http://www.jhpr.ir
Hosp Pract Res. 2022 Sep;7(3):90-96
doi 10.34172/hpr.2022.19
HPR
Hospital Practices and Research
Original Article

Blacks' Diminished Salience of Age as a Determinant of Chronic Obstructive Respiratory Disease

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Received April 8, 2022; **Accepted** July 28, 2022; **Online Published** August 12, 2022

Abstract

Background: Age is a major determinant of chronic respiratory disease (CRD). This is important because CRD have a main role in shaping morbidity and mortality of individuals and populations. However, less research is done on whether age-related changes in development of CRD differ across diverse racial groups.

Objectives: Using a conceptual model that considers race as a proxy of racism rather than genetics and attributing racial differences to sociological rather than biological differences, this study was conducted to explore racial differences in the effects of age on CRD. Based on Marginalization-related Diminished Returns (MDRs) framework, we expect diminished relevance of risk and resources for marginalized people due to racism, segregation, and social stratification.

Methods: Using data from baseline population assessment of tobacco and health (PATH) Adults data, we included 23761 adults. The independent variable was age treated as a categorical variable. The primary outcome was presence of any CRD including asthma, bronchitis, emphysema, and chronic obstructive pulmonary disease (COPD). Sex and education were the covariates. Race, as a proxy of racism, was the moderator. To analyze the data, we used logistic regression mode with and without interaction term between age and race.

Results: Higher age was associated with higher odds of CRD, while sex, and socioeconomic status (SES) was controlled. In line with the MDRs framework, the positive association between age and CRD was weaker for Black than White adults.

Conclusion: Under racism, age loses some of its effect as a major determinant of CRD across racialized groups.

Keywords: Health, Chronic Disease, Racism, Population Groups, Asthma, Respiratory Disease

1. Background

Our previous work on Marginalization-related Diminished Returns (MDRs)[1-3] suggests that due to racism, social stratification, segregation, and discrimination, resources and assets tend to show weaker effects on health and development of racialized and marginalized groups. Link attributes these observed MDRs to structural racism in the US society.[4] Recent work on the Adolescents Brain and Cognitive Development (ABCD) data has shown that MDRs at the level of human development may explain MDRs for health.[5-11] However, most of this literature is on MDRs of socioeconomic status (SES) rather than non-economic resources and assets such as age.[7,12-15] More recently, research has used national US data showing MDRs (diminished salience) of age as a determinant of health and behavior of Black populations, compared to White populations.[7,12-15]

According to MDRs, any deviation from social privilege and Whiteness (which is wrongfully considered a norm through history) is accompanied with a penalty which can be measured as diminished return of resources and assets. Thus, any visible or invisible marginalizing social identity will be associated with reduced health effects of resources and assets. It is proposed that discrimination, blocked opportunities, differential treatment, decreased access to safety nets and support systems in society, and other forms of marginalization generate MDRs in marginalized communities and individuals. MDRs are replicated regardless of type of social marginalization as they are shown for race,[16,17] ethnicity,[18-20] nativity,[21,22] sexual orientation,[23-25] and place.[26]

Although, Hispanic,[18-20] Native American,[27] Asian American,[28] immigrant,[21,22] lesbian gay bisexual (LGB),[23-25] and marginalized White[26] populations also show weaker than expected effects of resources and assets, these are most known for Black populations. In addition, while almost all resources such as SES,[21,22] coping,[29] and age[13] show these patterns, they are most frequently described for SES resources. Black populations were brought to the US by slavery 400 years ago and since then have suffered various forms of social and economic adversities. As similar MDRs are shown for all marginalized groups (including Whites),

we have concluded that social forces rather than biological traits explain MDRs.[1,2] However, due to unique aspects of anti-Black racism, slavery, and Jim Crow, MDRs are strongest in Black communities who have had a unique history.[1,2]

Due to MDRs, Black populations show poorer health, behavioral, and development, regardless of their economic and non-economic resources and assets.[30-34] For example, high SES Black people remain at risk of anxiety,[35] depression,[36] and suicide.[37-39] High SES Black youth show worse than expected school performance[16,17] and higher than expected high-risk behaviors such as smoking and externalizing behaviors.[40,41] As most of these observed diminished effects are shown for economic resources such as SES,[42-46] there is a need to replicate these MDRs for non-economic resources such as age. This can be hypothesized because high SES Black children and adults report higher-than expected risk of chronic conditions such as asthma, hypertension, obesity, heart disease, psychiatric conditions such as anxiety and depression, and neurological conditions such as attention-deficit/hyperactivity disorder (ADHD).[18,32,46,47]

Although there is an extensive body of research on weaker effects of family SES for racial minority groups, we are unaware of any previous studies that has compared racial differences in the effect of age on chronic respiratory disease (CRD). Therefore, we need to shift from studying MDRs of SES on a wide range of health, economic, and behavioral, outcomes, to studying racial variation in the effect of age on CRD.

MDRs are related to sociology of race(ism) rather than biology of race.[48-52] Due to racism, high SES Black communities remain at risk of depression, poor emotion regulation, and CRD. These can be due to environmental stressors that Black communities are exposed to across all SES spectrums. In this view, environmental hazards may confound, dilute, or change the effect of age on health. In a similar manner, we can expect racism-related diminished relevance of age as a determinant of CRD in Black communities. Again, this is because people would develop CRD regardless of their age in Black communities, which is due to racialization not race.[48-52]

2. Objectives

Built on MDRs, this study compared the salience of age as a determinant of CRD between Black and White individuals. MDRs framework conceptualizes race as a proxy of racism and marginalization rather than biology of group differences. Built on MDRs, we expected larger salience of age as a determinant of CRD for White than Black US adults. We expected positive effect of age on CRD overall, however, we expected racial variation in the magnitude of this effect. We expected weaker effect of age on CRD for Black communities, which is in line with the MDRs phenomenon.[1,2,34] Diminished age-related changes in CRD for Black adults would mean that other social factors, rather than biology of aging, make a larger contribution for Black than White people.

3. Methods

3.1. Design and Settings

This was a cross-sectional study. For this study, we conducted a secondary analysis of existing data. Data came from the baseline population assessment of tobacco and health (PATH) study (adults). Our analysis only used baseline (wave 1) of the PATH data. PATH is a national substance use survey of adults with a national sample. The PATH adults study has included a large and diverse national sample.

3.2. Analytical Sample

For this study, we only included non-Latino White or Black adults. Thus, any individual with Latino ethnic background or any other racial identification such as Asian, mixed, other, or unknown were excluded.

3.3. Study Variables

Dependent Variable. Our outcome variable was CRD. Participants were asked if they were told that they have our CRDs of interest namely asthma, bronchitis, emphysema, and chronic obstructive pulmonary disease (COPD). Prior studies have shown that self-reported CRD are valid and reliable.[53-59]

Moderator. Race was the moderator in this study. Race was self-identified by participants. Race was coded as 1 for Blacks and 0 for Whites (reference group).

Confounders. Sex was 1 for males and 0 for females. Education was a categorical measure ranging from 1 to 6.

Inependent Variable. Age was was a categorical variable with 10 categories.

3.4. Data Analysis

To analyze data, we used Statistical Package for the Social Sciences (SPSS). For univariate, we reported n (%) and mean [standard deviations (SDs). For bivariate analysis, we used Spearman correlations. We then used four logistic regression models for our multivariable analysis. In all regression models, the outcome variable was CRD, the main predictor (independent) was age. All models controlled for confounders such as sex and education. We used pooled sample for Model 1 and Model 2. No interaction term was included in *Model 1*. For *Model 2*, we included an interaction term between age and race (age X race). We fitted the next two models in stratified samples. This included Whites (Model 3) and Blacks (Model 4). We reported unstandardized regression coefficient (b), SE, and *P* values. We considered p-value less than or of equal to 0.05 as statistically significant.

4. Results

Table 1 presents the descriptive information in the pooled sample and by race. 23761 adults entered this analysis from whom 19277 (81.1%) were White and 4484 (18.9%) were Black.

Table 1. Descriptive data

	Mean	Standard Deviation
Age (1-7)	3.0980	1.77163
Education	3.6302	1.33188
	N	%
Gender		
Female	11824	49.8
Male	11937	50.2
Age		
1.00	6038	25.4
2.00	4532	19.1
3.00	3570	15.0
4.00	3757	15.8
5.00	3235	13.6
6.00	1806	7.6
7.00	823	3.5
Education		
1.00	2463	10.4
2.00	1607	6.8
3.00	5513	23.2
4.00	8613	36.2
5.00	3597	15.1
6.00	1882	7.9
Respiratory Condition		
No	19607	82.5
Yes	4154	17.5

Table 2. The Summary of Regression Model in the Pooled Sample

	B	SE	*P* Value	Exp(B)	95% CI for EXP(B)	
Model 1						
Race (Black)	-0.128	0.045	.005	0.880	0.806	0.962
Age (1-7)	0.086	0.010	0.000	1.089	1.069	1.110
Gender (Male)	-0.391	0.035	0.000	0.676	0.632	0.724
Education	-0.146	0.013	0.000	0.864	0.843	0.886
Constant	-1.097	0.061	0.000	0.334		
Model 2						
Race (Black)	0.189	0.088	0.032	1.208	1.017	1.436
Age (1-7)	0.104	0.010	0.000	1.109	1.087	1.132
Gender (Male)	-0.391	0.035	0.000	0.676	0.632	0.724
Education	-0.148	0.013	0.000	0.862	0.841	0.884
Age x Race	-0.105	0.026	0.000	0.901	0.857	0.947
Constant	-1.149	0.063	0.000	0.317		

Table 3. The Summary of Regression Model by Race

	B	SE	*P* Value	Exp(B)	95% CI for EXP(B)	
Model 1						
Age (1-7)	0.104	0.010	0.000	1.109	1.087	1.132
Gender (Male)	-0.407	0.038	0.000	0.666	0.617	0.718
Education	-0.160	0.014	0.000	0.852	0.829	0.876
Constant	-1.100	0.066	0.000	0.333		
Model 2						
Age (1-7)	0.002	0.023	0.930	1.002	0.957	1.049
Gender (Male)	-0.312	0.082	0.000	0.732	0.624	0.859
Education	-0.091	0.031	0.003	0.913	0.859	0.969
Constant	-1.179	0.135	0.000	0.308		

As Table 2 shows, age was a predictor of CRD overall, but we also found a statistical interaction suggesting a stronger effect for White than Black participants.

As Table 3 shows, age was a predictor of CRD for White but not Black participants.

5. Discussion

Our observation that older age is a risk factor for higher CRD was fully expected and can be explained by the widely recognized age-related health decline and process of ageing. However, the role of age as a determinant of normal health decline may have diminished for Black compared with White individuals. We explain this observation as a manifestation of structural racism in the US. We also see this finding in line with MDRs.

Our findings suggest that some of the racial variation in CRD and health may be due to diminished relevance of age and age-related change in health, in Black than White individuals. We interpret this finding through a sociological rather than a biological mechanism. Similar to the weaker effects of SES and psychological assets, the weakened effect of age for Black than White people may be due to widespread stressors and adversities in Black people lives.[60-62] In this view, high stress and adversity, unpredictable environment, and living in under-resourced areas with high crime rate and chronic poverty may hinder the normal age-related health changes for Black people.[63]

We see racism as the main driver of the inequalities observed here. Our work is different than previous work by Murry and others who attributed racial differences to genetic and innate differences.[64] Instead of a deterministic view, we attribute the observed differences to the variation in social environment, which hinders Blacks' daily lives. To undo the observed differences, we need to use social and economic and public policies that undo historic racism and legacy of slavery for Black communities. As income seems to generate equitable effects for Whites and Blacks, we need policies that increase minimum wage and increase income and wealth of Black families[9,65-67]

As shown by this study and previous work,[1] under racism, not only SES [30-33] but also coping [68,69] and age[13] show weaker influences on a wide range of health outcomes for Blacks than Whites. These patterns are robust and shown for children,[44,70] youth,[71] adults,[72] and older adults.[73,74] Under racism, all resources such as employment,[75] marital status,[45] parental education,[34] own education,[18,25,40] and coping style[68,69] lose some of their effects for Black communities. Given the systematic nature of these patterns, we have concluded that any resource may show diminished effects

for oppressed (Black) than privileged (White) people.[76-85] These are because of racism, segregation, discrimination, social stratification, and unequal treatment that are multilevel, deeply rooted, structural and societal causes of inequalities.

We cannot emphasize enough that this study conceptualized race as sociological force that reflects history of slavery, Jim Crow, equal but separate, and other unjust policies over four centuries. In our study race is a proxy of where people reside, what healthy options are available, and how much stress populations face. In our study, race is a proxy of treatment of the group by society. Our past results have suggested that, racism alters the implications of age for health, health care services, and brain development.[37-39] Similarly, racism reduces the salience of SES and other psychological resources as a determinants of outcomes of individuals.[86]

Under racism, the role of age as a normal, developmental, natural determinant of CRD is unequal across diverse racial groups. Due to the existing structural racism and inequalities that are present in the US, even age may not have the same health effects for Whites and Blacks, and the salience of age may be weaker for Black than White people. Therefore, policy solutions that wish to achieve racial health equality should go beyond just addressing SES inequalities and address the context in which health inequalities emerge. Economic, social, and policy solutions are needed to equalize the social environment of Black and White individuals so that age, SES, and other resources and assets generate equal health across all racial groups. The root cause of these inequalities is racism, as explained by Link.[87,88]

6. Conclusion

In a national sample of American adults, in line with other SES and psychological resources and assets, age shows a weaker association with CRD for Black than White Americans. This is also in line with previous research showing age is a less salient determinant of brain development for Blacks than Whites.[7,12-15] For Whites, predictability of environment increases the salience of healthy age-related development of CRD. In contrast, Blacks often experience adversities and stress, and live in less predictable environment, thus they show many other factors that may dilute the role of age as a determinant of CRD.

Author Contributions

Conceptualization: SA, AA, BN.
Formal Analysis: SA.
Writing – original draft: BN, AR.
Writing – review & editing: SA, AA, BN.
All authors approved the final draft.

Conflict of Interest Disclosures

Both authors declare no conflict of interests.

Ethical Approval

This study used publicly available PATH data. All data are fully deidentified. Thus, the study was not human subject research and, therefore, was exempt from a full IRB review.

Research Highlights

What Is Already Known?

- Age is a main determinant of CRD.
- Higher age is associated with a higher odd of CRDs such as COPD, emphysema, and chronic bronchitis.

What Does This Study Add?

- The association between age and CRD differs between White and Black individuals.
- Black individuals develop CRD at all ages. Whites are more likely to develop a CRD at higher age.

Funding/Support

Assari is funded and supported by the Tobacco-Related Disease Research Program (TRDRP) grant R00RG2347. Assari is also supported by NIH grant 5S21MD000103.

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