Social Science & Medicine xxx (2017) 1–9

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

Long-term physical health consequences of perceived inequality: Results from a twin comparison design

Joseph A. Schwartz*

School of Criminology and Criminal Justice, University of Nebraska at Omaha, Omaha, NE 68182-0149, USA

ARTICLE INFO

Article history:
Received 12 January 2017
Received in revised form
23 May 2017
Accepted 2 June 2017
Available online xxx

Keywords:
Inequality
Allostatic load
Twins
Stress

ABSTRACT

Rationale: Previous research has identified long-term exposure to stress as a risk factor for negative mental and physical health outcomes. This pattern of findings suggests that environmental stimuli that evoke feelings of stress or strain may also result in physiological responses, which may accumulate over the life course and ultimately increase the overall risk of various physical health conditions. This physiological "wear and tear" resulting from sustained levels of stress or strain has been previously operationalized as allostatic load (AL), a comprehensive indicator of stress exposure.
Objective: The current study examines the association between one potential environmental stressor—perceived inequality—and AL with a research design aimed at addressing both observed and unobserved sources of confounding; it also employs a more comprehensive AL measure (comprised of 24 biomarkers tapping seven physiological systems) than previous studies.
Method: The biomarker twin sample from the Midlife Development in the United States (MIDUS) study was used to estimate a series of twin comparison models, which include controls for latent sources of influence that cluster within families. The sibling comparison models also included additional controls for lifestyle choices, overall physical health, and demographics which may confound the examined associations.
Results: The results revealed significant associations between greater perceptions of inequality and greater overall levels of AL. The association persisted even after including controls for both observed and unobserved influences that may confound the examined associations but was limited to more recent measures of perceived inequality. Associations involving earlier measures of perceived inequality, along with a lifetime measure, failed to reach conventional levels of significance.
Conclusion: Perceived inequality appears to be a robust predictor of AL and potentially contributes to subsequent physical health problems, particularly for more proximate forms of perceived inequality.

1. Introduction

Physical health problems present a concerning economic challenge. During 2015, total health care spending in the United States reached $3.2 trillion, with the majority of the overall financial burden shouldered by the U.S. government (37%; Centers for Medicare and Medicaid Services, 2016). President Obama (2016) highlighted the immense cost of U.S. healthcare in a recent Special Communication in the *Journal of the American Medical Association,* which noted that 16% of the U.S. economy was spent on health care in 2008 (p. 526). Along with the financial burden of health care costs on the U.S. government, individuals are also tasked with substantial out-of-pocket costs totaling $338.1 billion in 2015, representing a 2.6% increase from the previous year. Physical health problems can also result in a number of indirect costs including a loss of productivity and absenteeism which may further compound the estimates reported above (Schultz et al., 2009).

Based on these observations, a substantial literature has focused on identifying factors that ultimately contribute to variation in health problems, with a particular emphasis on health disparities resulting from differences in socioeconomic status (SES; Dowd et al., 2009; Goldman, 2001; Sanders-Phillips et al., 2009). Taken together, the results of this expansive literature have revealed that physical health problems including obesity (Gordon-Larsen et al.,

* School of Criminology and Criminal Justice, University of Nebraska at Omaha, 6001 Dodge Street, 218 CPACS, Omaha, NE 68182-0149, USA.
E-mail address: jaschwartz@unomaha.edu.

http://dx.doi.org/10.1016/j.socscimed.2017.06.006
0277-9536/

2006), cardiovascular disease (Diez-Roux et al., 2000), and diabetes (Kumari et al., 2004) are more prevalent among individuals that occupy lower social positions. This pattern of results is so robust it has come to be referred to as a "social gradient in health" in the existing literature (Goldman, 2001, p. 119).

Drawing from Geronimus's (1991) "weathering hypothesis," previous studies have found evidence suggesting that individuals who experience social or economic adversity and political marginalization (such as racial minorities and African-Americans, more specifically) also experience more health problems. While the weathering hypothesis was originally formulated to explain racial differences in chronic morbidity (Geronimus, 1991; Geronimus et al., 2006), the underlying explanations may also provide a better understanding of the mechanisms driving the association between SES and physical health. The weathering hypothesis has enjoyed support in the existing literature, with previous studies reporting evidence of greater overall levels of stress among racial minorities (Brody et al., 2014; Geronimus et al., 2006, 2010; Green and Darity, 2010) and that such increases in stress are significantly associated with a wide range of physical health problems (Geronimus et al., 2010; Jackson et al., 2010; Turner and Avison, 2003). Previous studies have also revealed that greater overall perceptions of inequality and discrimination ultimately result in increased levels of stress (Brunner, 1997; Delhey and Dragolov, 2014). While racially motivated sources of discrimination are the most commonly examined in the existing literature (for example, see Brody et al., 2014; Geronimus et al., 2006), previous studies have also observed discriminatory treatment based on other characteristics including gender (Puhl et al., 2008), sexual orientation (Meyer, 2003), age (Oliver, 2009), and religious belief (Ghumman et al., 2013). Taken together, these findings indicate that inequality and discriminatory treatment, regardless of the impetus for such experiences, may have negative consequences and increase overall stress levels.

The underlying connection between stress and physical health has been inherently tied to the theoretical concept of *allostatic load* (AL), which refers to the cumulative wear and tear on various physiological systems due to exposure to chronic and acute sources of stress (McEwen, 1998; McEwen and Seeman, 1999; Sterling and Eyer, 1988). More specifically, AL is related to the process of *allostasis* (Sterling and Eyer, 1988), which refers to the activation of physiological systems including the cardiovascular, metabolic, immune, and endocrine systems based on demands stemming from encountered environmental experiences (McEwen, 1998; McEwen and Seeman, 1999). In most situations, allostasis is considered adaptive and provides the physiological responses necessary for encountering and overcoming an environmental obstacle. However, the process of allostasis is primed for acute stressors that persist for only a relatively short period of time, as physiological responses to such stressors are expected to be activated intermittently and normalize after the stressor subsides. When such physiological systems are activated more consistently and for more sustained periods of time, overall levels of allostatic load (or system dysregulation) may increase. Previous studies have indicated that greater overall levels of AL are associated with a wide range of deleterious physical and mental health outcomes (Juster et al., 2010; Mattei et al., 2010; Seeman et al., 2001).

Despite the connection between AL and such outcomes, the underlying process that ultimately links these two concepts is complex and involves multiple mediators. McEwen and Seeman (1999) provided an overview of these processes by distinguishing between *primary mediators*, *primary effects*, *secondary outcomes*, and *tertiary outcomes*. *Primary mediators* refer to stress hormones (i.e., cortisol and other glucocorticoids) released during the process of allostasis that have widespread influences on multiple physiological processes (or *primary effects*) and operate through "cellular events" which involve enzymes, receptors, ion channels, and structural proteins (Juster et al., 2010). Over time, downstream physiological systems attempt to compensate for the over- or underproduction of primary mediators to overcome the detrimental impact of primary effects, resulting in functional changes in such systems. These changes, or *secondary outcomes*, can impact cardiovascular, immune, and metabolic (among other) systems, causing such systems to operate at sub-optimal levels and ultimately resulting in dysfunction.

Directly in line with these findings, previous studies have demonstrated a positive and significant association between unequal treatment and physical health problems, such that individuals with greater perceptions of inequality display greater overall levels of inflammation (which has been linked to increased risk for cardiovascular disease and diabeates; Cunningham et al., 2012); increased levels of cortisol (Fuller-Rowell et al., 2012); increased sympathetic nervous system activity (Sawyer et al., 2012); and, greater overall levels of AL (which has been linked to neural atrophy, heart disease, and memory problems; Brody et al., 2014; Geronimus et al., 2006; Green and Darity, 2010; Gruenewald et al., 2012). In addition, the results of a recent meta-analysis synthesizing the results of 36 studies and 303 individual effects found a negative and significant association between perceived discrimination and physical health outcomes ($r = -0.13$), indicating that individuals who experienced more discrimination also exhibited poorer physical health outcomes (Pascoe and Smart Richman, 2009).

Despite the significant number of studies reporting associations between perceived inequality or discrimination and AL, determining whether such associations may be interpreted as causal effects represents a methodological challenge. The process of selection, whereby individual characteristics that increase the likelihood of physical health problems may also be more likely to elicit unfair treatment, remains particularly problematic. Although some previous studies have employed sophisticated multivariate research designs to limit selection effects, such designs are limited in that they only control for *observed* measures of selection. Additional latent or *unobserved* sources of selection remain particularly important, but are more difficult to address methodologically (Goldman, 2001). These sources of influence can encompass virtually any background variables that potentially impact the examined association (such as family experiences or genetic influences) and failing to take such influences into account may result in biased findings. For example, Hamdi et al. (2016) recently examined the association between educational attainment (a common measure of SES) and AL. The results revealed a significant association in preliminary models; but after including additional controls for unobserved sources of selection, including genetic and environmental influences that cluster within families, the association fell from significance.

Directly in line with these findings, the current study aims to examine the potential association between perceived inequality and AL with a twin comparison model, which includes controls for both observed and unobserved sources of selection (Hamdi et al., 2016; Turkheimer and Harden, 2014). In addition to the use of a twin comparison model, the current study also makes use of a comprehensive measure of AL. Few existing studies have employed comprehensive measures of AL when examining the potential ramifications of unequal or discriminatory treatment, with the majority of the existing literature focusing on dysfunction within a single physiological system or a subset of systems (Cunningham et al., 2012; Fuller-Rowell et al., 2012; Sawyer et al., 2012). Of the few studies that have employed a comprehensive measure of AL (Brody et al., 2014; Geronimus et al., 2006), the number of

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006

biomarkers used to measure AL is somewhat limited. The current study employs a measure of AL tapping seven physiological systems comprised of a total of 24 biomarkers.

2. Methods

2.1. Data

The current study uses data from the Survey of Midlife Development in the United States (MIDUS), a longitudinal study of a nationally representative sample of adults funded by the National Institute on Aging (Brim et al., 1996). The first wave of the study (MIDUS I) was conducted between 1995 and 1996 and included a sample of over 7000 participants (for additional information see Radler and Ryff, 2010). A second wave of data collection (MIDUS II), which included a total of 4963 participants from MIDUS I (70% retention rate; Love et al., 2010; Radler and Ryff, 2010), was conducted between 2004 and 2006 when participants were between 32 and 84 years old (Brim et al., 2004). A subsample of MIDUS II participants was asked to participate in the Biomarker Project ($n = 1255$), which included an extensive battery of mental and physical health assessments carried out over two days and included the collection of 12-h urine samples, fasting blood draws, and saliva samples (for additional information including more specific specimen collection protocols, see Love et al., 2010).

Nested within the full MIDUS sample is a nationally representative sample of twin pairs ($n = 1914$ [988 pairs] for MIDUS I and $n = 1484$ [742 pairs] for MIDUS II). Twin pairs were recruited into the sample using a two step process. First, sampled households were screened for the presence of a twin. Second, all respondents that identified the presence of a co-twin (14.8% of households) were asked to participate in the MIDUS study. For families with more than one twin pair, all pairs that agreed to participate were included in the study. A subsample of twins that participated in the MIDUS II also participated in the Biomarker Project ($n = 388$; Love et al., 2010). Directly in line with previous studies employing similar analytic procedures, the current study is restricted to same-sex monozygotic (MZ; $n = 164$) and dizygotic (DZ; $n = 126$) twin pairs that participated in the Biomarker Project ($n = 290$; Hamdi et al., 2016; Lahey and D'Onofrio, 2010; Turkheimer and Harden, 2014). The final analytic sample is limited to same-sex DZ twins since the employed analytic procedures effectively account for gender differences *between* but not *within* families.

While there were no significant differences in AL across the full biomarker sample and the final analytic sample ($t = 1.54$, $p = 0.123$), the Wave I ($t = 4.60$, $p < 0.001$), Wave II ($t = 4.48$, $p < 0.001$) and lifetime inequality ($t = 5.27$, $p < 0.001$) measures were significantly greater in the full sample compared to the analytic sample. In addition, there were significantly fewer Caucasian participants in the biomarker sample ($z = 2.33$, $p = 0.02$). There were no additional significant differences between the two samples across the remaining study measures (results are presented in the online supplement). Additional descriptive information (means and prevalence) of all study measures for the final analytic sample (and the MZ and DZ twin subsamples) appear in Table 1.

2.2. Measures

2.2.1. Allostatic load

AL was assessed using 24 biomarkers collected during the MIDUS biomarker project. Table 2 lists all 24 biomarkers (along with sample means, ranges, and high-risk cut points) tapping seven physiological systems: 1) cardiovascular functioning (three items; e.g., resting heart rate, systolic blood pressure); 2) glucose metabolism (three items; e.g., blood glucose, glycosylated hemoglobin); 3) lipid metabolism (five items; e.g., body mass index, low density lipoprotein cholesterol levels); 4) inflammation (five items; e.g., C-reactive protein, fibrinogen); 5) hypothalamic-pituitary-adrenal axis (HPA) activity (two items; e.g., urine cortisol, dehydroepiandresterone sulfate [DHEA-S]); 6) sympathetic nervous system activity (two items; e.g., urine epinephrine, urine norepinephrine); and 7) parasympathetic nervous system activity (four items; e.g., high-frequency heart rate variability; standard deviation of R-R intervals).

The allostatic load measure was created following a four-step process outlined in previous studies (Brooks et al., 2014; Friedman et al., 2015; Hamdi et al., 2016; Karlamangla et al., 2014). First, all 24 biomarkers were recoded into quartiles. Second, a series of dichotomous indicator variables were used to identify respondents that fell within the highest risk quartile of each biomarker. Depending on the biomarker examined, the high-risk quartile was defined as the highest (top 75th percentile) or lowest (bottom 25th percentile) observed quartile of each biomarker examined in the MIDUS sample. Third, system risk scores were calculated by averaging the resulting dichotomous measures within each physiological system to reflect the overall proportion of biomarkers that fell within the high-risk range among each of the examined physiological systems. Fourth, and finally, the resulting seven system risk scores were then summed to reflect an overall summary score of AL. The resulting AL measure had a possible range of 0–7 (but an observed range of 0–5.03), with higher scores reflecting higher levels of AL ($M = 1.63$) and was *z*-transformed.

2.2.2. Perceived inequality

Following the lead of previous studies analyzing the MIDUS sample (Kessler et al., 1999; Ryff et al., 2004; Williams et al., 1997), perceived inequality was assessed using nine items from the MIDUS I and MIDUS II general interviews tapping overall perceptions of unequal treatment across a variety of scenarios. During both interviews, respondents were asked to report the frequency in which they encountered different experiences in their day-to-day lives reflecting unequal treatment (e.g., you were treated with less courtesy than others). Responses were coded categorically and ranged between 1 (*never*) and 4 (*often*) and summed across all nine items at each wave to create the Wave I ($M = 11.56$; SD = 3.40; Range = 9–26) and Wave II ($M = 11.79$; SD = 3.76; Range = 9–30) perceived inequality measures. A lifetime measure was also created by taking the mean of the Wave I and Wave II measures ($M = 11.67$; SD = 3.03; Range = 9–23.5). For all three perceived inequality measures, greater scores reflected greater perceptions of the variety and frequency of unequal treatment. All three perceived inequality measures were *z*-transformed prior to the estimation of the employed statistical models to aid in the interpretation of the resulting coefficients.

2.2.3. Covariates

The employed statistical models also included nine lifestyle and demographic covariates. Since the employed perceived inequality measures are not confined to one specific form of unequal treatment, it remains possible that lifestyle choices, as well as the social and physiological consequences that accompany such choices (i.e., obesity, heavy drinking, etc.), may simultaneously elicit unequal treatment and impact overall levels of AL, effectively confounding the association. Based on this possibility, four lifestyle covariates were included in the estimated models. First, respondents were asked whether they typically exercised for at least 20 min three or more times per week. Responses were coded dichotomously such that 0 = *no* and 1 = *yes*. Second, to assess longitudinal cigarette use, respondents were asked whether they regularly smoked cigarettes

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006

Table 1
Descriptive statistics for the full sample and twin subsamples.

	Full Sample	MZ Twins	DZ Twins	*F*-Statistic
Perceived Inequality Measures				
Wave 1 (Mean)	11.56	11.26	11.95	1.71†
Wave 2 (Mean)	11.79	11.29	12.43	2.59*
Lifetime (Mean)	11.67	11.27	12.19	2.58*
Covariates				
Weekly Exercise				−1.26
Yes (%)	80.34	82.93	76.98	
No (%)	19.66	17.07	23.02	
Smoking Status				−0.63
Never (%)	65.17	64.63	65.87	
Previous Smoker (%)	25.17	22.56	28.57	
Current Smoker (%)	9.66	12.80	5.56	
Alcohol Use Frequency				−0.76
Never (%)	35.17	34.15	36.51	
Less than Once/Week (%)	29.66	28.05	31.75	
1 or 2 Days/Week (%)	13.10	13.41	12.70	
3 or 4 Days/Week (%)	11.38	14.02	7.94	
5 or 6 Days/Week (%)	3.79	4.88	2.38	
Everyday (%)	6.90	5.49	8.73	
Current Fast Food Consumption				−0.66
Never (%)	16.55	13.41	20.63	
Less than Once/Week (%)	37.24	40.24	33.33	
1–3 Times/Week (%)	38.97	39.02	38.89	
4–6 Times/Week (%)	6.21	6.10	6.35	
7 or more Times/Week (%)	1.03	1.22	0.79	
Difficulty in Paying Bills				0.89
Not at all Difficult (%)	40.09	40.61	39.68	
Not Very Difficult (%)	38.07	41.10	34.13	
Somewhat Difficult (%)	16.29	14.79	18.25	
Very Difficult (%)	5.55	3.72	7.94	
Educational Achievement				0.31
Less than High School (%)	4.14	1.83	7.14	
High School (%)	19.02	19.60	18.25	
Some College (%)	29.48	32.62	25.40	
4 Year College Degree (%)	25.26	26.98	23.02	
Graduate School (%)	22.10	18.96	26.19	
Race				−0.86
Caucasian (%)	96.21	95.73	97.62	
All Other Races (%)	3.79	4.27	2.38	
Age (Mean)	57.06	56.29	58.07	1.36
Sex				−0.86
Male (%)	39.31	41.46	36.51	
Female (%)	60.69	58.54	63.49	
N (Pairs)	290 (145)	164 (82)	126 (63)	

Note. F-statistic presents the results of *F*-tests comparing each study measure across zygosity.
MZ = monozygotic; DZ = dizygotic.
†$p < 0.10$; *$p < 0.05$; **$p < 0.01$.

(i.e., at least a few cigarettes every day) at the time of the biomarker interview or whether they had smoked cigarettes regularly in the past (regardless of whether they do currently). The responses from these two items were used to create a categorical smoking status measure coded such that 0 = *never smoked*, 1 = *smoked previously but not currently*, and 2 = *smokes currently*. Third, respondents were asked how often they used alcohol in the past month, with responses coded categorically and ranging between 1 (*never*) and 6 (*everyday*). Fourth, a measure tapping weekly consumption of fast food was also included and reflected the average number times each week respondents ate food from a fast food restaurant with responses ranging between 1 (*never*) and 5 (*seven or more times each week*).

In addition, a number of covariates tapping socioeconomic status (SES) were also included in the estimated models. These covariates were selected to prevent confounding stemming from issues related to the social gradient in health identified in previous studies (Goldman, 2001). In order to assess financial hardship, a single item from the general MIDUS II interview asking respondents to indicate the overall difficulty in paying their monthly bills was included and coded categorically with responses ranging between 1 (*not difficult at all*) and 4 (*very difficult*). In addition, a measure of overall academic achievement from the general MIDUS II interview was also included with responses ranging between 1 (*did not complete high school*) and 5 (*competed graduate degree*). Finally, race (coded such that 0 = *Caucasian* and 1 = *all other races*); age, assessed during the general MIDUS II interview (measured continuously); and sex (0 = *female* and 1 = *male*) were also included in the employed statistical models.

2.3. Plan of analysis

The analyses for the current study were carried out in a series of interconnected steps. The first step of the analysis involved the estimation of the bivariate correlations between the perceived inequality and AL measures (along with the included covariates). Second, a series of traditional multivariate linear regression models were estimated in which AL was regressed on each of the perceived inequality measures and the included covariates; separate models were used to assess the potential associations involving the Wave I,

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006

Table 2
Descriptive statistics for biomarker and allostatic load measures.

	Mean	Range	High-Risk Cut Point
Cardiovascular			
Resting Heart Rate (bpm)	73.04	45.20–108.60	≥87.65
Systolic Blood Pressure (mmHg)	129.95	83.00–191.00	≥156.34
Pulse Pressure (mmHg)	55.42	29.00–107.00	≥75.86
Cardiovascular Subscale	0.23	0.00–1.00	
Glucose Metabolism			
Hemoglobin A1c %	5.94	4.60–11.91	≥7.41
Blood Glucose (mg/dL)	99.55	5.00–418.00	≥129.67
Insulin Resistance (HOMA-IR)	3.23	0.04–53.73	≥7.86
Glucose Metabolism Subscale	0.18	0.00–1.00	
Lipid Metabolism			
Body Mass Index	28.38	18.29–53.07	≥37.51
Waist to Hip Ratio	0.89	0.66–1.14	≥1.03
LDL Cholesterol (mg/dL)	107.86	23.00–219.00	≥154.62
HDL Cholesterol (mg/dL)	57.13	22.00–121.00	≤35.96
Triglycerides (mg/dL)	126.00	25.00–765.00	≥228.79
Lipid Metabolism Subscale	0.22	0.00–1.00	
Inflammation			
C-Reactive Protein (mg/L)	2.76	0.14–32.10	≥7.82
Interleuken 6 (pg/mL)	2.64	0.28–21.68	≥6.41
E-Selectin (ng/Ml)	42.40	7.20–124.50	≥73.10
Intracellular Adhesion Molecule-1 (ng/Ml)	276.63	116.12–774.72	≥422.30
Fibrinogen (mg/dL)	347.41	101.00–759.00	≥463.05
Inflammation Subscale	0.22	0.00–1.00	
Hypothalamic-Pituitary-Adrenal Axis			
Urine Cortisol (ug/g)	20.36	1.50–725.00	≥41.64
Dehydroepiandresterone Sulfate (ug/dL)	100.87	3.00–408.00	≤30.78
Hypothalamic-Pituitary-Adrenal Axis Subscale	0.27	0.00–1.00	
Sympathetic Nervous System			
Urine Epinephrine (ug/g)	2.06	0.09–9.19	≥3.66
Urine Norepinephrine (ug/g)	27.91	8.11–101.91	≥44.48
Sympathetic Nervous System Subscale	0.25	0.00–1.00	
Parasympathetic Nervous System			
Low-Frequency Heart Rate Variability (msec2)	5.46	0.82–8.25	≤3.95
High-Frequency Heart Rate Variability (msec2)	4.85	1.63–8.38	≤3.20
RR Interval Standard Deviation (msec)	3.47	1.80–5.00	≤2.85
RMS Successive Differences	2.89	1.47–4.95	≤2.13
Parasympathetic Nervous System Subscale	0.22	0.00–1.00	
Allostatic Load			
Total Scale	1.63	0.00–5.03	

Note. Heart rate variability measures were log-transformed due to positive skew. High-risk cut points were estimated as scores that fell within the top or bottom (depending on the direction of risk) quartile of each measure within the MIDUS Biomarker Project sample. Units of measurement listed where appropriate.

Wave II, and lifetime inequality measures. Importantly, this analytic technique provides an estimate of the association between the perceived inequality measures and AL net of the included covariates, but additional sources of confounding including genetic and other familial influences are left uncontrolled, potentially resulting in a biased estimate of the causal effect (Barnes et al., 2014).

The third step of the analysis was aimed at addressing this potential limitation by employing a twin comparison design; previous studies have argued this design effectively strengthens causal inference by minimizing the extent to which genetic and additional familial influences may confound observed associations (Lahey and D'Onofrio, 2010; Turkheimer and Harden, 2014). The use of twin pairs allows for a closer examination of situations in which twins from a given pair have differing perceptions of inequality, with the expectation that the twin with greater overall perceptions of inequality would also have greater overall levels of AL compared to their co-twin. This feature of the twin comparison design offers a distinct advantage over more traditional regression-based techniques, since comparing between twins from the same pair effectively controls for all additional latent sources of influence that work to make twins similar to one another including genetic and familial influences.

The twin comparison design represents a special case of fixed-effects regression and has been presented in more detail previously (Hamdi et al., 2016; Lahey and D'Onofrio, 2010; Turkheimer and Harden, 2014). Briefly, the twin comparison design provides estimates of both "between-family" and "within-family" effects. A positive and significant between-family effect would indicate that families that have greater overall perceptions of inequality also have greater overall levels of AL. The reported between-family effects are similar to coefficients estimated using more traditional regression based techniques and are subject to confounding stemming from uncontrolled genetic and familial influences. Alternatively, a significant and positive within-family effect would indicate that twins who experienced greater levels of perceived inequality also experienced greater overall levels of AL compared to their co-twin. In this way, within-family effects are adjusted for the included individual-level covariates, but are also adjusted for latent sources of influence that ultimately work to make twins from the same family more similar to one another. A more detailed summary of the twin comparison design is also provided in the online supplement. Since the final analytic sample is comprised of twins nested within families, all models (including the baseline multivariate regression models) were estimated using generalized estimating equations (GEE) with robust standard errors.

Overall levels of missingness across the study variables were extremely low (between 1% and 8% of all cases, depending on the

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006

model estimated). To retain as many cases as possible, multiple imputation using chained equations was used (using the *mi* suite of commands in Stata 14.2 (StataCorp, 2015)) to generate 20 multiply imputed datasets (Graham et al., 2007). All analytic models were estimated using the *mi estimate* prefix in Stata, which combines estimates across all imputed datasets in accordance with Rubin's rules (Rubin, 1987).

3. Results

The plan of analysis began with the estimation of bivariate correlations between all study variables. The resulting correlation matrix is presented in the online supplement. The second step of the analysis involved the estimation of a series of baseline multivariate models aimed at assessing the potential association between the examined perceived inequality measures and AL (net of all included covariates). The first set of columns in Table 3 present the results from a model examining the association between perceived inequality at Wave I and AL. Although the results revealed that the association was positive, indicating that individuals who reported greater overall levels of inequality also experienced greater overall levels of AL, the association was small in magnitude and nonsignificant ($b = 0.08$, $p = 0.119$). The next two columns of the table present the results of a fixed-effects model examining the association between levels of inequality reported at Wave II and AL. The results indicated a positive and significant association ($b = 0.14$, $p = 0.005$), indicating that a one standard deviation increase in perceived inequality resulted in an increase in 0.14 standard deviation units in AL. The final two columns present the results of a fixed-effects equation examining the association between the lifetime perceived inequality measure and AL. The results indicated that individuals that reported overall greater levels of perceived inequality across Wave I and II also experienced greater overall levels of AL ($b = 0.14$, $p = 0.007$), even after controlling for the included covariates.

The results so far provide preliminary evidence suggesting that individuals who experience greater levels of inequality also experience greater overall levels of AL. In an effort to better estimate the causal effect of perceived inequality on AL, a series of twin comparison models were estimated, with the results presented in Table 4. Since the employed twin comparison approach estimates both *within* and *between* families, it was first necessary to estimate the extent to which the primary independent (perceived inequality) and dependent variables (AL) varied within and between families. To estimate the variability of the perceived inequality and AL measures a series of unconditional mixed models were estimated which allowed for the calculation of intraclass correlation (ICC) coefficients. In this context, the ICC can be interpreted as the proportion of overall variance in the examined measure explained by differences in examined families, with the residual variance explained by within-family differences. The resulting ICC coefficient for the AL measure was 0.47, indicating that approximately 47% of the overall variance in AL was explained by differences between families and the remaining 53% of the variance was explained by differences within families. The ICC coefficients for the perceived inequality measures were as follows: Wave I = 0.09; Wave II = 0.14; lifetime measure = 0.21. Collectively, these results indicate that the majority of the variance in the examined AL and perceived inequality measures is explained by within-family differences, effectively enabling the use of a twin comparison approach.

Table 3
Results of multivariate regression models examining the association between perceived inequality and allostatic load.

Study Measure	Wave 1 Inequality		Wave 2 Inequality		Lifetime Inequality	
	b	*SE*	*b*	*SE*	*b*	*SE*
Perceived Inequality	0.08	0.05	0.14**	0.05	0.14**	0.05
Weekly Exercise	-0.22†	0.12	-0.23†	0.12	-0.24*	0.12
Smoking Status	0.07	0.08	0.06	0.08	0.07	0.08
Alcohol Use Frequency	-0.08*	0.03	-0.08*	0.03	-0.08*	0.03
Current Fast Food Consumption	0.14*	0.06	0.13*	0.06	0.13*	0.06
Difficulty in Paying Bills	0.01	0.06	0.00	0.06	-0.01	0.06
Educational Achievement	-0.08	0.05	-0.06	0.05	-0.07	0.05
Age	0.04**	0.01	0.04**	0.01	0.04*	0.01
Male (female = 0)	-0.15	0.11	-0.15	0.11	-0.16	0.11
Race	0.02	0.10	0.00	0.10	0.01	0.10
N (Pairs)	290 (145)		290 (145)		290 (145)	

Note. Unstandardized regression coefficients (and accompanying standard errors) presented. General estimating equations (GEE) with robust standard errors were estimated to properly account for the nested nature of the data.
$^{\dagger}p < 0.10$; $^{*}p < 0.05$; $^{**}p < 0.01$.

Table 4
Results of sibling-comparison models examining the association between perceived inequality and allostatic load.

Study Measure	Wave 1 Inequality		Wave 2 Inequality		Lifetime Inequality	
	b	*SE*	*b*	*SE*	*b*	*SE*
Perceived Inequality						
Between-Family Effect	0.12	0.08	0.21**	0.08	0.18*	0.07
Within-Family Effect	0.10	0.09	0.19*	0.09	0.17†	0.09
Covariates						
Weekly Exercise	-0.19	0.12	-0.22†	0.12	-0.21†	0.12
Smoking Status	0.08	0.08	0.06	0.08	0.07	0.08
Alcohol Use Frequency	-0.08*	0.03	-0.08*	0.03	-0.08*	0.03
Current Fast Food Consumption	0.14*	0.06	0.14*	0.06	0.14*	0.06
Difficulty in Paying Bills	0.00	0.06	-0.01	0.06	-0.01	0.06
Educational Achievement	-0.08	0.05	-0.05	0.05	-0.06	0.05
Age	0.04**	0.01	0.04**	0.01	0.05**	0.01
Male (female = 0)	-0.15	0.11	-0.15	0.11	-0.15	0.11
Race	0.01	0.10	0.00	0.10	0.00	0.10
N (pairs)	290 (145)		290 (145)		290 (145)	

Note. Unstandardized regression coefficients (and accompanying standard errors) presented. General estimating equations (GEE) with robust standard errors were estimated to properly adjust for the nested nature of the data. Between-family effects provide an estimation of the association between perceived inequality and allostatic load across families. Within-family effects reflect differences between siblings from the same pair in examining the same association.
$^{\dagger}p < 0.10$; $^{*}p < 0.05$; $^{**}p < 0.01$.

The first two columns of Table 4 display the results of the twin comparison model examining the association between the Wave I perceived inequality measure and AL. The estimated between-family effect estimates the extent to which families with greater overall levels of perceived inequality also experience greater overall levels of AL. As can be seen in the table, the between-family effects for Wave I inequality were nonsignificant ($b = 0.12$, $p = 0.146$), indicating that while families that reported greater overall levels of inequality also had overall greater scores on the employed AL measure, the association was nonsignificant. Recall that between-family effects are similar to coefficients estimated from traditional regression-based techniques and do not include controls for genetic and environmental influences that cluster within families. The within-family effect for the Wave I inequality measure was also nonsignificant, which indicates that in situations in which twins from the same household experienced different levels of perceived inequality, the twin that reported greater levels of inequality had greater, but not significantly different, levels of AL ($b = 0.10$, $p = 0.220$).

The second set of columns in Table 4 present the results of a twin comparison model examining the association between perceived inequality reported at Wave II and AL. The between-family effects

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006

indicated that families with greater overall levels of perceived inequality also experienced greater overall levels of AL, even after including controls for various lifestyle and demographic factors ($b = 0.21$, $p < 0.009$). While the magnitude of the within-family effect was slightly attenuated ($b = 0.19$, $p = 0.027$), the examined association remained significant. Thus, even after controlling for latent sources of genetic and environmental influences (along with the observed covariates), twins who experienced greater overall levels of inequality also had greater overall levels of AL compared to their co-twin. The final two columns present the results of the twin comparison model examining the association between the lifetime perceived inequality measure and AL. Once again, the results revealed a significant between-family effect ($b = 0.18$, $p = 0.017$), but the more conservative within-family effect failed to meet the conventional level of statistical significance ($b = 0.17$, $p = 0.055$).

3.1. Sensitivity analyses

In an effort to examine the robustness of the findings from the primary analyses, a series of sensitivity analyses were also performed. The results of the sensitivity analyses appear in more detail in the online supplement. First, although the primary objective of the current study was to examine the association between perceived inequality and AL, supplemental twin comparison models were also estimated to examine the potential association between perceived inequality and each of the system risk scores used to create the overall AL measure. The overall findings failed to reveal any consistent pattern of significant associations. Second, the twin comparison models estimated in the primary analysis were estimated a second time with an alternative measurement strategy for the perceived inequality measures. Rather than focus on the frequency of perceived inequality (as in the primary analyses), the individual items used to create the perceived inequality measures were dichotomized such that 0 = *never or rarely* and 1 = *sometimes or often*. Responses to each item were summed to create a variety index, with higher scores indicating a greater variety of experiences. The results of twin comparison models that employed these alternative perceived inequality measures did not differ from the results of the primary analyses in any substantive way.

4. Discussion

Chronic health problems impact the lives of a sizable portion of U.S. residents (Centers for Disease Control, 2016). In 2012, nearly half of all U.S. adults suffered from at least one chronic health condition; during 2014 (the most recent year for which data were available), eight of the top ten leading causes of death were chronic diseases (Heron, 2016). Based on the alarming prevalence of chronic health conditions coupled with the extremely high monetary cost of addressing such issues ($3.2 trillion in 2015; Centers for Medicare and Medicaid Services, 2016), it comes as little surprise that a significant portion of the extant literature has been devoted to identifying factors that contribute to chronic morbidity and mortality. Inequality represents one source of environmental influence that has been previously linked to physical health problems, with previous research reporting a significant and positive association between greater levels of perceived inequality and AL (Brody et al., 2014; Geronimus et al., 2006; Green and Darity, 2010; Gruenewald et al., 2012). Despite this pattern of results, the existing literature has relied on measures of AL that are relatively limited and has failed to fully consider unobserved sources of selection that may confound the observed associations (Brody et al., 2014; Cunningham et al., 2012; Fuller-Rowell et al., 2012; Geronimus et al., 2006; Sawyer et al., 2012). The current study addressed these limitations by employing a more comprehensive measure of AL and making use of twin comparison models. The findings produced three key findings, all of which warrant additional explanation.

First, the results of the baseline multivariate regression models revealed significant associations between perceived inequality and AL, wherein individuals who reported greater levels of inequality at Wave II and across Waves I and II also had significantly higher scores on the AL measure. This finding falls directly in line with previous studies examining associations between perceived inequality and dysfunction within specific physiological systems (Cunningham et al., 2012; Fuller-Rowell et al., 2012; Sawyer et al., 2012) more restrictive measures of AL (Brody et al., 2014; Geronimus et al., 2006), and the results of a recent meta-analysis examining the association between perceived discrimination and overall health (Pascoe and Smart Richman, 2009). However, the baseline models did not include controls for unobserved or unmeasured sources of selection, rendering these findings preliminary and making it difficult to interpret the examined associations as causal.

Second, despite these significant associations, the bivariate correlations and baseline multivariate regression models failed to reveal a significant association between the Wave I perceived inequality measure and AL. This finding was somewhat unexpected, particularly in light of the significant associations involving the Wave II and lifetime inequality measures. While only speculation, this finding may be the result of the timing of the measurement of the inequality measures. More specifically, items used to create the Wave I and II measures ask respondents to report the amount of inequality they experience in their day to day lives, making it possible for experiences perceived as unequal treatment to accumulate during the time that elapsed between each wave. Another possibility pertains to the age of the MIDUS sample during each wave of data collection ($M = 53.70$ at Wave I and $M = 57.06$ at Wave II). Previous studies have reported findings indicating that as individuals age, they potentially become more susceptible to deleterious environments resulting in more pronounced changes in AL (Crimmins et al., 2003). Despite this possibility, supplementary analyses revealed that age did not significantly moderate any of the examined associations between perceived inequality and AL. Based on these findings, the potential longitudinal association between perceptions of inequality and AL appears to be unresolved and warrants additional attention.

The third finding to emerge from the current study was directly related to the results of the twin comparison models. As expected, the between-family effects, which are not adjusted for unobserved sources of selection, closely resembled the findings from the baseline multivariate regression models. The more unique set of findings flowing from the sibling comparison models are the within-family effects, which are adjusted for unobserved sources of genetic and familial confounding (Lahey and D'Onofrio, 2010; Turkheimer and Harden, 2014). The within-family effects indicated that among twins who were discordant on the Wave II perceived inequality measure, the twin that experienced greater levels of inequality also had greater overall levels of AL relative to their co-twin, providing strong evidence indicating that this association is robust. A similar pattern of findings was observed for the lifetime perceived inequality measure, but the resulting within-family effect did not reach conventional levels of statistical significance ($b = 0.17$, $p = 0.055$). This pattern of findings directly aligns with the results of a recent meta-analysis, indicating that more recent forms of discrimination resulted in slightly greater levels of health problems relative to lifetime discrimination (Pascoe and Smart Richman, 2009). These findings also demonstrate the importance of a further examination of the longitudinal association between perceived inequality and AL after accounting for within-family influences in future research, as such findings may further

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006

elucidate the underlying mechanisms that connect inequality and negative health outcomes.

4.1. Limitations

While the results of the current study illustrate the contributions of discriminatory treatment to the development of physical health problems, these findings should be interpreted with caution due to several limitations. First, the final sample size was limited, potentially resulting in restricted levels of statistical power and variability. While previous studies employing the Biomarker twin sample from the MIDUS have reported acceptable levels of power for the estimation of twin comparison models (Hamdi et al., 2016), future research would benefit from replicating these findings within a larger sample of twins or siblings. Second, the demographic composition of the MIDUS Biomarker twin subsample is limited, consisting of primarily older (M_{age} = 57.06), Caucasian (96.21%), female (60.69%) participants, potentially limiting the generalizability of the findings. The limited variation in the racial composition of the analytic sample is particularly problematic as the weathering hypothesis has directly implicated racially-motivated discrimination as a factor contributing to negative health outcomes (Geronimus et al., 2006, 2010). In addition, and directly related to these findings, it is expected that racially-motivated discriminatory treatment may effectively moderate the associations observed in the current study. In this way, future research would benefit from examining whether these findings extend to more heterogeneous samples, particularly samples comprised of a larger number of racial minoritites.

Third, and as indicated above, the current study was not able to fully explore the longitudinal association between perceived inequality and AL with the MIDUS Biomarker twin sample. The current analysis was limited to the use of two waves of data, making the estimation of more sophisticated longitudinal models (which typically require three waves of data; Nagin, 2005; Singer and Willett, 2003) impossible. While a third wave of the MIDUS data was recently made available (MIDUS III), the research protocol did not include the collection of biomarkers, preventing the measurement of AL. Finally, the current study was unable to make a more direct connection between specific physical health problems (or *tertiary outcomes*; e.g., cardiovascular disease, diabetes, cancer, etc.) and perceived inequality. While previous research has indicated that AL is an important determinate of physical and mental health problems (Juster et al., 2010; Mattei et al., 2010; Seeman et al., 2001), AL is expected to mediate the association between perceived inequality and physical health problems. The MIDUS contains a significant amount of information pertaining to overall physical health, but the data are not well-suited for examining this indirect pathway (i.e., from perceived inequality to AL to specific physical health problems). Temporal order cannot be effectively established since the biomarkers used to measure AL were contemporaneous and any questions regarding morbidity were retrospective. Future research would directly benefit from examining this potential indirect pathway more directly and with more detail.

5. Conclusion

Despite the limitations noted, the current study provides strong evidence indicating that increased perceptions of inequality can result in increased levels of AL. These findings, if replicated, have potentially important implications for the detection of future physical health problems and for intervention (or prevention) programming aimed at limiting such problems. While these findings do not provide unequivocal evidence of the link between inequality and AL, the employed methodological approach is rigorous and addresses many of the limitations of more traditional approaches. In this way, the current study not only demonstrates a potential pathway in which environmental stressors may have long-term physical health consequences, but also demonstrates the importance of employing research methodologies that take indirect sources of selection into account.

Author notes

I thank Kevin Beaver and two anonymous reviewers for their suggestions and insightful comments on previous drafts of this paper. The content of the article is my sole responsibility and any errors or omissions are mine alone. The MIDUS 1 study (Midlife in the U.S.) was supported by the John D. and Catherine T. MacArthur Foundation Research Network on Successful Midlife Development. The MIDUS 2 research was supported by a grant from the National Institute on Aging (P01-AG020166) to conduct a longitudinal follow-up of the MIDUS 1 investigation. The research was further supported by the following grants M01-RR023942 (Georgetown), M01-RR00865 (UCLA) from the General Clinical Research Centers Program and UL1TR000427 (UW) from the National Center for Advancing Translational Sciences (NCATS), National Institutes of Health.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2017.06.006.

References

Barnes, J.C., Boutwell, B.B., Beaver, K.M., Gibson, C.L., Wright, J.P., 2014. On the consequences of ignoring genetic influences in criminological research. J. Crim. Just. 42, 471–482.

Brim, O.G., Baltes, P.B., Bumpass, L.L., Cleary, P.D., Featherman, D.L., Hazzard, W.R., Kessler, R.C., Lachman, M.E., Markus, H.R., Marmot, M.G., Rossi, A.S., Ryff, C.D., Shweder, R.A., 1996. National Survey of Midlife Development in the United States (MIDUS), 1995–1996, 8th ICPSR version. Inter-university Consortium for Political and Social Research (distributor), Ann Arbor, MI.

Brim, O.G., Ryff, C.D., Kessler, R.C., 2004. The MIDUS national survey: an overview. In: Brim, O.G., Ryff, C.D., Kessler, R.C. (Eds.), How Healthy Are We? a National Study of Well-Being at Midlife. University of Chicago Press, Chicago, IL, pp. 1–34.

Brody, G.H., Lei, M.K., Chae, D.H., Yu, T., Kogan, S.M., Beach, S.R., 2014. Perceived discrimination among African American adolescents and allostatic load: a longitudinal analysis with buffering effects. Child. Dev. 85, 989–1002.

Brooks, K.P., Gruenewald, T., Karlamangla, A., Hu, P., Koretz, B., Seeman, T.E., 2014. Social relationships and allostatic load in the MIDUS study. Health Psychol. 33, 1373.

Brunner, E., 1997. Stress and the biology of inequality. Brit. Med. J. 314, 1472–1476.

Centers for Disease Control, 2016. Chronic disease overview. Retrieved from. https://www.cdc.gov/chronicdisease/overview/.

Centers for Medicare & Medicaid Services, 2016. National health expenditure 2015 highlights. Retrieved from. https://www.cms.gov/Research-Statistics-Data-and-Systems/Statistics-Trends-and-Reports/NationalHealthExpendData/Downloads/highlights.pdf.

Crimmins, E.M., Johnston, M., Hayward, M., Seeman, T., 2003. Age differences in allostatic load: an index of physiological dysregulation. Exp. Gerontol. 38, 731–734.

Cunningham, T.J., Seeman, T.E., Kawachi, I., Gortmaker, S.L., Jacobs, D.R., Kiefe, C.I., Berkman, L.F., 2012. Racial/ethnic and gender differences in the association between self-reported experiences of racial/ethnic discrimination and inflammation in the CARDIA cohort of 4 US communities. Soc. Sci. Med. 75, 922–931.

Delhey, J., Dragolov, G., 2014. Why inequality makes Europeans less happy: the role of distrust, status anxiety, and perceived conflict. Eur. Sociol. Rev. 30, 151–165.

Diez-Roux, A.V., Link, B.G., Northridge, M.E., 2000. A multilevel analysis of income inequality and cardiovascular disease risk factors. Soc. Sci. Med. 50, 673–687.

Dowd, J.B., Simanek, A.M., Aiello, A.E., 2009. Socio-economic status, cortisol and allostatic load: a review of the literature. Int. J. Epidemiol. 38, 1297–1309.

Friedman, E.M., Karlamangla, A.S., Gruenewald, T.L., Koretz, B., Seeman, T.E., 2015. Early life adversity and adult biological risk profiles. Psychosom. Med. 77, 176–185.

Fuller-Rowell, T.E., Doan, S.N., Eccles, J.S., 2012. Differential effects of perceived discrimination on the diurnal cortisol rhythm of African Americans and whites.

Psychoneuroendocrino 37, 107–118.
Geronimus, A.T., 1991. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. Ethn. Dis. 2, 207–221.
Geronimus, A.T., Hicken, M., Keene, D., Bound, J., 2006. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. Am. J. Public Health 96, 826–833.
Geronimus, A.T., Hicken, M.T., Pearson, J.A., Seashols, S.J., Brown, K.L., Cruz, T.D., 2010. Do US black women experience stress-related accelerated biological aging? Hum. Nat. 21, 19–38.
Ghumman, S., Ryan, A.M., Barclay, L.A., Markel, K.S., 2013. Religious discrimination in the workplace: a review and examination of current and future trends. J. Bus. Psychol. 28, 439–454.
Goldman, N., 2001. Social inequalities in health: disentangling the underlying mechanisms. Ann. NY. Acad. Sci. 954, 118–139.
Gordon-Larsen, P., Nelson, M.C., Page, P., Popkin, B.M., 2006. Inequality in the built environment underlies key health disparities in physical activity and obesity. Pediatrics 117, 417–424.
Graham, J.W., Olchowski, A.E., Gilreath, T.D., 2007. How many imputations are really needed? Some practical clarifications of multiple imputation theory. Prev. Sci. 8, 206–213.
Green, T.L., Darity, W.A., 2010. Under the skin: using theories from biology and the social sciences to explore the mechanisms behind the black-white health gap. Am. J. Public Health 100, S36–S40.
Gruenewald, T.L., Karlamangla, A.S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., Seeman, T.E., 2012. History of socioeconomic disadvantage and allostatic load in later life. Soc. Sci. Med. 74, 75–83.
Hamdi, N.R., South, S.C., Krueger, R.F., 2016. Does education lower allostatic load? A co-twin control study. Brain Behav. Immun. 56, 221–229.
Heron, M., 2016. Deaths: leading causes for 2014. Natl. Vital Stat. Rep. 65, 1–96.
Jackson, J.S., Knight, K.M., Rafferty, J.A., 2010. Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course. Am. J. Public Health 100, 933–939.
Juster, R.P., McEwen, B.S., Lupien, S.J., 2010. Allostatic load biomarkers of chronic stress and impact on health and cognition. Neurosci. Biobehav. R. 35, 2–16.
Karlamangla, A.S., Miller-Martinez, D., Lachman, M.E., Tun, P.A., Koretz, B.K., Seeman, T.E., 2014. Biological correlates of adult cognition: midlife in the United States (MIDUS). Neurobio. Aging 35, 387–394.
Kessler, R.C., Mickelson, K.D., Williams, D.R., 1999. The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. J. Health Soc. Behav. 40, 208–230.
Kumari, M., Head, J., Marmot, M., 2004. Prospective study of social and other risk factors for incidence of type 2 diabetes in the Whitehall II study. Arch. Intern. Med. 164, 1873–1880.
Lahey, B.B., D'Onofrio, B.M., 2010. All in the family: comparing siblings to test causal hypotheses regarding environmental influences on behavior. Curr. Dir. Psychol. Sci. 19, 319–323.
Love, G.D., Seeman, T.E., Weinstein, M., Ryff, C.D., 2010. Bioindicators in the MIDUS national study: protocol, measures, sample, and comparative context. J. Aging Health 22, 1059–1080.
Mattei, J., Demissie, S., Falcon, L.M., Ordovas, J.M., Tucker, K., 2010. Allostatic load is associated with chronic conditions in the boston puerto rican health study. Soc. Sci. Med. 70, 1988–1996.
McEwen, B.S., 1998. Stress, adaptation, and disease: allostasis and allostatic load. Ann. N. Y. Acad. Sci. 840, 33–44.
McEwen, B.S., Seeman, T., 1999. Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. Ann. N. Y. Acad. Sci. 896, 30–47.
Meyer, I.H., 2003. Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: conceptual issues and research evidence. Psychol. Bull. 129, 674–697.
Nagin, D.S., 2005. Group-based Modeling of Development. Harvard University Press, Cambridge, MA.
Obama, B., 2016. United States health care reform: progress to date and next steps. Amer. Med. Assoc. 316, 525–532.
Oliver, D., 2009. Age based discrimination in health and social care services. Brit. Med. J. 339, 643–644.
Pascoe, E.A., Smart Richman, L., 2009. Perceived discrimination and health: a meta-analytic review. Psychol. Bull. 135, 531–554.
Puhl, R.M., Andreyeva, T., Brownell, K.D., 2008. Perceptions of weight discrimination: prevalence and comparison to race and gender discrimination in America. Int. J. Obes. 32, 992–1000.
Radler, B.T., Ryff, C.D., 2010. Who participates? Accounting for longitudinal retention in the MIDUS national study of health and well-being. J. Aging Health 22, 307–331.
Rubin, D.B., 1987. Multiple Imputation for Nonresponse in Surveys. Wiley, New York, NY.
Ryff, C.D., Keyes, C.L.M., Hughes, D.L., 2004. Psychological well-being in MIDUS: profiles of ethnic/racial diversity and life-course uniformity. In: Brim, O.G., Ryff, C.D., Kessler, R.C. (Eds.), How Healthy Are We? a National Study of Well-being at Midlife. University of Chicago Press, Chicago, IL, pp. 398–422.
Sanders-Phillips, K., Settles-Reaves, B., Walker, D., Brownlow, J., 2009. Social inequality and racial discrimination: risk factors for health disparities in children of color. Pediatrics 124, S176–S186.
Sawyer, P.J., Major, B., Casad, B.J., Townsend, S.S., Mendes, W.B., 2012. Discrimination and the stress response: psychological and physiological consequences of anticipating prejudice in interethnic interactions. Am. J. Public Health 102, 1020–1026.
Schultz, A.B., Chen, C.Y., Edington, D.W., 2009. The cost and impact of health conditions on presenteeism to employers. Pharmacoeconomics 27, 365–378.
Seeman, T.E., McEwen, B.S., Rowe, J.W., Singer, B.H., 2001. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. P. Natl. Acad. Sci. U. S. A. 98, 4770–4775.
Singer, J.D., Willett, J.B., 2003. Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence. Oxford University Press, New York, NY.
StataCorp, 2015. Stata Statistical Software: Release 14. StataCorp LP, College Station, TX.
Sterling, P., Eyer, J., 1988. Allostasis: a new paradigm to explain arousal pathology. In: Fisher, S., Reason, J. (Eds.), Handbook of Life Stress, Cognition and Health. John Wiley & Sons, New York, NY, pp. 629–649.
Turkheimer, E., Harden, K.P., 2014. Behavior genetic research methods: testing quasi-causal hypotheses using multivariate twin data. In: Reis, H.T., Judd, C.M. (Eds.), Handbook of Research Methods in Social and Personality Psychology. Cambridge University Press, Cambridge, MA, pp. 159–187.
Turner, R.J., Avison, W.R., 2003. Status variations in stress exposure: implications for the interpretation of research on race, socioeconomic status, and gender. J.Health Soc. Behav. 44, 488–505.
Williams, D.R., Yu, Y., Jackson, J.S., Anderson, N.B., 1997. Racial differences in physical and mental health: socioeconomic status, stress and discrimination. J. Health Psychol. 2, 335–351.

Please cite this article in press as: Schwartz, J.A., Long-term physical health consequences of perceived inequality: Results from a twin comparison design, Social Science & Medicine (2017), http://dx.doi.org/10.1016/j.socscimed.2017.06.006