

Race and causality in health disparities research: time for a necessary paradigm shift

Emma K. T. Benn, DrPH, MPH (she/her)

Associate Professor, Center for Biostatistics & Department of Population Health Science & Policy

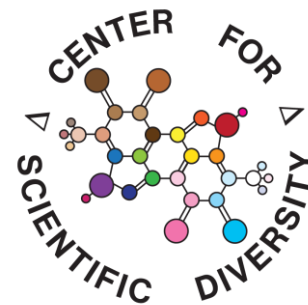
Founding Director, Center for Scientific Diversity

Associate Dean for Faculty Wellbeing and Development

Icahn School of Medicine at Mount Sinai

emma.benn@mountsinai.org

Twitter: @EKTBenn



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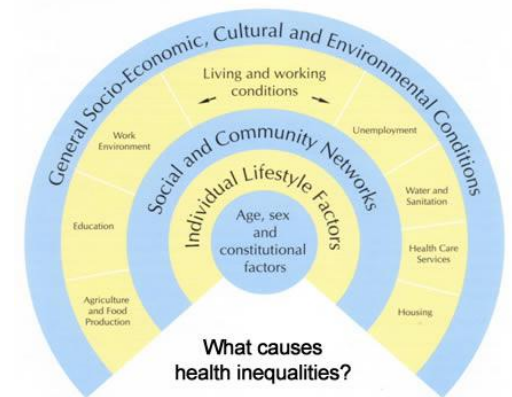
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I have no conflicts of interest to disclose.

Overview

- ▶ A little about me
- ▶ Motivating Example
- ▶ Define “Circular” Slump in Racial/Ethnic Disparities Research
- ▶ Brief Introduction to Causal Inference
- ▶ Important Considerations for Health Disparities Research

A little about me



Passion for research pertaining to racial/ethnic minorities and health disparities research continued...but hit with a philosophical dilemma

- ▶ Started questioning why we always start and end with race
 - We observe a racial/ethnic difference.
 - After adjustment for numerous covariates, difference remains. Further research needed.
- ▶ Describe racial/ethnic differences often but identification of effective targets for intervention less frequent.
- ▶ Searched the literature for guidance from statisticians and non-statisticians about how we should be more optimally operationalizing race.
 - Needed to fundamentally change my approach (unlearn the approach I had been taught) as it relates to examining race and health in research.

Motivating Example

A researcher sets out to estimate a measure of **the strength of the relationship between race and hypertension**. She considers factors (e.g. diet, educational attainment, income, access to healthcare, etc.) **associated with both race and hypertension** that might hinder her effort to get an **unbiased “effect” of race**.

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The researcher subsequently constructs her statistical model (accounting for diet, educational attainment, income, access to healthcare, etc.) and gets some **adjusted estimate of the effect of race on hypertension**.

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**FOCUS OF THIS
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BUT WHY NOT???

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- ▶ Next we power our study to detect a clinically meaningful difference (Δ), given our hypotheses.
- ▶ We sample from the population, collect data, conduct an appropriate analysis and conclude:
 - **We have sufficient evidence to suggest that the risk of disease differs between Blacks and Whites with magnitude $\geq \Delta$.**

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 - “In practice, however, **we observe that population distributions of disease vary on the basis of skin color.** If we pick a physical characteristic other than race at random, we cannot replicate the same degree of variation in disease occurrence. Blood-type, for example, is easily measured and could provide an alternate classification scheme, but there are few population differences in the distribution of common diseases along this axis. **To believe that skin color has a unique association to outcomes ranging from IQ to blood pressure to prostate cancer by sheer chance is a questionable, if not preposterous, proposition.**”

Kaufman JS, Cooper RS. In search of the hypothesis. *Public Health Reports*. 1995;110(6):662-666.

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- ▶ We are often stuck in this “**circular**” **slump** that gets us no closer to fixing the problem.
 - We hypothesize there are racial differences in the distribution of disease.
 - We reject H_0 , even after accounting for supposed confounders.
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- ▶ Templeton (2013) supports Kaufman & Cooper in their efforts to move us away from irrational circular arguments:
 - “Humans are an amazingly diverse species, **but this diversity is not due to a finite number of subtypes or races.** Rather, the **vast majority of human genetic diversity reflects local adaptations** and, most of all, our **individual uniqueness.**”

Templeton, A. R. (2013). Biological Races in Humans. *Studies in History and Philosophy of Biological and Biomedical Sciences*, 44(3), 262–271.

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 - **Experiences individuals undergo**, not attributes they possess.
 - Causal variables must reflect the possibility of **manipulation**.
 - While race is not a causal variable, it can play a crucial role in causal studies for a few reasons:
 - For **descriptive** reasons
 - “In my opinion, RACE can play an important descriptive role in identifying important societal differences such as those in wealth, education, and health care. **The attribution of cause to RACE as the producer of these differences is, to me, the most casual of causal talk** and does not lead to useful action.”
 - Understanding **whether an intervention works differently** across racial/ethnic groups
 - Attempting to delve deeper into the effects of important constructs like discrimination and bias

The “Circular” Slump of Racial/ethnic Disparities Research

Ascribing a causal role to race does not get us closer to change.

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- ▶ In the era of **#BlackLivesMatter**, if we can scrutinize the treatment of different groups in the US...
- ▶ As statisticians, mathematicians, data scientists, and clinical investigators, we must scrutinize the treatment of these groups in research too.

Brief Introduction to Causal Inference

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- ▶ But, an **association is only a statistical concept**.
 - *Let $E = \text{Exposure}$ and $D = \text{Disease}$.*
 - The statistical relationship will **look the same whether $E \rightarrow D$ or $D \rightarrow E$** .
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 - Also **if E and D, have the same underlying cause**, we will **still observe an association between E and D**.
- ▶ **Causation**, however, is not a statistical measure, but **stems from a rigorous underlying theory about the relationship between E and D**.

Brief Introduction to Causal Inference

- ▶ To test whether E causes D...
 - If we assume that E and D are both binary, then in theory:
 - We can conceive of a world where **every individual has a potential outcome D_E and $D_{E'}$** : outcome in the presence and absence of the exposure, respectively.
 - Thus the individual causal effect, **$CE_i = D_{E,i} - D_{E',i}$** .

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 - Thus the individual causal effect, $CE_i = D_{E,i} - D_{E',i}$.
 - Yet, in reality, we can only observe **D_E or $D_{E'}$** .
 - Thus, **we make strong assumptions** about the comparability of the exposed and unexposed groups, **so the unexposed are the counterfactual to the exposed**.

Brief Introduction to Causal Inference

- ▶ Given this fundamental problem, Holland (1985) suggests we are left with two solutions:
 - **Scientific Solution**
 - Assume that the response after exposure to the “control” at an earlier time for a given individual is the same as the response after exposure to the “control” for the same individual in the current experiment.
 - Only needs to expose the same individual to the treatment in the current experiment.
 - Underlying conditions may not hold true over time.
 - Difficult to translate to real-world of uncontrolled conditions.
 - **Statistical Solution**
 - Individual → Population
 - Estimate the average causal effect of the treatment/cause over a population
 - Contains individuals who are exposed to the treatment and individuals who are exposed to the control
 - Still requires good study design and that the exposed and unexposed are actually comparable

Holland, P. W. (1986). Statistics and causal inference. *Journal of the American statistical Association*, 81(396), 945-960.

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 - If our goal is **to reduce disease** instead of just describing differences, then...
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 - Causes, confounders, mediators, independent predictors, or effect modifiers???
 - **Reduce bias** due to unmeasured confounding.
 - For example, relationship between education and health may be confounded by air quality, Pb exposure, crime, collective efficacy, access to healthy food, etc.
 - **Impossible to measure everything w/ limited resources.**
 - Instead, study E-D relationship in subpopulations **homogenous with respect to a specific contextual construct.**

COVID-19 yields increased calls for naming and operationalizing constructs in disparities research

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Weber, E., Miller, S. J., Astha, V., Janevic, T., & Benn, E. (2020). Characteristics of telehealth users in NYC for COVID-related care during the coronavirus pandemic. *Journal of the American Medical Informatics Association*, 27(12), 1949-1954.

Nancy Krieger, 2020: ENOUGH: COVID-19, Structural Racism, Police Brutality, Plutocracy, Climate Change—and Time for Health Justice, Democratic Governance, and an Equitable, Sustainable Future. *American Journal of Public Health* 110, 1620-1623.

Hooper, M. W., Nápoles, A. M., & Pérez-Stable, E. J. (2020). COVID-19 and racial/ethnic disparities. *JAMA*, 323(24), 2466-2467.

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Almagro, M., & Orane-Hutchinson, A. (2020). JUE insight: The determinants of the differential exposure to COVID-19 in New York city and their evolution over time. *Journal of Urban Economics*, 103293.

Summary

- ▶ If we are to move from describing racial differences to identifying mutable targets for intervention, **then race CANNOT be our end point.**
- ▶ Difficult paradigm shift but necessary.
- ▶ Baby steps can go a long way.
 - **Real World Example: Black-White differences observed in 6-months postpartum depression treatment (prescription medication) acceptability**
 - A priori hypothesis that stigma was the culprit did not stand.
 - Medicaid and Black race walked hand in hand.
 - **“For example, the vast majority of women in our sample on Medicaid were black and some of our findings related to race may be more reflective of insurance status.”**

Bodnar-Deren S, Benn EKT, Balbierz A, Howell EA. Stigma and postpartum depression treatment acceptability among black and white women in the first six-months postpartum. *Matern Child Health J.* 2017; doi:10.1007/s10995-017-2263-6.

Race, Genetic Ancestry, and Clinical Algorithms

MEDICINE AND SOCIETY

Embracing Genetic Diversity to Improve Black Health

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Article

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40 References

Editors

Debra Malina, Ph.D., Editor

AS RESEARCHERS WHOSE WORK IS LARGELY FOCUSED ON GENETICS and who self-identify as Black men, arguably one of the most disadvantaged groups in the United States, we have had similar formative experiences during our training and careers. We have all dealt with aggressions and microaggressions, isolation, imposter syndrome, the Pygmalion effect or stereotype threat, gaslighting, and a lack of mentorship, especially Black mentorship. We have made our way in a field that has an alarming dearth of leaders and research participants of African descent — a common story among Black professionals in science, technology, engineering, and mathematics (STEM) fields.¹

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We do not believe that ignoring race will reduce health disparities; such an approach is a form of naive “color blindness” that is more likely to perpetuate and potentially exacerbate disparities. Although ignoring race could improve *equality* (by leading to identical medical treatment for everyone), we believe that *equity* is necessary to address disparities. We urge our colleagues in medicine and science to refrain from haphazardly removing race from clinical algorithms and treatment guidelines in response to recent initiatives attempting to combat anti-Black racism. The ultimate goal, we believe, would be to replace race with genetic ancestry in an evidence-based manner. But we have not yet reached a point where genetic-ancestry data are readily available in routine care or where clinicians know what to do with these data. Until we do, ignoring race and thereby reverting to the United States’ outdated system of health care, in which clinical research findings are generated in populations of European descent and extrapolated to the treatment of non-European populations, is neither equitable nor safe for those other populations.

QUESTIONS?