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Original Article

Childhood Misfortune and Handgrip Strength Among Black, White, and Hispanic Americans

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Abstract

Objective: Although early-life insults may affect health, few studies use objective physical measures of adult health. This study investigated whether experiencing misfortune during childhood is associated with handgrip strength (HGS) in later life.

Method: Data on childhood misfortune and adult characteristics from the Health and Retirement Study were used to predict baseline and longitudinal change in HGS among White, Black, and Hispanic American men and women.

Results: Regression analyses revealed that multiple indicators of childhood misfortune were related to HGS at baseline, but the relationships were distinct for men and women. Over the study, having one childhood impairment predicted steeper declines in HGS for men, but childhood misfortune was unrelated to HGS change among women. Hispanic Americans had lower baseline HGS than their non-Hispanic counterparts and manifested steeper declines in HGS.

Discussion: The relationship between childhood exposures and adult HGS varied by the type of misfortune, but there was no evidence that the relationship varied by race/ethnicity. The significant and enduring Hispanic disadvantage in HGS warrants greater attention in gerontology.

Keywords: Childhood disadvantage—Disability—Life course epidemiology

Research on the early origins of adult health has emerged as a notable theme in gerontology. There has always been interest in the health and functional problems of older people, but greater attention in recent years has been given to the development of those problems. Pioneering studies during the past two decades revealed a higher prevalence of adult health problems due to diseases during childhood (Blackwell, Hayward, & Crimmins, 2001) or harsh living conditions while growing up (O'Rand & Hamil-Luker, 2005). This line of research has provided evidence for the utility of a life-course approach to health and fostered fresh examination of resilience in the face of early-life insults (Seery, Holman, & Silver, 2010).

Two limitations of the literature on early origins merit attention. First, most research on the early origins of adult health is based on self-reported measures of adult health. Considerable research shows that these measures are generally valid and reliable (Ferraro & Farmer, 1999), but questions asking whether a "doctor ever told" the respondent they have a condition are contingent on the use of medical services. Self-reported measures will remain a central component of research on health, but objective measures of later-life functional health

are a necessary complement to self-reported measures. To our knowledge, relatively few studies of the early origins of adult health have used objective measures of physical performance as their outcome (e.g., Haas, Krueger, & Rohlfsen, 2012).

Second, few studies give attention to racial/ethnic differences in the study of adult health's early origins. Part of this may be due to the richness of longitudinal data in locations that had relatively modest racial diversity decades ago (e.g., British National Survey of Health and Development). Other studies may underrepresent population diversity or lack sufficient cases to test for racial/ethnic differences. It is clear that there is substantial racial and ethnic variation in stress exposure during early life (Turner & Avison, 2003). What is less clear is whether those differences in early stress exposure translate into compromised physical functioning in later life. There are many reasons why handgrip strength (HGS) may vary by race/ethnicity, but we focus on whether the influence of childhood misfortune operates differently for Black, White, and Hispanic Americans. To examine this question systematically, it is also important to consider adult status and resources, which can alter the potential consequences of early negative exposures. This study, therefore, examines the relationship between childhood misfortune and an objective performance measure within a diverse sample of older adults. We focus on HGS because studies show it can be used as a marker of late-life conditions such as frailty (Syddall et al., 2003) and sarcopenia (Sayer, Syddall, Gilbody, Dennison, & Cooper, 2004). Few researchers have investigated racial/ethnic variability in HGS; however, given the persistent racial/ethnic gaps in functional health outcomes associated with HGS such as disability (Warner & Brown, 2011), we examine potential racial/ethnic disparity in HGS. Moreover, if childhood misfortune is related to HGS, it may be a harbinger of other health problems and a way to detect emergent racial/ethnic disparities. We ask whether the relationship between childhood misfortune and HGS varies across non-Hispanic White Americans, non-Hispanic Black Americans, and Hispanic Americans. Owing to major differences in HGS between men and women, we also examine sex differences.

Significance and Theoretical Approach

Considerable research demonstrates that early-life misfortune raises the risk of health problems during later life, including heart attack (O'Rand & Hamil-Luker, 2005), cancer (Morton, Schafer, & Ferraro, 2012), and disability (Bowen & Gonzalez, 2010). Most studies examining morbidity and/or disability rely on self-reported adult measures, but some have examined performance measures—including HGS—and shown negative associations between childhood misfortune and adult functional ability. The type of childhood misfortune examined by most studies is socioeconomic status (SES; Birnie et al., 2011; Starr & Deary, 2011), but Haas and colleagues (2012) investigated the influence of both childhood SES and health on HGS. Their article is also important because the cross-sectional analyses revealed that Hispanic Americans have lower HGS than either White or Black Americans—one of the few studies to explore racial/ethnic variability in HGS. We seek to build on these studies by incorporating longitudinal analyses and examining a more comprehensive inventory of childhood misfortune. Performing both cross-sectional and longitudinal analyses will allow us to establish baseline models of HGS and then subsequently examine how HGS changes over time; steeper declines in HGS may indicate early-onset issues and reveal emerging racial/ethnic disparities.

Multiple Domains of Misfortune

Although the literature on the early origins of adult health has been greatly aided by studies examining a single negative exposure in childhood, negative experiences may be related. Failure to consider multiple types of misfortune may lead to overestimating the effect of a single exposure. For instance, some studies examine whether poor health during childhood influences health in later life, whereas others focus on the effects of financial strain on adult health. Although such studies are helpful, the poor health of a child (or other household member) may lead to or exacerbate financial strain (e.g., expenses of health care or caregiving). Alternatively, financial strain can lead to or exacerbate a child's health problem (e.g., nutritional deficiency). Drawing from cumulative inequality theory, disadvantage in one life domain may spill over to others (Ferraro & Shippee, 2009). Moreover, besides interfering with important child development processes, negative experiences may also be consequential because most children have limited ability to control undesirable situations. Some researchers argue for investigating the cumulative toll of negative experiences, reflecting a dose–response relationship (Brown et al., 2009). Others prefer to distinguish across domains (O'Rand & Hamil-Luker, 2005), but the overarching point is to consider multiple domains of early misfortune.

Social Stratification of Misfortune

The idea of multiple domains of childhood misfortune implies a consideration of accumulated difficulty. All of us will face hard times—consistent with Longfellow's "into each life some rain must fall" (*The Rainy Day*)—but such rain does not fall randomly. Some people face frequent hard times or a cascade of setbacks and catastrophic problems, whereas others live for long spells without major misfortune, even if beset by daily hassles. The fact that a child may have to face both a serious health condition and household financial strain is daunting enough, but the sequelae are predictably worse if accompanied by violence or substance abuse in the home or neighborhood.

The variability in exposure to misfortune is not due solely to personal choices but shaped by processes of social stratification. According to Max Weber (1978), social

stratification unfolds at the nexus of life choices and life chances. He defined the latter as related to one's class situation: "the typical chance for a supply of goods, external living conditions, and personal life experiences" (Weber, 1922/2001, p. 114). Especially for children, life chances are tightly linked to the social structure of their development, and early differences in status and quality of life lead to greater inequality within cohorts (Ferraro & Shippee, 2009).

Weber (1922/2001) also observed that these stratification processes are often more extreme when linked to racial and ethnic segregation. Race and ethnicity are major axes of stratification, differentiating life chances, either for privilege or hindrance, and thereby differentiating stress exposure (Turner & Avison, 2003). In the United States, childhood experiences are quite different by race/ethnicity. For instance, Black, Hispanic, and Native American children are more likely than White or Asian American children to live in poor households (Lichter, Qian, & Crowley, 2006; Macartney, 2011), which, in turn, means that they are more likely to attend schools with a higher dropout rate and be exposed to more risk in their neighborhoods (e.g., violence, air pollution; Williams & Collins, 2001). Poor childhood health, another type of misfortune, also varies by race/ethnicity (Flores, Olson, & Tomany-Korman, 2005). Thus, there is ample evidence to expect that Black, White, and Hispanic children have distinct early-life experiences that may lead to long-term effects on physical health. Moreover, the childhood of today's older Black and Hispanic Americans was generally harsher than that of more recent cohorts who grew up after the Civil Rights Movement.

Stratification processes likely mean that adult resources such as education, wealth, marital status, and a culture of health promotion are quite different by race/ethnicity, which, in turn, may influence one's ability to cope with childhood misfortune and its sequelae. Social stratification is related to access to health care and norms of daily physical activity—and racial/ethnic subcultures add another layer of meaning for whether and how to access those resources. Distinct childhood exposures coupled with differential access to adult resources to address misfortune may result in relationships between childhood disadvantage and functional health that are specific to race/ethnicity.

This study, therefore, poses two main research questions.

1. Is childhood misfortune associated with lower HGS in later life? Based on prior research on the early antecedents of adult health, we expect that childhood misfortune will be associated with poorer health in later life, specifically (a) lower initial levels of adult HGS and (b) more rapid declines in HGS (Starr & Deary, 2011).
2. Are there racial/ethnic differences in the relationship between childhood misfortune and HGS? Based on prior research documenting more misfortune among racial/ethnic groups as well as differential adult status and resources, we anticipate that (a) Hispanic and Black Americans will have lower HGS than White Americans and (b) the effect of childhood misfortune on HGS will be stronger for Hispanic and Black Americans than for White Americans (Haas et al., 2012). These anticipated relationships (a and b) require testing for main effects and interaction effects, respectively.

Method

Analyses used panel data from the 2004–2012 waves of the Health and Retirement Study (HRS, 2014). Multistage stratified panel data on individuals aged 50 and older are collected, with oversamples of Black adults, Hispanic adults, and Floridians. Supplementary Material contains a detailed description of the HRS.

Handgrip Strength

After a pilot sample in 2004, the HRS began collecting physical performance measures in 2006. Sample design specifies HGS measurement for a random half of the sample every 4 years. Therefore, one half of the sample was measured in 2006 and again in 2010; the other half was measured in 2008 and 2012. The 2006 and 2008 measurements were designated Time 1 (T1); the 2010 and 2012 measurements were Time 2 (T2). Longitudinal analyses are important to the study of the early origins of adult health to examine if potential effects are exhausted at the baseline HRS measurement or continue as people grow older (i.e., change over the study).

HGS measurements are recorded in kilograms for participants without medical issues (e.g., swelling) in both hands during the 6 months prior to the survey (Crimmins et al., 2008). A Smedley spring-type dynamometer was fit to the respondent to take two measurements on each hand; HGS for each survey occasion was the average of four measurements.

Childhood Misfortune Variables

We identified 27 dichotomous indicators of childhood misfortune; each represents a condition experienced before age 18 (1, experienced misfortune). Based on theory, confirmatory factor analyses, and prior literature (Felitti et al., 1998; Morton, Mustillo, & Ferraro, 2014), indicators were organized into five domains: infectious disease, chronic disease, impairment, SES, and risky parental behavior. The final domains were constructed as a count of indicators for each domain. Supplementary Material provides specific information on the indicators used in each domain. To examine the threshold effect of having at least two misfortunes compared with one, each count was top coded at 2, resulting in three categories of misfortune per domain: 0, 1, and 2+. Distributions of raw counts for each domain showed that in general few responses were changed by this top-coding scheme. We performed sensitivity analyses comparing this coding scheme with a binary variable for each

domain (1, if any misfortune experienced). The same trends were seen using dichotomous domains, but were driven by the influence of having two or more misfortunes, revealing the utility of three categories. This decision also emphasizes the concept of accumulated misfortune, a central tenet of cumulative inequality theory.

Covariates

Given that race and ethnicity are focal variables, all models include three dichotomous and mutually exclusive categories: non-Hispanic White Americans (reference group; hereafter White Americans), non-Hispanic Black Americans (hereafter Black Americans), and Hispanic Americans (including 26 respondents reporting Black and Hispanic). Preliminary analyses examined binary variables to differentiate Hispanics of Mexican, Puerto Rican, and Cuban descent, but there was no evidence that the conclusions varied across the groups.

Intermediate variables on the pathway relating childhood misfortune and HGS were taken from the baseline (2004) wave of the HRS and included wealth, educational attainment, marital status, depressive symptoms, physical activity, smoking status, body mass index, and chronic conditions. See Supplementary Material for detailed methodology on the measurement of these covariates.

Whereas HGS is dependent on age, we included age at the time of HGS measurement and an indicator variable for each time period to adjust for the staggered measurement within each (2006 or 2008; 2010 or 2012). Because the relationship between age and HGS may not be linear, we also included a squared term for age.

We identified potential confounding and intermediate variables on the relationship between childhood misfortune and HGS. To control for possible confounding variables, models were sex stratified and adjusted for immigration status (1, if born outside United States).

Analytic Design

After excluding participants who were deceased, age ineligible, or had validity issues (i.e., proxy response) in 2004, the sample size was 13,921. Regression analyses use individuals with complete HGS measurements at the time period analyzed, and descriptive statistics are presented for individuals included in regression models.

Descriptive statistics for HGS and childhood misfortune are stratified by race/ethnicity and sex (six groups) in Table 1; parallel information for all variables is presented in Supplementary Tables S1 and S2. Within gender, we performed analysis of variance or χ^2 tests for race/ethnic differences in variables (Table 1; Supplementary Tables S1 and S2) and estimated regression models using ordinary least squares (OLS) analyses in Stata 14. Analyses used the HRS physical measure weights corresponding to the time period when the respondent was measured to adjust for differential selection and nonresponse to physical measures.

We estimated three regression models per gender and time period. T1 (baseline) and T2 (residualized change) models were estimated using the two full sets of measurement. In the change model, HGS, T2 was regressed on HGS, T1 to study change over time (longitudinally).

Model 1 contains indicators of race/ethnicity, immigration status, linear and quadratic age terms (centered on average age), and an indicator of year of measurement within time period. Model 2 adds childhood misfortune indicators. Model 3 adds adult factors to examine their potential intermediate effects. Finally, product terms of

Table 1. Descriptive Statistics From the Health and Retirement Study, 2004–2012

Variable	White men	Black men	Hispanic men	White women	Black women	Hispanic women
HGS, Time 1 (kg)	*37.71*[a] (9.19)[b]	*37.25* (9.67)	*34.92* (8.63)	*22.13* (5.82)	*24.09* (6.27)	*20.78* (5.55)
HGS, Time 2 (kg)	*35.41* (9.13)	*35.81* (8.54)	*32.55* (8.35)	*21.20* (5.69)	*23.30* (5.85)	*19.41* (5.67)
Childhood misfortune[c]						
Infectious disease (= 1)	*0.13*	*0.16*	*0.23*	*0.12*	*0.18*	*0.25*
Infectious disease (= 2+)	*0.80*	*0.70*	*0.49*	*0.83*	*0.73*	*0.58*
Chronic disease (= 1)	0.21	0.17	0.24	*0.22*	*0.16*	*0.17*
Chronic disease (= 2+)	0.09	0.09	0.07	0.13	0.12	0.11
Impairment (= 1)	*0.19*	*0.16*	*0.11*	*0.15*	*0.13*	*0.09*
Impairment (= 2+)	0.04	0.03	0.02	0.02	0.02	0.01
SES (= 1)	*0.21*	*0.18*	*0.13*	*0.20*	*0.21*	*0.15*
SES (= 2+)	*0.55*	*0.68*	*0.76*	*0.59*	*0.65*	*0.76*
Parental behavior (= 1)	*0.59*	*0.51*	*0.57*	*0.55*	*0.47*	*0.51*
Parental behavior (= 2+)	0.14	0.11	0.11	*0.16*	*0.08*	*0.09*
Number of cases (Time 1)	3,713	438	308	5,046	855	502

Note: ANOVA = analysis of variance; HGS = handgrip strength; SES = socioeconomic status. Italics indicate significant ANOVA or χ^2 tests between Black, White, and Hispanic Americans for each sex.

[a]Mean. [b]Standard deviations are reported in parentheses. [c]Proportion with 0 childhood misfortune not shown (total adds to 1 for each misfortune).

childhood misfortune by race/ethnicity were tested in Model 3 to address our second research question. Twenty models, each with four product terms, were estimated to test for interactions at T1 and T2 for both men and women. Testing 20 models, each with four product terms, requires adjusting the significance values to avoid rejection by random chance. Due to the high number of related hypothesis tests being conducted, an adjusted significance cutoff (0.00256) was obtained using a step-down procedure to control for the family-wise error rate (Benjamini & Liu, 1999).

Supplementary Material also presents methods and results for additional analyses. These analyses tested corresponding male and female coefficients, estimated models using each domain of misfortune to independently predict HGS, and examined selection bias.

Results

Significant differences in HGS by race/ethnicity for both men and women are noted in Table 1 by italicized means. For men and women, Hispanic Americans had the lowest HGS among the three groups at T1 and T2. Black women had the highest HGS over both time periods. White men had the highest HGS at T1, but Black men had slightly higher HGS at T2.

Among both men and women, White Americans were most likely to report childhood infectious disease, impairment, and risky parental behavior. For both men and women, Hispanic Americans were most likely of the three groups to report SES misfortune. Descriptive statistics for all variables used in analyses are presented separately for men and women in Supplementary Tables S1 and S2, respectively.

Table 2 presents unstandardized OLS regression coefficients for focal variables and R^2 values. Within each gender, we present three models per time period (T1 and T2): Model 1 establishes a baseline relationship between demographic characteristics and the outcome; Model 2 adds childhood misfortune; and Model 3 adds adult covariates. The same models but with all variables and standard errors are shown in online Supplementary Tables S3 and S4 for men and women, respectively.

Men

Baseline models

Black and Hispanic men had significantly lower HGS than White men (Model 1 of Table 2 and Supplementary Table S3). Consistent with our hypotheses, Model 2 shows that HGS at T1 is associated with several indicators of childhood misfortune (2+ infectious diseases, 2+ chronic diseases, and 2+ SES disadvantages). However, differential exposure to childhood misfortune across race/ethnicity contributed little to race/ethnic disparities in HGS at T1. Specifically, adjusting for childhood misfortune in Model 2 explained just more than 14% of the Black-White difference and 9% of the Hispanic-White difference in HGS. In Model 3, we found that after adjusting for adult factors the Black-White difference was no longer significant ($p > .05$); 73% of the Black-White difference in Model 2 was due to adult resources and behaviors. By contrast, adjusting for adult resources and behaviors explained just 9% of the Hispanic-White difference in HGS. In Model 3, men who reported 2+ risky parental behaviors manifested higher HGS values.

Change models

Hispanic men exhibited steeper declines in HGS than White men, but change in HGS did not differ between Black and White men (Model 1). Again consistent with our hypotheses, Model 2 shows that steeper declines in HGS are associated with multiple domains of childhood misfortune (1 impairment and 2+ SES disadvantages). Differential exposure to childhood misfortune across ethnicity, however, contributed little to the Hispanic disparity in HGS change over time. Adjusting for childhood misfortune in Model 2 explained just 5% of the Hispanic-White difference. In Model 3, we found that adjusting for adult resources and behaviors explained more than 15% of the Hispanic-White difference in changing HGS.

Wealth, physical activity, and the absence of depressive symptoms emerged as significant adult factors in both the baseline and change analyses in men.

Women

Baseline models

Hispanic women had significantly lower HGS than White women (Model 1 of Tables 2 and Supplementary Table S4). Distinct from men, Model 2 shows that only one type of childhood misfortune (2+ chronic diseases) predicted HGS at T1. Adjusting for childhood misfortune in Model 2 increased the Hispanic-White difference in HGS by 1.5%. In Model 3, adjusting for adult factors revealed that Black women had higher HGS than White women at T1 and that the adult resources and behaviors explained about 30% of the Hispanic-White difference in HGS. Model 3 also showed that women with 2+ indicators of low SES reported higher HGS values.

Change models

Hispanic women manifested steeper declines relative to White women (Model 1). In contrast to our expectations, none of the indicators of childhood misfortune were associated with change in HGS (Models 2 and 3). In Model 3, we found that adjusting for adult resources and behaviors did not explain the Hispanic-White difference in changing HGS. Indeed, R^2 values changed very little across the three models; initial HGS was strongly related to HGS change by T2. Parallel to Model 3 for T1, we note that HGS change among Black women was more favorable than for White women.

Table 2. OLS Regression of Handgrip Strength in Men and Women

	Men, Time 1 (n = 4,459)			Men, Time 2 (n = 3,322)			Women, Time 1 (n = 6,403)			Women, Time 2 (n = 4,572)		
Independent variables	M1[a]	M2	M3[b]	M1	M2	M3	M1	M2	M3	M1	M2	M3
Black	*−1.79*‡	*−1.53**	−0.42	*0.25*	0.39	0.79	*0.62*	*0.57*	1.25†	0.38	0.40	0.50*
Hispanic	−3.98†	−3.61†	−3.28†	*−2.40*†	*−2.27*†	−1.92†	−2.66†	−2.70†	−1.89†	*−0.67**	*−0.70**	−0.70*
Immigrant	−1.81‡	*−1.55**	−1.88‡	*0.53*	*0.68*	*0.51*	*−0.15*	*−0.16*	*−0.15*	−0.18	−0.15	−0.18
Age	*−0.52*†	*−0.51*†	*−0.46*†	*−0.22*†	*−0.21*†	*−0.22*†	*−0.31*†	*−0.31*†	*−0.28*†	*−0.14*†	*−0.14*†	*−0.14*†
Age^2	−0.01‡	−0.01‡	−0.00*	−0.00	−0.00*	−0.00	−0.00*	−0.00*	−0.00	−0.00	−0.00	−0.00*
HGS, Time 1				0.69†	0.69†	0.67†				0.68†	0.67†	0.67†
Infectious disease (= 1)		0.86	0.62		−0.18	−0.40		0.17	−0.09		−0.27	−0.30
Infectious disease (= 2+)		1.49*	1.06		0.47	0.25		0.22	−0.04		−0.12	−0.14
Chronic disease (= 1)		−0.53	−0.48		−0.13	−0.08		−0.23	−0.12		−0.17	−0.14
Chronic disease (= 2+)		−1.62*	−1.24		0.33	0.34		−0.57‡	−0.27		−0.40	−0.35
Impairment (= 1)		−0.41	−0.09		−0.71*	−0.60*		−0.26	−0.19		0.04	0.05
Impairment (= 2+)		−1.27	−0.40		−0.21	0.21		−0.65	−0.25		−0.77	−0.68
SES (= 1)		−0.39	−0.19		−0.50	−0.30		0.08	0.41		−0.08	−0.02
SES (= 2+)		−0.83*	−0.38		−0.62*	−0.28		0.09	0.63‡		−0.06	0.03
Parental behavior (= 1)		0.07	0.27		−0.08	0.00		−0.05	−0.07		0.11	0.11
Parental behavior (= 2+)		0.50	0.91*		0.15	0.36		−0.18	−0.01		0.13	0.14
Constant	38.85†	38.34†	32.06†	9.17†	9.51†	8.95†	22.86†	22.82†	19.43†	6.08†	6.33†	6.64†
R^2	.28	.29	.34	.64	.64	.65	.28	.28	.31	.64	.64	.65

Note: BMI = body mass index; HGS = handgrip strength; OLS = ordinary least squares; SES = socioeconomic status. Unstandardized regression coefficients are presented. Italicized coefficients indicate significantly different coefficients for men and women. Reference group for each of the five domains is no misfortune.

[a]M1 = Model 1; M2 = Model 2; M3 = Model 3. [b]M3 adjusts for education, wealth, BMI, former smoking status, current smoking status, physical activity, depressive symptoms, marital status, and chronic conditions.

*$p < .05$; ‡$p < .01$; †$p < .001$.

Examination of residuals revealed that a higher percentage of Black women experienced an increase in HGS from T1 to T2, relative to White women (i.e., not just smaller declines).

Physical activity and fewer chronic conditions emerged as significant adult factors in both the baseline and change analyses in women.

Tests of Interactions

To address our second research question, we specified a series of product terms to examine race/ethnic differences in vulnerability to childhood misfortune. This involved testing interactions between race/ethnic groups and each domain of childhood misfortune. Results from the 20 models are summarized in Table 3. The complete results showing slopes and standard errors are presented Supplementary Tables S5–S8.

As shown in Table 3, there is no evidence that the effect of any domain of childhood misfortune differentially affects the HGS of Black, Hispanic, and White Americans.

Discussion

The study addressed two main research questions. The first probed whether childhood misfortune is associated with lower HGS in later life. Findings revealed that certain domains of misfortune are associated with HGS in adulthood, but relationships are distinct for men and women—and not always in the expected direction. Consistent with other studies, we anticipated that early misfortune would take a toll on the aging process and result in lower HGS (Birnie et al., 2011; Sayer et al., 2004). This was true for baseline HGS in men and women: Two or more chronic diseases in childhood predicted lower HGS, but the relationships were attenuated after adjustment for adult resources. A surprising conclusion was that some domains predicted better baseline HGS, and the findings were distinct for men and women.

In men, 2+ infectious diseases during childhood actually were predictive of better adult HGS before adjustment, suggestive of an acquired immunity thesis (Preston, Hill, & Drevenstedt, 1998). Few studies report that infectious diseases during childhood are associated with adult health (Blackwell et al., 2001); however, this study provides evidence that multiple infectious diseases during men's childhood are associated with better HGS. Having at least two of these infectious diseases (measles, mumps, and chickenpox) could be seen as an accumulated immune advantage at the time (vaccines for these diseases were not available until the late 20th century). After adjustment, multiple risky parental behaviors also predicted better HGS among men at baseline, suggesting a compensatory mechanism (Seery et al., 2010).

Table 3. Comparison of R^2 Values for Models Testing Four Interaction Terms for Each Domain[a]

Model	CM domain	Base R^2	Interaction R^2	p Value
Male	Infectious disease	.3379	.3392	.3637
Time 1	Chronic disease	.3379	.3385	.6061
	Impairment	.3379	.3385	.4308
	SES	.3379	.3386	.3579
	Parental behavior	.3379	.3390	.1641
Male	Infectious disease	.6518	.6522	.1642
Time 2	Chronic disease	.6518	.6523	.3275
	Impairment	.6518	.6519	.2593
	SES	.6518	.6521	.4846
	Parental behavior	.6518	.6520	.6617
Female	Infectious disease	.3148	.3155	.1940
Time 1	Chronic disease	.3148	.3156	.2384
	Impairment	.3148	.3153	.6356
	SES	.3148	.3154	.6205
	Parental behavior	.3148	.3156	.0573
Female	Infectious disease	.6453	.6455	.7395
Time 2	Chronic disease	.6453	.6457	.1701
	Impairment	.6453	.6456	.0204
	SES	.6453	.6454	.9617
	Parental behavior	.6453	.6456	.6461

Note: CM = childhood misfortune; SES = socioeconomic status.

[a]Four product terms were tested for each model (Black × CM1, Black × CM2+, Hispanic × CM1, Hispanic × CM2+). Base R^2 is the corrected R^2 from the fully adjusted M3. Each group of four product term interactions was entered into the adjusted model and tested for joint significance by assessing whether the set of interactions significantly increased the R^2 compared with the base model R^2. Due to the high number of related hypothesis tests being conducted, an adjusted significance cutoff (0.00256) was obtained using a step-down procedure to control for the family-wise error rate (Benjamini & Liu, 1999; multproc command in Stata 14 using method=liu1). None of the product term interaction groups were significant.

In contrast to men, SES misfortune (e.g., financial strain when growing up) predicted better HGS among Black, White, and Hispanic women after adjustment for adult factors. When comparing these results with prior studies, two points are noteworthy. First, it could be that the distinct relationships reported herein for men and women may not be discernible in studies combining the sexes. Second, the present study used a more comprehensive measure of childhood SES and uncovered the importance of accumulated misfortune on women's HGS. One might speculate that financially disadvantaged families were more likely to call on daughters to help with household tasks involving manual dexterity, but there are no data in the HRS to examine this thesis.

The analyses revealed the influence of early disadvantage on baseline HGS, but any additional declines over the study period appear to be largely contingent on adult resources and lifestyle. After accounting for adult factors, experiencing one impairment was the only type of childhood misfortune that predicted HGS declines in men. This finding was surprising; however, the same association was seen in the supplementary simple regressions.

Thus, rather than concluding that childhood misfortune leaves an amplifying imprint on lifelong health and function, this study provides evidence of early influence but also that such influence may operate through adult factors. Therefore, interventions and factors of adult health, SES, or psychosocial status may be able to constrain negative consequences associated with early insults. There is plasticity involved in the influence of the types of early misfortune considered; some types of presumed disadvantage (low SES) may lead to compensatory mechanisms to ward off unfavorable health outcomes (Elder & Liker, 1982). It is tempting to think of accumulated disadvantage as universally detrimental, but that is an oversimplification. This study revealed multiple exceptions to the presumption that early misfortune leads to more health problems and as such, investigators should anticipate a wider range of outcomes.

Future research on the early origins of adult health should also give more attention to formally analyzing adult factors as mediators. The present study identified that some adult factors were associated with better HGS in both the T1 and T2 analyses. Physical activity was the only adult factor that was associated with better HGS for both men and women at baseline and over time. Although people may regularly engage in physical effort to "stay active"—whether via walking, golf, or dancing—HGS may be a health dividend realized through a wide range of activities.

The second research question addressed whether the relationship between childhood misfortune and HGS varied across racial/ethnic groups. We began by estimating main effects for the influence of race/ethnicity on HGS, then turned to testing interaction effects. After estimating 20 models with multiple product terms to test for interactions, there was no evidence that the relationship between childhood misfortune and HGS varied by race/ethnicity. We anticipated that Black and Hispanic Americans would suffer more misfortune during childhood, which was the case for SES disadvantage, but it did not translate into distinctive effects on HGS due to early misfortune. Nevertheless, the magnitude of the main-effect differences by race/ethnicity is notable.

Consistent with prior research, this study revealed disparities in HGS among White, Black, and Hispanic Americans, even after adjusting for adult factors. As expected, Hispanic Americans manifested lower baseline HGS than their White American counterparts, but the present study also observed steeper declines in HGS for Hispanic Americans over the observational period. Although consistent with prior research, these observed disparities are surprising given that the average age of Hispanic Americans in this sample is younger than either White or Black Americans. When interpreting these results, it is critical to note that the mean educational attainment for Hispanic Americans was at least 2 years less than White or Black Americans. We interpret this educational disparity as likely resulting in fewer resources such as health insurance coverage, access to quality medical care, and other amenities that enhance healthful aging. The results also show that immigrant men also have lower HGS, again likely related to SES resources prior to immigration.

The data also showed racial differences between Black and White Americans. Lower baseline HGS was observed in Black men, but relative to White women, Black women had better initial levels of HGS and slower declines in HGS over time. The baseline finding for women was somewhat surprising given the extensive literature documenting higher levels of disability in the Black population (Haas et al., 2012; Hirsch et al., 2006; Warner & Brown, 2011). When examining potential explanations for the slower declines in HGS, supplementary analyses revealed that a higher percentage of Black women actually experienced an increase in HGS from T1 to T2, relative to White women.

Three limitations of the current study are notable. First, retrospective reports of childhood may contain bias. The HRS avoided types of questions known to be highly unreliable (e.g., asking for the date or age of an experience), and we excluded respondents with poor memory. Nevertheless, recall bias is possible. A review of validity studies showed that retrospective reports in adulthood of adverse childhood events generally have a higher rate of false negatives than false positives (Hardt & Rutter, 2004). False negatives would likely lead to an underestimation of the relationship between early misfortune and HGS; thus, caution is warranted when interpreting the results. Gender bias in reporting of childhood misfortune is also possible.

Second, whereas this study uses respondents at least 51 years of age, the findings should be interpreted in light of selective survival. Although we took steps to reduce selective sample survival bias (adjustments for sample weights and mortality), there could be population survivorship bias that we were unable to adjust for in our

analyses. Given our findings, this may apply particularly to Black Americans and the oldest members of our sample (Markides & Machalek, 1984). Adults who experienced the most childhood misfortune, along with higher risk of premature mortality, may not survive to be eligible for the HRS (Brown et al., 2009).

Third, although we examined potential differences among Mexican, Cuban, and Puerto Rican respondents, the relatively small numbers of the latter two categories did not allow for detailed sex-stratified analyses.

Despite the limitations, the current study contributes to the literature regarding childhood misfortune and adult health. Using a longitudinal approach enabled investigation of the influence of childhood misfortune and race/ethnicity on both baseline and change in HGS. These findings reveal the long-term influence of early insults on adult functioning and the plasticity involved. There was little evidence that childhood misfortune resulted in late-onset declines in HGS; rather, its influence is likely to be earlier in the life course.

Racial and ethnic differences, by contrast, influenced not only initial HGS but also change in HGS. Hispanic American men and women were notably disadvantaged, despite accounting for childhood misfortune and adult factors. Hispanic Americans manifested lower baseline HGS and steeper declines in HGS over time. Compared with their White counterparts, Black women manifested higher baseline HGS and Black men showed lower baseline HGS. Additionally, Black women have slower age-related declines than White women. This finding was surprising given that previous studies generally show Black Americans, namely women, to be disadvantaged on other measurements of muscle strength such as knee extension (Goodpaster et al., 2006). Thus, future research is needed to probe racial and ethnic differences in upper- and lower-body strength, especially among women.

Understanding the properties of a simple, objective, and cost-effective performance measure such as HGS is important to aid its utility in epidemiologic and clinical settings. HGS is a useful marker of frailty (Hirsch et al., 2006; Syddall et al., 2003) and also predicts mortality (Gale et al., 2007), speed of aging (Sanderson & Scherbov, 2014), and cognitive decline (Alfaro-Acha et al., 2007). With better understanding of pathways influencing HGS as well as its racial/ethnic disparities, clinicians can use HGS to document function before and after interventions or identify patients at risk. Given our findings that modest forms of childhood misfortune are associated with better baseline HGS, research is needed to investigate pathways, especially compensatory mechanisms.

Supplementary Material

Please visit the article online at http://psychsocgerontology.oxfordjournals.org/ to view supplementary material.

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