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Jason T. Newsom

Portland State University, newsomj@pdx.edu

Emily C. Denning

Portland State University, edenning@pdx.edu

Miriam R. Elman

OHSU-PSU School of Public Health

Anda Botoseneanu

University of Michigan

Heather G. Allore

Yale University

See next page for additional authors

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Authors

Jason T. Newsom, Emily C. Denning, Miriam R. Elman, Anda Botoseneanu, Heather G. Allore, Corey L. Nagel, David A. Dorr, and Ana R. Quiñones

Physical Activity as a Mediator between Race/Ethnicity and Changes in Multimorbidity

¹Jason T. Newsom, PhD, ¹Emily C. Denning, MS, ²Miriam R. Elman, ³Anda Botoseneanu, MD, PhD, ⁴Heather G. Allore, PhD, ⁵Corey L. Nagel, PhD, MPH, RN, ⁶David A. Dorr, MD, ⁷Ana R. Quiñones, PhD

¹Department of Psychology, Portland State University

²School of Public Health, Oregon Health & Science University/Portland State University

³Department of Health and Human Services and Institute of Gerontology, University of Michigan-Dearborn and Ann Arbor

⁴Internal Medicine and Department of Biostatistics, Yale University

⁵College of Nursing, University of Arkansas for Medical Sciences

⁶Department of Medical Informatics and Clinical Epidemiology, School of Medicine, Oregon Health and Science University

⁷Family Medicine, Oregon Health and Science University

Corresponding author: Jason T. Newsom, PhD, Professor, Department of Psychology,

Portland State University, P.O. Box 751, 97207. Email: newsomj@pdx.edu

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Jason T. Newsom planned the research, directed the analyses and was the primary contributor to the manuscript. Emily C. Denning collaborated on the research plan, conducted the analyses, prepared the tables, and contributed to manuscript writing. Miriam R. Elman assisted with data management and data analysis. Anda Botoseneanu collaborated on the research plan and manuscript writing. Heather G. Allore collaborated on the research plan and manuscript writing. Corey L. Nagel collaborated on the research plan and manuscript writing. David A. Dorr collaborated on the research plan and manuscript writing. Ana R. Quiñones is the principal investigator of the project and collaborated on the research plan and manuscript writing.

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Abstract

Objectives. Studies report racial/ethnic disparities in multimorbidity (≥ 2 chronic conditions) and their rate of accumulation over time as well as differences in physical activity. Our study aimed to investigate whether racial/ethnic differences in the accumulation of multimorbidity were mediated by physical activity among middle-aged and older adults. *Methods.* We assessed racial/ethnic differences in the accumulation of multimorbidity (of nine conditions) over twelve years (2004-2016) in the Health and Retirement Study (HRS; N = 18,264, mean age = 64.4 years). Structural equation modeling was used to estimate latent growth curve models of changes in multimorbidity and investigate whether the relationship of race/ethnicity (non-Hispanic Black, Hispanic, non-Hispanic White participants) to changes in the number of chronic conditions was mediated by physical activity after controlling for age, sex, education, marital status, household wealth, insurance coverage, smoking, alcohol, and body-weight. *Results.* There was a significant increase in multimorbidity over time. Initial levels and changes in multimorbidity over time varied significantly across individuals. Indirect effects of the relationship between race/ethnicity and changes in multimorbidity as mediated by physical activity were significant, consistent with the mediational hypothesis. Black respondents engaged in significantly lower levels of physical activity than White respondents after controlling for covariates, but there were no differences between Hispanic and White respondents once education was included. These results provide important new information for understanding how modifiable lifestyle factors may help explain disparities in multimorbidity in mid-to-late life, suggesting greater need to intervene to reduce sedentary behavior and increase physical activity.

Keywords: exercise, chronic illness, disparities

Importance of Multimorbidity

Multimorbidity, often defined as the co-existence of two or more chronic diseases, commonly occurs among middle-aged and older adults, and its prevalence increases with age (Goodman et al., 2016).¹ Multimorbidity has additional negative health consequences that are not explained by single disease models (Zeng et al., 2014) and is costly for patients and the health care system (Goodman et al., 2016). Some of these additional consequences include increased mortality risk and poor quality of life (Wei & Mukamal, 2018), as well as reduced life satisfaction (Marques et al., 2018). As the single disease approach fails to recognize the unique impact of experiencing concurrent multiple chronic diseases, it is important to consider how multimorbidity changes over time, as well as the factors that contribute to accumulation of chronic diseases over time (Quiñones et al., 2014, 2019).

Racial/Ethnic Disparities in Multimorbidity

Racial/ethnic differences in health and mortality in middle and older age have been well established (National Center for Health Statistics, 2016). Compared with non-Hispanic Whites, non-Hispanic Blacks have a higher prevalence of chronic disease (e.g., Davis et al., 2017) and higher risk of mortality (Masters et al., 2014). A variety of distinct hypotheses have been posited to explain racial/ethnic health disparities in middle and older age. One explanation is the cumulative disadvantage or cumulative inequality hypothesis, which posits that disadvantages in factors such as health care access accumulate over time, resulting in greater adverse effects for certain groups and intergenerational socioeconomic inequality (Yearby, 2018). This hypothesis states that disease incidence increases over time at a faster rate for minorities than for Whites. The persistent inequality hypothesis, in contrast, posits that racial/ethnic disadvantages occur steadily across the lifespan, so that increases in multimorbidity occur in parallel and at similar rates among race/ethnicity groups (Henretta & Campbell, 1976). The age-as-leveler hypothesis (Kim & Miech, 2009) posits a contrasting

hypothesis that health disparities exist among groups at earlier ages but that disparities decrease over time because disease risk is high at older ages among all groups. Economic differences also tend to be compressed at older age groups because of retirement (Robert & House, 1996), which may reduce health disparities. Although economic disadvantages are a central component to most of these explanations, all of the frameworks either incorporate or are consistent with identifying discrimination as a root cause of inequality that may operate through several pathways, including the effect of chronic stress through discrimination (e.g., National Urban League, 1964), discrimination in the health care setting, and unequal access to high-quality care, among others.

Although the same theoretical explanations are sometimes applied to disparities between Hispanic and non-Hispanic White adults, different patterns emerge, suggesting a more complex picture. Compared with non-Hispanic Whites, Hispanic older adults are sometimes found to be at higher risk (Angel et al., 2015) and sometimes at lower risk for disease or mortality in later life (e.g., Davis et al., 2017), with the latter phenomenon often referred to as the “Hispanic paradox” (Markides & Coreil, 1986).

Understanding racial/ethnic inequalities in multimorbidity is of particular importance because of the enhanced risk of multimorbidity for institutionalization and mortality. Research by Quiñones and colleagues (2019), for example, indicates that Blacks have higher multimorbidity compared with Whites beginning in middle age that is carried forward into older age. This pattern is consistent with the cumulative inequality hypothesis. Hispanics, however, have lower multimorbidity beginning in middle age and more rapid accumulation of chronic conditions than Whites (Quiñones et al., 2011, 2019). This pattern appears to be consistent with both the Hispanic paradox hypothesis and the cumulative inequality hypothesis. Sauver and colleagues (2015) examined incident multimorbidity of two or three chronic conditions and found that incidence of two conditions among Black patients

increased more rapidly than among White patients, with initial differences beginning in the 30-40 years of age range. This finding is consistent with the cumulative inequality hypothesis. The incidence of having any three conditions, however, showed initial differences beginning in this age range, but disappeared at approximately age 70, which is consistent with the age-as-leveler hypothesis. There are fewer studies that have compared changes in multimorbidity between Hispanics and non-Hispanic Whites.

Physical Activity as Predictor of Multimorbidity

Previous work has demonstrated that a number of lifestyle factors, or health behaviors, have an influence on multimorbidity (Canizares et al., 2018), such as smoking, obesity, and a sedentary lifestyle, with sedentary behavior specifically associated with a 14% increase in the likelihood of having multimorbidity. One health behavior that appears to be consistently related to multimorbidity is physical activity (Dhalwani et al., 2016). A study of English older adults not only showed an important difference between those who were inactive and those who were not inactive, but also suggested a dose-response gradient, such that more moderate or vigorous physical activity was associated with lower risks of multimorbidity (Dhalwani et al., 2016). This finding is consistent more generally with a wide variety of studies suggesting any degree of increase in physical activity is associated with reduced risks of adverse health outcomes and mortality (Lin et al., 2015).

Race/Ethnicity and Physical Activity

The apparent protective role that physical activity plays in reducing multimorbidity risk raises the question as to whether racial/ethnic disparities in multimorbidity may be partially attributable to group differences in physical activity. Studies have been fairly consistent in showing that non-Hispanic Black and Hispanic groups are less likely to report moderate and vigorous leisure time activity than Non-Hispanic Whites (Saffer et al., 2013), as well as more likely to report sedentary behavior (Marquez et al., 2010). It should be noted

that, in the study by Marquez and colleagues (2010), differences in leisure-time activity between Hispanics and non-Hispanic Whites were eliminated after controlling for education. Although prior studies have examined race/ethnicity differences in physical activity and multimorbidity, to date, no studies have investigated physical activity as a potential mediational pathway between race/ethnicity and multimorbidity. Establishing such a link would provide a potential explanation for disparities in the accumulation of multimorbidity in older age.

Contributions of the Study

The present study seeks to expand prior research by examining the relationship between physical activity and changes in multimorbidity in middle and later adulthood—one pathway by which racial/ethnic disparities in multimorbidity may occur. We hypothesized that physical activity would be an important factor in accounting for the relationship between race/ethnicity and changes in multimorbidity over time as exhibited by significant indirect effects. The possible role of physical activity in at least partially explaining racial/ethnic differences in multimorbidity disparities in middle age and later life has yet to be examined. The present study, thus, seeks to expand our knowledge of health disparities by investigating changes in multimorbidity over a 12-year period and the mediational role of physical activity in the association between race/ethnicity and multimorbidity in a population-based sample of U.S. middle-aged and older adults.

Method

Sample

Participants were from the Health and Retirement Study (HRS; Heeringa & Conner, 1995) using data from 2004 to 2016. Participants who responded to the 2004 HRS questionnaire were included in this study, including proxy responses (8.95%). Those who were residing in nursing homes were excluded. Participants were also excluded if they did

not report their race/ethnicity (N = 497) or they indicated American Indian, Asian, or “other” race/ethnicities due to small sample sizes for these groups (N = 1,088, across these other groups). The final analytic sample included 18,264 respondents.

Design

The HRS is a longitudinal study of a nationally representative sample of Americans over age 50 (Sonuga & Weir, 2014). The study includes a wide array of health, socioeconomic, and psychosocial variables assessed in biennial interviews. For the present study, seven waves of data (years 2004 through 2016) were used. The 2004 interview was used as the first time point because not all physical activity variables were consistent in prior years. Although participants with missing data were included in the analyses, data from participants added to the study after 2004 were not included in order to minimize cohort effects.

Measures

Multimorbidity. To create a multimorbidity score for each participant for each wave (from 2004 to 2016), chronic disease variables were summed such that respondent multimorbidity score (range 0-9) indicated the total number of chronic conditions. This measure is consistent with the approach to measurement of multimorbidity in a large number of prior studies (Johnston et al., 2019; Cezard et al., 2021). Seven self-reported somatic conditions were each prompted by “Since we last talked to you, that is since [last interview date], has a doctor told you that you have ...” or, if the interview was the first interview for the respondent, “Has a doctor ever told you that you have...” and included: hypertension (i.e., high blood pressure), diabetes, cancer (including any malignant tumors, but excluding skin cancer), lung disease (e.g., chronic bronchitis, or emphysema, but excluding asthma), heart disease (including myocardial infarction, congestive heart failure, angina, and other heart problems), stroke (excluding TIA), and arthritis. Two mental health conditions, high

depressive symptoms (CESD 8-item score ≥ 4), and cognitive impairment (≤ 10 or less on the 35-point scale for the Telephone Interview for Cognitive Status) were also included. To settle clinically-inconsistent patterns of chronic disease reports over time for the somatic conditions, we applied a previously developed multistep adjudication method (Cigolle et al., 2018) that resolves inconsistencies using disease-specific follow-up questions (i.e., “evidence” of disease such as reporting disease-specific medications or treatments).

Physical Activity. Physical activity was measured with three separate items (Jenkins et al., 2008) from the 2004 wave assessing the frequency to which participants engaged in mildly energetic, moderately energetic, and vigorous levels of physical activity. These items were rated on a 5-point scale of frequency, ranging from “hardly ever or never” to “every day”. Question wording is provided in the online appendix.

Race/ethnicity. Race and ethnicity were coded as mutually exclusive categories: Non-Hispanic White, Non-Hispanic Black, and Hispanic older adults. Dummy variables were created to allow for comparison between racial groups, with Non-Hispanic Whites as the referent group.

Covariates. Structural models controlled for sociodemographic, socioeconomic, and health behavior variables that might be potentially confounding factors that would account for race and ethnicity differences. Covariates included sex (female = 1, male = 0), age in 2004, marital status in 2004 (married = 1, not married = 0), insurance status from 2004 to 2016 (two dummy variables: continuous vs. intermittent, continuous vs. no insurance), net worth in 2004 (in US dollars), education level (number of years in school), smoking (two dummy variables: never smoked vs. ever smoked, never smoked vs. currently smoke), and alcohol use as measured by the number of reported drinks per week. Body mass index (kg/m^2 ; BMI) at each time point (from 2004 to 2016) was included as a time-varying covariate to account for the changes in multimorbidity that might be due to weight changes.

Analysis Overview

Baseline (2004) sample weights and sampling design adjustments appropriate for the HRS were used for all analyses in accordance with recommendations for the study (Heeringa & Conner, 1995; see the online appendix for additional information). All analyses were conducted with Mplus version 8.5 (Muthén & Muthén, 1998-2017) using full information maximum likelihood for missing data with robust estimation (Yuan & Bentler, 2000), which has been shown to produce less biased parameter estimates and more accurate standard errors than listwise deletion (Collins et al., 2001). The sample size varied somewhat across analyses when data were missing on covariates, because Mplus excludes cases with missing data on exogenous measured variables. Indirect effects tests were based on 10,000 bootstrap samples of the maximum likelihood estimates.

Prior to tests of the hypothesized model, we conducted a confirmatory factor analysis (CFA) of the physical activity items to assess the measurement portion of the model. An unconditional latent growth curve model was then tested using the multimorbidity scores from 2004 to 2016 to investigate change in multimorbidity over time. The main research questions were then investigated with a structural equation model in which physical activity was a mediator in the association between race/ethnicity and growth curve factors. Figure 1 illustrates the basic mediational model (without covariates) in which race and ethnicity variables predict physical activity, which, in turn, was a predictor of baseline multimorbidity (intercept factor) and changes in multimorbidity (slope or rate of growth factor). Subsequent models included covariates. All covariates, except BMI, were included as time-invariant covariates as predictors of physical activity. BMI was included as a time-varying predictor, with multimorbidity regressed on BMI measured at each corresponding time point, to examine changes in multimorbidity over time after accounting for any changes in body weight over time (Newsom, 2015). Three variations of this model were used to better

understand the extent to which covariates might account for the differences between Non-Hispanic Whites and Hispanic older adults—first without covariates (Model 1), then adding all covariates except education (Model 2), and finally including all covariates (Model 3).

The amount of attrition, attrition patterns, and analyses describing which participants were most likely to drop out of the study are described in the online appendix. Models using listwise deletion did not change the conclusions.

Results

Preliminary descriptive analyses and correlations among variables used in the subsequent models are presented in Table A1 in the online appendix.

Confirmatory Factor Analysis of Physical Activity Measure

A single-factor CFA was tested with the three physical activity items (mild, moderate, and vigorous activity) to investigate the interrelations between these three items and the appropriateness of the latent variable. As this model only had three indicators, it is just-identified and no information about model fit is available. Standardized loadings were acceptable (mild = .484, moderate = .851, and vigorous = .437, all p -values < .001) with the highest loading being that of moderate physical activity. Further details of the confirmatory factor analyses are provided in the online appendix.

[Figure 1 about here]

Unconditional Growth Curve Model

An unconditional latent growth curve model was tested to examine linear changes in multimorbidity between 2004 and 2016. Intercept and slope factors were specified using commonly employed values for the loadings (all 1 for the intercept factor and 0, 1, 2, 3, 4, 5, 6 for the slope factor), so that the intercept factor mean represents baseline multimorbidity,

and the slope factor mean represents the change in multimorbidity across each wave, respectively. Both factors were allowed to vary freely, providing information about individual variation in baseline scores and change in multimorbidity (see Figure 1). Although the chi-square for the model was significant, $\chi^2 (N = 18,264, df = 23) = 2920.86, p < .001$, given the large sample size, the alternative fit indices indicated the model fit well, CFI = .951, SRMR = .049, according to commonly used criteria for fit (Hu & Bentler, 1999). The average intercept was equal to 1.889, indicating participants had less than two chronic conditions at baseline. The intercept factor variance was significant, $\psi = 2.035, SE = .030, p < .001$, indicating baseline values varied across individuals. The average slope also was significant, $\alpha = .195, SE = .002, p < .001$, which indicates that, on average, participants had approximately .2 additional chronic conditions every two years. Individuals with fewer chronic conditions at baseline had more rapid increases in multimorbidity, as shown by the negative correlation between intercept and slope, $\psi = -.028, SE = .004, \psi^* = -.106, p < .001$.

Mediational Models

To investigate whether physical activity mediates the relationship between race/ethnicity and changes in multimorbidity, a set of structural equation models were tested, beginning with a model with no covariates (Model 1), followed by a model with all covariates except education (Model 2), and, finally, a model with all covariates (Model 3).

Table 1 summarizes the results.

[Table 1 about here]

Model 1 (N = 18,264) specified the two race/ethnicity dummy variables, as predictors of the latent variable for physical activity, which, in turn, predicted the multimorbidity intercept and growth curve factors (see Table 1). The indirect effect of the race/ethnicity dummy variables on the growth factor represents an estimate of the degree to which differences among Black, Hispanic, and White participants in the rate of multimorbidity

changes over time are due to differences in the amount of physical activity. The direct effect paths between the race/ethnicity variables and the growth curve factors were included to appropriately compute and test the indirect effect (MacKinnon, 2008). The direct effects of the race/ethnicity variables on the growth factor represent the degree to which there are remaining differences in the rate of change in multimorbidity that are not accounted for by physical activity. The initial model without direct effects fit the data well according to commonly used cutoffs for alternative fit indices, CFI = .960, SRMR = .036, though the Satorra-Bentler adjusted chi-square was significant, $\chi^2(56) = 3598.83$, $p < .001$. Both paths between race/ethnicity variables and physical activity were significant. Non-Hispanic Blacks were less likely to engage in physical activity than non-Hispanic Whites, and Hispanics were significantly less likely to engage in physical activity than non-Hispanic Whites. Physical activity also was a significant predictor of both initial multimorbidity and change in multimorbidity. Although the direct effects of race/ethnicity variables were significant for initial multimorbidity levels, neither direct effect reached conventional significance levels when predicting slopes (non-Hispanic Black vs. non-Hispanic White predicting slope). These results are thus consistent with a full mediational effect, in which differences in trajectories across groups is accounted for entirely by differences in physical activity across groups. Bootstrap tests of the significance of the indirect effects between the race/ethnicity variables and the intercept and slope growth factors were all significant, supporting the mediational hypothesis: non-Hispanic Black vs. non-Hispanic White for the intercept, $\beta = .295$, SE = .029, 95% CI [.238, .351], $\beta^* = .061$; Hispanic vs. non-Hispanic White for the intercept, $\beta = .133$, SE = .023, 95% CI [.085, .174], $\beta^* = .025$; non-Hispanic Black vs. non-Hispanic White for the slope, $\beta = .005$, SE = .002, 95% CI [.002, .008], $\beta^* = .007$; and Hispanic vs. non-Hispanic White for the slope, $\beta = .002$, SE = .001, 95% CI [.001, .004], $\beta^* = .003$.

Model 2 (N = 18,057) included all covariates except education as predictors of physical activity. Although the Satorra-Bentler adjusted chi-square was significant, χ^2 (305) = 8175.15, $p < .001$, the alternative fit indices were below or very near the commonly used cutoffs indicating acceptable fit, CFI = .948, SRMR = .071. Results of this model were largely consistent with Model 1. Non-Hispanic Blacks were less likely to engage in physical activity than non-Hispanic Whites, and Hispanics were significantly less likely to engage in physical activity than non-Hispanic adults. Physical activity also was a significant predictor of both initial multimorbidity and change in multimorbidity. Contrary to the results from Model 1, the direct effect of the non-Hispanic Black vs. non-Hispanic White comparison predicting intercept values was not significant for initial multimorbidity levels. The direct effect of the Hispanic vs. non-Hispanic White comparison predicting the intercept remained significant, however. Also, as in Model 1, neither direct effect reached conventional significance levels when predicting slopes (non-Hispanic Black vs. non-Hispanic White predicting the slope). These results are thus consistent with a full mediational effect. Bootstrap tests of the significance of the indirect effects between the race/ethnicity variables and the intercept and slope growth factors were all significant, supporting the mediational hypothesis: non-Hispanic Black vs. non-Hispanic White for the intercept, $\beta = .384$, SE = .035, 95% CI [.315, .451], $\beta^* = .079$; Hispanic vs. non-Hispanic White for the intercept, $\beta = .262$, SE = .041, 95% CI [.180, .343], $\beta^* = .048$; non-Hispanic Black vs. non-Hispanic White for the slope, $\beta = .011$, SE = .002, 95% CI [.007, .016], $\beta^* = .018$; and Hispanic vs. non-Hispanic White for the slope, $\beta = .008$, SE = .002, 95% CI [.005, .012], $\beta^* = .011$. Among the covariates, those who were older, female, unmarried, had intermittent insurance coverage, and lower income, were less likely to engage in physical activity. Those who reported ever smoking or currently smoking at baseline also were less likely to engage in physical activity,

while drinking alcohol was associated with greater physical activity. Having no insurance coverage was not significantly predictive of physical activity.

Model 3 (N = 18,035) tested the same model with the same covariates but also controlled for education. Although the Satorra-Bentler adjusted chi-square was significant, $\chi^2(321) = 8393.01$, $p < .001$, the alternative fit indices were below or near the commonly used cutoffs indicating acceptable fit, CFI = .948, SRMR = .070. The results of this model were consistent with those from Model 2 with one important exception. Although non-Hispanic Blacks were less likely to engage in physical activity than non-Hispanic Whites, as in the previous model, Hispanics were no longer significantly less likely to engage in physical activity at baseline or increase in multimorbidity than non-Hispanic Whites when education was added to the model. The other covariates remained significant predictors of physical activity. Bootstrap tests of the significance of the indirect effects between non-Hispanic Blacks vs. non-Hispanic Whites were significant for the intercept and the slope, $\beta = .336$, SE = .035, 95% CI [.268, .403], $\beta^* = .069$, and $\beta = .010$, SE = .002, 95% CI [.006, .014], $\beta^* = .016$, respectively. As would be expected from the non-significant direct effect in Model 3 for the comparison between Hispanics and non-Hispanic Whites, the indirect coefficients for the intercept or slope factors were no longer significant when education was added to the model.

Discussion

The findings from this study indicate that engaging in greater physical activity is related to lower initial levels of multimorbidity, as well as slower accumulation of chronic disease over a period of 12 years, above the effects of a number of relevant covariates including net worth and insurance coverage. These results were consistent with our initial hypothesis that physical activity is a mediator in the relationship between race/ethnicity and multimorbidity. Our study goes beyond prior work that documents disparities in multimorbidity by investigating one explanation for how such disparities occur. Significant

indirect effects highlight the importance of physical activity in older adulthood as an explanation for racial/ethnic differences in initial levels of multimorbidity, as well as changes in multimorbidity. The results, therefore, help inform our understanding of health disparities and the potential role that physical activity can play contributing to accumulation of chronic disease later in life.

Results from the present study may help explain some discrepancies in the literature regarding whether differences in multimorbidity across race/ethnicity groups change at similar rates (i.e., the persistent inequality hypothesis; Henretta & Campbell, 1976) or increase with time (i.e., the cumulative disadvantage hypothesis; Yearby, 2018). Our results indicate that differences across groups in the level of physical activity, which are consistent with those reported elsewhere (e.g., Saffer et al., 2013), accounted for differences in multimorbidity rates of increase over twelve years during middle and late adulthood. Physical activity is known to be a strong mitigator of a variety of physiological aging processes (Harridge & Lazarus, 2017). Differences in physical activity among race/ethnicity groups across the lifespan may account for the existing mid-life differences in multimorbidity evident at the beginning of this study's observation period, as well as provide an explanation for differences in the rate of accumulation of chronic conditions observed in some studies (Sauver et al., 2015; Quiñones et al., 2013). Interestingly, we found that the differences between Hispanic and non-Hispanic Whites in physical activity levels disappeared once education—but not other covariates such as wealth, insurance status, age, and gender—was controlled. The connection to education may have to do with occupation type, a variable found to be related to physical activity differences between Hispanics and non-Hispanic Whites because of work-related physical activity (Chung et al., 2009).

One component of most theories concerning racial/ethnic health disparities is that discrimination plays a leading or substantial role in the underlying reasons for health

differences across groups. Where access to healthcare, chronic stress associated with discrimination, or financial inequities are the culprits (Office of Disease Prevention and Health Promotion, 2018), it may be easier to see a direct link between these factors and health outcomes. Where the mechanism involves group differences in health behaviors, such as physical activity, however, the process by which discrimination plays a role is perhaps less obvious. Nevertheless, discrimination in access to health care, sustained chronic stress experiences, or financial inequities may still play a central or important role in processes that have been more commonly delineated for some health behaviors, such as smoking (e.g., Kendzor et al., 2014). Race/ethnicity differences in physical activity levels may be a function of many of these same factors.

Although engaging or not engaging in physical activity is often seen as a product of motivation or intention (Fishbein & Azjen, 1975), and, thus, on the surface, it may appear that racial/ethnic differences in activity are a result of choice, there are a wider array of sociological and economic factors that may contribute importantly to differences in physical activity that are not a function of individual choice. Resource differences, involving available leisure time, financial means (e.g., gym memberships, exercise equipment; Reichert et al., 2007), and geographic factors (e.g., access to local parks, neighborhood safety or walkability; Estabrooks et al., 2003) as well as differences in interpersonal support, social norms, and access to health information (Newsom et al., 2020) are all likely to be factors that contribute to differences in whether an individual is able or likely to engage in physical activity. Disparities across race and ethnic groups in any of these areas are mechanisms that operate outside of or limit personal choice.

As multimorbidity can accumulate slowly over the life span, one major strength of the present study was the use of a large, population-based sample that followed participants' occurrence of chronic disease for over a decade, specifically at the time in the life course

when multimorbidity is more likely to develop. The use of longitudinal data allowed for the examination of the relationship between physical activity and baseline multimorbidity levels, but also investigate how physical activity relates to accumulation of multimorbidity over time. There are some potential limitations, however. This study makes exclusive use of self-report measures. Self-report of chronic diseases may not always be the most reliable, and it would be beneficial for future work to include chronic conditions reported from different sources (e.g., linked Medicare records). We also used a short self-report measure for physical activity, which would be optimally measured using a more extensive self-report assessment or physiological measures. A more extensive and more reliable measure of physical activity might be expected to show stronger relationships to multimorbidity. Another limitation of the present study was the use of the broad Hispanic category, which was mostly comprised of Mexican-American individuals, but also included Puerto Rican, Cuban, and others with South American heritage, who may be better examined as separate groups when sample sizes are sufficient. Previous work on the healthy immigrant hypothesis (e.g., Rodriguez et al., 2014) has noted clear differences in the health of people of Hispanic heritage who were born in the US or migrated to the US. It will be important for future work to examine differences within the group of individuals who report Hispanic ethnicity, to better elucidate the potential differences in physical activity and multimorbidity between those who were born in the US and those who migrated to the US.

Additional research is needed to understand how specific multimorbidity combinations and patterns might differ for diverse groups of older adults in the US and how differences in social needs inform divergence in multimorbidity accumulation. Particular multimorbidity combinations may have different consequences for health outcomes and mortality (Quiñones et al., 2020). Although the focus was on overall multimorbidity, given its demonstrated importance for mortality and other outcomes, specific multimorbidity

combinations (for instance, arthritis and heart disease) may show different patterns of disparities across race and ethnicity groups and some conditions in combinations may be more and less related to physical activity. These hypotheses deserve greater attention in future research. More also needs to be learned about the potential limiting effects that health conditions may have on physical activity. Our analyses were designed to investigate whether initial differences in the level of physical activity among racial and ethnic groups would be predictive of later changes in multimorbidity, predicated on a wealth of findings establishing that exercise impacts health (e.g., Lin et al., 2015). It is possible that experiencing multimorbidity may result in subsequent physical inactivity (the reverse effect compared to our models), a phenomenon that deserves additional study. Additionally, the association between physical activity and multimorbidity appears to differ based on factors such as sex or age (e.g., Cimarras-Otal et al., 2014).

Public health, policy-based, and clinical interventions with the aim of reducing disparities in multimorbidity may come in a variety of forms (e.g., Olanrewaju, et al., 2016). Public health initiatives can address discrepancies in pro-exercise norms and increase awareness that physical activity constitutes an important pathway by which disparities in multimorbidity may occur. Policy changes could help reduce geographic disparities in access to gyms or safe and walkable areas, for example. Clinical interventions also are needed, particularly those aimed at correcting any existing differences in the form or frequency of recommendations made to patients. Although interventions will be most effective and economical earlier in life, as lifelong habits are difficult to change, behavioral change in later life can and does occur, and racial and ethnic differences in behavioral change as secondary prevention after chronic conditions occur also can play a critical role (Quiñones et al., 2017).

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List of Tables & Figures

Table 1. *Mediation Model Results.*

Figure 1. *Full mediation model.*

Caption: *Note.* MM = multimorbidity. Coefficients are results from Model 1. Model 2 included gender (female = 1, male = 0), age in 2004, marital status in 2004 (married = 1, not married = 0), insurance status from 2004 to 2016 (two dummy variables: continuous vs. intermittent, continuous vs. no insurance), net worth in 2004 (in US dollars), smoking (two dummy variables: never smoked vs. ever smoked, never smoked vs. currently smoke) in 2004, and alcohol use (drinks per week) in 2004 as time-invariant predictors of physical activity; and body mass index at each time point (from 2004 to 2016) was included as a time-varying covariate predicting multimorbidity at each wave. Model 3 added education level (number of years in school) as a time-invariant predictor of physical activity.

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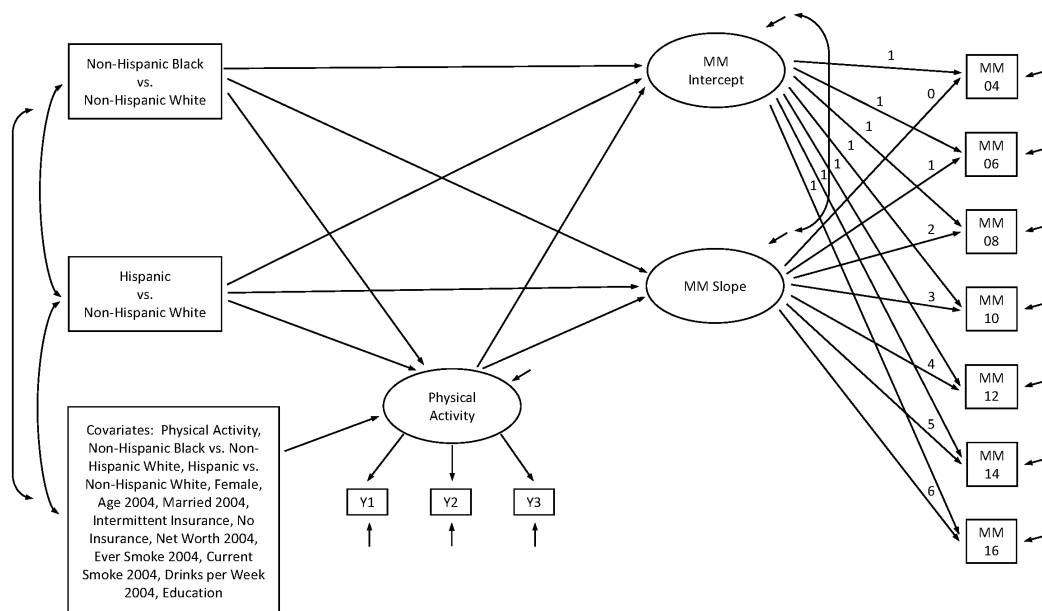
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Table 1
Mediational Model Results

	Dependent Variables		
	Physical Activity	Intercept	Slope
Model 1			
Average	--	1.852	1.042
Physical Activity	--	-.426***	-.049**
Non-Hispanic Black vs. Non-Hispanic White	-.135***	.025***	.005
Hispanic vs. Non-Hispanic White	-.063***	-.028**	.017
Model 2			
Average	--	1.880	1.058
Physical Activity	--	-.547***	-.122***
Non-Hispanic Black vs. Non-Hispanic White	-.145***	-.005	-.006
Hispanic vs. Non-Hispanic White	-.089***	-.045***	.013
Female	-.026*	--	--
Age 2004	-.452***	--	--
Married 2004	.051***	--	--
Intermittent Insurance	-.037***	--	--
No Insurance	.002	--	--
Net Worth 2004	.131***	--	--
Ever Smoke 2004	-.083***	--	--
Current Smoke 2004	-.120***	--	--
Drinks per Week 2004	.033*	--	--
Model 3			
Average	--	1.906	1.062
Physical Activity	--	-.565***	-.128***
Non-Hispanic Black vs. Non-Hispanic White	-.123***	-.010	-.007
Hispanic vs. Non-Hispanic White	-.019	-.048***	.013
Female	-.018	--	--
Age 2004	-.400***	--	--
Married 2004	.052***	--	--
Intermittent Insurance	-.012	--	--
No Insurance	.006	--	--
Net Worth 2004	.099***	--	--
Ever Smoke 2004	-.077***	--	--
Current Smoke 2004	-.100***	--	--
Drinks per Week 2004	.030*	--	--
Education	.217***	--	--

Note. All coefficients are standardized values, except average intercept values, which are unstandardized. All values are based on robust maximum likelihood estimation for missing data and sampling design adjustments; * $p < .05$, ** $p < .01$, *** $p < .001$; Model 1 $N = 18,264$, Model 2 $N = 18,057$, Model 3 $N = 18,035$.

Figure 1



ⁱ Multimorbidity, as defined as two or more chronic health conditions, occurs in 61.1% of the sample of participants in the HRS.

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