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Structural Inequity and Socioeconomic Status Link to Osteoporosis Diagnosis in a Population-Based Cohort of Middle-Older-Age Americans

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Abstract

Socioeconomic status (SES) is an important social determinant of health inequities that has been linked to chronic conditions, including osteoporosis, but research tends to focus on socioeconomic disadvantage rather than how socioeconomic advantage may facilitate these inequities. This study accounts for structural inequities and assesses the relationship between early-life and later-life SES, and risk of osteoporosis diagnosis. Data come from the nationally representative, population-based cohort Health and Retirement Study and include individuals ages 50 to 90. The outcome variable is osteoporosis diagnosis. Logistic regression models of the relationship between SES and osteoporosis diagnosis are estimated, accounting for demographic, health, and childhood variables. Higher levels of childhood and adult SES link to lower odds of osteoporosis diagnosis. Structural inequities in income and underdiagnosis of osteoporosis among persons identifying as Black/African American were detected. Accounting for bone density scan access, inequities in osteoporosis diagnosis appear to stem from barriers to accessing health care due to financial constraints. The important role of SES and evidence of structural inequities leading to underdiagnosis suggest the critical importance of clinicians receiving Diversity, Equity, and Inclusion training to reduce health inequities.

Keywords

bone mineral density, income, childhood adversity, health inequalities, socioeconomic disparities in health, osteoporosis, structural inequities, socioeconomic status

What do we already know about this topic?
Social inequalities and systemic racism are barriers to accessing health care in the United States, and socioeconomic status is a fundamental cause of health disparities.

How does this research contribute to the field?
We estimate the impacts from socioeconomic inequalities when parsing out the effects of systemic racism, showing systemic racism is a greater barrier to accessing osteoporosis-diagnosis-related care than socioeconomic status.

What are the research's implications toward theory, practice, or policy?
Inequities in osteoporosis diagnosis stem more from structural racism than from socioeconomic status; clinicians must receive Diversity, Equity, and Inclusion training to reduce health inequities.

Socioeconomic status (SES) is an important determinant of health disparities in the United States and is linked to systemic inequities, including those deriving from racial inequity.[1] Health disparities are a significant economic burden; as of 2018, health disparities in the United States were estimated to cause $93 billion in excess medical costs and an additional $42 billion in lost labor market productivity.[2] This underscores the critical need to research the causes of, and solutions to, this financial catastrophe.

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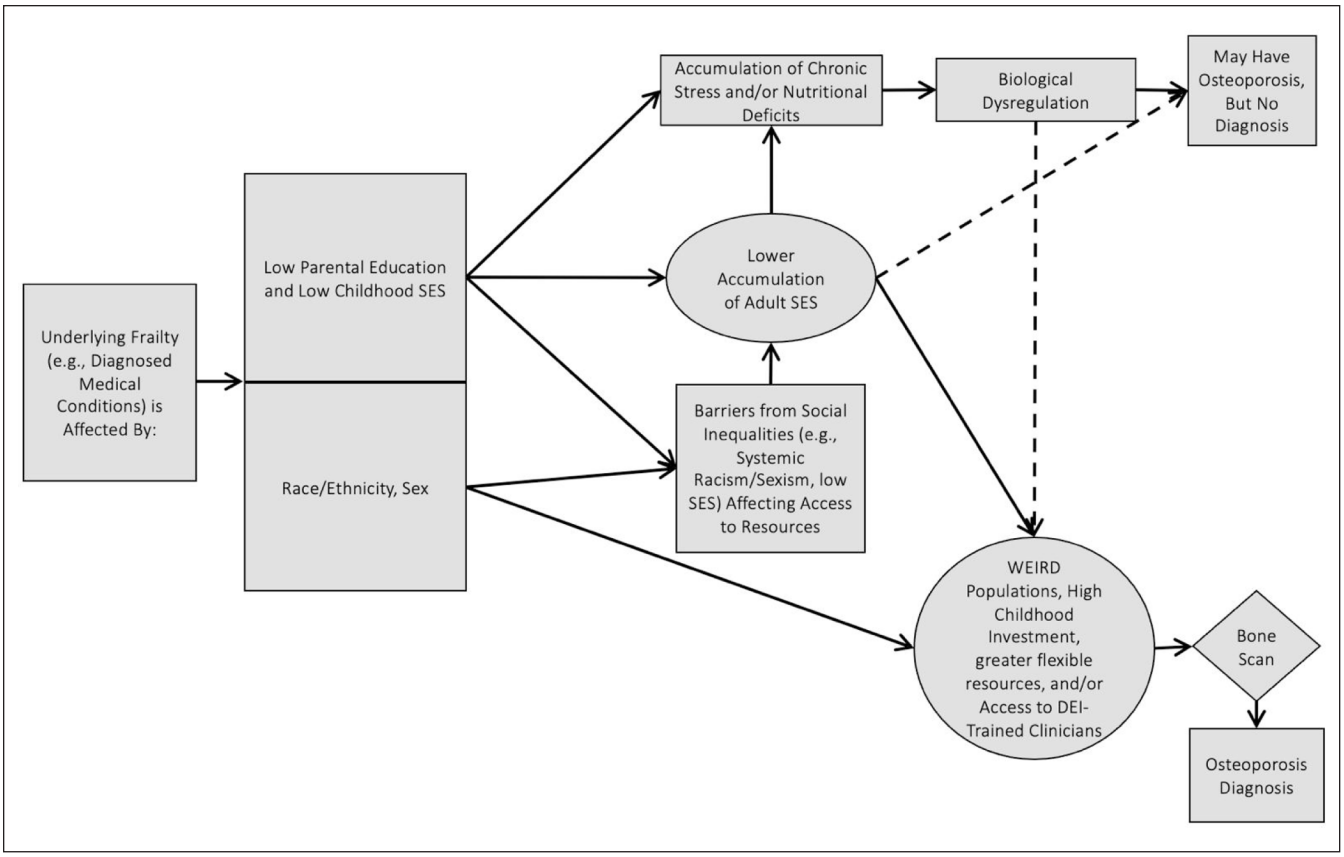

Figure 1. Conceptual model derived from Barr,[4] Kubzansky et al,[5] Riancho and Brennan-Olsen,[7] Gough Courtney et al,[14] Godde et al,[13] and Phelan et al.[9] WEIRD = Western, educated, industrialized, rich, and democratic societies.

SES has been conceptualized and measured in a number of ways. Common approaches include reliance on income (eg, family or household level), educational attainment, wealth, occupation, parental education or income (in the case of measuring children's SES), and sometimes socioeconomic position or social class (eg, low-income, middle class, upper class, or high-income).[3] There are strengths and weaknesses of each, but for health disparities the measures usually lead to similar results, broadly speaking.[3] It is established that current and prior SES in adulthood influence health, mortality, and risk of disease, but little is known about the role of adult SES while accounting for the childhood SES environment, and how these are linked with adult health and demographic factors.

The objective of this study is to examine how childhood SES and adult SES are together related to risk of osteoporosis diagnosis in a sample of middle-older age adults in the longitudinal Health and Retirement Study (HRS), while accounting for relevant adult health and demographic characteristics. Furthermore, we study these relationships in the context of understanding the key role of structural and systemic inequities in generating differences in SES and health care access, and cognizant of the importance of conceptualizing socioeconomic advantage for health, not only socioeconomic disadvantage. We hypothesize that lower childhood SES is a key predictor of higher risk of osteoporosis (with a potentially lower likelihood of actual diagnosis) but that higher adult SES is a key predictor of lower risk of osteoporosis (and an improved likelihood of diagnosis when the condition is present). The findings shed light on how inequities in osteoporosis diagnosis could be mitigated despite entrenched socioeconomic disparities.

Background and Theory

We describe 2 major pathways through which SES might affect health outcomes. First, socioeconomic strain and broad social inequities can lead to physiological responses in the body that precipitate negative health outcomes (eg, chronic inflammation, poorer bone health) for example as described by Barr[4] and Kubzansky et al.[5] Social epidemiological conceptual models explicate how environmental and social factors can lead to physiological changes in the body, which have downstream consequences for a person's health[4-7] (see upper pathway of Figure 1). In this framework, structural and systemic inequities influence access to resources but also cause chronic stress and strain, which initiates a response from the hypothalamic-pituitary-adrenal (HPA) axis, leading

to a rise in allostatic load within the body, increased levels of inflammatory biomarkers,[4] and potentially, increased risk of osteoporosis and other disease outcomes. Early life experiences, along with adult SES, may be important determinants of health outcomes. The precipitating structural and systemic inequities are produced and maintained by advantaged social groups. Discrimination based on SES may occur similar to discrimination based on race or ethnicity,[8] which could also influence HPA dysregulation. This pathway focuses on the role of socioeconomic disadvantage for health.

The second pathway details a lack of socioeconomic resources that can limit an individual's ability to achieve health goals, access health care, and make healthy choices,[1,9] while the opposite is also true. Fundamental causes of health disparities identify SES as one fundamental cause with 4 distinct features[9]: (1) Inclusion of flexible resources (ie, resources that can be used to "avoid risks or to minimize the consequences of disease once it occurs," including "money, knowledge, power, prestige, and the kinds of interpersonal resources embodied in the concepts of social support and social network"[10] (p. 87)) that influence multiple disease outcomes[9]; (2) It affects disease through multiple risk factors[9]; (3) It is used to minimize or avoid risk and complications[9] as socioeconomic resources may be used to reduce disease exposures and improve disease outcomes through access to better health care; and (4) The effects are reproduced over time, although mechanisms can change.[9] Bone densitometry for osteoporosis screening is one such mechanism; it is a diagnostic tool that was not available to everyone when it was first introduced, and those with greater socioeconomic resources had more access, although this may still be an issue.[11] Over time, access became more universal through Medicare, reducing unequal access to a scan. Yet, while access to scans improved, SES can still result in disparities related to osteoporosis, such as through access to treatment, ability to see specialists, and related issues.[12-14] The fundamental cause framework differs from the first potential pathway as it suggests SES will affect health outcomes, regardless of whether it results in chronic stress, in large part because flexible resources (lower right in Figure 1) provide important advantages for health.

Both potential pathways (chronic strain/physiological and SES as a fundamental cause) are rooted in an understanding of underlying structural and systemic inequities in society. Levels of SES are racially patterned in the United States, and in the same way that SES is a fundamental cause of health inequities, so too is racism.[1] Phelan and Link[1] argue that racism influences health outcomes primarily through SES, but not entirely; thus, racial differences in health extend beyond those that can be traced to SES. Both pathways are specifically related to systemic racism, which is based on the idea of the involvement of entire systems, such as the health care system or the economic system, including the structures that make up the systems; by comparison structural racism occurs at the structural level.[15] Dennis et al[16] (p. 302) define structural racism as: ". . .a constellation of macro-level systems, social forces, institutions, ideologies, and interactive processes that generate and reinforce inequities among racial and ethnic groups." Evidence of the role of structural and systemic racism in health has expanded considerably in recent years, and specifically in osteoporosis diagnosis and care (see discussion in Godde et al.)[13] Systemic discrimination is one mechanism through which structural racism plays out, with policies that advantage those in the majority group (ie, people identifying as Non-Hispanic White in the United States) to the detriment of other racial and ethnic groups.[15,16] Systemic and structural discrimination constrain access to the flexible resources described by Phelan and Link (eg, socioeconomic resources),[1,9] and contribute to health inequities by limiting opportunities and constraining life chances.[16] (p. 302).

Often, research using the 2 theoretical frameworks described focuses on disadvantage. Recently, Link and García[8] called for greater focus on how social and socioeconomic *advantage* influences outcomes and how the actions of advantaged groups can produce inequities in health outcomes, consistent with fundamental cause theory. Our study draws on fundamental cause theory,[10] to consider more explicitly the role of advantages and flexible resources as contributors to disparities in osteoporosis diagnosis. This is denoted in the Figure 1 bubble containing "WEIRD Populations, High Childhood Investment, greater flexible resources, and/or Access to DEI-Trained Clinicians." We focus on osteoporosis as an outcome because it is a common, progressive disease[17] that is underdiagnosed[18] and leads to a significant morbidity and mortality burden, including reductions in quality of life.[19]

Prior Research

Numerous studies link SES to risk of osteoporosis, though these studies are often narrowly focused. Following from Figure 1, early life SES may play an important role for later life disease risk. Lower bone mass deposition and the building of inadequate bony architecture leads to osteoporosis.[20] Research suggests that socioeconomic advantage in childhood may have long-term benefits for bone health, with more childhood advantage being related to higher lumbar spine bone mineral density (BMD) in adulthood.[21] Karlamangla et al[22] find higher childhood SES associated with increased femoral neck strength in persons identifying as White men, but not in people identifying as White women, nor persons identifying as women or men of a non-White race or ethnicity. This suggests an intersectional advantage whereby the benefit of higher SES is for individuals from both the more advantaged gender and more advantaged race/ethnicity. Conversely, Pearce[23] notes that higher social status (parental occupational social class) at birth is related to increased femoral neck-shaft angle, a predictor of fracture risk, in persons identifying as female in adulthood at ages 49 to 51 years old.

Childhood exposures can also be negatively related to bone mineral density. Nabulsi et al[24] show that lower maternal SES during pregnancy is significantly associated with low BMD in offspring. Furthermore, adults who lived in single-parent households during childhood have lower femoral neck bending strength, compression strength, and impact strength relative to load,[25] which translates to poorer bone health. In general, this suggests lower childhood SES is a risk factor for bone health.

Research on adult household income's relationship to osteoporosis and resulting fracture is mixed. Lotfi et al[26] uncover that adults with higher levels of education, and higher SES more broadly, experience lower odds of osteoporosis. Similarly, lower SES (both education and income measures), especially extreme socioeconomic disadvantage, is associated with lower BMD in adults.[27] Swedish women with high and medium household income have a lower risk of hip fracture than those with low household income.[28] In Korean men, having a low income and low education is associated with a higher risk of osteoporosis.[29] However, other work show no relationship between income and fracture risk in young adults and community-dwelling older adults[30,31] or BMD in adults 25 to 75 years of age.[21] One caveat is the impact of household income differs by population as they have differential access to medical care (eg, free medical care), food, and other resources, so comparing across populations is problematic. Domestically, Lyles et al[32] observed that income is an independent risk factor for osteoporosis in American adults 50 years and older; those at or below 200% of the Federal Poverty Line (FPL) had a 90% higher risk of osteoporosis than those with incomes above 200% of the FPL. Furthermore, income below the FPL appears to be a risk factor for lower BMD, specifically in the spine[33,34] and femur[33] and increases the risk of fracture among postmenopausal women.[34] Thus, research on adulthood SES indicates that higher levels may be protective (and lower levels a risk factor) for bone health.

Aims

Our prior work[14] found the relationship between osteoporosis and social determinants to revolve around access to healthcare and other resources, and underlying frailty. This research examines the relationship between childhood SES and adult SES, as indicators of exposure to long-term structural inequities, and osteoporosis diagnosis, while accounting for additional measures of childhood social environment, and health and demographic factors. We test 2 hypotheses using data from the nationally representative, longitudinal 2012 to 2016 waves of the cohort-based Health and Retirement Study: 1. In the presence of potentially confounding/mediating demographic and health variables, lower childhood SES will be a key predictor of higher risk of osteoporosis (with a potentially lower likelihood of actual diagnosis), consistent with the upper pathway of Figure 1. In the presence of potentially confounding/mediating demographic and health variables, higher adult household income will be a key predictor of lower risk of osteoporosis (and an improved likelihood of diagnosis when the condition is present), consistent with the fundamental cause framework (lower pathway in Figure 1). We include several potential mediators that have been found to relate to osteoporosis diagnosis in previous work,[14] including age, sex, race/ethnicity, marital status, education, weight, thyroid disease, and allostatic load.

Method

Study Design and Data

We employ nationally representative, longitudinal data from the 2012 to 2016 waves of the HRS core, which represent the same sample (less any respondent dropout and the addition of new participants), cleaned and merged by RAND in the Longitudinal File and Fat Files.[35,36] Further, we incorporate data from the associated Life History Mail Survey (fielded in 2015 and 2017 to half of the respondents each year),[37,38] HRS Cross-Wave Race and Ethnicity File,[39] together with data from the Biomarker Study (collected from half of the respondents each wave)[40] and the validated measures of childhood socioeconomic status.[41] The first wave in which data on osteoporosis diagnosis was collected is 2012. Information on study design and response rates are reported by others.[42,43] The analytic samples of the population-based cohort study include 11 637 to 18 572 individuals (depending on analysis; Figure 2) who were community dwellers or assisted living residents (previously community dwelling) between the ages of 50 and 90. The inclusion criteria were self-reported osteoporosis diagnosis status and self-reported/measured responses to the variables described below, along with non-zero biomarker weights (see Figure 2). This secondary data research follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines and was issued an exemption after review by the University of La Verne Institutional Review Board (2019-13-CAS).

Measures

The outcome variable is whether the respondent reported ever being diagnosed by a doctor with osteoporosis (0 = not diagnosed, 1 = diagnosed). The key socioeconomic exposures are the childhood variables from the validated measures of childhood socioeconomic status (some variables are imputed),[41] and total (adult) household income in dollars (both continuous). The childhood variables include: (1) an index of SES built from many questions in the HRS (for more detail on index construction and validation, see Vable et al[41]); (2) average financial resources (index of four measures described and

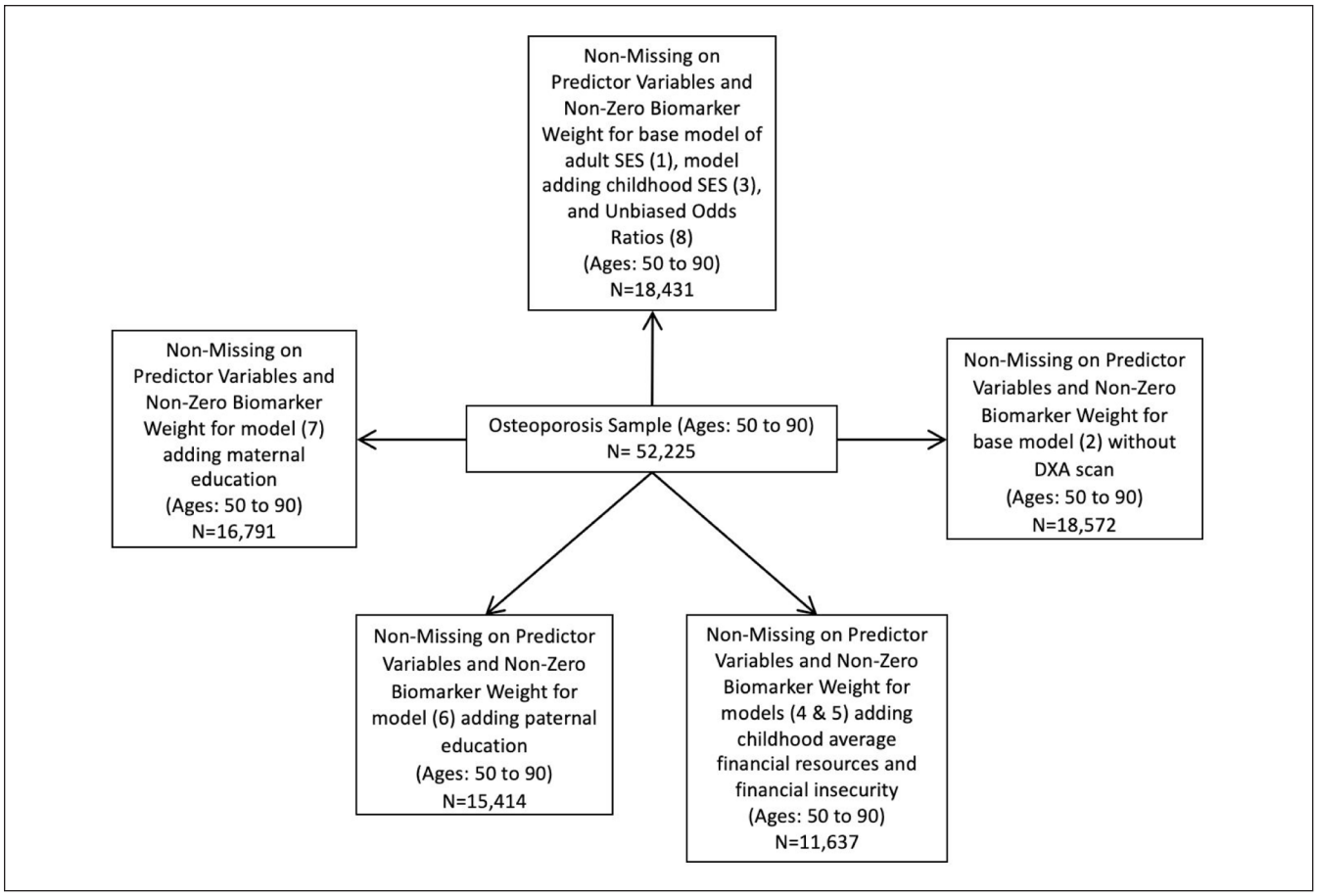

Figure 2. Sample size flow chart. The chart demonstrates how the analytic samples were derived.

validated in Vable et al[41]); (3) financial instability (also an index of 4 measures described and validated in Vable et al[41]); (4) father's education (in years; from HRS core); and (5) mother's education (in years; from HRS core). Each of these socioeconomic factors is potentially influenced by structural and systemic inequalities in society, across dimensions such as social class and race as described in the theoretical framework. As socioeconomic status is tied to health insurance in the United States, a variable for having insurance (at least one of the following types: private, VA, Medicaid, Medicare) was added (binary; 0 = no insurance, 1 = has insurance).

The potential socioeconomic mediator included is the respondent's highest educational level completed (categorical: less than high school, GED [a high school equivalency exam], high school graduate, some college, college degree and above). For potential demographic exposures and mediators, the natural log of age (continuous; in years), sex (binary; 0 = male, 1 = female), and race/ethnicity (sample sizes restricted the categories to identifying as 0 = White/European American, 1 = Black/African American, 2 = Another race/ethnicity), as exposures, and marital status (categorical: married/cohabiting, separated/divorced, widowed, and never married), as a potential mediator, were assessed. We include a number of control variables that have demonstrated importance in prior work and also through our larger variable selection process,[14] although some variables that were expected to be associated with osteoporosis were not important in our sample, including amount of alcohol consumed, smoking, and others. We include measured allostatic load and weight in kilograms, both continuous health-related mediators, along with a categorial health-related mediator: thyroid disease (binary; 1 = thyroid disease, 5 = no thyroid disease). Allostatic load represents an index based on McCrory et al.[44] derived from continuous measures of systolic and diastolic blood pressure (mg/dL), pulse (bpm), high-density lipoprotein (HDL) cholesterol (mg/dL), total cholesterol (mg/dL), high-sensitivity C-reactive protein (mg/L), A1c (%), cystatin C (mg/L), and waist circumference (in inches; >35 for women, >40 for men). Values greater than the 75th percentile were scored as 1, and the values were summed. As described in the theoretical background and depicted in the upper pathway of Figure 1, allostatic load and other health-related factors may be influenced by exposure to structural and systemic inequalities. As

osteoporosis diagnoses are tied to systemic/structural racism, and accessing a bone density scan tempers that effect,[13] we use a variable for whether the individual has had a bone density scan (binary; 0 = no, 1 = yes).

Statistical Analysis

A series of models was estimated to examine the childhood environment and access to resources. Model 1 provides a base model of adult demographic, health, and SES factors to which the childhood SES components will be compared: natural log of age, sex, race/ethnicity, respondent's education level, marital status, allostatic load, weight, thyroid disease, household income, whether the respondent has medical insurance, and whether the respondent has had a bone density scan. Model 2 removed the bone density scan variable to demonstrate how it provides greater health equity and tempers the effects of systemic racism from our models. Model 3 adds childhood SES to Model 1. Models 4 and 5 separately examine childhood average financial resources and childhood financial instability in relation to the base model (1), rather than Model 3, as there is an issue of multicollinearity with childhood SES in both models. By estimating separate models, we can examine the potential roles of childhood SES, as measured by the indices of childhood average financial resources and childhood financial instability, as each is an important, but distinct, way of characterizing the experience of SES during childhood. Models 6 and 7 include paternal and maternal education with Model 3, respectively as paternal and maternal education have distinct relationships with osteoporosis that should be examined separately. Finally, Model 8 retains only significant variables to furnish unbiased odds ratios.

Because the outcome of interest, osteoporosis diagnosis, is a binary measure, we estimate the machine learning multivariable logistic regression model:

$$logit\left(p\right) = B_0 + B_1X_1 + B_2X_2 + \ldots B_kX_k \quad (1)$$

where p is the probability the outcome variable equals 1. The model produces an odds ratio, which is a measure of the association between the exposure and outcome variables. The models' multicollinearity is assessed by the variance inflation factor (VIF), discrimination of the model by a C statistic, and fit by McFadden's adjusted pseudo R^2. The HRS uses a complex survey sample design. Therefore, to account for this design and reduce response bias, we estimated the logistic regression model using survey weighting (individual-level biomarker weight), accounting for stratum ID, and sampling error computation unit. As in our prior work,[14] we made computations using the survey package[45] in R,[46] which increases the robustness of model estimates by producing Horvitz-Thompson standard errors and handles non-linearity between the continuous predictors and log odds. This approach, combined with a wave variable included in all models to proxy for year of data collection, accounts for repeated measures in the data. Statistical significance was judged at $\alpha = 0.05$. Listwise deletion was implemented to address missing data, leading to the final sample sizes depicted in Figure 2.

Results

Descriptive Statistics

Approximately 7% to 9% of the sample reported an osteoporosis diagnosis over the period, as seen in Table 1. Approximately half of the sample is female, around 80% to 90% identify as White/European American, around 10% as Black/African American, and the remainder identify as another race/ethnicity. Most respondents are married with at least a high school degree. The approximate average household income is $81 000 to $94 000. Models 4 and 5 had substantially smaller samples and therefore the summary statistics are slightly different than the other models. The VIFs for all models did not exceed 2, indicating multicollinearity was not an issue in these models.

Model 1 Results

Results from Model 1 are shown in Table 2. The pseudo R^2 for the first model is .21, which indicates the model is excellent at accounting for the variation in the data,[47] and the C statistic indicated similarly excellent discrimination of the outcome variable (0.827). The odds for the exposure variable of household income are lower for an osteoporosis diagnosis when household income is higher. The exposures of the natural log of age and identifying as female (vs male) are associated with higher odds of osteoporosis diagnosis. For mediators, lower odds of osteoporosis were found for having a high school degree (as compared to some high school), for having a college degree or above (as compared to some high school) and for not having a bone density scan (as opposed to having one). Two health-related mediators were linked to lower odds of diagnosis: greater allostatic load yielded a lower odds of osteoporosis diagnosis, which is likely driven by the inclusion of the waist circumference variable (and its relationship to weight); higher weight was also linked to lower odds of osteoporosis diagnosis. The remaining variables were not significant.

Model 2 Results

Model 2 (Table 2), which dropped the bone density scan variable, demonstrated a large decrease in both the McFadden's Pseudo R^2 (.15) and C statistic (0.776), indicating a substantial loss of fit and discrimination. In this model several variables changed significance or signs, including

Table 1. Descriptive Statistics of Models 1 to 8.

Variable	Mean (SE)/ frequency (%) Models 1-3 (N = 18431)	Mean (SE)/ frequency (%) Models 4 and 5 (N = 11637)	Mean (SE)/ frequency (%) Model 6 (N = 15414)	Mean (SE)/ frequency (%) Models 7 (N = 16791)	Mean (SE)/ frequency (%) Models 8 (N = 18572)
Osteoporosis					
No	17062 (92.57)	10548 (90.64)	14287 (92.69)	15528 (92.48)	17196 (92.59)
Yes	1369 (7.43)	1089 (9.36)	1127 (7.31)	1263 (7.52)	1376 (7.41)
Childhood socioeconomic status index	0.17 (0.01)		0.21 (0.01)	0.19 (0.01)	0.16 (0.01)
Total household income in dollars	88538 (2367.0)	81382 (2697.0)	93833 (2610.9)	91778 (2455.5)	89324 (2571.7)
Childhood average financial resources		–0.0001 (0.005)			
Childhood financial instability		0.03 (0.01)			
Father's education level			10.56 (0.08)		
Mother's education level				10.71 (0.09)	
Has medical insurance					
Yes	17310 (92.94)	11204 (96.28)	14420 (93.55)	15651 (93.21)	
No	1301 (7.06)	433 (3.72)	994 (6.45)	1140 (6.79)	
Education level					
Less than high school	2317 (12.57)	1565 (13.45)	1518 (9.85)	1793 (10.68)	2348 (12.64)
GED	881 (4.78)	506 (4.35)	657 (4.26)	752 (4.48)	888 (4.78)
High school grad	4947 (26.84)	3388 (29.11)	4038 (26.20)	4458 (26.55)	4987 (26.85)
Some college	4932 (26.76)	2899 (24.91)	4265 (27.67)	4626 (27.55)	4968 (26.75)
College and above	5352 (29.04)	3279 (28.18)	4934 (32.01)	5160 (30.73)	5382 (28.98)
Age in years	64.90 (0.22)	69.75 (0.22)	64.92 (0.24)	64.78 (0.24)	64.88 (0.22)
Sex					
Female	9317 (50.55)	5883 (50.55)	7732 (50.16)	8553 (50.94)	9392 (50.57)
Male	9114 (49.45)	5754 (49.45)	7682 (49.84)	8238 (49.06)	9180 (49.43)
Race and ethnicity					
White/European American	15003 (81.40)	10144 (87.17)	13020 (84.47)	13871 (82.61)	
Black/African American	1978 (10.73)	944 (8.11)	1289 (8.36)	1667 (9.93)	
Another race/ethnicity	1452 (7.88)	549 (4.72)	1105 (7.17)	1251 (7.45)	
Marital status					
Married	12345 (66.98)	7776 (66.82)	10568 (68.56)	11393 (67.85)	
Separated/divorced	2748 (14.91)	1442 (12.39)	2190 (14.21)	2467 (14.69)	
Widowed	2070 (11.23)	1907 (16.39)	1648 (10.69)	1798 (10.71)	
Never married	1268 (6.88)	513 (4.41)	1010 (6.55)	1133 (6.75)	
Allostatic load	2.19 (0.02)	2.21 (0.02)	2.15 (0.02)	2.18 (0.02)	2.19 (0.02)
Weight in kilograms	83.91 (0.22)	82.11 (0.26)	83.82 (0.24)	83.85 (0.23)	83.90 (0.22)
Thyroid disease (ever)					
Yes	547 (2.97)	419 (3.60)	473 (3.07)	512 (3.05)	
No	17884 (97.03)	11218 (96.40)	14941 (96.93)	16279 (96.95)	
Has had a bone density scan					
Yes	7103 (38.54)	5368 (46.13)	6061 (39.32)	6604 (39.33)	7152 (38.51)
No	11328 (61.46)	6269 (53.87)	9355 (60.69)	10189 (60.68)	11420 (61.49)
Wave	12.02 (0.01)	11.90 (0.01)	12.03 (0.01)	12.03 (0.01)	12.03 (0.01)
Osteoporosis diagnosis by doctor 2012					
No	16026 (86.95)	9816 (84.35)	13410 (87.00)	14554 (86.68)	16152 (86.97)
Yes	2405 (13.05)	1821 (15.65)	2004 (13.00)	2237 (13.32)	2420 (13.03)
Osteoporosis diagnosis by doctor 2014					
No	17554 (95.24)	11017 (94.67)	14688 (95.29)	15988 (95.22)	17692 (95.26)
Yes	877 (4.76)	620 (5.33)	726 (4.71)	803 (4.78)	880 (4.74)
Osteoporosis diagnosis by doctor 2016					
No	17642 (95.72)	11021 (94.71)	14780 (95.89)	16069 (95.70)	17777 (95.72)
Yes	789 (4.28)	616 (5.29)	634 (4.11)	722 (4.30)	795 (4.28)

SE = standard error.

Table 2. Logistic Regression Results of Models Estimating Odds of Osteoporosis Diagnosis. Model 1: Base Model; Model 2: Without Bone Density Scan; and Model 3: Adding Childhood SES (N = 18431).

	OR (95% CI)	OR (95% CI)	OR (95% CI)
Variable	Model 1	Model 2	Model 3
Intercept	7.076 (0.554-90.300)	0.048* (0.004-0.513)	6.417 (0.491-83.941)
Childhood socioeconomic status index			0.916* (0.845-0.994)
Total household income in dollars	0.999998** (0.999997-0.9999996)	0.999999* (0.999998-0.9999998)	0.999999* (0.999997-0.9999996)
Has medical insurance			
Yes	1.082 (0.727-1.609)	1.417 (0.962-2.088)	1.081 (0.729-1.604)
Education			
GED	1.362 (0.954-1.946)	1.445* (1.043-2.003)	1.375 (0.960-1.971)
High school grad	0.779* (0.612-0.992)	0.944 (0.753-1.183)	0.814 (0.638-1.040)
Some college	0.887 (0.7163408-1.099)	1.164 (0.952-1.423)	0.939 (0.752-1.173)
College and above	0.629*** (0.502-0.788)	0.856 (0.703-1.042)	0.678** (0.534-0.862)
Natural log of age	2.639*** (1.511-4.610)	6.214*** (3.764-10.257)	2.676*** (1.525-4.696)
Sex			
Female	2.220*** (1.723-2.859)	5.840*** (4.645-7.342)	2.219*** (1.724-2.856)
Race and ethnicity			
Black/African American	0.787 (0.596-1.039)	0.673** (0.512-0.883)	0.774 (0.585-1.023)
Another race/ethnicity	1.178 (0.834-1.664)	1.088 (0.801-1.478)	1.149 (0.814-1.622)
Marriage status			
Separated/divorced	1.146 (0.952-1.381)	1.113 (0.935-1.324)	1.142 (0.947-1.376)
Widowed	1.126 (0.949-1.337)	1.022 (0.866-1.206)	1.120 (0.943-1.331)
Never married	0.871 (0.611-1.240)	0.815 (0.589-1.128)	0.870 (0.611-1.237)
Allostatic load	0.942* (0.897-0.990)	0.921*** (0.879-0.965)	0.940* (0.895-0.988)
Weight in kilograms	0.988*** (0.983-0.992)	0.988*** (0.984-0.993)	0.988*** (0.983-0.992)
Thyroid disease (ever)			
No	0.812 (0.631-1.044)	0.703** (0.556-0.889)	0.811 (0.631-1.043)
Has had a bone density scan			
No	0.153*** (0.122-0.192)		0.152*** (0.121-0.191)
Wave	0.559*** (0.508-0.614)	0.547*** (0.497-0.602)	0.559*** (0.509-0.615)
VIF (min/max/avg)	1.02/1.55/1.21	1.02/1.32/1.15	1.02/1.55/1.21

Note. Model 1 depicts adulthood socioeconomic environment for osteoporosis diagnosis outcome. Model 2 depicts socioeconomic environment without DXA scan variable to demonstrate access by race for osteoporosis diagnosis outcome. Model 3 depicts adulthood and childhood socioeconomic environment for osteoporosis diagnosis outcome.
OR = odds ratio; CI = confidence interval; N = sample size.
*$P < .05$. **$P < .01$. ***$P < .001$.

identifying as Black/African American and not having thyroid disease, which were linked to lower odds of having an osteoporosis diagnosis, while having a GED (in comparison to some high school) yielded higher odds of an osteoporosis diagnosis. The direction and significance for age, sex, allostatic load, household income, and weight remained the same as in Model 1.

Model 3 Results

Model 3 (Table 2) added childhood SES to Model 1 and was largely parallel with an excellent McFadden's Pseudo R^2 of .21 and C statistic of 0.827. Model 3 was similar to Model 1 with the exception of having a high school degree being no longer significant. Greater childhood SES was associated with lower odds of osteoporosis diagnosis.

Model 4 and 5 Results

Models 4 and 5 (Table 3) had nearly identical results; the McFadden's Pseudo R^2 were both .22 and each had C statistics of 0.821. The results of the base model variables were very close to Model 3, with the exceptions of allostatic load and household income, which were no longer significant, and separated/divorced, which was newly linked to higher odds of osteoporosis diagnosis, compared to married/cohabiting.

Table 3. Logistic Regression Results of Models Estimating Odds of Osteoporosis Diagnosis. Model 4: With Average Childhood Financial Resources; and Model 5: With Childhood Financial Instability (N = 11 637).

	OR (95% CI)	OR (95% CI)
Variable	Model 4	Model 5
Intercept	6.187 (0.186-205.270)	6.229 (0.188-206.236)
Total household income in dollars	0.999999 (0.999997-1.0000004)	0.999999 (0.999997-1.0000003)
Childhood average financial resources	1.035 (0.808-1.326)	
Childhood financial instability		0.979 (0.850-1.127)
Has medical insurance		
Yes	0.945*** (0.562-1.589)	0.944 (0.562-1.588)
Education		
GED	1.387 (0.923-2.084)	1.388 (0.923-2.087)
High school grad	0.861(0.677-1.095)	0.861(0.678-1.092)
Some college	0.858 (0.683-1.078)	0.858 (0.685-1.075)
College and above	0.749* (0.603-0.930)	0.749* (0.603-0.930)
Natural log of age	3.477** (1.530-7.903)	3.472** (1.527-7.892)
Sex		
Female	2.708*** (1.923-3.814)	2.708*** (1.923-3.815)
Race and ethnicity		
Black/African American	0.715 (0.495-1.033)	0.715 (0.495-1.0315)
Another race/ethnicity	1.123 (0.800-1.577)	1.123 (0.800-1.578)
Marriage status		
Separated/divorced	1.306* (1.039-1.640)	1.306* (1.039-1.641)
Widowed	1.166 (0.951-1.430)	1.167 (0.951-1.431)
Never married	1.059 (0.648-1.732)	1.059 (0.648-1.732)
Allostatic load	0.948 (0.893-1.006)	0.948 (0.893-1.006)
Weight in kilograms	0.988*** (0.983-0.993)	0.988*** (0.983-0.993)
Thyroid disease (ever)		
No	0.813 (0.605-1.093)	0.813 (0.605-1.093)
Has had a bone density scan		
No	0.178*** (0.134-0.237)	0.178*** (0.134-0.237)
Wave	0.501*** (0.452-0.556)	0.501*** (0.452-0.556)
VIF (min/max/avg)	1.02/1.76/1.22	1.02/1.76/1.21

Note. Model 4 depicts adulthood socioeconomic environment with childhood average financial resources for osteoporosis diagnosis outcome. Model 5 depicts adulthood socioeconomic environment and childhood financial instability for osteoporosis diagnosis outcome.
OR = odds ratio, CI = confidence interval, N = sample size.
*$P < .05$. ** $P < .01$. *** $P < 0$ 0.001.

Neither exposure variable of childhood average financial resources, nor financial instability, was significant.

Model 6 and 7 Results

Models 6 (Table 4) and 7 (Table 5) had McFadden's Pseudo R^2 of .19 and .20, respectively. The C statistics were 0.831 and 0.826 for Models 6 and 7. Neither parental education exposure variable is significant. Models 6 and 7 were similar to Model 3 except allostatic load is not a significant predictor of osteoporosis diagnosis, and not having thyroid disease led to lower odds of osteoporosis diagnosis in Model 6 only. Also in Model 6 that evaluates father's education, although the bone density scan variable is included, persons who identify as Black/African American have significantly lower odds of osteoporosis diagnosis. Household income yielded lower odds of osteoporosis diagnosis for Model 6 and was not significant in Model 7. Childhood SES was not significant in either model.

Model 8 Results

Unbiased odds ratios are reported in Model 8 (Table 5). The McFadden's Pseudo R^2 is excellent at .21 as is the C statistic of 0.826. The only variables significant in predicting osteoporosis diagnosis and producing higher odds are: identifying as female and greater age (natural log). Significantly lower odds were calculated for: having a college education or above, greater allostatic load (likely due to waist circumference; see Discussion), higher weight, greater household income (exposure variable), not having a bone density scan, and higher childhood SES (exposure variable).

Table 4. Logistic Regression Results of Models Estimating Odds of Osteoporosis Diagnosis. Model 6: With Father's Education (N = 15414).

	OR (95% CI)
Variable	Model 6
Intercept	3.702 (0.214-63.941)
Childhood socioeconomic status	0.937 (0.847-1.035)
Total household income in dollars	0.999999* (0.999999-0.9999998)
Father's education (in years)	1.006 (0.982-1.029)
Has medical insurance	
Yes	1.200 (0.745-1.933)
Education	
GED	1.447 (0.959-2.185)
High school grad	0.827 (0.628-1.089)
Some college	0.879 (0.671-1.151)
College and above	0.690* (0.514-0.925)
Natural log of age	3.129*** (1.667-5.876)
Sex	
Female	2.427*** (1.882-3.131)
Race and ethnicity	
Black/African American	0.717* (0.543-0.948)
Another race/ethnicity	1.107 (0.8074-1.519)
Marriage status	
Separated/divorced	1.184 (0.963-1.456)
Widowed	1.178 (0.958-1.450)
Never married	0.835 (0.570-1.222)
Allostatic load	0.962 (0.907-1.020)
Weight in kilograms	0.987*** (0.9821-0.992)
Thyroid disease (ever)	
No	0.736* (0.573-0.946)
Has had a bone density scan	
No	0.153*** (0.120-0.194)
Wave	0.546*** (0.495-0.603)
VIF (min/max/avg)	1.02/1.68/1.29

Note. OR = odds ratio, CI = confidence interval, and N = sample size. Model 6 depicts adulthood socioeconomic environment and father's education level for osteoporosis diagnosis outcome.
*$P < .05$. ***$P < .001$.

Discussion

In this study we tested 2 hypotheses. Hypothesis 1 stated that in the presence of potentially confounding/mediating demographic and health variables, lower childhood SES would be a key predictor of higher risk of osteoporosis (with a potentially lower likelihood of actual diagnosis), potentially consistent with both the social epidemiological model pathway and the fundamental cause pathway depicted in Figure 1. We find evidence to support this hypothesis in Model 3 and Model 8; higher childhood SES links to lower odds of osteoporosis diagnosis, whereas poorer childhood SES associates with higher odds. Hypothesis 2 stated that in the presence of potentially confounding/mediating demographic and health variables, higher adult SES would be a key predictor of lower risk of osteoporosis (and an improved likelihood of diagnosis when the condition is present), consistent with the fundamental cause framework. We also find evidence to support this hypothesis, with higher levels of income and education linked to lower odds of osteoporosis diagnosis in Models 1, 3, 6, 7, and 8. Thus, we agree with several studies that have found childhood socioeconomic status and household income to be related to the risk of low BMD and osteoporosis—typically, higher levels of these resources are associated with lower risk—our results support this even after accounting for important demographic and health factors. In other words, long-term lack of resources (ie, present during childhood) appears to increase the likelihood of osteoporosis diagnosis in this sample, while socioeconomic advantage across the life course decreases the likelihood of osteoporosis diagnosis. Similarly, lower current household income likely reduces access to health care, resulting in a lower likelihood of preventing osteoporosis. These findings indicate that socioeconomic factors are relevant when considering the odds of osteoporosis diagnosis, net of key demographic characteristics. As our data set is nationally representative and does not exclude any particular subgroup, our findings are generalizable to middle-older adults in the greater United States.

We found some relationships between potential mediators and risk of osteoporosis diagnosis. The negative relationship that we find between allostatic load and odds of osteoporosis diagnosis may be due to the inclusion of waist circumference. Weight is associated with a lower risk of osteoporosis diagnosis in several studies[48]; it is well-established that weight is positively correlated with BMD, regardless of whether it is fat mass or lean mass.[49] Our allostatic load finding contradicts prior work that related higher allostatic load to increased risk of frailty for older women,[50] of which osteoporosis is one component. However, Szanton et al's[50] allostatic load measurement uses BMI as a proxy for waist/hip ratio, which may have led to the opposite result as BMI introduces heterogeneity to unhealthy waist size because athletes and people with generally larger body sizes, but smaller waist circumferences, are assessed similarly. BMI is further problematic due to its biased theoretical foundation and development on people who identified as White/European men, which limits applicability to persons who identify as women and/or persons of color.[51-57]

In a few models the direction and significance of odds ratios change. For example, in 2 models, identifying as Black/African American is linked to lower odds of osteoporosis diagnosis. In Model 2 this is likely attributable to the exclusion of the bone density scan variable, which previous research has shown eliminates the racial/ethnic disparity in diagnosis[13] and was purposely added to show this phenomenon. However, this result also shows up in Model 6, which includes the bone density scan variable. Ultimately, we suspect this is evidence of the strength of structural racism in society that limits access to diagnosis and treatment and is a

Table 5. Logistic Regression Results of Models Estimating Odds of Osteoporosis Diagnosis. Model 7: With Mother's Education; and Model 8: Unbiased Odds Ratios.

	OR (95% CI)	OR (95% CI)
Variable	Model 7 (N = 16791)	Model 8 (N = 18572)
Intercept	6.673 (0.425-104.836)	3.756 (0.329-42.896)
Childhood socioeconomic status	0.963 (0.864-1.074)	0.917* (0.848-0.993)
Total household income in dollars	0.999999* (0.999997-0.9999998)	0.999998** (0.999997-0.9999995)
Mother's education level	0.990 (0.962-1.019)	
Has medical insurance		
Yes	1.028 (0.683-1.548)	
Education		
GED	1.489* (1.012-2.190)	1.383 (0.976-1.961)
High school grad	0.840 (0.640-1.101)	0.818 (0.648-1.033)
Some college	0.964 (0.744-1.249)	0.936 (0.763-1.149)
College and above	0.701* (0.531-0.925)	0.678*** (0.543-0.846)
Natural log of age	2.756** (1.513-5.020)	2.983*** (1.793-4.963)
Sex		
Female	2.224*** (1.698-2.912)	2.276*** (1.773-2.921)
Race and ethnicity		
Black/African American	0.798 (0.615-1.034)	
Another race/ethnicity	1.153 (0.812-1.637)	
Marriage status		
Separated/divorced	1.145 (0.929-1.412)	
Widowed	1.162 (0.959-1.408)	
Never married	0.864 (0.597-1.252)	
Allostatic load	0.947 (0.893-1.004)	0.941* (0.896-0.989)
Weight in kilograms	0.988*** (0.983-0.993)	0.987*** (0.983-0.992)
Thyroid disease (ever)		
No	0.847 (0.653-1.100)	
Has had a bone density scan		
No	0.158*** (0.127-0.198)	0.150*** (0.120-0.187)
Wave	0.554*** (0.506-0.606)	0.559*** (0.509-0.615)
VIF (min/max/avg)	1.02/1.67/1.28	1.01/1.48/1.21

Note. Model 7 depicts adulthood socioeconomic environment and mother's education level for osteoporosis diagnosis outcome. Model 8 depicts unbiased odds ratios of socioeconomic environment for osteoporosis diagnosis outcome.
OR = odds ratio; CI = confidence interval; N = sample size.
*$P < .05$. **$P < .01$. ***$P < .001$.

key predictor of socioeconomic disparities.[1] Thus, although racial biases or inappropriate assumptions in the health care system play a key role in underdiagnosis of osteoporosis for individuals with non-White/European American identities, structural racism also patterns access to socioeconomic resources.[58] Flexible resources, as defined by Link and Phelan,[10] can be used to avoid disease or improve outcomes when disease occurs across racial identities, yet they are not equally distributed within the population, and race is a key dimension across which inequities in these resources occur.[1] In Models 4 and 5 the socioeconomic variables are not statistically significant, but the sample size is smaller. However, these changes across models are not reflected in the final model of unbiased odds ratios, suggesting an important role for confounding.

The study has limitations, including a simplified categorization of race/ethnicity due to sample size. In addition, the HRS measures binary sex, which does not account for the complete continuum of sex. Further, the osteoporosis diagnosis variable could be subject to recall bias and is also self-reported. Respondents are asked whether a doctor has ever told them they have osteoporosis. Given the age of some of the respondents, some respondents may have forgotten a diagnosis, and some may have confused an osteoporosis diagnosis with another condition, such as osteoarthritis. The self-report nature also means that respondents must be connected with the health care system and have received osteoporosis screening to be diagnosed with osteoporosis. Recall bias is also a concern for the childhood exposure variables as so much time has transpired. Finally, it is probable that

respondents reporting no osteoporosis diagnosis may have osteoporotic changes to bone that have not been diagnosed. Thus, the undiagnosed respondent sample may have additional heterogeneity that minimizes the differences between the diagnosed and undiagnosed samples. Nonetheless, this study offers an important examination of the role of socioeconomic factors in the risk of osteoporosis diagnosis, after accounting for basic demographic factors.

Conclusion

In totality, our results demonstrate the minimum strength of structural inequities and structural racism for increased odds of an osteoporosis diagnosis. These findings, and the support we show for our conceptual model, highlight the critical need for clinicians to receive Diversity, Equity, and Inclusion (DEI) training as a key step toward reducing health inequities. In this study the focus was on osteoporosis diagnosis, but receipt of comprehensive DEI training would likely reduce inequity in health outcomes across multiple disease domains, helping to address poorer access to prevention, diagnosis, and treatment for persons from low SES background and for persons of color. Furthermore, we join Link and García's[8] call for additional research into the role of advantaged groups in perpetuating and exacerbating existing health inequities in U.S. society. Finally, these findings are applicable to past populations as they shed light on how the contextual environment affects access to care (however organized in the past) and the development and persistence of frailty.

Author Note

Access to the Health and Retirement Study data used in this study are available through a restricted-use data agreement with the Health and Retirement Study. Statistical code is available after appropriate vetting by the Health and Retirement Study staff. Our study was declared exempt by the University of La Verne Institutional Review Board (2019-13-CAS). Consent was collected from participants in the initial data collection process by the Health and Retirement Study (see https://hrs.isr.umich.edu/sites/default/files/biblio/HRS_IRB_Information-10-2017.pdf).

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