

UCLA

UCLA Previously Published Works

Title

Chronic Physiologic Effects of Stress Among Lesbian, Gay, and Bisexual Adults

Permalink

<https://escholarship.org/uc/item/8xj6j6vg>

Journal

Psychosomatic Medicine, 80(6)

ISSN

0033-3174

Authors

Mays, Vickie M

Juster, Robert-Paul

Williamson, Timothy J

et al.

Publication Date

2018-07-01

DOI

10.1097/psy.0000000000000600

Peer reviewed



HHS Public Access

Author manuscript

Psychosom Med. Author manuscript; available in PMC 2019 July 01.

Published in final edited form as:

Psychosom Med. 2018 ; 80(6): 551–563. doi:10.1097/PSY.0000000000000600.

Chronic physiologic effects of stress among lesbian, gay, and bisexual adults: Results from the National Health and Nutrition Examination Survey

Vickie M. Mays, PhD, MSPH,

Department of Psychology and Department of Health Policy and Management, Fielding School of Public Health University of California, Los Angeles

Robert-Paul Juster, PhD,

Columbia University, New York State Psychiatric Institute, New York, NY

Timothy J. Williamson, MPH, M.A.,

University of California, Los Angeles

Teresa E. Seeman, PhD, and

UCLA School of Medicine and Fielding School of Public Health, University of California, Los Angeles

Susan D. Cochran, PhD, MS

Department of Epidemiology, Fielding School of Public Health and Department of Statistics, University of California, Los Angeles

Abstract

Objectives: Social disadvantage is associated with markers of physiological dysregulation, which is linked to disease trajectories. Chronic experiences with discrimination are thought to result in the accumulation of physiological “wear and tear” known as allostatic load among socially marginalized populations such as sexual minorities. Using a nationally-representative United States sample, we examined whether: (1) people who self-identified as homosexual or bisexual display higher levels of AL than heterosexual individuals and (2) subgroups of sexual identity would further differ from each other as a consequence of distinct experiences of marginalization.”

Methods: We use data from the 2001–2010 National Health and Nutrition Examination Survey. Employing multivariate regression methods with sex-specific analyses, we examined AL score differences among lesbian/gay ($n = 211$), bisexual ($n = 307$), homosexually experienced ($n = 424$), and exclusively heterosexual ($n = 12,969$) individuals, adjusting for possible confounding due to demographics, health indicators, and, among men, HIV infection status.

Correspondence concerning this article should be addressed to: Vickie M. Mays, Ph.D., MSPH 405 Hilgard Avenue, 1285 Franz Hall, Box 951563, Los Angeles, CA 90095-1563, 310-206-5159, mays@ucla.edu.

Conflicts of Interest

The authors have no conflicts of interest to report.

Results: Results indicate that elevated AL was more common in bisexual men compared to exclusively heterosexual men (adjusted $\beta = 0.25$, 95% CI = 0.05, 0.44), with significantly higher levels of glycosylated hemoglobin A1c (adjusted OR = 3.51, 95% CI = 1.46, 7.92) and systolic blood pressure (adjusted OR = 2.07, 95% CI = 1.02, 4.18). Gay-identified men evidenced significantly lower AL (adjusted $\beta = -0.22$, 95% CI = -0.41 , -0.04). No significant differences in AL were observed among women.

Conclusions: These findings indicate that physiological dysregulation is more common in bisexual males compared to all other men. The results are discussed with regard to differences in health outcomes between individuals with different sexual orientations.

Keywords

sexual orientation; sexual minority stress; allostatic load; National Health and Nutrition Examination Study; bisexuality

INTRODUCTION

A growing body of research documents that sexual minorities experience stress associated with stigma, prejudice, and discrimination that predispose them to negative physical and mental health outcomes (1–7). To date, however, the consequences of these experiences on the health of lesbian, gay, and bisexual (LGB) individuals outside of HIV research have seldom been studied using biological approaches commonly employed in biobehavioral studies. In order to better understand how physiological indicators of chronic stress operate by sexual minority status differences among LGB subgroups, the current study aims to assess how sexual orientation status relates to allostatic load.

Allostatic load (AL) refers to the multi-systemic ‘wear and tear’ that chronic stress exacts on the brain and body (8). AL is often used to measure this physiological “wear and tear” from the body’s efforts to maintain its internal response to stress throughout life (9, 10). Seeman and her colleagues (11) through a series of pioneering studies demonstrated that over time the strain of trying to maintain homeostasis in the face of chronic stress can result in the dysregulation of several physiological parameters. In particular these include inflammatory, cardiovascular, endocrine, metabolic, and autonomic systems (12).

Theoretically under cumulative strain, the biphasic effects of numerous biomarkers lead to AL and disease as follows: (i) over-activation of *primary mediators* such as stress hormones (e.g., cortisol) and pro- and anti-inflammatory cytokines (e.g., interleukin-6) induce *primary effects* on cellular activities (13); (ii) leading to *secondary outcomes*, whereby metabolic, cardiovascular, and second-order immune biomarkers become dysregulated; and (iii) culminate as *tertiary outcomes* or clinical endpoints (14). The MacArthur Studies on Successful Aging first indexed AL using 10 neuroendocrine, immune, metabolic, and cardiovascular biomarkers that were predictive of increased physical/cognitive declines and incident cardiovascular disease (11). After nearly two decades of research, AL algorithms have been robustly related to numerous social antecedents in dozens of studies worldwide (for reviews, see (15, 16)). In particular, social disadvantage is associated with elevated AL which is linked to various physical and mental disease trajectories (17).

Epidemiological evidence that AL is an effective tool to monitor population level chronic stress and health associations has come from the National Health and Nutrition Examination Survey (NHANES). Over the lifespan, Americans living in poverty manifest the sharpest increases in AL up until middle and older age, when AL levels plateau (18). This plateau is due, in part, to selective mortality among the most socially disadvantaged. Furthermore, life expectancy is six years shorter for those with the most elevated AL levels, as compared to those who evidence lower AL levels (19). Understanding how social inequalities in populations relate to AL provides insights into the pathways whereby the social determinants of health lead to physiological dysregulation and subsequent clinical endpoints (20)

One rationale for the current study on sexual minority statuses (e.g., LGB) is supported by findings of the relationship between experiences of discrimination and stress in racial/ethnic minorities and negative health related physiological outcomes. Studies support that AL is elevated among racial/ethnic minorities often with African Americans experiencing some of the highest odds of adverse health outcomes (21). In a multi-race/ethnic NHANES analysis, Black men and women evidenced higher AL levels than White individuals, which in turn contributed to an overall greater risk for cardiovascular- and diabetes-related mortality (22). These findings and others demonstrate that social inequalities experienced by racial/ethnic minorities contribute to cumulative stress that can be captured with AL algorithms (23–25). Similar to racial/ethnic minorities in which high levels of discrimination and hostility significantly predicted higher levels of AL (23), sexual minorities are expected here to experience chronic stress based on prejudice, stigma, and discrimination.

Social inequalities are related to AL because they represent cumulative adversities that strain the body and mind over time (20). Cumulative disadvantage theory describes the systemic tendency for inter-individual divergence in a given characteristic – such as social or health status – to be experienced in a socially unjust way over time (26). For instance, race/ethnic inequalities are linked to cumulative disadvantage that is associated with an accumulation of negative health outcomes throughout life (12, 27). Depending on the socio-cultural contexts, these adverse outcomes may also manifest themselves at specific ages among vulnerable populations (28). This is consistent with the “weathering hypothesis” (29) that states that Black women’s health deteriorates earlier in adulthood as the physical consequence of cumulative social disadvantage. Using the NHANES to confirm weathering health inequality, Black women were indeed shown to have the most consistently elevated AL across age groups (9). The experiences of minority status represent a cumulative strain that shapes stress sensitivities, which can exacerbate AL further and promote disease.

Consistent with literature on cumulative disadvantage as a social determinant of health, the LGB health literature has been framed according to minority stress theory that is only beginning to be assessed using stress biomarkers as indicators of cumulative strain. Sexual minority stress models (5, 30, 31) propose that the stress experienced by LGB individuals comes from two sources of stigma over and above general life stressors experienced by everybody (32). First at the individual level, *proximal minority stress processes* refers to internalized homophobia and concealment of one’s sexual orientation or gender identity for transgender individuals (33). Second at the social level, *distal minority stress processes*

refers to stressors like discrimination and violence that disproportionately affect LGB individuals.

Two recent studies exemplify how both distal and proximal stress processes influence physiological outcomes in LGB samples. First, Doyle and Molix showed that discrimination predicts elevated interleukin-6 levels in gay men; however, this relationship was present only among gay men who engaged in less covering, a strategy that involves downplaying one's stigmatized identity (34). Second, Parra and colleagues showed that LGB-related stressful life events, internalized homonegativity, and flatter diurnal cortisol slopes were positively associated with depressive symptoms (35). Apart from these studies, it is unknown to what extent distal (e.g., macro-level stigma) and proximal (e.g., micro-level distress) sexual minority stress processes affect multisystemic biomarker profiles among LGB subgroups.

Emerging research shows that AL may differ by sexual orientation. In a convenience sample of 87 Canadians, Juster and colleagues (36) first showed that sexual minorities do not manifest heightened stress pathophysiology when compared to heterosexuals. Quite to the contrary, sexual minority men had lower AL levels than heterosexual men, but no such differences were found among women (36). Lower AL among the sexual minority men was driven by lower values of triglycerides, BMI, and tumor necrosis factor- α in comparison to heterosexual men. Interestingly, LGB participants who had fully disclosed their sexual orientation to family and friends showed significantly lower symptoms of depression, anxiety, and burnout as well as lower concentrations of the stress hormone cortisol thirty minutes post-awakening compared to LGB individuals who had not completely disclosed (36). A separate analysis of only LGB participants revealed that those who engaged in avoidance coping strategies during their sexual identity formation and disclosure processes evidenced elevated AL, while those who sought social support experienced less perceived stress (37).

Stigma-related stress can promote adaptive behavioral responses among stigmatized individuals that successfully appropriate their identities, which may in fact render some more resilient (38). Despite this possibility for gay men, a key limitation in Juster and co-authors' 2013 study was that bisexual men were underrepresented and were therefore collapsed in analyses with gay men. Likewise, lesbian and bisexual women were combined due to restricted power that may have compromised the ability to detect AL differences among women. While this analytic approach is common in small studies, it is important to investigate potential differences between LGB subgroups. In particular, there is evidence that bisexual individuals experience the greatest health disparities (1).

Bisexuality is a minority within the sexual minority population. It is possible that bisexual individuals experience alienation and stigmatization from both heterosexual and homosexual communities (39). Consistent with this hypothesis, research has shown that bisexual men and women report significantly lower levels of connection to their community than their lesbian and gay peers (40). Similarly, "homosexually experienced heterosexual" individuals (41) are those who fall between heterosexual and bisexual individuals on spectrum of sexual orientation, attractions, and behaviors. This represents another understudied group with their own unique experiences that have yet to be investigated using stress biomarkers. According

to a systemic review, homosexually experienced heterosexuals also experience psychological and physical health problems that are greater than heterosexual individuals, but lower than bisexual individuals (42).

The current study investigated AL differences as a function of sexual orientation using the population sample public data NHANES while adjusting for key covariates. First, we hypothesized that bisexual men and women would evidence higher AL than heterosexual men and women based on studies indicating high levels of stress in bisexual individuals (1). Second, we explored whether gay men differed in AL, as compared to heterosexual and bisexual men. Third, we explored whether lesbians would show higher AL than heterosexual women consistent with sexual minority stress theory (5, 31, 43). Finally, we included a fourth stratification of homosexually experienced individuals of both sexes that otherwise identified as heterosexual to contrast potential gradients in AL as a function of sexual behavior. These hypotheses are based on studies of racial/ethnic minorities in which findings indicate frequent activation of the physiological stress response systems that can be manifested as AL (44).

METHODS

Data Source and Sample

We use publicly available data from the 2001–2010 National Health and Nutrition Examination Survey (NHANES). The NHANES is a continuous population-based health survey conducted by the National Center for Health Statistics (NCHS) and released in two-year cycles. The NHANES sample is representative of the civilian, non-institutionalized U.S. population ages 2 months and older. Beginning in 2001, the NHANES included assessments of sexual orientation identity for individuals age 14 years and older with varying upper age limits depending on the survey cycle.

Availability of sexual orientation assessment varies in the publicly released survey cycles across different age ranges. As such, we limit the current analysis to participants between ages 20 to 59 years ($n = 18,014$), as this is the age cohort consistently included in all 5 of the NHANES cycles. Of those age-eligible individuals, 15,361 were administered the sexual behavior modules described more fully below. From this latter group, we excluded 870 women who were pregnant at the time of the NHANES examination, as this may have affected biological markers key to the current study. An additional 580 persons were excluded because they did not have their blood drawn ($n = 519$), were not measured for height, weight, and blood pressure ($n = 13$), or provided insufficient information to be coded for sexual orientation (e.g., denied being sexually active and did not report a heterosexual, gay, or bisexual identity; $n = 48$). This resulted in a final sample size of 13,911. Further information on the NHANES datasets are described elsewhere (30).

Sexual Orientation

The NHANES assessed both sexual orientation identity (e.g., heterosexual, lesbian/gay, bisexual) and the sex of sexual partners since age 18 and in the year prior to interview. Following procedures suggested by the NCHS, we logically recoded several individuals who

were skipped out of the detailed sexual history assessment ($n = 492$). These persons did not affirmatively acknowledge being sexually experienced but were queried as to their sexual orientation identity. Those who reported a current marital status most likely reflective of past or current heterosexuality (i.e., married, widowed, divorced, separated: $n = 339$) or, for women, a history of being pregnant ($n = 153$) were coded as having a positive lifetime history of opposite-sex sexual partners.

Participants were next grouped as follows: (i) those reporting a lesbian or gay identity, regardless of sexual history ($n = 211$); (ii) those reporting a bisexual identity, regardless of sexual history ($n = 307$); (iii) those indicating positive lifetime histories of same-sex sexual partners (homosexually experienced; $n = 424$) in the absence of a current lesbian, gay or bisexual identity (92% currently identified as heterosexual); or (iv) exclusively heterosexual ($n = 12,969$) including those who explicitly self-identified as heterosexual ($n = 12,671$) or reported no same-sex sexual partners or gay/bisexual identity ($n = 282$) or, barring that, evidenced marital and reproductive histories consistent with heterosexuality ($n = 15$)(45). While we did not include participants who reported “something else”, “not sure”, “don’t know”, or “refused”, these subgroups represent yet another layer of complexity in sexual orientation of significance (46).

Allostatic Load

Across the five survey cycles of interest, the NHANES consistently measured nine biomarkers that are commonly used to index AL (15). These represent cardiovascular (systolic and diastolic blood pressure, resting heart rate), metabolic (glycosylated hemoglobin, body mass index (BMI), total cholesterol, high density lipoprotein (HDL) cholesterol), and immune (serum albumin, C-reactive protein) functioning. Consistent with previously reported strategies that index physiological dysregulations using clinical reference ranges (36, 47), we first scored individuals as positive or not for each of the nine biomarkers individually using standard clinical cutoffs as previously applied in NHANES analyses of AL (18, 19, 21). Clinical ranges were provided by NHANES laboratory protocol manuals as well as supplemental documents routinely used (48–50). AL was then indexed by a count of positive biomarkers (range = 0 to 9).

The cut-offs used are as follows: systolic blood pressure ≥ 140 mm, diastolic blood pressure ≥ 90 mm, resting heart rate ≥ 90 beats/minute, glycosylated hemoglobin $\geq 6.4\%$, BMI ≥ 30 kg/m^2 , total cholesterol ≥ 240 mg/dL, HDL cholesterol < 40 mg/dL, serum albumin < 3.8 g/dL, and C-reactive protein > 0.3 mg/dL. Respondents who reported that they were currently taking medication for high blood pressure, cholesterol lowering drugs, or diabetes medication or insulin injections were scored positive for the two blood pressure biomarkers, total cholesterol, and/or glycosylated hemoglobin, respectively, regardless of laboratory values.

Detailed information for the NHANES examination and laboratory protocols are available on the Center for Disease Control’s website (<https://www.cdc.gov/nchs/nhanes/index.htm>). As part of the examination, three to four resting blood pressure and heart rate measurements were taken in the mobile examination center and during home examinations on all eligible individuals using the Baumanometer® calibrated mercury true gravity wall model or

portable desk model sphygmomanometer along with the LittmanTM Cardiology III stethoscopes. Height and weight used to calculate BMI were obtained by trained health technicians who recorded values as a team in a specially equipped room of the NHANES mobile examination center.

The following information summarizes laboratory protocols. Whole blood glycohemoglobin measurements were done using the A1c 2.2 Plus Glycohemoglobin Analyzer and during the survey cycle by A1c G7 HPLC Glycohemoglobin Analyzer (Tosoh Medics Inc., San Francisco, CA). Specimens destined for cholesterol and albumin measurement were processed, stored and shipped to the University of Minnesota, Minneapolis, MN for analysis. Cholesterol was analyzed using the Roche Modular P chemistry analyzer using protocols specified by the manufacturer. Albumin was quantified with solid-phase fluorescent immunoassay with a standard curve ranging from 0.5–20 µg/mL. Blood specimens destined for c-reactive protein measurement were processed, stored and shipped to University of Washington, Seattle, WA. Serum ultra-sensitive c-reactive protein was quantified using the Behring latex-enhanced nephelometric technique that yields a lower detection limit of 0.02 ng/mL.

Health Indicators

The NHANES also measured several health-related indicators that are robust covariates in studies of both sexual orientation (1) and AL (15). These included health insurance status (coded as has current coverage or not), tobacco smoking (coded as current smoker or not), and levels of mental distress.

Mental distress was assessed in the NHANES using a single item from the Center for Disease Control and Prevention HRQOL-4 “Healthy Days Measure” (51). Respondents reported how many days in the past 30 days that their mental health was “not good.” Those reporting 14 or more days were coded as experiencing frequent mental distress.

In addition, the NHANES measured reports of leisure time exercise that has been shown in previous studies to be associated with AL levels (52) though its association with sexual orientation is somewhat unclear (53–55). Respondents who reported that they had not engaged in either vigorous and/or moderate leisure time exercise lasting 10 minutes or more were coded as not exercising. Over the 5 survey cycles, the time frame for the questions varied between 30 days prior to interview (2001–2006) and “in a typical week” (2007–2010).

Finally, information on prevalent HIV infection is also available in the public dataset, but only for individuals ages 20 to 49 years old. As HIV infection was quite rare among sexual minority women (only 2 cases are reported across 10 years of NHANES data), analyses focusing on the possible contribution of HIV infection to AL were limited to men in the sample.

Demographics

The NHANES also collected information on respondents’ sex and race/ethnicity. The latter was coded as non-Hispanic White vs. racial/ethnic minority. Several other demographic

characteristics, causally unrelated to sexual orientation in AL studies (15), were also considered as possible confounders. These included age, foreign birth, and educational attainment. All have been shown to be associated with sexual orientation (56, 57), as well indicators of mental health morbidity (58–64) and AL (15). We also took into consideration possible measurement and temporal variance over the two survey cycles.

Analytic Approach

Analyses were conducted in Stata 11 (65) using design information and sample weights. Missing data were imputed by ICE methods. In the first set of analyses, we used linear or logistic regression, as appropriate, to evaluate sexual orientation-linked differences in demographic characteristics, discrimination experiences, mental health morbidity, and substance use behaviors. In conducting analyses of discrimination and morbidity measures, we adjusted for possible demographic (age, race/ethnicity, educational attainment, and foreign birth) and survey cycle confounding effects. For analyses of summary AL counts, we used negative binomial regression methods for men and women separately, as well as for men, age 20–49 years who were assessed for HIV infection. We present two sets of models. The first adjusts for confounding due to demographic factors and survey cycle. The second model further adjusts for health indicators. In the text, we report weighted prevalences and means, and their standard errors, standardized betas, adjusted odds ratios, and results from Wald F Tests. Significance of all tests, was evaluated at $p < 0.05$ level. All reported confidence intervals are at 95% confidence. Given our focus on within-sex/gender variation as a function of sexual orientation, our statistical analyses were conducted for men and women separately as previously justified (66).

RESULTS

Individual Characteristics Associated With Sexual Orientation

Approximately 6.8% (95% CI: 6.2%–7.5%) of the weighted respondents reported either a lesbian/gay (1.7%, 95% CI: 1.3%–2.0%) or bisexual identity (2.1%, 95% CI: 1.8%–2.4%), or, in their absence, same-sex sexual partners since age 18 (3.1%, 95% CI: 2.6%–3.5%) (see Table 1). Several characteristics that might confound associations between measures of AL and sexual orientation varied significantly by sexual orientation status. Such characteristics include sex (adjusted Wald $F(3) = 26.76$, $p < 0.001$), age (adjusted Wald $F(9) = 2.88$, $p < 0.05$), level of education (adjusted Wald $F(9) = 6.12$, $p < 0.001$), foreign birth (adjusted Wald $F(3) = 5.90$, $p < 0.05$), family income (adjusted Wald $F(12) = 3.22$, $p < 0.001$), and survey cycle (adjusted Wald $F(12) = 2.71$, $p < 0.05$). Notably, significant differences in racial/ethnic backgrounds were not observed (adjusted Wald $F(9) = 0.67$, $p = 0.74$).

Sexual Orientation Differences in Health Indicators

Sexual orientation among men was associated with differences in prevalence of frequent mental distress (adjusted Wald $F(3) = 13.17$, $p < 0.001$) and weekly binge drinking (adjusted Wald $F(3) = 2.73$, $p = 0.05$) (see Tables 2 and 3). However, similar effects were not observed for prevalence of health insurance coverage (adjusted Wald $F(12) = 1.39$, $p = 0.25$), leisure time exercise (adjusted Wald $F(3) = 0.43$, $p = 0.73$) or current smoking (adjusted Wald $F(3) = 1.96$, $p = 0.13$) though focused contrasts indicate that gay men were

significantly more likely to be current smokers than exclusively heterosexual men. Prevalence of HIV infection was strongly associated with sexual orientation among men age 20 to 49 years (adjusted Wald $F(12) = 58.77$, $p < 0.001$). Among women, health insurance coverage (adjusted Wald $F(3) = 2.76$, $p < 0.05$), frequent mental distress (adjusted Wald $F(3) = 7.14$, $p < 0.001$), weekly binge drinking (adjusted Wald $F(3) = 12.33$, $p < 0.001$) and reports of current smoking (adjusted Wald $F(3) = 17.56$, $p < 0.001$) were associated with sexual orientation though similar to men leisure time exercise (adjusted Wald $F(3) = 0.42$, $p = 0.74$) was not.

Sexual Orientation Differences in Allostatic Load

Figure 1 illustrates the weighted mean AL for the sample (Table 3). Among men, sexual orientation was associated with AL (adjusted Wald $F(3) = 3.75$, $p < .05$). After we adjusted for confounding, gay men had significantly lower levels of AL compared with men who identified as exclusively heterosexual (see Table 4). In contrast, bisexual men evidenced significantly higher levels of AL compared with exclusively heterosexual men. When we restricted our sample to men ages 20–49, this relationship was attenuated for gay men but not for bisexual men.

Among specific biomarkers comprising AL, there were statistically significant sexual orientation related differences in high systolic blood pressure (adjusted Wald $F(3) = 3.37$, $p < .05$), elevated glycosolated hemoglobin (adjusted $F(3) = 5.37$, $p < .05$), and high diastolic blood pressure (adjusted Wald $F(3) = 3.80$, $p < .05$). Specifically, gay men evidenced significantly lower levels of glycosolated hemoglobin (see Table 3) and systolic blood pressure compared with exclusively heterosexual men. By contrast, bisexual men had significantly higher levels of glycosolated hemoglobin and systolic blood pressure than men who identified as exclusively heterosexual. Homosexually experienced men evidenced significantly lower levels of diastolic blood pressure compared with exclusively heterosexual men.

Among women, sexual orientation was not associated with AL (adjusted Wald $F(3) = 0.51$, $p = .67$). There were no statistically significant differences in AL for lesbian women, bisexual women, or homosexually experienced women, compared with exclusively heterosexual women. However, there were significant sexual orientation related differences among specific indices of AL, including BMI consistent with obesity (adjusted Wald $F(3) = 1.91$, $p = .08$), and low albumin (adjusted Wald $F(3) = 2.21$, $p = .09$). Specifically, bisexual women were more likely to have a higher BMI and lesbian women were more likely to have lower levels of albumin compared with women who identified as exclusively heterosexual.

DISCUSSION

The current study assessed whether physiological dysregulations measured using AL indices differs by sexual orientation in a large population-based sample. We found sub-group differences in AL only among men where gay men showed the lowest AL levels and bisexual men showed the highest AL in comparison to exclusively heterosexual men. No differences in AL were found among women or among homosexually experienced heterosexuals of either sex. We theorize that social marginalization affects both pathogenic

and/or salutogenic processes that contribute to AL profiles in unique ways within subgroups of sexual minorities.

Our results are consistent with an earlier Juster and colleagues' study (36) showing that gay men evidence lower AL levels compared to heterosexual men and where women show no AL differences. These results do not, however, concur with the only other known published study of AL and sexual orientation from the United States. In an analysis by Hatzenbuehler and colleagues (67) of 306 LGB and 6667 heterosexual young adults from the National Longitudinal Study for Adolescent Health (ADD HEALTH), LGB individuals did not show differences in AL compared to heterosexuals. Among LGB individuals only, more stressful life events spanning childhood to emerging adulthood predicted elevated AL based on blood pressure, pulse, c-reactive protein, glycosylated hemoglobin, and waist circumference (67). Another ADD HEALTH analysis of individual biomarkers found that gay/bisexual men had higher c-reactive protein, diastolic blood pressure, and pulse, but lower glycosylated hemoglobin compared to heterosexual men (68). These studies did not, however, examine cardiometabolic biomarkers or AL indices differentially between LGB subgroups. Our study expands measurement factors in this literature and indicates a need for future studies to analytically divide within-sex (if sufficiently powered to do so) when assessing stress biomarkers and AL.

The findings from the current study and those of Juster and colleagues (36) suggest that gay men evidence lower levels of AL compared to heterosexual men. This is not consistent with a sexual minority stress framework. There may be a number of ways that the status of being a sexual minority presents a unique set of conditions accounting for differences both between sexual minorities and heterosexuals and within sexual minorities by sex. It may be the case that the health disparities experienced by gay men are not mediated by physiological dysregulation per se, but may be influenced through other psychosocial pathways. Indeed, a critical feature in the Hatzenbuehler and colleagues AL study (67) was the analytic combination of stressful life events in conjunction with sexual minority status that together were associated with cardiometabolic risk factors.

An often-unaddressed factor to consider in the minority stress literature is life-course perspectives. Within racial/ethnic minority groups, for example, the stress of being treated badly, differently, or poorly begins in early childhood. The stress-health dysregulating hypothesis in racial/ethnic minorities, particularly African Americans, is also highly related to macroeconomic conditions which may be quite different for non-racial/ethnic minority gay men. Brody, Yu & Beach (12) found that for African American adolescents, societal-level economic conditions are related to immune and physiological processes (e.g., AL, cellular epigenetic aging). Examining within gay men the extent to which these socioeconomic conditions are present and play a role may be important in future efforts to identify how stress, minority, sex/gender (69), objective and subjective SES statuses (70), and physiologic processes cluster to protect or confer risk for negative physiological health outcomes. Another reason for the lack of elevated AL findings may be related to the length of time of being or identified with a sexual minority status as AL is based on a 'wear and tear' premise over time. Knowing more about age of 'coming out', recognition of sexual

minority status as well as how milestones of the sexual minority development process (71) are implicated in the AL process would be helpful.

We also know that psychosocial resources like support networks can also influence AL (72) in ways that can promote risk and/or protection. Brody, Yu & Beach's (12) study of racial/ethnic minority adolescents found that even in the face of difficult socioeconomic conditions, strong parental emotional support served to offset some risk for cardiovascular disease, inflammatory, neuroendocrine, and metabolic risk for diseases and disorders (44). Including peer networks and social capital of neighborhoods in gay men may be useful areas for future consideration. In our study, we adjusted for psychological distress and key health and demographic factors; however, the NHANES does not measure many of the factors that we advocate would benefit in better understanding AL, sex/gender, and sexual minority status to refining our knowledge of gay men's stress and AL.

There is also another explanation that may be a factor in accounting for why gay men show lower levels of AL which involves their experiences of socially reinforced ideals of body thinness and muscularity that influence their health behaviors. Compared to exclusively heterosexual men, gay-identified men evidenced lower BMI (73) and in the current study glycosylated hemoglobin while adjusting for exercise and other health behaviors. While this represents a more favorable metabolic profile, these differences may be related to sociocultural beliefs regarding body image ideals among gay men (74). Indeed, gay men are more likely to endorse a muscular physique (75), disordered eating (76, 77), and experience body dissatisfaction compared to both heterosexual men (54, 55) and bisexual men (56). As early as adolescence, being a sexual minority male influences attitudes and perspectives about weight, muscularity, and body image (78). Future studies may consider investigating sub-group differences in body image related behaviors (e.g., exercise, body fat, eating patterns, eating disorders) to further explicate influencing factors in AL variations among gay men.

In our study, bisexual men evidenced significantly higher levels of AL compared to exclusively heterosexual men. A growing number of studies suggest that among sexual minorities, bisexual individuals experience higher levels of psychological distress and are at greater risk for poor health outcomes compared to other sexual minorities and heterosexuals who have poor health status (79–81). Bisexual men show the poorest self-rated health (82) and engage in more unhealthy behaviors that increase risk of cardiovascular disease (83). Elevated AL among bisexual men in the current study may represent their elevated levels of stress associated with their minority status and lower levels of support within diverse communities.

A recent study revealed that bisexual individuals were more likely to report lower levels of community connection and self-disclosure and higher levels of identity confusion (40). In a 2015 Pew Research Center survey, bisexuals were significantly less likely than gay men or lesbians to be out to people important to them (84). Only 28% of bisexuals say people in their life know they are bisexual which stood in comparison to 77% of gay men and 71% of lesbians. Similarly, studies suggest that bisexuals have higher proximal stressors associated with concealment of their bisexuality status (81). In addition to experiencing the typical

sexual minority stressors associated with heterosexism and homophobia, it has been reported that bisexual individuals also face unique forms of hostility, prejudice, stigma, and discrimination based on attitudes in both the heterosexual and lesbian/gay community (81, 85). They are perceived as unable to commit, disloyal, sexually promiscuous, confused about their sexual orientation, and/or immoral or unstable (81) that could compound their stress and AL.

In stark contrast to results among men, we found no sexual orientation differences in AL among women. Compared to exclusively heterosexual women, bisexual women did, however, have higher BMI and lesbian women had lower levels of albumin. The lack of AL differences among women is again consistent with the findings of Juster and colleagues (36). It is noteworthy that secondary analysis of Juster and colleagues' sample revealed that lesbian/bisexual women showed higher dehydroepiandrosterone-sulphate (antagonist of cortisol) and lower low-density lipoprotein ("bad") cholesterol than heterosexual women (86), which denotes a healthier metabolic profile.

The current stress biomarker findings are inconsistent with research indicating that lesbian and bisexual women report poorer overall physical health (87) and evidence more risk factors for disease (83, 88) than exclusively heterosexual women. We believe that this inconsistency with other studies showing greater physical health risk (e.g., smoking, alcohol) may be related to metabolic mechanisms that are poorly understood. In the current study and many other AL studies, the majority of biomarkers comprising AL algorithms are related in some way to obesity. As early as adolescence, obesity is more prevalent among sexual minority women (89, 90). And yet, a systemic review of 20 studies concluded that the prevalence of physical health disorders is not higher among these women (91).

There is substantial literature showing that obese sexual minority women can be physiologically fit (92) and emerging literature that they may show dampened inflammatory markers (68). With the exception of an increased risk of asthma, there is some evidence that sexual minority women do not in fact show increased risk for diabetes, hypertension, cardiovascular disease, and most cancers (93). This is in spite of greater overall risk factors for cardiovascular disease (e.g., smoking, heavy alcohol consumption, and obesity)(94). In an ADD HEALTH analysis, lesbian/bisexual women indeed evidenced greater BMI than heterosexual women; however, they also showed lower levels of c-reactive protein (68) involved in acute phase inflammatory reactions. Interestingly, another study showed that lesbian women experiencing greater discrimination had lower levels of the pro- and anti-inflammatory cytokine interleukin-6 levels (34). While we did not detect differences in c-reactive protein by sexual orientation, it is possible that unmeasured upstream processes (e.g., cytokines) may have influenced differences among women. Further research that assess psychosocial characteristics of sexual minority women in relation to stress biomarkers are needed to help solve this puzzle.

Ours is not the only study to not detect within-sex diversity in AL among women. Using a "sex-specific" AL formulation – as opposed to the traditional "all-inclusive" formulation that ignores sex differences in individuals biomarkers – a recent study found within-sex differences in AL only among working men but not women (66). Independent of sexual

orientation, androgynous men reporting both high masculinity (e.g., independent) and high femininity (e.g., sympathetic) evidenced protection against AL, but this difference was not present among women who must juggle more work/home responsibilities and who may not garner the same health benefits of androgynous adaptability that men do. From a cumulative disadvantage perspective, women worldwide experience social inequalities that may affect their AL more than men irrespective of their sexual orientation. The compounding effects of multiple marginalized identities include the pernicious effects of gender inequities (20) that have not been directly assessed in AL studies.

Theoretically, biological sex- and sociocultural gender-based differences influence patterns of physiological stress responsivity (95, 96). For instance, women display increased cortisol reactivity when facing social rejection (97), whereas men mount an increased stress response when confronted with social-evaluative threat (98). Taylor and colleagues' evolutionary proposal states that men and women cope differently to stressful situations (99). Whereas men are more likely to engage in "fight-or-flight" responses, women are more likely to engage in "tend-and-befriend" responses that involve nurturing and affiliation-based behaviors that protect against the demands of pregnancy, nursing, and child care (99). But how does this theory apply to sexual minority women and men? Within the lifetime of sexual minorities, stress and resilience processes may uniquely influence their stress-related biobehavioral mechanisms.

Our results support thinking that sexual orientation status can be related to within-sex variations in biobehavioral stress responses linked to AL that differs from patterns theorized with heterosexuals in mind. In accordance, Juster and colleagues showed that cortisol reactivity to a social-evaluative stressor is gender inversed (100). Specifically, lesbian/bisexual women showed higher cortisol concentrations than heterosexual women, while gay/bisexual men showed lower overall cortisol production than heterosexual men in response to the Trier Social Stress Test. Allostatic mechanisms (101) thus span a wide spectrum of response patterns within-sex. It follows that stress-related physiological functions will recalibrate to match the needs of unique circumstances over time. Sexual minority status and the psychosocial processes therein may therefore embed biobehavioral patterns in unique ways for each LGB sub-group according to distinct sociocultural pressures (e.g., fitness, diet, partnership, social spaces) to be explored.

Strengths and Limitations

There are several limitations to the current study centered upon four areas: (i) potential response bias, (ii) temporality, (iii) AL formulations, and (iv) LGB-related psychosocial correlates. First, it is possible that participants' response bias and their willingness to disclose their sexual minority status during the NHANES interview may have confounded the generalizability of our findings. Research suggests that LGB individuals who have 'come out' have lower AL compared to their non-disclosed peers (36). Willingness to disclose sexual minority status may be indicative of a well-adjusted, highly resilient sub-sample of LGB individuals. If so, this could have resulted in a response bias that could explain why gay men show the lowest AL in our study.

Second, the NHANES is a cross-sectional survey. Longitudinal research is the best approach to elucidate the pathways through which variations in sexual orientation are associated with vulnerability and/or resilience to physiological dysregulation. Other studies have noted that the individual biological pathways through which AL is facilitated differ by race/ethnicity, SES, and education (102). It is therefore possible that sexual orientation, particularly when combined with race/ethnicity and any of these other variables, may also affect the pathways. Results of our research suggest that it is important to demarcate intersecting statuses (103). Intersectionality recognizes that individuals are members of multiple social groups with diverse societal responses that determine contextual experiences, opportunities, stress exposures and ultimately health and wellness (104). For example, diurnal cortisol differs by race/ethnicity among sexual minority men (105) and by stigma exposure among transgender men (106). While power was not sufficient in the current study to properly employ an intersectional approach, future prospective studies could use moderation analyses of race/ethnicity or other identities in interaction with sexual orientation to further nuance the biological footprints of stigmatized identities/statuses.

Third, it is possible that ascribing clinical AL cut-offs without regard for sex differences does not fully capture meaningful associations among women (66). Clinical norms that can identify meaningful biomarker cut-offs to better predict sex-specific disease processes are needed for the advancement of gender medicine. It is also noteworthy that while sex differences exist in individual biomarkers, few AL studies account for these (66). Furthermore in NHANES, individual biomarkers used to calculate AL show factorial unidimensionality; however, subtle variations exist by race/ethnicity (102), speaking potentially to unique experiences of “weathering” among marginalized social groups. Moreover, of the 26 different biomarkers that have been used in 21 NHANES AL studies spanning 1988 to 2010, many do not have population-specific clinical guidelines (107). Lastly, the NHANES does not include neuroendocrine biomarkers such as cortisol. This limitation is not uncommon in the AL literature and does not pose a major limitation given the heterogeneity of biomarkers used in AL studies (15). Nevertheless, it is promising that our population-level findings concord with those of Juster and colleagues (36) that did include neuroendocrine biomarkers in a 21 biomarker AL index.

Fourth, incorporating stress biomarkers may not necessarily provide additional means from which to differentiate AL by sexual orientation without also considering psychosocial variables that can accurately capture individual differences in stress and coping among LGB subgroups. In accordance, a recent study using a nationally-representative sample of young American adults ($N=1670$) reported no differences in diurnal cortisol as a function of sexual orientation alone. Likewise in ADD HEALTH, sex differences in c-reactive protein and Epstein-Barr virus were reversed among sexual minorities compared to heterosexuals and not explained by known risk factors such as victimization, alcohol and tobacco use, and BMI (108). It may be that stress biomarkers are more strongly correlated to proximal stress processes specific to LGB subgroups (e.g., concealment, disclosure, body image) than to distal stress processes (e.g., victimization, discrimination) that are often assumed to exist by virtue of sexual orientation grouping, but that have seldom been actually measured in relation to stress biomarkers. Lastly, the experiences of transgender individuals has received

limited attention (109). Emerging research suggest that transgender-specific stressors modulate stress-related biomarkers (106, 110) that should be explored in future AL studies.

Despite these limitations, our multi-systemic findings complement emerging research showing that LGB individuals may manifest distinct biological profiles as a function of sexual minority stress processes. Specifically, stigma generated by distal processes (e.g., structural stigma) and proximal processes (e.g., ‘coming out’, internalized homophobia) are associated with either up-regulation or down-regulation of cortisol functioning (36, 111, 112), a primary mediator of AL. Future research would do well to nuance distal from proximal stress processes central to sexual minority stress theory since the unique experiences of subgroups (e.g., bisexuals) may obscure differences in their unique biological signatures and AL trajectories. Lastly, we encourage disaggregation by sex when assessing sexual orientation subgroups following new NIH recommendations (113). Similar to previously raised concerns (102), if efforts to address health disparities are to be successful studies – such as ours and others that suggest that specific stressors associated with the wear and tear of AL or differences in the pathways that lead to AL – we may need to rethink our clinical intervention and health policies. Launching large scale untailed interventions may fail to effectively address the way that AL is expressed in some sub-group populations such as male and female LGB members.

Conclusions

To summarize, our findings indicate that bisexuality among men is associated with elevated physiological dysregulation measured using multi-systemic AL indices. By contrast, gay men evidenced the lowest AL, while no AL differences were detected among women. These findings underscore the importance of examining stress biomarkers and AL differentially among subgroups of sexual minorities. Additional research is also needed to elucidate the socio-cultural pathways that contribute to distinct AL profiles among subgroups of sexual minorities. The impact of minority stressors and unique exposures to stigma experienced by each LGB subgroup differently may drive distinct biobehavioral stress and coping responses that ultimately increase and/or decrease one’s risk of developing physical and mental disease.

Acknowledgments

Sources of Funding:

This research was funded by grants from the National Institute on Drug Abuse (DA20826) and the National Center for Minority Health and Health Disparities (MD006923), National Institute of Mental Health Predoctoral Fellowship (MH 15750) and Quebec Fonds de recherche Santé Post-Doctoral Fellowship (FRQS 31783).

ACRONYMS:

AL	allostatic load
BMI	body mass index
HDL	high-density lipoprotein

LGB	lesbian, gay, and bisexual
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey

References

1. IOM. The Health of Lesbian, Gay, Bisexual, and Transgender People: Building a Foundation for Better Understanding Washinton, DC: The National Academies Press; 2011.
2. Cochran SD, Mays VM. Burden of psychiatric morbidity among lesbian, gay, and bisexual individuals in the California Quality of Life Survey. *J Abnorm Psychol* 2009;118:647–58. [PubMed: 19685960]
3. Frost DM, Lehavot K, Meyer IH. Minority stress and physical health among sexual minority individuals. *J Behav Med* 2015;38:1–8. [PubMed: 23864353]
4. Hsieh N, Ruther M. Sexual Minority Health and Health Risk Factors: Intersection Effects of Gender, Race, and Sexual Identity. *Am J Prev Med* 2016.
5. Meyer IH. Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: conceptual issues and research evidence. *Psychol Bull* 2003;129:674–97. [PubMed: 12956539]
6. Mays VM, Cochran SD. Mental health correlates of perceived discrimination among lesbian, gay, and bisexual adults in the United States. *Am J Public Health* 2001;91:1869–76. [PubMed: 11684618]
7. Sandfort TG, Bakker F, Schellevis FG, Vanwesenbeeck I. Sexual orientation and mental and physical health status: findings from a Dutch population survey. *Am J Public Health* 2006;96:1119–25. [PubMed: 16670235]
8. McEwen BS, Stellar E. Stress and the individual. Mechanisms leading to disease. *Arch Intern Med* 1993;153:2093–101. [PubMed: 8379800]
9. Geronimus AT, Hicken M, Keene D, Bound J. “Weathering” and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health* 2006;96:826–33. [PubMed: 16380565]
10. McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol Rev* 2007;87:873–904. [PubMed: 17615391]
11. Seeman E, Singer BH, Rowe J, Horwitz RI, McEwen B. Price of adaptation - allostatic load and its health consequences. *Archives of Internal Medicine* 1997;157:2259–68. [PubMed: 9343003]
12. Brody GH, Yu T, Beach SR. Resilience to adversity and the early origins of disease. *Dev Psychopathol* 2016;28:1347–65. [PubMed: 27692007]
13. Picard M, Juster RP, McEwen BS. Mitochondrial allostatic load puts the ‘gluc’ back in glucocorticoids. *Nature Reviews Endocrinology* 2014;10:303–10.
14. McEwen BS. Protective and damaging effects of stress mediators. *The New England Journal of Medicine* 1998;338:171–9. [PubMed: 9428819]
15. Juster RP, McEwen BS, Lupien SJ. Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews* 2010;35:2–16. [PubMed: 19822172]
16. Beckie TM. A systematic review of allostatic load, health, and health disparities. *Biological research for nursing* 2012;14:311–46. [PubMed: 23007870]
17. Juster RP, Bizik G, Picard M, Arseneault-Lapierre G, Sindi S, Trepanier L, Marin MF, Wan N, Sekerovic Z, Lord C, Fiocco AJ, Plusquellec P, McEwen BS, Lupien SJ. A transdisciplinary perspective of chronic stress in relation to psychopathology throughout life span development. *Dev Psychopathol* 2011;23:725–76. [PubMed: 21756430]
18. Crimmins EM, Johnston M, Hayward M, Seeman T. Age differences in allostatic load: an index of physiological dysregulation. *Experimental Gerontology* 2003;38:731–4. [PubMed: 12855278]
19. Crimmins EM, Kim JK, Seeman TE. Poverty and biological risk: the earlier “aging” of the poor. *J Gerontol A Biol Sci Med Sci* 2009;64:286–92. [PubMed: 19196637]

20. Juster RP, Seeman T, McEwen BS, Picard M, Mahar I, Mechawar N, Sindi S, Smith NG, Souza-Talarico J, Sarnyai Z, Lanoix D, Plusquellec P, Ouellet-Morin I, Lupien SJ. Social inequalities and the road to allostatic load: From vulnerability to resilience. In: Cicchetti D, editor. *Developmental Psychopathology Handbook Third ed*: Cambridge Press; 2016.
21. Merkin SS, Basurto-Davila R, Karlamangla A, Bird CE, Lurie N, Escarce J, Seeman T. Neighborhoods and cumulative biological risk profiles by race/ethnicity in a national sample of U.S. adults: NHANES III. *Ann Epidemiol* 2009;19:194–201. [PubMed: 19217002]
22. Duru OK, Harawa NT, Kermah D, Norris KC. Allostatic load burden and racial disparities in mortality. *J Natl Med Assoc* 2012;104:89–95. [PubMed: 22708252]
23. Upchurch DM, Stein J, Greendale GA, Chyu L, Tseng CH, Huang MH, Lewis TT, Kravitz HM, Seeman T. A Longitudinal Investigation of Race, Socioeconomic Status, and Psychosocial Mediators of Allostatic Load in Midlife Women: Findings From the Study of Women’s Health Across the Nation. *Psychosom Med* 2015;77:402–12. [PubMed: 25886828]
24. Parente V, Hale L, Palermo T. Association between breast cancer and allostatic load by race: National Health and Nutrition Examination Survey 1999–2008. *Psychooncology* 2013;22:621–8. [PubMed: 22290849]
25. Tomfohr LM, Pung MA, Dimsdale JE. Mediators of the relationship between race and allostatic load in African and White Americans. *Health Psychol* 2016;35:322–32. [PubMed: 27018723]
26. Dannefer D Cumulative advantage/disadvantage and the life course: cross-fertilizing age and social science theory. *J Gerontol B Psychol Sci Soc Sci* 2003;58:S327–37. [PubMed: 14614120]
27. DiPrete T, Eirich GM. Cumulative advantage as a mechanism for inequality: A review of theoretical and empirical developments. *Annual Review of Sociology* 2006;32.
28. Leopold L Cumulative advantage in an egalitarian country? Socioeconomic health disparities over the life course in Sweden. *Journal of Health and Social Behavior* 2016;57:257–73. [PubMed: 27284078]
29. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis* 1992;2:207–21. [PubMed: 1467758]
30. Brooks VR. *Minority stress and lesbian women* Lexington, USA: Lexington Books, D. C. Health and Co.; 1981.
31. Meyer IH. Minority stress and mental health in gay men. *J Health Soc Behav* 1995;36:38–56. [PubMed: 7738327]
32. White Hughto JM, Reisner SL, Pachankis JE. Transgender stigma and health: A critical review of stigma determinants, mechanisms, and interventions. *Soc Sci Med* 2015;147:222–31. [PubMed: 26599625]
33. Herek GM. Sexual stigma and sexual prejudice in the United States: A conceptual framework. In: Hope DA, editor. *Contemporary perspectives on lesbian, gay, and bisexual identities* New York, NY: Springer Science + Business Media; 2009 p. 65–111.
34. Doyle DM, Molix L. Minority stress and inflammatory mediators: covering moderates associations between perceived discrimination and salivary interleukin-6 in gay men. *J Behav Med* 2016;39:782–92. [PubMed: 27534538]
35. Parra LA, Benibgui M, Helm JL, Hastings PD. Minority stress predicts depression in lesbian, gay, and bisexual emerging adults via elevated diurnal cortisol. *Emerging Adulthood* 2016;4:1–8.
36. Juster RP, Smith NG, Ouellet E, Sindi S, Lupien SJ. Sexual orientation and disclosure in relation to psychiatric symptoms, diurnal cortisol, and allostatic load. *Psychosom Med* 2013;75:103–16. [PubMed: 23362500]
37. Juster RP, Ouellet E, Lefebvre-Louis JP, Sindi S, Johnson PJ, Smith NG, Lupien SJ. Retrospective coping strategies during sexual identity formation and current biopsychosocial stress. *Anxiety, stress, and coping* 2016;29:119–38.
38. Hatzenbuehler ML. How does sexual minority stigma “get under the skin”? A psychological mediation framework. *Psychol Bull* 2009;135:707–30. [PubMed: 19702379]
39. Persson TJ, Pfaus JG, Ryder AG. Explaining mental health disparities for non-monosexual women: Abuse history and risky sex, or the burdens of non-disclosure? *Soc Sci Med* 2014.
40. Balsam KF, Mohr JJ. Adaptation to sexual orientation stigma: A comparison of bisexual and lesbian/gay adults. *Journal of Counseling Psychology* 2007;54:306–19.

41. Savin-Williams RC, Vrangalova Z. Mostly heterosexual as a distinct sexual orientation group: A systematic review of the empirical evidence. *Developmental Review* 2013;33:55–88.
42. Vrangalova Z, Savin-Williams RC. Psychological and physical health of mostly heterosexuals: a systematic review. *J Sex Res* 2014;51:410–45. [PubMed: 24754361]
43. Lick DJ, Durso LE, Johnson KL. Minority stress and physical health among sexual minorities. *Perspectives on Psychological Science* 2013;8:521–48. [PubMed: 26173210]
44. Miller GE, Chen E, Parker KJ. Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychol Bull* 2011;137:959–97. [PubMed: 21787044]
45. Cochran SD, Bandiera FC, Mays VM. Sexual orientation-related differences in tobacco use and secondhand smoke exposure among US adults aged 20 to 59 years: 2003–2010 National Health and Nutrition Examination Surveys. *Am J Public Health [Comparative Study Research Support, N.I.H., Extramural]* 2013;103:1837–44. [PubMed: 23948019]
46. Eliason MJ, Radix A, McElroy JA, Garbers S, Haynes SG. The “Something Else” of Sexual Orientation: Measuring Sexual Identities of Older Lesbian and Bisexual Women Using National Health Interview Survey Questions. *Womens Health Issues* 2016;26 Suppl 1:S71–80. [PubMed: 27397920]
47. Juster RP, Sindi S, Marin MF, Perna A, Hashemi A, Pruessner JC, Lupien SJ. A clinical allostatic load index is associated with burnout symptoms and hypocortisolemic profiles in healthy workers. *Psychoneuroendocrinology* 2011;36:797–805. [PubMed: 21129851]
48. Jeppsson JO, Kobold U, Barr J, Finke A, Hoelzel W, Hoshino T, Miedema K, Mosca A, Mauri P, Paroni R, Thienpont L, Umemoto M, Weykamp C. Approved IFCC reference method for the measurement of HbA1c in human blood. *Clinical chemistry and laboratory medicine : CCLM / FESCC [Guideline Practice Guideline Research Support, Non-U.S. Gov't]* 2002;40:78–89.
49. NCEP. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA : the journal of the American Medical Association [Guideline Practice Guideline]* 2001;285:2486–97. [PubMed: 11368702]
50. Sacks DB, Bruns DE, Goldstein DE, Maclaren NK, McDonald JM, Parrott M. Guidelines and recommendations for laboratory analysis in the diagnosis and management of diabetes mellitus. *Clin Chem* 2002;48:436–72. [PubMed: 11861436]
51. Hagerty M, Cummins R, Ferriss A, Land K, Michalos A, Peterson M, Sharpe A, Sirgy J, Vogel J. Quality of life indexes for national policy: review and agenda for research. *Social Indicators Research* 2001;55:1–96.
52. Seeman T, Merkin SS, Crimmins E, Koretz B, Charette S, Karlamangla A. Education, income and ethnic differences in cumulative biological risk profiles in a national sample of US adults: NHANES III (1988–1994). *Soc Sci Med* 2008;66:72–87. [PubMed: 17920177]
53. Zaritsky E, Dibble S, inventors; - Risk factors for reproductive and breast cancers among older lesbians
54. Boehmer U, Miao X, Ozonoff A. Health behaviors of cancer survivors of different sexual orientations. *Cancer Causes Control* 2012;23:1489–96. [PubMed: 22752329]
55. Boehmer U, Miao X, Linkletter C, Clark M. Adult health behaviors over the life course by sexual orientation. *Am J Public Health* 2012;102:292–300. [PubMed: 22390443]
56. Butler AC. Trends in same-gender sexual partnering, 1988–1998. *J Sex Res* 2000;37:333–43.
57. Carpenter C, Gates GJ. Gay and lesbian partnership: Evidence from California. *Demography [Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't]* 2008;45:573–90. [PubMed: 18939662]
58. Breslau J, Aguilar-Gaxiola S, Borges G, Kendler KS, Su M, Kessler RC. Risk for psychiatric disorder among immigrants and their US-born descendants: Evidence from the National Comorbidity Survey Replication. *J Nerv Mental Dis* 2007;195:189–95.
59. Kessler RC, Berglund P, Demler O, Jin R, Koretz D, Merikangas KR, Rush AJ, Walters EE, Wang PS. The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *JAMA* 2003;289:3095–105. [PubMed: 12813115]

60. Cochran SD, Mays VM, Sullivan JG. Prevalence of mental disorders, psychological distress, and mental health services use among lesbian, gay, and bisexual adults in the United States. *J Consult Clin Psychol* 2003;71:53–61. [PubMed: 12602425]
61. Cochran SD, Keenan C, Schober C, Mays VM. Estimates of alcohol use and clinical treatment needs among homosexually active men and women in the U.S. population. *J Consult Clin Psychol* [Comparative Study Research Support, Non-U.S. Gov't Research Support, U.S. Gov't, P.H.S.] 2000;68:1062–71. [PubMed: 11142540]
62. Gilman SE, Cochran SD, Mays VM, Hughes M, Ostrow D, Kessler RC. Risk of psychiatric disorders among individuals reporting same-sex sexual partners in the National Comorbidity Survey. *Am J Public Health* 2001;91:933–9. [PubMed: 11392937]
63. Sandfort TG, de Graaf R, Bijl RV. Same-sex sexuality and quality of life: Findings from the Netherlands Mental Health Survey and Incidence Study. *Arch Sex Behav* [Research Support, Non-U.S. Gov't] 2003;32:15–22. [PubMed: 12597268]
64. Sandfort TG, de Graaf R, Bijl RV, Schnabel P. Same-sex sexual behavior and psychiatric disorders: Findings from the Netherlands Mental Health Survey and Incidence Study (NEMESIS). *Arch Gen Psychiatr* [Comparative Study Research Support, Non-U.S. Gov't] 2001;58:85–91. [PubMed: 11146762]
65. Stata Corporation. *Stata 11 User's Guide* College Station, TX: Stata Press; 2010.
66. Juster RP, Preussner JC, Desrochers AB, Bourdon O, Durand N, Wan N, Tourjman V, Kouassi E, Lesage A, Lupien SJ. Sex and gender-roles in relation to mental health and allostatic load. *Psychosomatic Medicine* 2016;78:788–804. [PubMed: 27359170]
67. Hatzenbuehler ML, Slopen N, McLaughlin KA. Stressful life events, sexual orientation, and cardiometabolic risk among young adults in the United States. *Health Psychol* 2014;33:1185–94. [PubMed: 25133830]
68. Hatzenbuehler ML, McLaughlin KA, Slopen N. Sexual orientation disparities in cardiovascular biomarkers among young adults. *Am J Prev Med* 2013;44:612–21. [PubMed: 23683979]
69. Pelletier R, Ditto B, Pilote L. A Composite Measure of Gender and Its Association With Risk Factors in Patients With Premature Acute Coronary Syndrome. *Psychosom Med* 2015.
70. Freeman JA, Bauldry S, Volpe VV, Shanahan MJ, Shanahan L. Sex Differences in Associations Between Subjective Social Status and C-Reactive Protein in Young Adults. *Psychosom Med* 2016;78:542–51. [PubMed: 26910797]
71. Calzo JP, Antonucci TC, Mays VM, Cochran SD. Retrospective recall of sexual orientation identity development among gay, lesbian, and bisexual adults. *Dev Psychol* 2011;47:1658–73. [PubMed: 21942662]
72. Wiley JF, Bei B, Bower JE, Stanton AL. Relationship of Psychosocial Resources With Allostatic Load: A Systematic Review. *Psychosom Med* 2017;79:283–92. [PubMed: 27768647]
73. Deputy NP, Boehmer U. Weight status and sexual orientation: differences by age and within racial and ethnic subgroups. *Am J Public Health* 2014;104:103–9. [PubMed: 24228650]
74. Lanzieri N, Cook BJ. Examination of muscularity and body fat depictions in magazines that target heterosexual and gay men. *Body Image* 2013;10:251–4. [PubMed: 23352323]
75. Kaminski PL, Chapman BP, Haynes SD, Own L. Body image, eating behaviors, and attitudes toward exercise among gay and straight men. *Eat Behav* 2005;6:179–87. [PubMed: 15854864]
76. Russell CJ, Keel PK. Homosexuality as a specific risk factor for eating disorders in men. *Int J Eat Disord* 2002;31:300–6. [PubMed: 11920991]
77. Yelland C, Tiggemann M. Muscularity and the gay ideal: body dissatisfaction and disordered eating in homosexual men. *Eat Behav* 2003;4:107–16. [PubMed: 15000974]
78. Calzo JP, Corliss HL, Blood EA, Field AE, Austin SB. Development of muscularity and weight concerns in heterosexual and sexual minority males. *Health Psychol* 2013;32:42–51. [PubMed: 23316852]
79. Kim HJ, Fredriksen-Goldsen KI. Hispanic lesbians and bisexual women at heightened risk for [corrected] health disparities. *Am J Public Health* 2012;102:e9–15.
80. McNair R, Szalacha LA, Hughes TL. Health status, health service use, and satisfaction according to sexual identity of young Australian women. *Womens Health Issues* 2011;21:40–7. [PubMed: 21185989]

81. Mereish EH, Katz-Wise SL, Woulfe J. Bisexual-Specific Minority Stressors, Psychological Distress, and Suicidality in Bisexual Individuals: the Mediating Role of Loneliness. *Prev Sci* 2017;18:716–25. [PubMed: 28593529]
82. Gorman BK, Denney JT, Dowdy H, Medeiros RA. A New Piece of the Puzzle: Sexual Orientation, Gender, and Physical Health Status. *Demography* 2015;52:1357–82. [PubMed: 26126883]
83. Conron KJ, Mimiaga MJ, Landers SJ. A population-based study of sexual orientation identity and gender differences in adult health. *Am J Public Health* 2010;100:1953–60. [PubMed: 20516373]
84. PEW. Among LGBT Americans, bisexuals stand out when it comes to identity, acceptance <http://www.pewresearch.org/fact-tank/2015/02/20/among-lgbt-americans-bisexuals-stand-out-when-it-comes-to-identity-acceptance/>; 2015.
85. Bostwick W, Hequembourg A. 'Just a little hint': bisexual-specific microaggressions and their connection to epistemic injustices. *Cult Health Sex* 2014;16:488–503. [PubMed: 24666221]
86. Juster RP, Almeida D, Cardoso C, Raymond C, Johnson PJ, Pfaus JG, Mendrek A, Duchesne A, Pruessner JC, Lupien SJ. Gonads and strife: Sex hormones vary according to sexual orientation for women and stress indices for both sexes. *Psychoneuroendocrinology* 2016;72:119–30. [PubMed: 27398882]
87. Cochran SD, Mays VM. Physical health complaints among lesbians, gay men, and bisexual and homosexually experienced heterosexual individuals: results from the California Quality of Life Survey. *Am J Public Health* 2007;97:2048–55. [PubMed: 17463371]
88. Case P, Austin SB, Hunter DJ, Manson JE, Malspeis S, Willett WC, Spiegelman D. Sexual orientation, health risk factors, and physical functioning in the Nurses' Health Study II. *J Womens Health (Larchmt)* 2004;13:1033–47. [PubMed: 15665660]
89. Fogel SC, McElroy JA, Garbers S, McDonnell C, Brooks J, Eliason MJ, Ingraham N, Osborn A, Rayyes N, Redman SD, Wood SF, Haynes SG. Program Design for Healthy Weight in Lesbian and Bisexual Women: A Ten-City Prevention Initiative. *Womens Health Issues* 2016;26 Suppl 1:S7–S17. [PubMed: 27397919]
90. McElroy JA, Haynes SG, Eliason MJ, Wood SF, Gilbert T, Barker LT, Minnis AM. Healthy Weight in Lesbian and Bisexual Women Aged 40 and Older: An Effective Intervention in 10 Cities Using Tailored Approaches. *Womens Health Issues* 2016;26 Suppl 1:S18–35. [PubMed: 27397912]
91. Eliason MJ, Ingraham N, Fogel SC, McElroy JA, Lorvick J, Mauery DR, Haynes S. A systematic review of the literature on weight in sexual minority women. *Womens Health Issues* 2015;25:162–75. [PubMed: 25747521]
92. McElroy JA, Gilbert T, Hair EC, Mathews KJ, Redman SD, Williams A. Obese But Fit: The Relationship of Fitness to Metabolically Healthy But Obese Status among Sexual Minority Women. *Womens Health Issues* 2016;26 Suppl 1:S81–6. [PubMed: 27397921]
93. Eliason MJ. Chronic Physical Health Problems in Sexual Minority Women: Review of the Literature. *LGBT Health* 2014;1:259–68. [PubMed: 26789854]
94. Caceres BA, Brody A, Luscombe RE, Primiano JE, Marusca P, Sitts EM, Chyun D. A Systematic Review of Cardiovascular Disease in Sexual Minorities. *Am J Public Health* 2017;107:570.
95. Dedovic K, Wadiwalla M, Engert V, Pruessner JC. The role of sex and gender socialization in stress reactivity. *Developmental Psychology* 2009;45:45–55. [PubMed: 19209989]
96. Kajantie E, Phillips DI. The effects of sex and hormonal status on the physiological response to acute psychosocial stress. *Psychoneuroendocrinology* 2006;31:151–78. [PubMed: 16139959]
97. Stroud LR, Salovey P, Epel ES. Sex differences in stress responses: social rejection versus achievement stress. *Biological Psychiatry* 2002;52:318–27. [PubMed: 12208639]
98. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull* 2004;130:355–91. [PubMed: 15122924]
99. Taylor SE, Klein LC, Lewis BP, Gruenewald TL, Gurung RA, Updegraff JA. Biobehavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychological Review* 2000;107:411–29. [PubMed: 10941275]
100. Juster RP, Hatzenbuehler ML, Mendrek A, Pfaus JG, Smith NG, Johnson PJ, Lefebvre-Louis JP, Raymond C, Marin MF, Sindi S, Lupien SJ, Pruessner JC. Sexual orientation modulates endocrine stress reactivity. *Biological Psychiatry* 2015;77:668–76. [PubMed: 25444167]

101. Sterling P, Eyer J. Allostatic: A new paradigm to explain arousal pathology. In: Fisher S, Reason J, editors. *Handbook of Life Stress, Cognition and Health* New York: John Wiley; 1988 p. 629–49.
102. Howard JT, Sparks PJ. Does allostatic load calculation method matter? Evaluation of different methods and individual biomarkers functioning by race/ethnicity and educational level. *Am J Hum Biol* 2016.
103. Carbado DW, Crenshaw KW, Mays VM, Tomlinson B. INTERSECTIONALITY: Mapping the Movements of a Theory. *Du Bois Rev* 2013;10:303–12. [PubMed: 25285150]
104. Johnson JL, Repta R, Kaylan S. Implications of sex and gender for health research. In: Oliffe JL, Greaves L, editors. *Designing and Conducting Gender, Sex, and Health Research*: SAGE Publications, Inc.; 2011 p. 39–64.
105. Cook SH, Juster RP, Calebs BJ, Heinze J, Miller AL. Cortisol profiles differ by race/ethnicity among young sexual minority men. *Psychoneuroendocrinology* 2017;75:1–4. [PubMed: 27768979]
106. DuBois LZ, Powers S, Everett BG, Juster RP. Stigma and diurnal cortisol among transitioning transgender men. *Psychoneuroendocrinology* 2017;82:59–66. [PubMed: 28511045]
107. Duong MT, Bingham BA, Aldana PC, Chung ST, Sumner AE. Variation in the Calculation of Allostatic Load Score: 21 Examples from NHANES. *J Racial Ethn Health Disparities* 2017;4:455–61. [PubMed: 27352114]
108. Everett BG, Rosario M, McLaughlin KA, Austin SB. Sexual orientation and gender differences in markers of inflammation and immune functioning. *Ann Behav Med* 2014;47:57–70. [PubMed: 24347405]
109. van de Grift TC, Elaut E, Cerwenka SC, Cohen-Kettenis PT, De Cuypere G, Richter-Appelt H, Kreukels BPC. Effects of Medical Interventions on Gender Dysphoria and Body Image: A Follow-Up Study. *Psychosom Med* 2017;79:815–23. [PubMed: 28319558]
110. Dubois LZ. Associations between transition-specific stress experience, nocturnal decline in ambulatory blood pressure, and C-reactive protein levels among transgender men. *Am J Hum Biol* 2012;24:52–61. [PubMed: 22120883]
111. Huebner DM, Davis MC. Gay and bisexual men who disclose their sexual orientations in the workplace have higher workday levels of salivary cortisol and negative affect. *Ann Behav Med* 2005;30:260–7. [PubMed: 16336077]
112. Hatzenbuehler ML, McLaughlin KA. Structural Stigma and Hypothalamic-Pituitary-Adrenocortical Axis Reactivity in Lesbian, Gay, and Bisexual Young Adults. *Ann Behav Med* 2013;47:39–47.
113. Clayton JA, Collins FS. Policy: NIH to balance sex in cell and animal studies. *Nature* 2014;509:282–3. [PubMed: 24834516]

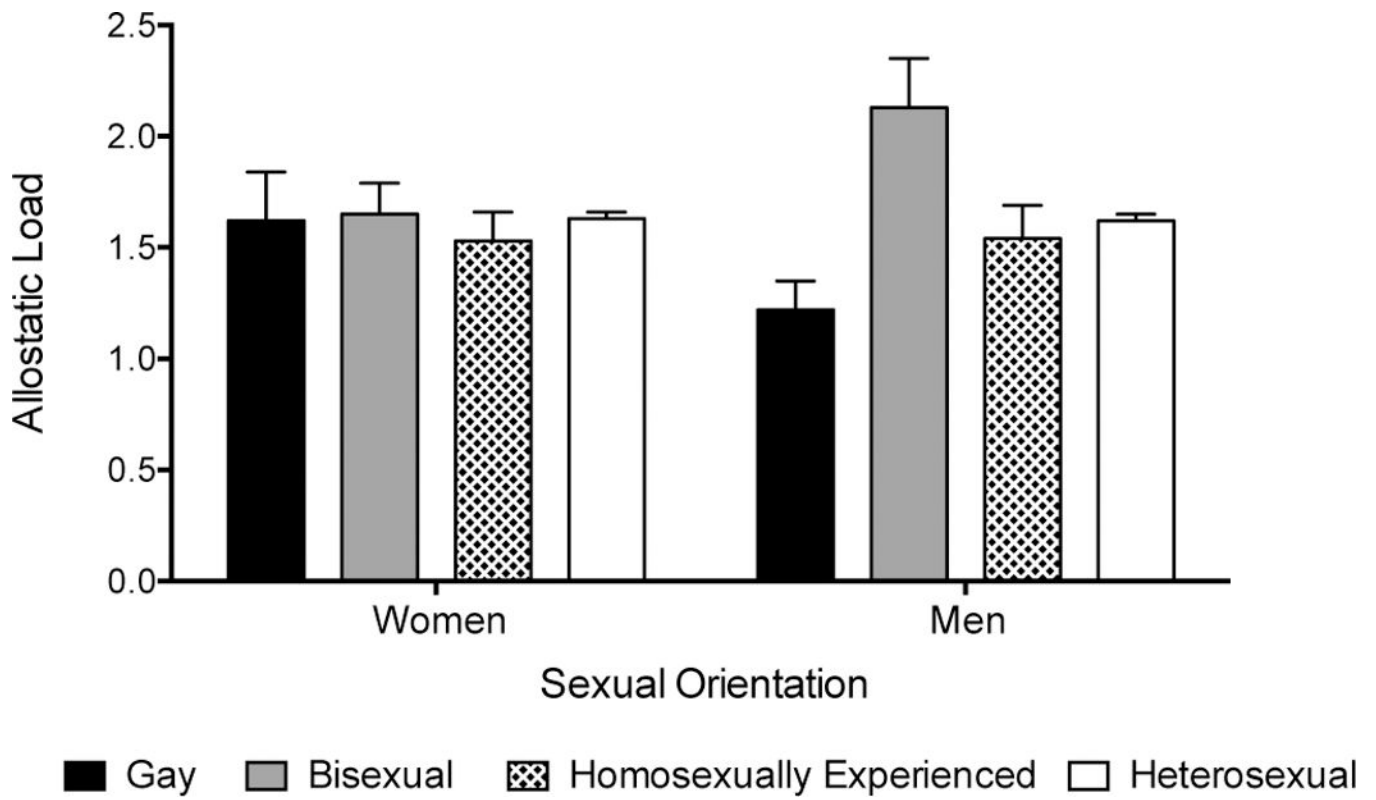


FIGURE 1. Weighted mean (SE) allostatic load as a function of sexual orientation stratified by sex.

TABLE 1.

Demographic characteristics of U.S. adults, age 20 to 59 years, by sexual orientation, NHANES (2001–2010)

<i>Characteristics</i>	<i>Gay</i> (<i>n</i> = 211)		<i>Bisexual</i> (<i>n</i> = 307)		<i>Homosexually</i> <i>experienced</i> (<i>n</i> = 424)		<i>Exclusively</i> <i>Heterosexual</i> (<i>n</i> = 12,969)	
	%	(SE)	%	(SE)	%	(SE)	%	(SE)
Female gender **	38.8	(5.4)	68.2	(3.2)	67.1	(2.6)	48.2	(0.4)
<u>Age **</u>								
20–29 years	20.8	(2.8)	35.8	(3.7)	23.4	(2.3)	23.3	(0.6)
30–39 years	31.6	(3.7)	27.9	(3.3)	26.3	(2.5)	23.9	(0.5)
40–49 years	39.8	(4.4)	21.6	(2.6)	27.4	(2.8)	28.5	(0.5)
50–59 years	17.8	(4.3)	14.6	(2.2)	22.8	(2.8)	24.2	(0.6)
<u>Educational attainment</u>								
Less than high school	7.1	(1.9)	18.5	(3.0)	12.6	(1.6)	16.0	(0.6)
High school degree	12.0	(2.6)	23.2	(2.4)	17.2	(2.5)	24.5	(0.6)
Some college	33.8	(4.7)	37.8	(3.2)	43.2	(3.2)	32.3	(0.6)
College degree	47.2	(5.3)	20.5	(3.0)	27.0	(2.4)	27.2	(0.9)
<u>Race/ethnicity</u>								
Non-Hispanic White	73.4	(3.6)	72.3	(2.9)	72.1	(2.6)	69.6	(1.4)
Hispanic	9.6	(1.8)	10.6	(1.7)	11.0	(1.4)	14.1	(1.1)
Non-Hispanic Black	9.8	(1.7)	14.2	(1.9)	11.2	(1.4)	11.0	(0.8)
Non-Hispanic other/multiracial	7.1	(2.2)	2.9	(1.0)	5.7	(1.2)	5.3	(0.4)
<u>Family income as percent of federal poverty level (FPL) *</u>								
Below FPL	10.3	(1.9)	24.2	(2.9)	15.3	(1.9)	13.4	(0.5)
100–199% of FPL	16.4	(2.6)	22.2	(2.4)	20.3	(1.9)	18.1	(0.5)
200–299% of FPL	15.0	(3.0)	20.6	(2.9)	15.1	(2.4)	14.3	(0.5)
300–300% of FPL	11.4	(2.4)	10.4	(2.1)	13.5	(2.0)	14.8	(0.5)
400% or more of FPL	46.8	(5.6)	22.6	(3.0)	35.8	(3.2)	39.3	(1.0)
Foreign birth *	10.7	(2.4)	8.0	(1.6)	10.5	(1.5)	16.2	(1.0)

Note. N = 13,959. Percentages sum to 100% except for rounding error. Statistical significance evaluated by multinomial regression regressing sexual orientation status on all demographic characteristics and survey cycle considered simultaneously. SE = Standard error; NHANES = National Health Nutrition and Examination Survey.

*
p < 0.05

**
p < 0.001.

Health indicators and markers of allostatic load among U.S. adults, age 20 to 59 years, by gender and sexual orientation: Weighted estimates and standard errors shown, NHANES (2001–2010)

TABLE 2.

Health indicators, %	Women				Men			
	Gay (n = 87)	Bisexual (n = 201)	Homosexually experienced (n = 272)	Heterosexual (n = 6,230)	Gay (n = 124)	Bisexual (n = 106)	Homosexually experienced (n = 152)	Heterosexual (n = 6,739)
	% (SE)	% (SE)	% (SE)	% (SE)	% (SE)	% (SE)	% (SE)	% (SE)
Currently insured	70.6 (5.1)	67.4 (3.8)	80.0 (2.7)	81.0 (0.8)	88.6 (2.9)	65.8 (4.9)	70.5 (3.9)	74.5 (0.7)
Lack of exercise	38.4 (5.3)	37.5 (2.9)	39.0 (4.0)	36.4 (0.9)	19.6 (4.2)	38.1 (5.5)	32.4 (4.5)	34.3 (1.1)
Current smoker	37.5 (5.9)	46.1 (3.4)	42.8 (3.7)	23.0 (0.8)	29.8 (5.1)	40.2 (6.1)	30.5 (4.5)	29.5 (0.8)
Frequent mental distress	13.0 (3.6)	30.0 (3.5)	20.2 (3.1)	14.8 (0.5)	12.4 (3.4)	23.7 (4.3)	18.5 (3.5)	9.3 (0.4)
Weekly binge drinking	10.9 (3.3)	11.9 (2.3)	9.2 (2.1)	3.3 (0.2)	3.4 (1.7)	23.2 (5.5)	16.0 (3.3)	16.5 (0.7)
Prevalent HIV infection ¹					14.7 (3.8)	6.7 (2.1)	0.7 (0.7)	0.2 (0.1)
Allostatic load indicators, %								
Albumin < 3.8 g/dL	2.0 (1.2)	6.2 (2.0)	7.2 (1.7)	7.5 (0.4)	1.7 (1.2)	2.5 (2.0)	1.1 (0.6)	1.5 (0.2)
C-reactive protein > 0.3 mg/dL	41.0 (6.2)	45.2 (4.1)	38.8 (3.2)	40.0 (0.9)	18.4 (4.3)	36.5 (6.7)	20.2 (4.0)	25.5 (0.7)
BMI 30 kg/m ²	41.3 (5.3)	44.5 (4.8)	29.0 (3.0)	34.5 (0.8)	24.4 (4.3)	38.6 (5.2)	27.8 (4.0)	32.0 (0.8)
Total cholesterol 240 mg/dL	10.1 (3.2)	13.3 (3.2)	17.1 (2.4)	13.4 (0.6)	13.6 (3.5)	13.1 (3.8)	19.7 (3.2)	15.5 (0.6)
HDL cholesterol < 40 mg/dL	15.1 (3.8)	16.1 (2.9)	13.3 (3.2)	10.6 (0.5)	32.5 (4.8)	36.3 (6.2)	26.1 (4.0)	30.2 (0.7)
Glycosolated hemoglobin 6.4%	6.3 (4.7)	3.4 (1.7)	5.8 (1.8)	4.6 (0.3)	0.6 (0.4)	16.4 (4.8)	4.7 (2.0)	5.3 (0.4)
Resting heart rate 90 beats/min	11.6 (3.5)	14.8 (3.2)	13.6 (3.0)	11.1 (0.4)	7.7 (2.6)	14.4 (4.4)	11.9 (3.5)	7.9 (0.4)
Systolic blood pressure 140mm	11.0 (4.2)	5.8 (2.2)	4.1 (1.3)	7.6 (0.4)	2.2 (1.3)	17.6 (4.7)	9.2 (3.0)	9.0 (0.4)
Diastolic blood pressure 90mm	4.9 (2.9)	3.7 (1.8)	2.0 (1.1)	3.8 (0.3)	3.9 (1.5)	8.1 (3.4)	2.8 (1.2)	7.5 (0.4)
Allostatic load, ² mean	1.62 (0.22)	1.65 (0.14)	1.53 (0.13)	1.63 (0.03)	1.22 (0.13)	2.13 (0.22)	1.54 (0.15)	1.62 (0.03)

Note. N = 13,959. Percentages sum to 100% except for rounding error. Numbers in parentheses are standard errors; NHANES = National Health Nutrition and Examination Survey.

¹ Among men age 20–49 years only. Females not shown due to rarity of HIV infection among sexual minority women; sample includes gay, bisexual men, homosexually experienced, and exclusively heterosexual men only.

² Allostatic load estimated as a sum of biologic indicators scored positive.

TABLE 3.

Sexual orientation differences in health indicators and markers of allostatic load among U.S. adults, age 20 to 59 years, by gender: Adjusted odds ratios and 95% confidence intervals shown, NHANES (2001–2010)

Health status	Women			Men		
	Gay	Bisexual	Homosexually experienced	Gay	Bisexual	Homosexually experienced
<u>Health indicators</u>						
Currently insured	0.57 (0.33–0.97)	0.61 (0.41–0.92)	0.92 (0.64–1.32)	1.50 (0.80–2.84)	0.76 (0.42–1.34)	0.70 (0.41–1.21)
Lack of exercise	1.15 (0.90–1.49)	0.96 (0.74–1.26)	1.19 (0.83–1.70)	0.80 (0.45–1.40)	1.07 (0.68–1.69)	0.92 (0.62–1.34)
Current smoker	1.91 (1.17–3.14)	2.18 (1.56–3.04)	2.56 (1.82–3.62)	1.82 (1.03–3.08)	1.52 (0.91–2.55)	1.20 (0.78–1.85)
Frequent mental distress	0.80 (0.42–1.54)	2.16 (1.52–3.08)	1.38 (0.90–2.12)	1.90 (0.98–3.67)	2.79 (1.76–4.44)	2.26 (1.40–3.65)
Weekly binge drinking	3.19 (1.54–6.61)	2.58 (1.68–3.96)	2.50 (1.41–4.42)	0.25 (0.09–0.72)	1.50 (0.78–2.87)	1.07 (0.66–1.75)
Prevalent HIV infection (age 20–49 men only)*				395.38 (144.01–1085.51)	47.66 (18.46–123.09)	3.16 (0.34–29.42)
<u>Allostatic load indicators</u>						
Albumin < 3.8 g/dL	0.22 (0.07–0.75)	0.74 (0.36–1.55)	0.91 (0.53–1.56)	1.36 (0.35–5.25)	1.31 (0.24–7.25)	0.66 (0.23–1.89)
C-reactive protein > 0.3 mg/dL	1.03 (0.60–1.74)	1.22 (0.86–1.72)	0.98 (0.75–1.28)	0.77 (0.43–1.39)	1.59 (0.85–2.95)	0.69 (0.41–1.16)
BMI 30 kg/m ²	1.34 (0.82–2.19)	1.51 (1.01–2.26)	0.76 (0.57–1.02)	0.71 (0.44–1.15)	1.32 (0.85–2.05)	0.75 (0.50–1.12)
Total cholesterol 240 mg/dL,	0.79 (0.36–1.69)	1.35 (0.75–2.43)	1.48 (1.01–2.19)	0.85 (0.46–1.57)	0.81 (0.42–1.60)	1.27 (0.84–1.92)
HDL cholesterol < 40 mg/dL	1.43 (0.78–2.61)	1.27 (0.83–1.94)	1.25 (0.73–2.13)	1.33 (0.84–2.10)	1.30 (0.75–2.26)	0.81 (0.53–1.24)
Glycosylated hemoglobin 6.4%	1.75 (0.41–7.46)	1.27 (0.40–4.02)	1.58 (0.76–3.30)	0.13 (0.26–0.66)	3.51 (1.46–7.92)	0.75 (0.30–1.90)
Resting heart rate 90 beats/min	0.96 (0.49–1.88)	1.07 (0.63–1.81)	1.12 (0.69–1.82)	1.20 (0.58–2.50)	1.80 (0.86–3.78)	1.54 (0.79–3.01)
Systolic blood pressure 140mm	1.83 (0.74–4.51)	1.38 (0.54–3.50)	0.57 (0.29–1.14)	0.24 (0.07–0.86)	2.07 (1.02–4.18)	0.92 (0.44–1.92)
Diastolic blood pressure 90mm	1.33 (0.41–4.32)	1.36 (0.46–3.98)	0.58 (0.19–1.74)	0.36 (0.12–1.02)	1.02 (0.42–2.46)	0.31 (0.13–0.74)

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Note. N = 13,959. Numbers in parentheses are confidence intervals; NHANES = National Health Nutrition and Examination Survey. Sexual orientation differences estimated by logistic regression, adjusted for age, race/ethnicity, family income, educational attainment, foreign birth, and insurance status.

¹Referent is exclusive heterosexuals.

TABLE 4. Predictors of allostatic load among U.S. adults age 20 to 59 years, by gender, NHANES (2001–2010): Partial results shown

	Women		Men			
	Model 1 Adj β (CI)	Model 2 Adj β (CI)	Total sample Model 1 Adj β (CI)	Model 2 Adj β (CI)	Age 20–49 only Model 1 Adj β (CI)	Model 3 Adj β (CI)
<u>Sexual orientation</u> ¹						
Gay/lesbian	0.00 (-0.20,0.21)	0.02 (-0.19,0.23)	-0.21 (-0.42,-0.01)	-0.22 (-0.41,-0.04)	-0.19 (-0.43,0.06)	-0.21 (-0.45,0.02)
Bisexual	0.10 (-0.08,0.29)	0.11 (-0.08,0.30)	0.25 (0.05,0.45)	0.25 (0.05,0.44)	0.30 (0.06,0.54)	0.29 (0.04,0.53)
Homosexually experienced	-0.03 (-0.19,0.13)	-0.03 (-0.19,0.12)	-0.12 (-0.31,0.07)	-0.12 (-0.30,0.06)	-0.20 (-0.43,0.04)	-0.20 (-0.43,0.04)
<u>Health indicators</u>						
Currently insured		0.17 (0.09,0.25)		0.16 (0.09,0.23)		
Current smoker		0.01 (-0.05,0.07)		-0.05 (-0.11,0.01)		
Weekly binge drinking		-0.10 (-0.28,0.08)		-0.03 (-0.10,0.04)		
Frequent mental distress		0.11 (0.06,0.18)		0.13 (0.05,0.21)		
Lack of exercise		0.20 (0.14,0.26)		0.21 (0.15,0.28)		
HIV infection						0.15 (-0.25,0.55)

Note. N = 13,959. Allostatic load estimated as a sum of biologic indicators scored positive. Numbers in parentheses are confidence intervals (CI); NHANES = National Health Nutrition and Examination Survey. Sexual orientation differences estimated by negative binomial regression. Model 1 adjusted for age, race/ethnicity, family income, educational attainment, foreign birth, and survey cycle. Model 2 additionally adjusted for health indicators shown. Model 3 adjusted for HIV infection status, in addition to Model 1 confounders.

¹Referent is exclusive heterosexuals.