systems and their structures that create health inequities and the integration of quantitative and qualitative evidence from across multiple disciplines and sources of knowledge to inform development of more equitable public health policies.

Recommendation #5. Develop Guidelines on the Use of Race and Ethnicity within EHS

The previous recommendations were about furthering the development of the science of racism within the EHS context; this recommendation focuses on the use of race and ethnicity within EHS research. Unfortunately, race is notoriously poorly measured. One study of 995 medical articles found that race was defined in only 4.5% of the studies and the method for racial classification mentioned in only 10.3% (Bokor-Billmann et al. 2020). We would not accept such lax reporting for any environmental exposure, nor should we accept it when researching disparities. We should demand rigor in the study of race and ethnicity. Many disciplines have thus developed guidelines to clarify the meaning and use of race and ethnicity in research and publications, including the following:

- APA Guidelines on Race and Ethnicity in Psychology (APA Task Force on Race and Ethnicity Guidelines in Psychology 2019)
- American Anthropological Association statement on race (Executive Board of the American Anthropological Association 1998)

- Race, Ethnicity, and Language Data: Standardization for Health Care Quality Improvement (Institute of Medicine Subcommittee on Standardized Collection of Race/Ethnicity Data for Healthcare Quality Improvement 2009)
- "The impact of racism on child and adolescent health" (Trent et al. 2019)
- "On racism: a new standard for publishing on racial health inequities" (Boyd et al. 2020)

Environmental health scientists should follow suit and develop our own set of guidelines in collaboration with community members, policy makers, social scientists, and representatives from all of the other disciplines that contribute to EHS research. Professional societies, including the Society of Toxicology, the International Society for Environmental Epidemiology (ISEE), and the International Society of Exposure Science could also further this effort. For example, ISEE recently established an antiracism task force whose goals include promoting consideration of structural racism as a mechanism for inequities in environmental health and encouraging more rigorous use and interpretation of race in environmental health research (D. Payne-Sturges and M. Hicken, personal communication). Such guidelines would call upon the researchers to

- Provide the scientific rationale for examining race as an exposure, confounder, or modifier
- Describe how race was measured and operationalized
- Integrate the role of racism and its various components (e.g., discrimination, prejudice) into research, and in doing so, to consider how racism operates at multiple levels (Bailey et al. 2017; Williams et al. 2019) and across the life course (Gee et al. 2012, 2019).

Summary

As environmental health researchers, we are accustomed to making the invisible visible, be it the health impacts of tiny particles or epigenetic changes from exposures to chemicals. If we are serious about ending environmental racism, we must also direct our attention to making more visible the social mechanisms and systems that create racialized disparities in environmental health and how they influence the toxicity of chemical exposures. This means looking farther upstream and asking different research questions related to disproportionate environmental health impacts of practices such as redlining, predatory lending, and the siting of polluting industries, as well as so-called race-neutral policies and lax environmental enforcement. We should also work to increase collaboration with social scientists, be explicit about our reasons for using race in research, measure and classify race in a precise and rigorous way, and increase community-based participatory research. Our recommendations are not meant to be exhaustive but, rather, a starting point for future discussion and research.

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