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Accelerated Aging: The Role of Socioeconomic, Social, Demographic, and Biological Factors on Bone Mineral Density

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Abstract

We investigate socioeconomic, social, demographic, and biological variables that are associated with chronic stress in the body to assess whether they have an effect on bone mineral density (BMD) as an indicator of accelerated aging. Multiple regression models were derived from data in the Midlife in the United States: A National Longitudinal Study of Health and Well-Being, Waves 1 (1995–1996) and 2 (2004–2006). Three BMD variables were used as outcomes: femoral, femoral neck, and spine *T* scores. The sample included 333 men and women aged 34–80. Poverty and C-reactive protein were related to BMD for spine *T* scores, partially consistent with hypotheses. But, marital status, perceived support of a partner, and education were not associated. Friend strain was discovered to have a relationship with BMD. More variables, and from a broader context, need to be examined together to understand what affects reduced BMD, given the high costs of accelerated aging.

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Many factors including social factors, such as poverty (as opposed to low socioeconomic status), violence exposure, and familial caregiving (Oliveira et al., 2016) can contribute to prolonged stress. Social stressors affect telomere length (a measure of cellular aging) through a stress response in the body (Oliveira et al., 2016). Chronic inflammation results from a prolonged increase in stress hormones (Barr, 2014) and is linked to negative health outcomes (e.g., cardiovascular disease [CVD]; Kubzansky, Seeman, & Glymour, 2014), many of which are usually associated with advanced age and are associated with chronic stress (e.g., Williamson, Porges, Lamb, & Porges, 2015). Inflammation has been linked to osteoporosis (Ginaldi, Di Benedetto, & De Martinis, 2005), a condition characterized by reduced bone mineral density (BMD) and associated with advanced age.

Life history stress (perceived and chronic) can result in shorter telomere length, among other outcomes, which is in turn linked to accelerated aging (Epel et al., 2004). Reduced BMD is one marker of accelerated aging as demonstrated in murine (mouse) models (cf., Jilka, Weinstein, Takahashi, Parfitt, & Manolagas, 1996), although these direct biological models have yet to be extended to humans in the literature. Researchers have instead considered a variety of socioeconomic, social, and biological stressors that contribute to reduced BMD in humans but questions still remain. The literature reveals a gap in our knowledge at the intersection of biological outcomes of decreased BMD (e.g., fracture risk) and social factor independent variables (e.g., socioeconomic status [SES], education level), as research does not typically consider both combined or across a range of social variables. Instead, most, but not all, studies focus on a single social factor as the independent variable in statistical models. It is perhaps the differences in approaches across the literature that lead to conflicting results among researchers and studies.

To address this gap in the literature, we answer the following research question in this project: What are the stressors from the four domains of socioeconomic, social, demographic, and biological factors that individuals face in America currently, that lead to changes in the aging process, as detected in BMD, and that may ultimately affect long-term health and quality of life? From this research, question stems a series of hypotheses we seek to test as we examine how BMD is affected by stress. Each hypothesis will be tested accounting for demographic characteristics, and

positive and negative health behaviors that influence health outcomes: (1) Poverty, specifically childhood poverty, will show an inverse relationship with BMD, (2) marital status (men: marriage under the age of 25 and marital changes will display an inverse relationship) and perceived support from a significant other will be associated with BMD (increased marital support in women has a positive relationship with BMD), (3) education will be inversely associated with BMD, (4) other markers of demographic, socioeconomic, social, and biological factors generally not considered in the literature will be associated with BMD, and (5) CRP, as a measure of inflammation, will be strongly associated with BMD, displaying an inverse relationship. The hypotheses will not be tested with separate models, but rather, they will be tested by looking at individual coefficients within models that contain variables from all four domains.

Literature Review

Definitive findings for a relationship between SES and BMD are lacking, possibly due to limited literature on the subject (Brennan, Pasco et al., 2009); however, several trends have been identified. A bimodal distribution exists in the greater adult population where the lowest and highest quantiles of SES (five quantiles) are at greater risk of decreased BMD (Brennan, Henry et al., 2009). Women are most affected by SES, as adult women with incomes lower than USD20,000 a year experience a decrease in BMD (Du, Zhao, Xu, Wu, & Deng, 2017). Moreover, low SES (Alver, Søgaard, Falch, & Meyer, 2007; Brennan, Leslie, & Lix, 2013) and poverty (Amiri et al., 2008) are associated with decreased BMD in postmenopausal women, and low SES and food insecurity with osteoporosis later in life (Lyles, Schafer, & Seligman, 2014). Poverty, differentiated from low SES studies by additional nonmonetary factors, is associated with increased risk of fracture in older adults (Quah, Boulton, & Moran, 2011). Childhood SES is also associated with BMD in select adult samples (e.g., Karlamangla et al., 2013), showing that experiences in childhood persist into adulthood. Furthermore, having been raised in a single-parent household (but not the event leading to this, e.g., death, divorce, abandonment) is associated with lower femoral neck bone strength in adults, which is one measure of BMD (Crandall et al., 2015).

The literature on social factors is generally narrow. For men, marriage under the age of 25 and marital changes (e.g., divorce, separation) show an inverse relationship with BMD, while women show an increase in BMD with increased marital support (Miller-Martinez et al., 2014). Education is most commonly associated with BMD levels (e.g., Du et al., 2017) and fracture

risk in minority women (Crandall et al., 2014), with lower education levels being associated with lower bone density and higher fracture risk. Certain social factors have also been associated with increased inflammation, though not yet directly associated with BMD. For example, caregiving has been shown to increase inflammation (Kiecolt-Glaser et al., 2011), along with being a racial/ethnic minority (Uchino et al., 2016), and childhood adverse events (Runsten et al., 2014).

The relationship of specific biological factors to reduced BMD is problematic in the literature. CRP is an inflammatory marker that has been explored in relation to BMD because it is a convenient and cost-effective biomarker to jointly research inflammation (e.g., from stress processes) and potential outcomes on BMD. The mechanism relating CRP and BMD has been explored at the cellular level (see Discussion) and is well-documented in the physiological literature, but the outcomes of increased CRP in models predicting BMD are inconsistent. Increased CRP levels have been linked to decreased BMD in postmenopausal women (Ferrari, Ahn-Luong, Garnero, Humphries, & Greenspan, 2003; Ganesan, Teklehaimanot, Tran, Asuncion, & Norris, 2005; Sponholtz et al., 2014), and pre-and postmenopausal women (dePablo, Cooper, & Buckley, 2012; Koh et al., 2005). Yet, in other studies, postmenopausal women were shown to not experience a decrease in BMD with elevated CRP (Dahl et al., 2015), and men have displayed an inverse relationship between CRP and BMD (Dahl et al., 2015). These conflicting results suggest researchers should use caution when employing CRP as an indicator of BMD.

The outcomes of reduced BMD in older adults include an increase in the risk of fracture, although not all studies have detected this (Berglundh, Malmgren, Luthman, McGuigan, & Åkesson, 2015). As a biological risk factor for fracture, increased CRP could lead to an increase in risk of fracture (e.g., Dahl et al., 2015), but the results conflict across studies and are not definitive (Wu et al., 2015). The risk of fracture appears to be associated with social and socioeconomic factors. Globally, some studies have reported increases in fracture risk in lower SES older adults (e.g., Brennan et al., 2013), while others found no increase in the risk of fracture (Vestergaard, Rejnmark, & Mosekilde, 2006). Antithetically, a higher risk of fracture has been detected in higher SES individuals (Reyes et al., 2015). Increased fractures have also been associated with lower education levels (Benetou et al., 2015; Wilson, Chase, Chrischilles, & Wallace, 2006), living alone (Benetou et al., 2015), and, for women, marital status (Farahmand et al., 2000), among others. Because fractures, as a measure of poor health, lead to a lower quality of

life, a more complete evaluation of nonbiological risk factors for decreased BMD is necessary.

In sum, the relationships of socioeconomic, social, demographic, and biological factors on decreased BMD and resulting fractures are not straightforward. A limitation to the greater body of research is the investigation of only one or a few indicators and/or domains on decreased BMD; an examination of the relationships among all of the variables identified in the literature is lacking, but necessary, because as Riancho and Brennan-Olsen (2017) point out, osteoporosis is determined by a combination of genetic and environmental factors that is not yet fully understood. Moreover, attention has not yet been focused upon the uncovering of other stress-related variables that could yield a similar negative outcome on BMD or whose effects might be masking the associations of previously studied factors with BMD. Therefore, by examining all of the stressors that are identified in the literature, combined with additional factors that could lead to prolonged stress and chronic inflammation, we aim to uncover a better understanding of how socioeconomic, social, demographic, and biological factors are related to decreased BMD. To graphically represent the conceptual model for how the variables we are investigating affect inflammation, leading to an outcome of reduced BMD, we developed Figure 1 from the models by Barr (2014), Kubzansky, Seeman, and Glymour (2014), and Riancho and Brennan-Olsen (2017).

Research Design

Data

The longitudinal data used in this study were obtained from Midlife in the United States: A National Longitudinal Study of Health & Well-Being (MIDUS). MIDUS (2011) was developed to "investigate the role of behavioral, psychological, and social factors in accounting for age-related variations in health and well-being in a national sample of Americans". The first wave of MIDUS (MIDUS 1) was collected in 1995 and 1996 by the MacArthur Foundation Research Network on Successful Midlife Development. The second wave of MIDUS (MIDUS 2) was collected between 2004 and 2006 and was a longitudinal follow-up of the original sample. We use MIDUS 1 for background variables, and MIDUS 2 for measures collected at, and around, the time of the biomarker data collection. MIDUS 2 includes a reassessment of data collected at MIDUS 1 with some expanded questions about stressful life events, caregiving, and other topics, along with the

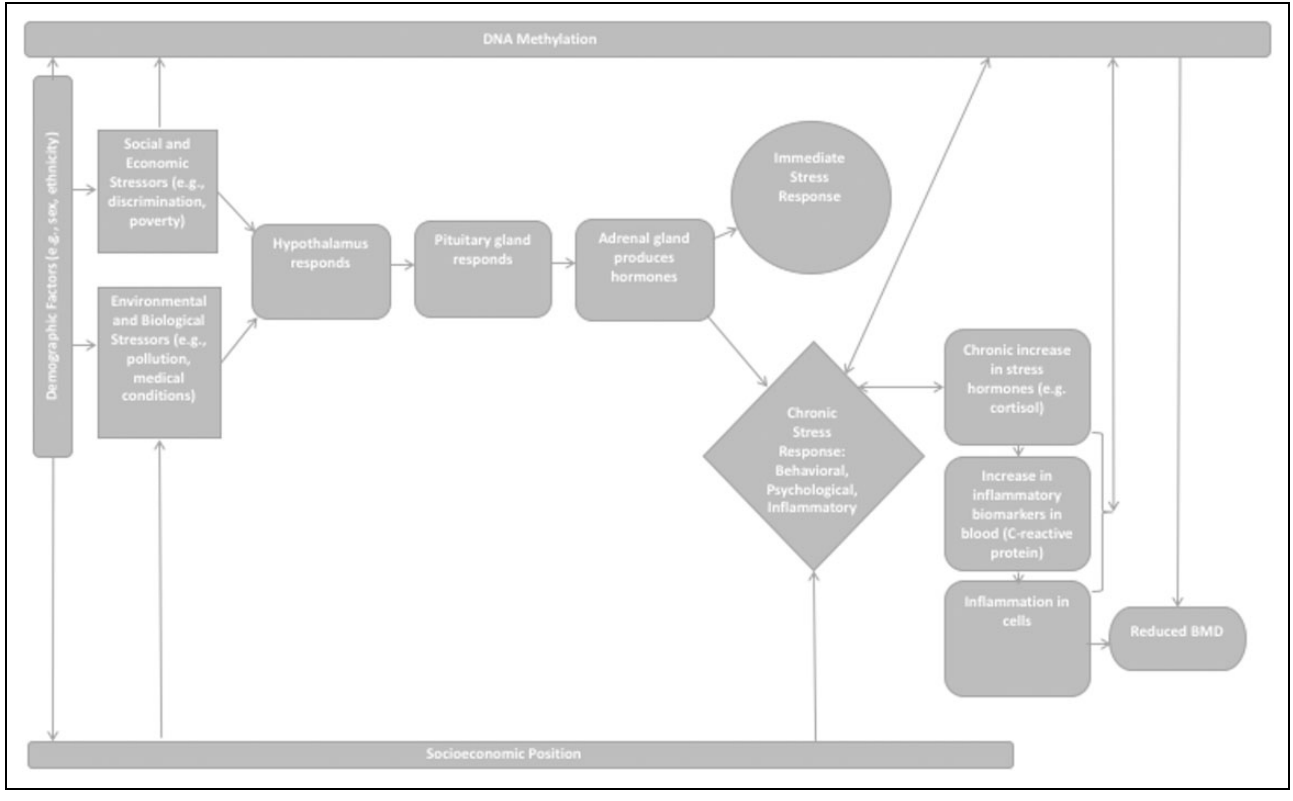

Figure 1. Conceptual model of nonbiological stressors' influence on biological processes and bone mineral density.

biomarker study of MIDUS. The biomarker study includes up to 1,054 respondents who participated in the MIDUS 1 and MIDUS 2 interviews. As these data were previously collected by another source and provided to us as deidentified, secondary data, the research was not considered human subjects research by the institutional review board at University of La Verne, and approval was not required.

Sample

The sample includes all participants who responded to MIDUS 1 and the MIDUS 2 reinterview and biomarker studies after screening for the following criteria. Only respondents with complete data on the outcomes (349 cases dropped) and key independent variables (61 cases dropped) were included. Further, individuals were removed who had exclusionary health conditions/treatments that would be linked to decreased BMD, including tuberculosis, which causes alteration to the vertebral bodies; arthritis (as rheumatoid arthritis [RA] is not separated from remaining diagnoses of arthritis in the data; treatments for RA affect BMD [Blalock, Vellis, DeVellis, & Sauter, 1988]), autoimmune/lupus disease (treatment for Lupus can affect BMD; Kipen et al., 1997) or abnormally high CRP (e.g., from burns; 311 cases dropped). The total sample size is 333. Although the sample size is modest, to date this is the best available longitudinal data that include BMD, CRP, and a multitude of socioeconomic and social variables.

Measurements

In accordance with the literature review, we include three main sets of variables: those related to SES, other social factors, and biological factors. Possible confounders are also addressed. The first variable related to SES is poverty, measured at both waves, as calculated through an income-to-needs ratio (values <1 indicate poverty) and through a subjective question about whether the respondent has enough money for needs. The second variable related to SES is perceived neighborhood quality (both waves) to proxy for exposure to negative environmental conditions, such as exposure to violence, that are often determined by SES. We include two SES-related measures from childhood: Childhood SES and time living in a single-parent family (both measured at Wave 1). Childhood SES is measured through the use of two variables: financial level growing up (a relative measure) and whether the family was on welfare. Length of time spent in

a single-parent family is calculated as age 18 minus the age at which the parents divorced or one parent died.

Along with SES-related factors, other social factors, both positive and negative, have been linked to chronic inflammation and/or osteoporosis in the literature. The first of these measures is caregiving (both waves), which is measured through two variables: caring for a minor child and caring for an aging adult. A second variable associated with caregiving, number of children (at home under the age of 18), was added to examine the stress associated with caregiving of minor children. The second and third measures are education and racial/ethnic minority status (both from Wave 1). We also included the measures of spousal, friend, and family strain (both waves). In addition, measures of age at marriage (Wave 1) and whether the respondent has experienced a negative marriage transition (separation, divorce, widowhood) were included (both waves). Finally, childhood adverse events were examined. Childhood adverse events is a score combining the childhood trauma questionnaire and indicators of parental drug and alcohol problems (Wave 1). In terms of potentially positive social factors, we included measures of marital support (and marital quality; both waves) and measures of social support from family and friends (both waves).

For biological and health factors, we included measures of negative health behaviors known to affect health outcomes including smoking and alcohol consumption (both waves); the number of health conditions reported (both waves), and biomarkers related to inflammation and chronic disease (Wave 2), including high CRP (indicated by CRP levels >3 mg/L and <10 mg/L), and hemoglobin A1c (a marker of diabetes). Total cholesterol, high-density lipoproteins (HDL), low-density lipoproteins (LDL), triglycerides, insulin, and cortisol were also incorporated. The biomarker samples were collected at three General Clinic Research Centers: University of California, Los Angeles, University of Wisconsin, and Georgetown University. Participants spent 2 days at the clinics, staying overnight (Dienberg Love, Seeman, Weinstein, & Ryff, 2010). In terms of potentially positive health behaviors, we included a measure of physical activity (both waves). Physical activity is a yes/no question about whether the respondent engages in regular exercise or activity for 20 min or more at least 3 times per week.

We examined changes in neighborhood quality, family and friend support, family and friend strain, and poverty across the waves as well as at each wave. The underlying motivation is a concern that chronic strain, poverty, and so forth may have compounding effects on health that are not captured in the point-in-time variables. For poverty, the variable has four categories: not in poverty at both waves, in poverty at Wave 1 only, in poverty at Wave 2

only, and in poverty at both waves. The other "change" variables have three categories: a reference category of the variable (e.g., family strain) staying the same across waves, and two comparison categories indicating improvements over time and worsening over time.

Control variables for sex (Wave 1) and menopause status for women (Wave 2) were included as potential confounders. Menopause status is measured based on a question about whether the respondent's menstrual period stopped because of menopause. If the respondent answers "yes" they have a 1 value for menopause status. Men, women who reported a menstrual period for all of the prior 12 months, and women who reported their menstrual periods stopped for other reasons (e.g., medication induced) have a value of 0 for menopause status. Body mass index (BMI; both waves) was also controlled.

The outcome variable for these models is BMD, which is measured at Wave 2. The variables labeled in the data set as femur *T* score, femur neck *T* score, and spine *T* score were used as measures of BMD. *T* scores were utilized because the MIDUS 2 bone density data were collected on Lunar and Hologic machines, and only the *T* score values from the two machines can be directly compared according to the MIDUS documentation (Ryff, Seeman, & Weinstein, 2012). *T* scores are defined in relation to the mean bone density of a young adult, such that a *T* score of -1 indicates a bone density of one standard deviation below the young adult mean.

Data Analysis

The original group of variables described above was selected for evaluation as they have been identified in the literature to have an effect on BMD or are known stressors that could cause inflammation if there is chronic exposure. We included all variables at once because the potential pathway structure is unknown (theorized in the conceptual model but practically speaking, unknown), and we have a limited sample size that does not allow us to test possible pathway structures. Further, only a few upstream variables were statistically significant predictors of BMD in preliminary analyses. To narrow down the variables for constructing our models, independent and confounding variables were evaluated for significance (setting our significance level at $\alpha = 0.05$) and using the change-in-estimate method. In the latter, the main independent variable coefficient was assessed when a previously nonsignificant variable was added to the model. If it was impacted by 10% or more when the variable was added, the variable was left in the model (Maldonado & Greenland, 1993). Variable selection routines are an important

facet of epidemiological research as they can identify confounders that are not readily apparent. Further, they help to prevent variables masking the effects of others, something separating analyses by domains cannot accomplish. Therefore, only the necessary variables that directly affect the levels of BMD due to external factors are evaluated, which allows for targeted treatment/discussion by public health and medical practitioners. Because the BMD *T* scores are continuous, linear multiple regression was employed. All analyses were completed in Stata, version 13.1 (StataCorp, 2013).

Results

Descriptive statistics for the sample are shown in Table 1. The final sample includes 333 individuals with no missing data on key independent or outcome variables. A little more than half of the sample was female (53%), and the mean age was 50.09 at Time 2. Mean BMI was 28.09 at Time 2, which falls into the overweight category. The mean insulin score was 11.88, mean HDL cholesterol was 55.33, and at Time 1 the mean number of health conditions was 0.72. Nineteen percent of the sample had experienced menopause. Twenty-one percent of the sample had high CRP levels. The mean total femur *T* score in the sample was −0.30, that is, 0.30 standard deviations below the young adult mean. The mean femur neck *T* score was −0.74 (indicating 0.74 standard deviations below the young adult mean). The mean spine *T* score was −0.41 (indicating 0.41 standard deviations below the young adult mean). Osteopenia (*T* score between −1 and −2.5) was adequately represented in the sample with a prevalence of 24% in femoral *T* score, 37% in femoral neck *T* score, and 27% in the spine *T* score. Osteoporosis (*T* score below −2.5) was less prevalent with only 2% of respondents scoring as osteoporotic from the femoral *T* score, 4% from the femoral neck *T* score, and 7% from the spine *T* score. Mean number of children under the age of 18 at Time 2 was 0.83, 95% of the sample reported some exercise or regular activity at Time 1, and at Time 2, 49% of the sample had consumed alcohol in the past 30 days.

There are some changes in variables over time. Twenty-six percent of the sample had friend strain that stayed the same across both waves, while 42% saw improvements over time, and 32% saw friend strain worsen over time. With regard to changes in poverty status over time, 81% of the sample reported no poverty at either wave, 14% reported being in poverty at Time 1 but not Time 2, 4% reported being in poverty at Time 2 but not Time 1, and less than 1% reported being in poverty at both waves.

Table 1. Descriptive Statistics—Sample Means and Percentages for Key Independent and Outcome Variables, MIDUS.

Variable	Mean	Range
Female	0.53	0–1
Age at Wave 2 (years)	50.09 (10.34)	34–80
Body mass index at Wave 2	28.09 (5.41)	14.99–49.76
Insulin (μIU/ml)	11.88 (10.35)	2–74
Number of health conditions at Wave 1	0.72 (1.00)	0–4
Menopause	0.19	0–1
Alcohol consumption at Wave 2		0–1
Ever drinker, last 30 days	0.49	
3+ drinks/week, last 30 days	0.22	
Never drinker, last 30 days	0.29	
Exercise at Wave 1	0.95 (1.21)	0–1
Number of children under 18 at Wave 2	0.83 (1.24)	0–7
HDL cholesterol	55.33 (17.97)	22–121
Friend strain across waves		0–1
Same strain at Waves 1 and 2	0.26	
Improved strain from Wave 1 and Wave 2	0.42	
Worsened strain from Wave 1 to Wave 2	0.32	
Poverty across waves		0–1
Not in poverty at either wave	0.81	
In poverty at Wave 1, not Wave 2	0.14	
In poverty at Wave 2, not Wave 1	0.04	
In poverty at both waves	0.01	
High CRP	0.21	0–1
Femoral *T* score	−0.30 (1.02)	−3.11–2.55
Femoral neck *T* score	−0.74 (0.99)	−3.22–2.16
Spine *T* score	−0.41 (1.39)	−3.70–5.49
Percent w/osteopenia-level *T* scores		0–100
Femoral (g/cm^2)	24%	
Femoral neck (g/cm^2)	37%	
Spine (g/cm^2)	27%	
Percent w/osteoporosis-level *T* scores		0–100
Femoral (g/cm^2)	2%	
Femoral neck (g/cm^2)	4%	
Spine (g/cm^2)	7%	

Note. MIDUS = Midlife in the United States: A National Longitudinal Study of Health & Well-Being. $N = 333$. Mean (*SD*) values are presented for continuous variables. Units are expressed in parentheses after variable name.

Table 2. Coefficients and Standardized β from Linear Regression Model Predicting Femoral *T* Score.

Variable	*B (SE)*	β	*p*
Age at Wave 2	−0.01 (.005)	−.14	*.007*
Body mass index at Wave 2	0.09 (.01)	.47	*<.001*
Number of health conditions at Wave 1	−0.16 (.05)	−.15	*.001*
Menopause	−0.74 (.15)	−.29	*<.001*
Female	0.15 (.11)	.08	*.166**
Insulin	−0.01 (.005)	−.12	*.029*
Constant	−1.78 (.38)	0	*<.001*

Note. $N = 333$. $R^2 = 0.3198$. Adjusted $R^2 = 0.3072$. *p* values are presented in italics.
*indicates not significant but changed the main independent variable by 10% or more.

Model 1 selected independent variables of femoral *T* score included age at Time 2, BMI at Time 2, the number of health conditions the respondent reported at Time 1, menopause, female, and insulin. For Model 2, femoral neck *T* score outcome, the model-selected independent variables included age at Time 2, alcohol consumption at Time 2, BMI at Time 2, insulin level, menopause, and female. Finally, Model 3, which used spine *T* score as the outcome variable, had model-selected independent variables including age at Time 2, changes in friend strain across waves, changes in poverty across waves, high CRP, the number of minor children in the household at Time 2, HDL cholesterol, and menopause.

The results for Model 1 are shown in Table 2. The multiple R^2 of 0.32 is moderate for explaining variance in femoral *T* score. Each additional year of age is associated with 0.01 points lower femoral *T* score ($p = .007$), while one point higher BMI at Time 2 is associated with 0.09 points higher femoral *T* score ($p < .001$). Furthermore, each additional health condition at Time 1 is associated with 0.16 points lower femoral *T* score ($p = .001$). Menopause was associated with 0.74 points lower femoral *T* score ($p < .001$), and a one point higher insulin level is associated with a 0.01 point lower femoral *T* score ($p = .029$). Finally, from the model selection procedure, we included the variable for female as it adjusted the main independent variable by 10% or more, but it was not significantly related to femoral *T* score.

In Model 2, we estimated bone density using the femoral neck *T* score. The results are shown in Table 3. The multiple R^2 of 0.26 was again in the moderate category for the independent variables explaining variance in femoral neck *T* score. Each additional year of age is associated with 0.02 points lower femoral neck *T* score ($p = .001$), while one point higher BMI at

Table 3. Coefficients and Standardized βs From Linear Regression Model Predicting Femoral Neck *T* score.

Variable	*B (SE)*	β	*p*
Age at Wave 2	−0.02 (.005)	−.18	*.001*
Body mass index at Wave 2	0.07 (.01)	.38	*<.001*
Alcohol consumption at Wave 2 (ever drinker, last 30 days omitted)			
3+ drinks/week, last 30 days	0.12 (.12)	.12	*.333*
Never drinker, last 30 days	−0.25 (.11)	−.26	*.025*
Insulin	−0.01 (.006)	−.13	*.025*
Menopause	−0.65 (.15)	−.26	*<.001*
Female	0.23 (.12)	.12	*.049*
Constant	−1.67 (.39)	.05	*<.001*

Note. $N = 333$. $R^2 = 0.2580$. Adjusted $R^2 = 0.2420$. *p* values are presented in italics.
*indicates not significant but changed the main independent variable by 10% or more.

Time 2 is associated with 0.07 points higher femoral neck *T* score ($p < .001$). Both of these results are very consistent with the femoral *T* score model. However, for femoral neck *T* score, alcohol consumption at Time 2 is a significant independent variable. Specifically, compared to those who have had a drink in the last 30 days, those who have not had a drink in the last 30 days have lower *T* scores ($p = .025$). Additionally, having higher insulin levels is associated with lower *T* scores ($p = .025$). As in the prior model, menopause was associated with 0.65 points lower femoral neck *T* score ($p < .001$), but unlike the prior model, being female was associated with 0.23 points higher femoral neck *T* score ($p = .049$).

The Model 3 results are shown in Table 4. The multiple R^2 of 0.15 is weak and indicates the explanatory variables do not explain variability in spine *T* score as well as the prior models accounted for variability in femoral and femoral neck *T* scores. Unlike with the prior models, menopause was only marginally significantly associated with lower spine *T* score ($p = .054$). For spine *T* score, compared to femoral and femoral neck *T* score, a few additional variables are relevant. For biological/health variables, high CRP is associated with higher spine *T* scores ($p < .001$), while higher HDL cholesterol is associated with lower *T* scores ($p = .041$). For social/socioeconomic variables, having a reduction in friend strain between waves is associated with lower *T* scores (a paradoxical reaction; $p = .015$) and being in poverty at Time 2, when compared to never being in poverty, is associated with lower spine *T* scores ($p = .019$). Finally, from the model selection procedure, we included the

Table 4. Coefficients and Standardized βs From Linear Regression Model Predicting Spine *T* Score.

Variable	*B (SE)*	β	*p*
Age at Wave 2	−0.01 (.01)	−.05	*.466**
Friend strain across waves (same strain omitted)			
Strain improved from Wave 1 to Wave 2	−0.44 (.18)	−.32	*.015*
Strain worsened from Wave 1 to Wave 2	−0.29 (.19)	−.21	*.136*
Poverty across waves (not in poverty both waves omitted)			
Poverty at Wave 1 but not Wave 2	−0.40 (.21)	−.29	*.062*
Poverty at Wave 2 but not Wave 1	−0.86 (.36)	−.62	*.019*
Poverty at both waves	0.73 (.94)	.53	*.437*
High CRP	0.79 (.18)	.23	*<.001*
Menopause	−0.41 (.21)	−.12	*.054*
HDL cholesterol	−0.01 (.004)	−.11	*.041*
Number of children under 18 at Wave 2	0.07 (.07)	.06	*.296**
Constant	0.61 (.56)	.26	*.281*

Note. CRP = C-reactive protein. $N = 333$. $R^2 = 0.1465$. Adjusted $R^2 = 0.1199$. *p* values are presented in italics.
*indicates not significant but changed the main independent variable by 10% or more.

variables for age at Time 2 and number of children under 18 in the household at Time 2 as they adjusted the main independent variable by 10% or more, but they did not demonstrate a significant relationship to spine *T* scores.

Discussion

In this project, we tested for diminished BMD, when compared to the normal, osteopenia, and osteoporosis ranges, as one way of examining accelerated aging under the conceptual model that osteopenia and osteoporosis are indicators of aging (see Figure 1). The results from our analysis were mixed; some hypotheses were supported while others were not. We did find some support for Hypothesis 1 as regards spine *T* scores and poverty. Exposure to poverty at Time 2 was associated with lower BMD, but there was no association with childhood exposure to poverty, unlike prior research (Karlamangla et al., 2013). Unfortunately, there was no support for Hypotheses 2 or 3: marital status and education did not appear to be related to BMD. Hypothesis 4 was supported as friend strain, number of children under the age of 18 (as a confounder), BMI, alcohol consumption, insulin, HDL cholesterol, number

of health conditions, and age were all associated with at least one of the BMD outcome variables. Finally, Hypothesis 5 stated that CRP would be strongly associated with BMD, and there would be an inverse relationship. While high CRP was a significant independent variable for spine *T* score, the relationship was positive, and it did not appear to be related to femoral and femoral neck *T* scores. Thus, Hypothesis 5 was not supported. While, in general, our initial hypotheses were minimally supported, the results suggest controlling for all available domains when testing social variables as indicators of stress and stress mediators will elucidate a different understanding of BMD and accelerated aging.

An association of poverty was not forthcoming in either femoral BMD measurement; however, poverty was associated with spine *T* score. The pattern of the relationship of BMD to the different categories of the poverty measure appears to be derived from the demographics and representativeness of the sample. The slopes of the poverty categories indicate an inverse relationship between BMD and being in poverty in either Wave 1 or Wave 2, demonstrating that being in poverty in either wave is associated with lower BMD at the second wave. Most notably, being in poverty at Wave 2 was associated with much lower spine *T* scores compared to the young adult mean: 0.86, which is 43% of the normal range of BMD (−1 to 1). The sample was too small to assess the role of being in poverty at both time points.

Holding all else constant, poverty could be a factor that drives osteopenic (usually associated with aging) *T* scores compared to those not in poverty, especially in light of further analysis of poverty and spine *T* score; however, definitive conclusions regarding causality cannot be made. First, poverty appears to exacerbate bone loss in adults over the age of 50; an analysis of variance (ANOVA) test comparing individuals who experienced poverty (in either or both waves) to those who have not (F value = 8.325, $df = 160$, $p = .004$) found lower spine *T* scores in older adults who have experienced poverty (either at one or both waves). This pattern is not unexpected; poverty has been found in conjunction with very high levels of CRP (>10 mg/L), an indicator of CVD, (Alley et al., 2006). Second, when adults under the age of 50 were similarly analyzed, the ANOVA results were not significant (F value = 0.732, $df = 169$, $p = .393$). Upon examining simple linear regression coefficients for the model on individuals under age 50, we found the standard error was similar in magnitude to the slope of poverty ($SE = 0.226$, $b = 0.194$), which indicates imprecise estimation, and thus inconclusive results. Of the adults under age 50 who experienced poverty ($N = 41$), 27% had scores in the osteopenia and osteoporosis range, suggesting there might be a

detectable relationship with poverty on such an age-associated condition if the sample size were larger.

A trend in education level may not be present in these data due to the sample being skewed toward a more educated participant. More education has long been thought to be related to better health outcomes (e.g., Cutler & Lleras-Muney, 2006), but in this sample, that is, not the case. This may be due to the already high level of education of the participants: 19% were high school graduates, 2% did not graduate from high school, 28% had some college, and 51% were college graduates.

A new social variable found to be associated with bone health, friend strain, was a significant independent variable for vertebral BMD. Decreases in strain across the two waves were significantly associated with reduced BMD, compared to unchanging strain levels. While an increase in strain being associated with worse outcomes would be consistent with expectations, the improvement in strain having a negative value is not. Because MIDUS did not ask for reasons why strain may have changed over time, we can only speculate that some unknown factor was at play. This effect may have been due to the perception of the question by the respondents. We also considered the possibility that changes in the respondent's health were associated with changes in friend strain (i.e., worsening health reduced friend strain), but we did not find any evidence to suggest this was the case. Ultimately, we believe this is an area that requires further research because the causes of these changes are unknown and thus cannot be fully tested in our data.

An expected outcome of our models is the relationship of moderate alcohol consumption to higher BMD. An examination of health behaviors in our models indicates that alcohol consumption has a relationship to femoral neck *T* score; not drinking in the last 30 days at Wave 2, versus having had a drink at some point in the last 30 days, was associated with lower BMD, and thus higher risk. This suggests that at least moderate consumption of alcohol may be protective against low BMD, which is supported by the literature (Berg et al., 2008). Alternatively, people with low BMD may have multiple health problems and therefore be less likely to consume alcohol.

Several of the other results in the BMD models are expected as they relate to confounding factors, although the relationship is not necessarily clear. Having experienced menopause is associated with lower *T* scores, which is a result we would expect from the literature. The negative association of insulin with BMD is unexpected given insulin's relationship with BMI; obesity is a risk factor for insulin resistance (Ye, 2013). Although significant, the coefficients for insulin are small in this sample, indicating this

relationship may be an artifact of sample size/representation. Likewise, high cholesterol is linked to lower BMD (Mandal, 2015). Number of minor children living at home is also a confounder, likely with age, as parents with minor children at home tend to be younger. *T* tests confirm this is the case ($t = 10.29$, $df = 331$, $p < .001$), with parents of minor children in the household being 10 years younger than those without.

Interestingly, but not unexpectedly, the number of health conditions variable was related to lower BMD in our femoral *T* score model. Because we removed individuals with known health conditions that affect BMD (e.g., tuberculosis), the number of health conditions variable should reflect an index of biological stress from battling a number of medical conditions. But, it may just point to a lack of understanding of the effects on BMD that some medical conditions exert.

The general lack of association between CRP and BMD, and the paradoxical reaction between them (higher CRP associated with higher BMD in the spine *T* score model), is not unexpected; the literature has pointed to contradictions that suggest there is not a straightforward relationship between the two markers. Our results are similar to Ganesan, Teklehaimanot, Tran, Asuncion, and Norris (2005); the two markers are generally not correlated with these data, and there is no predictive relationship between the two. However, studies of correlation and regression do not directly test the underlying biological processes, and we should look to the bone biology literature for support of these findings.

When examining the bony microstructure literature, it is found that CRP is reportedly regulated by cytokines including interleukin (IL)-1, IL-6, and tumor necrosis factor (Koh et al., 2005). Therefore, scholars have hypothesized that increased CRP is related to bone resorption through IL-6 polymorphisms that are independently linked to levels of CRP and osteoclastic activity (Ferrari et al., 2003). IL-6 then may promote osteoclastogenesis (Hofbauer, Khosla, Dunstan, Lacey, Boyle, & Riggs, 2000), and osteoclastic activity (Hofbauer et al., 2000), as well as regulate CRP production (in the liver; Ferrari et al., 2003). Further, it is thought that inflammation is linked to cytokines such as IL-6, which leads to an upregulation in IL-6, and an increase in CRP and bone resorption, which may lead to a decrease in BMD (Koh et al., 2005). This model of CRP's relationship to BMD has been the conventional wisdom for some time, but a recent paper showed that increased CRP was associated with decreased osteoclastogenesis (Cho et al., 2016). Collectively, the findings in the literature, coupled with the lack of correlation between CRP and BMD, indicate there is likely more to the process than originally documented, and further research is needed.

The main bias in this study is the lack of representativeness of the MIDUS sample; to conduct this research, we had to greatly reduce the sample size. Moreover, the respondents represent a wealthier, more educated, and less ethnically diverse segment of the U.S. population, making the generalizability of our results limited to the demographics described by MIDUS. We do note that our analytic sample is quite similar to the full MIDUS biomarker sample, though the analytic sample is slightly younger, slightly more educated, and slightly healthier (see Supplementary Table S1). The analytic sample is also somewhat more likely to have had strain measures improve across waves. Our sample size precluded us from running separate models of BMD across meaningful subsets in our data (e.g., by race/ethnicity) to test our hypotheses. As the type of independent variables used in BMD studies vary so much in the literature, it is not surprising that early life experiences, some individual experiences, and levels of CRP were not associated with BMD in the expected manner. A variety of factors not explored here may have influenced the results, including the government's ability to deal with social and health disparities (Gortmaker et al., 2011), because politics influence public health policy (Navarro et al., 2006), which in turn may shape social environments and the resulting trends in these data. Furthermore, evidence has emerged that experiences in utero are strong determinants of BMD and osteoporosis later in life (see Riancho & Brennan-Olsen, 2017), an aspect the current study could not examine.

One new variable that is associated with BMD was identified in this research: friend strain. Decreasing friend strain was associated with lower BMD, which is counterintuitive but may make sense in relation to the broadness of the question respondents were asked. Ultimately, the findings from this study suggest a need to rethink some of the prior assumptions in the literature about what factors are associated with decreased BMD and accelerated aging, to consider a wider range of factors, and to specifically examine how different combinations of factors influence outcomes. The health costs of accelerated aging are high, so a broader understanding of contributing factors is essential in potentially mitigating future need for treatment, while also protecting the quality of life for aging adults. The results of this study can be used to inform future research and by clinicians as they consider the constellation of risk factors for their patients.

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Supplemental Material

Supplemental material for this article is available online.

References

Alley, D. E., Seeman, T. E., Kim, J. K., Karlamangla, A., Hu, P., & Crimmins, E. M. (2006). Socioeconomic status and C-reactive protein levels in the US population: NHANES IV. *Brain, Behavior, and Immunity*, *20*, 498–504.

Alver, K., Søgaard, A. J., Falch, J. A., & Meyer, H. E. (2007). The Oslo health study: Is bone mineral density higher in affluent areas? *International Journal for Equity in Health*, *6*, 19.

Amiri, M., Nabipour, I., Larijani, B., Beigi, S., Assadi, M., Amiri, Z., & Mosadeghzadeh, S. (2008). The relationship of absolute poverty and bone mineral density in postmenopausal Iranian women. *International Journal of Public Health*, *53*, 290–296. doi:10.1007/s00038-008-8033-x

Barr, D. A. (2014). *Health disparities in the United States* (2nd ed.). Baltimore, MD: Johns Hopkins University Press.

Benetou, V., Orfanos, P., Feskanich, D., Michaëlsson, K., Pettersson-Kymmer, U., Ahmed, L. A., … Trichopoulou, A. (2015). Education, marital status, and risk of hip fractures in older men and women: The CHANCES project. *Osteoporosis International*, *26*, 1733–1746. doi:10.1007/s00198-015-3054-9

Berg, K. M., Kunins, H. V., Jackson, J. L., Nahvi, S., Chaudhry, A., Harris, K. A., … Arnsten, J. H. (2008). Association between alcohol consumption and both osteoporotic fracture and bone density. *The American Journal of Medicine*, *121*, 406–418.

Berglundh, S., Malmgren, L., Luthman, H., McGuigan, F., & Åkesson, K. (2015). C-reactive protein, bone loss, fracture, and mortality in elderly women: A

longitudinal study in the OPRA cohort. *Osteoporosis International*, *26*, 727–735. doi:10.1007/s00198-014-2951-7

Blalock, S. J., Vellis, B. M. D., DeVellis, R. F., & Sauter, S. V. H. (1988). Self-evaluation processes and adjustment to rheumatoid arthritis. *Arthritis & Rheumatism*, *31*, 1245–1251.

Brennan, S. L., Henry, M. J., Wluka, A. E., Nicholson, G. C., Kotowicz, M. A., Williams, J. W., & Pasco, J. A. (2009). BMD in population-based adult women is associated with socioeconomic status. *Journal of Bone and Mineral Research*, *24*, 809–815. doi:10.1359/jbmr.081243

Brennan, S. L., Leslie, W. D., & Lix, L. M. (2013). Associations between adverse social position and bone mineral density in women aged 50 years or older: Data from the Manitoba bone density program. *Osteoporosis International*, *24*, 2405–2412. doi:10.1007/s00198-013-2311-z

Brennan, S. L., Pasco, J. A., Urquhart, D. M., Oldenburg, B., Hanna, F., & Wluka, A. E. (2009). The association between socioeconomic status and osteoporotic fracture in population-based adults: A systematic review. *Osteoporos International*, *20*, 1487–1497. doi:10.1007/s00198-008-0822-9

Cho, I. J., Choi, K. H., Oh, C. H., Hwang, Y. C., Jeong, I. K., Ahn, K. J., & Chung, H. Y. (2016). Effects of C-reactive protein on bone cells. *Life Sciences*, *145*, 1–8. doi:10.1016/j.lfs.2015.12.021

Crandall, C., Karlamangla, A., Merkin, S., Binkley, N., Carr, D., Greendale, G., & Seeman, T. (2015). Adult bone strength of children from single-parent families: The Midlife in the United States study. *Osteoporosis International*, *26*, 931–942. doi:10.1007/s00198-014-2990-0

Crandall, C. J., Han, W., Greendale, G. A., Seeman, T., Tepper, P., Thurston, R., … Karlamangla, A. S. (2014). Socioeconomic status in relation to incident fracture risk in the study of women's health across the nation. *Osteoporosis International*, *25*, 1379. doi:10.1007/s00198-013-2616-y

Cutler, D. M., & Lleras-Muney, A. (2006). *Education and health: Evaluating theories and evidence*. Cambridge, MA: National Bureau of Economic Research.

Dahl, K., Ahmed, L. A., Joakimsen, R. M., Jørgensen, L., Eggen, A. E., Eriksen, E. F., & Bjørnerem, Å. (2015). High-sensitivity C-reactive protein is an independent risk factor for non-vertebral fractures in women and men: The tromsø study. *Bone*, *72*, 65–70. doi:10.1016/j.bone.2014.11.012

dePablo, P., Cooper, M. S., & Buckley, C. D. (2012). Association between bone mineral density and C-reactive protein in a large population-based sample. *Arthritis & Rheumatism*, *64*, 2624–2631. doi:10.1002/art.34474

Dienberg Love, G., Seeman, T. E., Weinstein, M., & Ryff, C. D. (2010). Bioindicators in the MIDUS national study: Protocol, measures, sample, and comparative context. *Journal of Aging and Health*, *22*, 1059–1080.

Du, Y., Zhao, L. J., Xu, Q., Wu, K. H., & Deng, H. W. (2017). Socioeconomic status and bone mineral density in adults by race/ethnicity and gender: The Louisiana osteoporosis study. *Osteoporosis International*, *28*, 1699–1709. doi:10.1007/s00198-017-3951-1

Epel, E. S., Blackburn, E. H., Lin, J., Dhabhar, F. S., Adler, N. E., Morrow, J. D., & Cawthon, R. M. (2004). Accelerated telomere shortening in response to life stress. *Proceedings of the National Academy of Science USA*, *101*, 17312–17315.

Farahmand, B. Y., Persson, P. G., Michaëlsson, K., Baron, J. A., Parker, M. G., & Ljunghall, S., & Swedish Hip Fracture Group. (2000). Socioeconomic status, marital status and hip fracture risk: A population-based case–control study. *Osteoporosis International*, *11*, 803–808. doi:10.1007/s001980070060

Ferrari, S. L., Ahn-Luong, L., Garnero, P., Humphries, S. E., & Greenspan, S. L. (2003). Two promoter polymorphisms regulating interleukin-6 gene expression are associated with circulating levels of C-reactive protein and markers of bone resorption in postmenopausal women. *Journal of Clinical Endocrinology and Metabolism*, *88*, 255–259. doi:10.1210/jc.2002-020092

Ganesan, K., Teklehaimanot, S., Tran, T. H., Asuncion, M., & Norris, K. (2005). Relationship of C-reactive protein and bone mineral density in community-dwelling elderly females. *Journal of the National Medical Association*, *97*, 329.

Ginaldi, L., Di Benedetto, M. C., & De Martinis, M. (2005). Osteoporosis, inflammation and ageing. *Immunity and Ageing*, *2*, 14. doi:10.1186/1742-4933-2-14

Gortmaker, S. L., Swinburn, B. A., Levy, D., Carter, R., Mabry, P. L., Finegood, D. T., … Moodie, M. L. (2011). Changing the future of obesity: Science, policy, and action. *The Lancet*, *378*, 838–847. doi:10.1016/S0140-6736(11)60815-5

Hofbauer, L. C., Khosla, S., Dunstan, C. R., Lacey, D. L., Boyle, W. J., & Riggs, B. L. (2000). The roles of osteoprotegerin and osteoprotegerin ligand in the paracrine regulation of bone resorption. *Journal of Bone and Mineral Research*, *15*, 2–12.

Jilka, R. L., Weinstein, R. S., Takahashi, K., Parfitt, A. M., & Manolagas, S. C. (1996). Linkage of decreased bone mass with impaired osteoblastogenesis in a murine model of accelerated senescence. *The Journal of Clinical Investigation*, *97*, 1732–1740.

Karlamangla, A. S., Mori, T., Merkin, S. S., Seeman, T. E., Greendale, G. A., Binkley, N., & Crandall, C. J. (2013). Childhood socioeconomic status and adult femoral neck bone strength: Findings from the Midlife in the United States study. *Bone*, *56*, 320–326. doi:10.1016/j.bone.2013.06.021

Kiecolt-Glaser, J. K., Gouin, J. P., Weng, N. P., Malarkey, W. B., Beversdorf, D. Q., & Glaser, R. (2011). Childhood adversity heightens the impact of later-life

caregiving stress on telomere length and inflammation. *Psychosomatic Medicine*, *73*, 16–22. doi:10.1097/PSY.0b013e31820573b6

Kipen, Y., Buchbinder, R., Forbes, A., Strauss, B., Littlejohn, G., & Morand, E. (1997). Prevalence of reduced bone mineral density in systemic lupus erythematosus and the role of steroids. *The Journal of rheumatology*, *24*, 1922–1929.

Koh, J. M., Khang, Y. H., Jung, C. H., Bae, S., Kim, D. J., Chung, Y. E., & Kim, G. S. (2005). Higher circulating hsCRP levels are associated with lower bone mineral density in healthy pre- and postmenopausal women: Evidence for a link between systemic inflammation and osteoporosis. *Osteoporosis International*, *16*, 1263–1271. doi:10.1007/s00198-005-1840-5

Kubzansky, L., Seeman, T. E., & Glymour, M. M. (2014). Biological pathways linking social conditions and health: Plausible mechanisms and emerging puzzles. In L. Berkman, I. Kawachi, & M. M. Glymour (Eds.), *Social epidemiology: New perspectives on social determinants of global population health* (pp. 512–561). Oxford, England: Oxford University Press.

Lyles, C. R., Schafer, A. L., & Seligman, H. K. (2014). Income, food insecurity, and osteoporosis among older adults in the 2007–2008 National Health and Nutrition Examination Survey (NHANES). *Journal of Health Care for the Poor and Underserved*, *25*, 1530–1541. doi:10.1353/hpu.2014.0174

Maldonado, G., & Greenland, S. (1993). Simulation study of confounder-selection strategies. *American Journal of Epidemiology*, *138*, 923–936.

Mandal, C. C. (2015). High cholesterol deteriorates bone health: New insights into molecular mechanisms. *Frontiers in Endocrinology*, *6*, 165.

Midlife in the United States: A National Longitudinal Study of Health & Well-Being. (2011). History & overview of MIDUS. Retrieved from http://midus.wisc.edu/scopeofstudy.php

Miller-Martinez, D., Seeman, T., Karlamangla, A., Greendale, G., Binkley, N., & Crandall, C. (2014). Marital histories, marital support, and bone density: Findings from the midlife in the United States study. *Osteoporosis International*, *25*, 1327–1335. doi:10.1007/s00198-013-2602-4

Navarro, V., Muntaner, C., Borrell, C., Benach, J., Quiroga, Á., Rodríguez-Sanz, M., … Pasarín, M. I. (2006). Politics and health outcomes. *The Lancet*, *368*, 1033–1037. doi:10.1016/S0140-6736(06)69341-0

Oliveira, B. S., Zunzunegui, M. V., Quinlan, J., Fahmi, H., Tu, M. T., & Guerra, R. O. (2016). Systematic review of the association between chronic social stress and telomere length: A life course perspective. *Ageing Research Review*, *26*, 37–52. doi:10.1016/j.arr.2015.12.006

Quah, C., Boulton, C., & Moran, C. (2011). The influence of socioeconomic status on the incidence, outcome and mortality of fractures of the hip. *Journal of Bone Joint Surgery British*, *93*, 801–805.

Reyes, C., García-Gil, M., Elorza, J. M., Fina-Avilés, F., Mendez-Boo, L., Hermosilla, E., . . . Prieto-Alhambra, D. (2015). Socioeconomic status and its association with the risk of developing hip fractures: A region-wide ecological study. *Bone*, *73*, 127–131. doi:10.1016/j.bone.2014.12.019

Riancho, J. A., & Brennan-Olsen, S. L. (2017). The epigenome at the crossroad between social factors, inflammation, and osteoporosis risk. *Clinical Review of Bone Mineral Metabolism*, *15*, 59–63. doi:10.1111/j.1365-2028.2006.00707.x

Runsten, S., Korkeila, K., Koskenvuo, M., Rautava, P., Vainio, O., & Korkeila, J. (2014). Can social support alleviate inflammation associated with childhood adversities? *Nordic Journal of Psychiatry*, *68*, 137–144.

Ryff, C. D., Seeman, T., & Weinstein, M. (2012). *Documentation for bone health project in MIDUS II biomarker project (P4), Midlife in the United States (MIDUS 2): Biomarker project, 2004-2009*. Madison: University of Wisconsin Institute on Aging.

Sponholtz, T. R., Zhang, X., Fontes, J. D. T., Meigs, J. B., Cupples, L. A., Kiel, D. P., . . . McLean, R. R. (2014). Association between inflammatory biomarkers and bone mineral density in a community-based cohort of men and women. *Arthritis Care & Research*, *66*, 1233–1240. doi:10.1002/acr.22270

StataCorp. (2013). *Stata statistical software: Release 13*. College Station, TX: StataCorp LLC.

Uchino, B. N., Ruiz, J. M., Smith, T. W., Smyth, J. M., Taylor, D. J., Allison, M., & Ahn, C. (2016). Ethnic/racial differences in the association between social support and levels of C-reactive proteins in the North Texas heart study. *Psychophysiology*, *53*, 64–70.

Vestergaard, P., Rejnmark, L., & Mosekilde, L. (2006). Socioeconomic aspects of fractures within universal public healthcare: A nationwide case-control study from Denmark. *Scandinavian Journal of Public Health*, *34*, 371–377. doi:10.1080/14034940500441223

Williamson, J. B., Porges, E. C., Lamb, D. G., & Porges, S. W. (2015). Maladaptive autonomic regulation in PTSD accelerates physiological aging. *Frontiers in Psychology*, *5*, 1571.

Wilson, R. T., Chase, G. A., Chrischilles, E. A., & Wallace, R. B. (2006). Hip fracture risk among community-dwelling elderly people in the United States: A prospective study of physical, cognitive, and socioeconomic indicators. *American Journal of Public Health*, *96*, 1210–1218.

Wu, Z. J., He, J. L., Wei, R. Q., Liu, B., Lin, X., Guan, J., & Lan, Y. B. (2015). C-reactive protein and risk of fracture: A systematic review and dose–response meta-analysis of prospective cohort studies. *Osteoporosis International*, *26*, 49–57. doi:10.1007/s00198-014-2826-y

Ye, J. (2013). Mechanisms of insulin resistance in obesity. *Frontiers of medicine*, *7*, 14–24.

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