

# Stressful Life Events, Sexual Orientation, and Cardiometabolic Risk Among Young Adults in the United States

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**Objective:** The goal of the present study was to examine whether sexual minority young adults are more vulnerable to developing cardiometabolic risk following exposure to stressful life events than heterosexual young adults. **Method:** Data came from the National Longitudinal Study for Adolescent Health (Shin, Edwards, & Heeren, 2009; Brummett et al., 2013), a prospective nationally representative study of U.S. adolescents followed into young adulthood. A total of 306 lesbian, gay, and bisexual (LGB) respondents and 6,667 heterosexual respondents met inclusion criteria for this analysis. Measures of cumulative stressful life events were drawn from all 4 waves of data collection; sexual orientation and cardiometabolic biomarkers were assessed at Wave 4 (2008–2009). **Results:** Gay/bisexual men exposed to 1–2 ( $\beta = 0.71, p = .01$ ) and 5+ ( $\beta = 0.87, p = .01$ ) stressful life events had a statistically significant elevation in cardiometabolic risk, controlling for demographics, health behaviors, and socioeconomic status. Moreover, in models adjusted for all covariates, lesbian/bisexual ( $\beta = 0.52, p = .046$ ) women with 5+ stressful life events had a statistically significant elevation in cardiometabolic risk. There was no relationship between stressful life events and cardiometabolic risk among heterosexual men or women. **Conclusion:** Stressful life events during childhood, adolescence, and young adulthood place LGB young adults at heightened risk for elevated cardiometabolic risk as early as young adulthood. The mechanisms underlying this relationship require future study.

**Keywords:** sexual orientation, cardiometabolic biomarkers, stressful life events, young adults

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Exposure to a wide range of stressful life events—including high job strain (Bosma, Peter, Siegrist, & Marmot, 1998), caregiving for an ill family member (Lee, Colditz, Berkman, & Kawachi, 2003), loss of a loved one (Kaprio, Koskenvuo, & Rita, 1987), exposure to a life-threatening traumatic event (Kark, Goldman, & Epstein, 1995), and childhood adversities (Slopen, Kubzansky, McLaughlin, & Koenen, 2013)—has been consistently linked to risk for cardiovascular disease (CVD). Despite numerous studies documenting these relationships, several important gaps in the literature remain. First, the degree to which associations between stressful life events and CVD risk are consistent across the life

course is largely unknown. Many behavioral (e.g., smoking, diet) and social (e.g., childhood adversity) risk factors for adult CVD emerge early in development (e.g., Adair & Dahly, 2005), suggesting that relationships between stressful life events and CVD risk might be evident in young adulthood. Scant research has examined this possibility. Indeed, the vast majority of work examining stressful life events and CVD risk has been conducted among middle-aged and older adults (Everson-Rose & Lewis, 2005). Second, the relationship between stressors and CVD risk is likely to vary across sociodemographic groups. For instance, psychosocial stressors, such as marital stress, are more strongly associated with CVD outcomes among women compared with men (Iso et al., 2002; Orth-Gomér et al., 2000). Further, the association between stressful events and subclinical CVD risk also varies by race/ethnicity (Slopen et al., 2010; Troxel, Matthews, Bromberger, & Sutton-Tyrrell, 2003). Although there is emerging evidence for sexual orientation disparities in CVD-related outcomes (Cochran & Mays, 2007; Conron, Mimiaga, & Landers, 2010; Everett & Mollborn, 2013; Hatzenbuehler, McLaughlin, & Slopen, 2013), we are unaware of studies that have examined whether the relationship between stressful life events and CVD risk varies by sexual orientation. For the current study, we sought to address these gaps in the literature.

Differential vulnerability models (Nolen-Hoeksema & Girgus, 1994) provide a potential framework for understanding how sexual

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orientation could moderate the relationship between stressful life events and CVD risk. Specifically, differential vulnerability models posit that members of certain social groups are rendered more vulnerable to the negative effects of stressful life events because they have a lower threshold for developing adverse reactions to these events (Nolen-Hoeksema & Girgus, 1994). These preexisting vulnerabilities, in turn, are exacerbated by the heightened degree of stressful life events that socially disadvantaged groups experience compared with more advantaged groups.

Why might lesbian, gay, and bisexual (LGB) individuals be more vulnerable to the negative health consequences of stressful life events than heterosexuals? Managing a stigmatized identity disrupts development of a variety of cognitive (e.g., negative self-schemas), emotion-regulation (e.g., maladaptive coping such as rumination and suppression), and neurobiological (e.g., hypothalamic–pituitary–adrenal axis-functioning) processes (Hatzenbuehler, 2009; Hatzenbuehler & McLaughlin, 2014; Inzlicht, McKay, & Aronson, 2006; Major & O'Brien, 2005). In turn, these biopsychosocial processes affect future susceptibility to poor health, including CVD risk (Everson-Rose & Lewis, 2005; Miller, Chen, & Cole, 2009; Repetti, Taylor, & Seeman, 2002). Differential vulnerability might also emerge due to differences in the developmental timing of exposure to stressors. Individuals who have been exposed to early life stressors are more likely to develop depression (Hammen, Henry, & Daley, 2000) and posttraumatic stress disorder (McLaughlin, Conron, Koenen, & Gilman, 2010) following exposure to stressors in adulthood than individuals who have not experienced early life stressors. Multiple studies have documented that LGB adolescents and young adults are more likely than their heterosexual peers to be victimized (Bontempo & D'Augelli, 2002), and to experience childhood maltreatment and homelessness (McLaughlin, Hatzenbuehler, Xuan, & Conron, 2012). This differential exposure might be directly linked to greater vulnerability to subsequent stressful life events.

Based on differential vulnerability models (Nolen-Hoeksema & Girgus, 1994; Hammen et al., 2000), we hypothesized that stressful life events would be more strongly associated with cardiometabolic risk among LGB young adults compared with their heterosexual peers. In particular, for the reasons stated above, LGB young adults were expected to have more preexisting characteristics that render them more vulnerable to CVD risk; when these characteristics interact with stressful life events, LGB young adults may be more likely to develop cardiometabolic risk than heterosexuals. We examined this question using a cumulative CVD risk score, designed to characterize overall functioning across multiple measures of cardiovascular activity (Seeman et al., 2004). We opted to use a measure of cumulative CVD risk rather than examine individual biomarkers in light of allostatic load research, which suggests that adverse risk factors across multiple biological systems predict morbidity and mortality risk better than individual components (Poulter, 2003; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). There is limited knowledge about the relationship between stressful life events and CVD risk in younger populations, particularly using a cumulative cardiometabolic risk score. Consequently, the study makes several unique contributions to the literature on stressful life events and CVD risk. We examined our research questions using the only existing data set (from the National Longitudinal Study of Adolescent Health; Add Health; Shin, Edwards, & Heeren, 2009; Brummett et al., 2013)

with a representative sample of young adults that simultaneously measured sexual orientation, stressful life events over multiple waves, cardiometabolic biomarkers, and established CVD risk factors. This data set therefore offered us a rare opportunity to address the role of stressful life events in cardiometabolic risk among both LGB and heterosexual young adults in the United States.

## Method

Data were drawn from Add Health (Shin, Edwards, & Heeren, 2009; Brummett et al., 2013), an ongoing nationally representative study of adolescents and young adults. Add Health researchers recruited a school-based sample of adolescents in Grades 7 through 12 in 1994 and have followed respondents into young adulthood. To date, there have been four waves of data collection. Wave 1 (1994–1995) utilized a multistage sampling design to enroll adolescents. A systematic random sample of 80 high schools was selected proportional to enrollment size and stratified by region, urbanicity, school type, and percentage of White students; the largest feeder school for each high school was also invited to participate. A total of 134 schools (79%) participated. An in-school survey was completed by 90,118 students, and 20,745 students participated in a more detailed in-home interview (75.6% and 79.5% of eligible students, respectively). Adolescents in the Wave-1, home-interview subsample were contacted to complete additional in-home interviews at Wave 2 in 1996 ( $N = 14,738$ ; 88.2% response rate), Wave 3 in 2001–2002 ( $N = 15,197$ ; 76.0% response rate), and Wave 4 in 2008–2009 ( $N = 15,701$ ; 80.25% response rate). The current study utilized data on stressful life events from all four waves (described below) and information on cardiometabolic biomarkers was obtained at Wave 4 when participants were 24 to 32 years of age. Details about Add Health have been described previously and can be found at (<http://www.cpc.unc.edu/projects/addhealth/design>).

To be included in our analyses, we required that respondents: (a) participate in all four waves of Add Health, (b) had complete data for all components of the cardiometabolic risk score, (c) had at least one complete measure for each component of the stressful life events inventory, (d) had complete data on all covariates, and (e) had complete data on sexual orientation at Wave 4. We excluded respondents who (a) did not have information on sample weights, (b) reported having HIV/AIDS or a Hepatitis-C infection, or (c) were pregnant at Wave 4; these latter two factors might have affected the components of the cardiometabolic risk score. As described below, we also omitted individuals who identified as “mostly heterosexual” or who reported that they were neither attracted to boys/men nor girls/women. Those who were excluded (see Appendix 1 of the online supplemental materials) were more likely to be female, older, non-White, to binge drink less often, and to have a slightly higher mean number of “high-risk” cardiometabolic biomarkers (1.34 vs. 1.28,  $p = .04$ ).

There were 9,422 respondents who were present in all four waves of data collection. Of these respondents, 7,821 provided complete data on the predictor (stressful life events) and components of the cardiometabolic risk score (six biomarkers), were not pregnant, and did not self-report HIV or Hepatitis-C infection. After further excluding participants who identified as “mostly heterosexual” ( $n = 776$ ) or asexual ( $n = 19$ ; see below), and

further excluding individuals with missing data on any of the covariates ( $n = 146$ ), the final analytic sample included 6,973 respondents (306 LGB; 6,667 heterosexual). The mean age of the final analytic sample was 28.54 years ( $SE = 0.12$ ); on average, they were 15.53 years old ( $SE = 0.12$ ) when they entered the study and had been in the study for 13 years ( $SE = 0.01$ ).

## Measures

**Sexual orientation.** Self-identified sexual orientation was assessed at Wave 4 with an item asking respondents to “Please choose the description that best fits how you think about yourself.” Six response options were given (numbers provided correspond to the final sample who met the above inclusion criteria): 100% heterosexual (straight;  $n = 6,667$ ); mostly heterosexual but somewhat attracted to people of their own sex (some attraction;  $n = 776$ ); bisexual ( $n = 121$ ); mostly homosexual, but somewhat attracted to people of the opposite sex ( $n = 73$ ); 100% homosexual ( $n = 112$ ); and not sexually attracted to either males or females ( $n = 19$ ). Due to the small sample size of LGB individuals, we present results aggregated across lesbian, gay, and bisexual respondents ( $n = 306$ ). Because studies on sexual orientation disparities in cardiometabolic biomarkers have not included a “mostly heterosexual” or asexual groups (Hatzenbuehler et al., 2013), we did not have an a priori hypothesis about these groups, and therefore omitted them from analyses.

**Stressful life events.** Table 1 depicts the list of 19 stressful life events that were included in the current analyses and the waves in which they were assessed. We created an additive index to measure cumulative exposure to a wide range of stressful life events across all four waves of Add Health, based on previous studies on stressful life events in the Add Health sample (Adkins, Wang, & Elder, 2009) and on prior research examining stressful life events in LGB adolescents (e.g., McLaughlin et al., 2012). As shown in Table 1, some stressful life events were assessed more than once; other stressors were assessed only one time. Reliability

of the stressful life events measure is suggested by the fact that the majority of respondents consistently answered the subset of stressful life events that were assessed more than once and that used the same screening items across waves. For instance, among individuals who reported that they had ever spent time in jail at Wave 3, 92.5% reported having spent time in jail at Wave 4.

We created a single indicator variable for each of the 19 stressful life events; each indicator reflected the positive endorsement of the particular event at any time point. The 19 indicator variables were summed to create the stressful life-events score. For the present study, we chose to combine events across all four waves of Add Health because we were interested in cumulative exposure to stressful life events in relation to cardiometabolic dysfunction, and because we did not have specific hypotheses with regard to differing associations for recent versus distal events. If a respondent had missing data on a stressful life event that was asked about at more than one wave, and he or she had complete information at another wave, he or she was retained in the sample. Based on the distribution, stressful life events were examined as an ordinal variable, with the following groups: 0 events, 1–2 events, 3–4 events, 5 or more (5+) events (results were similar when stressful life events were examined as a continuous measure).

**Cardiometabolic risk score.** Several cardiovascular biomarkers were collected from Wave-4 respondents, including systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse rate, C-reactive protein (CRP), glycosylated hemoglobin (Hb1Ac), and waist circumference (WC).

SBP and DBP were measured using an oscillometric blood-pressure (BP) monitor with an appropriately sized cuff placed on the right upper arm. Three BP measurements were taken, separated by 30-s intervals. SBP and DBP values represent the average of the second and third measurements (in mmHg). The BP monitor also provided information on pulse rate at each of the three measure-

Table 1  
Timing of Items in the Stressful Life Events Index

Item	Wave 1	Wave 2	Wave 3	Wave 4
1. Childhood physical abuse (before 18)				✓
2. Childhood sexual abuse (before 18)				✓
3. Expelled from school (Waves 1 and 2: current school year; Wave 3: ever)	✓	✓	✓	
4. Lived in foster home (ever)			✓	
5. Kicked out of home (ever)			✓	
6. Homeless (ever)			✓	
7. Friend committed suicide (past 12 months)	✓	✓	✓	
8. Relative committed suicide (past 12 months)	✓	✓	✓	
9. Witnessed violence (past 12 months)	✓	✓	✓	
10. Threatened by knife or gun (past 12 months)	✓	✓	✓	✓
11. Shot or stabbed (past 12 months)	✓	✓	✓	✓
12. Death of a parent (ever)	✓	✓	✓	✓
13. Criminal conviction (ever)			✓	✓
14. Served time in jail or detention (ever)			✓	✓
15. Parent incarcerated (ever)				✓
16. Physical intimate partner violence (ever) <sup>a</sup>		✓	✓	✓
17. Sexual intimate partner violence (ever) <sup>a</sup>			✓	✓
18. Physical forced sex (ever)				✓
19. Non-physical forced sex (ever)				✓

<sup>a</sup> At Wave 3, physical and sexual intimate-partner violence questions were only administered to individuals who reported an intimate relationship.

ments. Pulse-rate values represented the average of the second and third measurements in beats per min (bpm).

Blood-spot samples were obtained using a finger prick and were submitted for laboratory analysis of high-sensitivity CRP (mg/L), a marker of systemic inflammation, tissue damage, and infection, and Hb1Ac (%), a measure of long-term glycemic control, reflecting average blood glucose over the preceding 8–12 weeks (American Diabetes Association, 2011). Following a standard protocol, trained interviewers collected blood spots on standardized filter paper using a sterile disposable lancet. Blood spots were dried overnight and then sealed at  $-70^{\circ}\text{C}$  in Ziploc bags until laboratory analysis. CRP was assayed from blood spots using a highly sensitive standardized enzyme-immunoassay protocol. Previous validation studies have indicated high correlations between CRP values from blood-serum and blood-spot samples (McDade, Burhop, & Dohnal, 2004), and recent analyses from the Add Health sample indicated that the correlation (Pearson  $r$ ) between dried blood spots and plasma was 0.98 (Brummett et al., 2013).

Blood spots were assayed for Hb1Ac using an immunoturbidimetric method for HbA1c quantitation and a colorimetric method for released hemoglobin (Hb) quantitation. HbA1c was calculated based on the HbA1c:Hb ratio using the formula  $\text{HbA1c (\%)} = 2.27 + 87.6 \times (\text{HbA1c} \div \text{Hb})$  (Rohlfing et al., 2002). A validation study compared whole blood values of HbA1c with paired dried blood spots from 115 Wave-4 respondents. Blood-spot values and a conventional HbA1c assay were strongly associated ( $r = .99$ ,  $p < .001$ ).

Finally, WC was measured using a SECA 200 metric-increment circumference tape measure (Seca Corp., Hanover, MD). Field interviewers measured WC to the nearest 500 cm at the superior border of the iliac crest for all respondents capable of standing unassisted.

A cardiometabolic risk score based on these six cardiometabolic biomarkers was created using two strategies that represented the concept of allostatic load (e.g., Seeman et al., 2004). First, for the primary analyses, we used the continuous values of each marker to construct the cumulative biological risk scores applying a modification of methods conducted in prior research (Karlmanjla, Singer, & Seeman, 2006). Specifically, we created sex-standardized  $z$  scores for each marker, summed the markers, and then restandardized the resulting values to  $z$  scores. Second, for the sensitivity analyses, we created a cumulative biological risk score by counting the number of biological markers that met a clinically defined high-risk criterion (King, Morenoff, & House, 2011). The criterion for “high risk” was defined as: (a) SBP of 140 mmHg or higher (Chobanian et al., 2003); (b) DBP of 90 mmHg or higher (Chobanian et al., 2003); (c) resting pulse rate of 90 bpm or more (Chobanian et al., 2003); (d) HbA1c of 6.4% or higher (Osei, Rhinesmith, Gaillard, & Schuster, 2003); (e) CRP of 3 mg/DL or higher (Ridker, 2003); and (f) WC of more than 102 cm for men and 88 cm for women (Guagnano et al., 2001). Individuals received a value of 1 if they were above the threshold of risk (range: 0–6).

**Covariates.** Three sets of covariates, including demographics, socioeconomic status (SES), and health behaviors were chosen because prior studies have shown that these characteristics are robustly associated with cardiometabolic risk (Hubert, Feinleib, McNamara, & Castelli, 1983; Neaton & Wentworth, 1992; Roerecke, & Rehm, 2010; Thompson, 2003) and are an established

set of covariates that are commonly used in research on cardio-metabolic risk scores (e.g., King et al., 2011). Consistent with previous literature (Kubzansky, Koenen, Jones, & Eaton, 2009; Kubzansky, Koenen, Spiro, Vokonas, & Sparrow, 2007), we used measures of all covariates at the most recent time point (i.e., Wave 4).

Demographic controls included age (continuous measure), race/ethnicity (White vs. non-White), and nativity status (foreign-born vs. not, derived from the question “Were you born a U.S. citizen?”). SES indicators included annual household income ( $\leq \$39,999$  vs.  $\geq \$40,000$ ) and educational attainment (less than or equal to a high school degree vs. more than a high school degree). Controls for health behaviors included physical activity (reporting 5+ bouts of moderate to vigorous physical activity in the past week vs. not (Ornelas, Perreira, & Ayala, 2007), cigarette smoking, and binge drinking. Smoking was categorized as a three-level variable: current (daily smoking for the past 30 days); intermittent or previous (smoking on 1–29 of the past 30 days or previous regular smoking); and none (Hatzenbuehler et al., 2013). Past-year binge drinking was coded present for respondents who reported more than two episodes per month of drinking 5+ drinks (for men) or 4+ drinks (for women) in a single sitting (Shin, Edwards, & Heeren, 2009).

For sensitivity analyses, we controlled for a dichotomous indicator of the presence of self-reported illness in the past 2 weeks—including cold or flu symptoms, fever, nausea, vomiting, diarrhea, night sweats, blood in stool or urine, frequent urination, or skin rash—which could have affected the cardiometabolic risk score (Hatzenbuehler et al., 2013).

## Analysis Plan

Analyses proceeded in two steps. First, analyses examined sexual orientation differences in exposure to stressful life events, stratified by sex. To address this aim, we conducted  $\chi^2$  tests for categorical variables (i.e., the categories for number of stressful life events) and an analysis of variance (ANOVA) test for the continuous variable (i.e., mean cumulative score of stressful life events). Second, we examined the associations between stressful life events (entered as a categorical variable) and the biological risk score (entered as a continuous  $z$  score) in a series of progressive models adjusting for potential confounders, stratified by sexual orientation status and sex, after previous research showing sex differences in measured cardiometabolic biomarkers based on sexual orientation (e.g., Everett & Mollborn, 2013; Hatzenbuehler et al., 2013). Model 1 showed the unadjusted relationships between stressful life events and the cardiometabolic risk score. Model 2 examined the age-adjusted association, controlling for race/ethnicity and nativity. Models 3 and 4 added controls for SES and health behaviors (smoking status, past-year binge drinking, and low physical activity), respectively. The final model included all controls simultaneously.

As noted above, the primary analyses yielded a continuous outcome, and in sensitivity analyses we evaluated a count outcome. The three-way interaction between stressful life events, sexual orientation, and sex was not statistically significant for the continuous measure ( $p = .21$ ); however, this interaction was statistically significant for the count outcome ( $p < .01$ ), suggesting that a 3-way interaction was present on a multiplicative scale, but

not on an additive scale (Bauer, 2014). Given that tests of interaction may be statistically underpowered in smaller subsets of participants (Conron et al., 2010), we proceeded to estimate sex-stratified models for all analyses, consistent with previous work on sexual orientation and health in population-based samples (e.g., Bostwick, Boyd, Hughes, & McCabe, 2010; Cochran & Mays, 2007; Sandfort, de Graaf, Bijl, & Schnabel, 2001). In sex-stratified models predicting the continuous outcome, the two-way interaction between sexual orientation and stressful life events was statistically significant for both men ( $p = .03$ ) and women ( $p = .04$ ). These interaction tests provided evidence that among both men and women, the relationship between stressful life events and cardiometabolic risk differs by sexual orientation.

In all analyses, we applied poststratification weights to adjust for selection probabilities and nonresponse, account for the complex sample design, and generate nationally representative estimates of association. Analyses were completed using SAS 9.2 and SUDAAN 10.0.1.

## Results

### Descriptive Statistics

Table 2 summarizes the descriptive statistics for cardiometabolic biomarkers and study covariates, stratified by sexual orientation and sex. Mean levels of the cardiometabolic risk score did not differ between LGB and heterosexuals. There were no sexual orientation differences in the demographic variables (i.e., age, race, or nativity). However, group differences were observed in SES among the women; lesbian/bisexual women had lower educational attainment and household income than heterosexual women. No sexual orientation

differences in SES were observed among the men. For the health behaviors, heterosexual men were more likely to report low physical activity than gay/bisexual men. Among women, lesbian/bisexual women were more likely to smoke and to report past-year binge drinking than were heterosexual women.

Table 3 depicts the prevalence of stressful life events stratified by sexual orientation status and sex. Neither the mean number of stressful life events nor the distribution of these events differed significantly between heterosexual and gay/bisexual men ( $p$  values = .92 and  $p = .72$ , respectively). In contrast, the mean number of stressful life events was significantly higher ( $p < .0001$ ) among lesbian/bisexual women than among heterosexual women (3.45 vs. 2.26, respectively). Related, the distribution of stressful life events also differed between lesbian/bisexual and heterosexual women ( $p < .001$ ); for example, heterosexual women were more likely to report no stressors than were lesbian/bisexual women (23.98% vs. 15.34%, respectively), and lesbian/bisexual women were more likely to report five or more stressors than were heterosexual women (32.31% vs. 14.54%, respectively).

### Associations Between Stressful Life Events and Cardiometabolic Risk

Stressful life events were robustly associated with the cardiometabolic risk score among gay/bisexual men (see Table 4). Across all models, a greater number of stressful life events predicted elevated cardiometabolic risk scores for gay/bisexual men, and this was evident for each level of stressful life events examined (i.e., 1–2, 3–4, and 5+). In the final model that controlled for all covariates simultaneously, gay/bisexual men with 1–2 ( $\beta =$

Table 2

Weighted Sample Characteristics by Sexual Orientation Status: National Longitudinal Study of Adolescent Health ( $N = 6,973$ )<sup>a</sup>

Characteristic	Males ( $N = 3,415$ )			Females ( $N = 3,558$ )		
	Gay or bisexual ( $n = 117$ ) % (SE) or mean (SE)	Heterosexual ( $n = 3,298$ ) % (SE) or mean (SE)	$p$ value <sup>a</sup>	Lesbian or bisexual ( $n = 189$ ) % (SE) or mean (SE)	Heterosexual ( $n = 3,369$ ) % (SE) or mean (SE)	$p$ value <sup>a</sup>
Cardiometabolic risk $z$ score (mean)	-0.02 (0.13)	0.00 (0.03)	0.89	0.14 (0.08)	0.00 (0.03)	0.11
Number of cardiometabolic biomarkers > "high risk" values	1.32 (0.20)	1.15 (0.03)	0.40	1.48 (0.11)	1.41 (0.03)	0.51
Age (mean)	28.72 (0.23)	28.59 (0.12)	0.53	28.38 (0.18)	28.49 (0.12)	0.45
Race (% White)	68.04 (6.27)	68.30 (2.86)	0.96	70.17 (4.83)	65.93 (3.26)	0.37
Nativity (% born outside of U.S.)	6.48 (3.52)	4.44 (0.86)	0.46	2.39 (1.30)	4.92 (0.98)	0.15
Education (% with high school degree or less)	20.52 (5.27)	30.98 (1.85)	0.08	29.51 (4.19)	20.57 (1.33)	0.02
Household income (%)						
Missing	4.47 (2.63)	6.69 (0.74)	0.48	7.49 (2.78)	6.72 (0.69)	0.01
Low ( $\leq$ \$39,999)	34.58 (5.43)	27.87 (1.47)		47.19 (5.41)	33.53 (1.59)	
High ( $\geq$ \$40,000)	60.95 (6.39)	65.45 (1.64)		45.31 (4.79)	59.75 (1.61)	
Smoking status (%)						
Current	24.49 (4.61)	27.11 (1.29)	0.63	31.60 (5.35)	20.62 (1.36)	0.0002
Past/intermittent	28.37 (5.15)	23.41 (0.98)		31.70 (5.04)	20.05 (1.09)	
Never	47.14 (7.09)	49.47 (1.52)		36.70 (4.66)	59.33 (1.72)	
Past-year binge drinking (%) <sup>b</sup>	23.32 (5.33)	29.26 (1.25)	0.31	31.75 (4.3)	13.58 (0.86)	<0.0001
Low physical activity (%) <sup>c</sup>	29.63 (6.29)	43.33 (1.43)	0.05	53.73 (4.99)	51.47 (1.30)	0.67

<sup>a</sup> Table presents weighted means and percents and standard errors, taking into account the complex sample design;  $p$  values are derived from chi-square tests for categorical variables and an analysis of variance test for the cardiometabolic risk scores and age (i.e. the only continuous variables). <sup>b</sup> Binge drinking was defined as consuming four (female) or five (males) or more drinks in a row at least 2–3 times per month in the past year. <sup>c</sup> Low physical activity was defined as less than 5 bouts of moderate or vigorous physical activity during the past 7 days.

Table 3

*Distribution of Stressful Life Events by Sexual Minority Status, % Reporting Exposure in at Least One Wave (N = 6,973)*

Event	Males (N = 3,415)			Females (N = 3,558)		
	Gay or Bisexual (n = 117) % (SE)	Heterosexual (n = 3,298) % (SE)	p value <sup>a</sup>	Lesbian or bisexual (n = 189) % (SE)	Heterosexual (n = 3,369) % (SE)	p value <sup>a</sup>
Mean cumulative score of events (SE)	2.81 (0.28)	2.84 (0.08)	0.92	3.45 (0.26)	2.26 (0.06)	<0.0001
Exposure categories: Number of events						
0	15.35 (3.74)	16.65 (0.90)	0.72	15.34 (3.41)	23.98 (0.97)	<0.0001
1–2	37.69 (7.02)	36.94 (1.25)		24.59 (4.04)	37.70 (1.24)	
3–4	29.04 (6.15)	23.91 (0.90)		27.76 (3.88)	23.78 (1.01)	
5 or more	17.92 (4.52)	22.50 (1.16)		32.31 (4.45)	14.54 (0.96)	

<sup>a</sup> p values are derived from analysis of variance tests for the mean cumulative score, and from chi-square tests for exposure as a categorical variable.

0.71,  $p = .01$ ) and 5+ ( $\beta = 0.87$ ,  $p = .01$ ) stressful life events had a statistically significant elevation in cardiometabolic risk. In contrast, there was no relationship between stressful life events and cardiometabolic risk among heterosexual men (see Table 5).

Among the women, in models adjusted for all covariates, lesbian/bisexual women with 5+ stressful life events had a statistically significant elevation in cardiometabolic risk ( $\beta = 0.52$ ,  $p = .046$ ). Although heterosexual women exposed to 5+ stressful life events had greater cardiometabolic risk in models adjusted for demographic characteristics, these relationships were no longer statistically significant once SES was included in the model.

### Sensitivity Analyses

Two sensitivity analyses were performed. In one set, we controlled for self-reported illness in the past 2 weeks in addition to all covariates used in Model 5; a similar pattern of results was maintained, although slightly attenuated for lesbian/bisexual women (results not shown, but available upon request).

In the second set of sensitivity analyses, we examined cardiometabolic risk using the dichotomous threshold score (see Appendix 2 of the online supplemental materials). The two differences to emerge were among the women. In the unadjusted model, lesbian/bisexual women experiencing five or more stressful life events had elevated cardiometabolic risk, incidence rate ratio (IRR) = 1.69, 95% confidence interval (CI) [1.00, 2.84]. However, after additional control for demographics, SES, and health behaviors, experiencing five or more stressful life events was no longer statistically significantly associated with cardiometabolic risk among lesbian/bisexual women (IRR = 1.41, 95% CI [0.87, 2.27]). In contrast, heterosexual women experiencing five or more stressful life events had elevated cardiometabolic risk in fully adjusted models (IRR = 1.13, 95% CI [1.01, 1.27]).

### Discussion

The current study examined whether sexual orientation influences the relationship between stressful life events and cardiometabolic risk using data from a national longitudinal sample of young adults in the United States. Contrary to previous studies of individual CVD-risk biomarkers among young adults (Everett & Mollborn, 2013; Hatzenbuehler et al., 2013), we did not observe sexual orientation disparities in cumulative cardiometabolic risk when using either a continuous cumulative risk

score or an alternative conceptualization that used clinically defined thresholds of “high risk.” However, previous studies have examined individual CVD-risk biomarkers separately (e.g., CRP, hypertension). The application of a composite, cumulative approach to modeling cardiometabolic biomarkers used in the current study therefore may have contributed to discrepancies between this report and earlier studies.

Although there was no evidence for disparities in cumulative cardiometabolic risk between LGB and heterosexual young adults, sexual orientation status moderated the relationship between stressful life events and CVD risk. In particular, stressful life events predicted higher cardiometabolic risk scores among gay/bisexual young adult men and lesbian/bisexual young adult women, controlling for multiple potential confounders, including demographic factors, SES, health behaviors, and self-reported illness. Among gay/bisexual men, the relationship between stressful life events and CVD risk was apparent at both low and high levels of exposure to stressful life events. Among lesbian/bisexual women, a relationship between stressful life events and CVD risk was apparent only at the highest level of exposure (i.e., five or more stressful life events), and this finding was less robust in sensitivity analyses that used a threshold risk score. In contrast, there was no association between stressful life events and CVD risk among heterosexual young adult men or women (although sensitivity analyses showed a small but heightened risk among heterosexual women with five or more stressful life events using an alternative measure of cardiometabolic risk). Previous studies among adults have documented greater CVD risk associated with stress for heterosexual women than for heterosexual men (Iso et al., 2002; Orth-Gomér et al., 2000), suggesting that this relationship may emerge later in the life course for this population.

Our results are consistent with a differential vulnerability or stress-sensitization model. That is, LGB young adults may be more vulnerable to the negative health consequences associated with stressful life events than heterosexuals, perhaps due to prior or concurrent exposure to stigma-related stressors. Such stressors disrupt cognitive, affective, and neurobiological processes (Hatzenbuehler, 2009; Hatzenbuehler & McLaughlin, 2014; Inzlicht et al., 2006) that could lower the threshold for developing negative reactions to stressors, placing LGB young adults at heightened risk for CVD in the context of exposure to stressful life events. However, the current study did not test

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**Table 4**  
*Results of Linear Regression Models Predicting Cardiometabolic Risk Score: LGB Participants Stratified by Sex*

Stress score by sex	Model 1: Unadjusted		Model 2: Adjusted for age, nativity, and race/ethnicity		Model 3: Adjusted for covariates in Model 2 + education and income		Model 4: Adjusted for covariates in Model 2 + alcohol consumption, smoking, and physical activity		Model 5: Adjusted for all covariates in Models 2, 3, and 4	
	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value
Gay/Bisexual males ( <i>N</i> = 117)										
1-2	0.52 (0.26)	.045	0.62 (0.25)	.01	0.71 (0.27)	.01	0.61 (0.26)	.02	0.71 (0.28)	.01
3-4	0.73 (0.36)	.04	0.74 (0.32)	.02	0.73 (0.37)	.048	0.75 (0.28)	0.01	0.73 (0.34)	.03
5 or more	0.68 (0.33)	.04	0.81 (0.32)	.01	0.74 (0.30)	.02	0.84 (0.32)	0.01	0.87 (0.31)	.01
None (reference)	—	—	—	—	—	—	—	—	—	—
Lesbian/Bisexual females ( <i>N</i> = 189)										
1-2	0.39 (0.36)	.28	0.32 (0.36)	.36	0.34 (0.32)	.29	0.34 (0.35)	.33	0.33 (0.32)	.31
3-4	0.08 (0.29)	.79	-0.07 (0.28)	.82	-0.03 (0.28)	.91	-0.06 (0.28)	.82	-0.05 (0.27)	.85
5 or more	0.72 (0.28)	.01	0.62 (0.28)	.03	0.54 (0.27)	.048	0.62 (0.28)	.03	0.52 (0.26)	.046
None (reference)	—	—	—	—	—	—	—	—	—	—

*Note.* All models take into account the complex sample design and sample weights. SE = standard error. Covariates were modeled as follows: race/ethnicity (White, non-White; reference = White); nativity (born in U.S., born outside of U.S.; reference = born in U.S.); high school degree or less (yes/no; reference = no); household income (missing, <\$39,999, >\$40,000; reference = >\$40,000); smoking status (never, previous/intermittent, regular smoker; reference = never); low physical activity (yes/no; reference = no); and past-year binge drinking (yes/no; reference = no).

**Table 5**  
*Results of Linear Regression Models Predicting Cardiometabolic Risk Score: Heterosexual Participants Stratified by Sex*

Stress score by sex	Model 1: Unadjusted		Model 2: Adjusted for age, nativity, and race/ethnicity		Model 3: Adjusted for covariates in Model 2 + education and income		Model 4: Adjusted for covariates in Model 2 + alcohol consumption, smoking, and physical activity		Model 5: Adjusted for all covariates in Models 2, 3, and 4	
	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value	$\beta$ (SE)	<i>p</i> value
Males ( <i>N</i> = 3,298)										
1-2	0.05 (0.08)	.52	0.05 (0.08)	.52	0.02 (0.07)	.76	0.03 (0.08)	.73	0.01 (0.07)	.91
3-4	0.11 (0.08)	.16	0.09 (0.08)	.26	0.04 (0.08)	.59	0.06 (0.08)	.46	0.03 (0.08)	.75
5 or more	0.12 (0.08)	.14	0.08 (0.07)	.31	-0.01 (0.07)	.89	0.02 (0.08)	.82	-0.04 (0.08)	.58
None (reference)	—	—	—	—	—	—	—	—	—	—
Females ( <i>N</i> = 3,369)										
1-2	0.07 (0.06)	.23	0.02 (0.05)	.71	-0.01 (0.05)	.85	0.01 (0.06)	.90	-0.01 (0.05)	.80
3-4	0.10 (0.06)	.12	0.05 (0.06)	.46	-0.00 (0.07)	.96	0.03 (0.06)	.65	-0.01 (0.07)	.93
5 or more	0.25 (0.07)	.001	0.17 (0.08)	.03	0.09 (0.08)	.27	0.14 (0.07)	.06	0.08 (0.08)	.29
None (reference)	—	—	—	—	—	—	—	—	—	—

*Note.* All models take into account the complex sample design and sample weights. SE = standard error. Covariates were modeled as follows: race/ethnicity (White, non-White; reference = White); nativity (born in U.S., born outside of U.S.; reference = born in U.S.); high school degree or less (yes/no; reference = no); household income (missing, <\$39,999, >\$40,000; reference = >\$40,000); smoking status (never, previous/intermittent, regular smoker; reference = never); low physical activity (yes/no; reference = no); and past-year binge drinking (yes/no; reference = no).

specific stress-sensitization mechanisms with regard to the developmental timing of stressors, which remains an important avenue for future research.

The stronger association between stressful life events and cardiometabolic risk for LGB young adults is notable, given our focus on exposure to stressful life events that are not specific to sexual minorities. Although the study included a wide range of stressful life events that were based on prior literature (e.g., Adkins et al., 2009; McLaughlin et al., 2012), there are many LGB-specific stressors that were not assessed in Add Health that might be important predictors of cardiometabolic risk (e.g., concealment, disclosure, stigma consciousness). The degree to which exposure to the types of stressors that are unique to LGB populations plays a role in explaining differential vulnerability to other types of stressful life events is an important question that warrants examination in future research.

Limitations of the study include a small sample size of LGB respondents who met criteria for study inclusion, which required us to use relatively crude categorical variables as covariates to avoid oversaturating the models. Related, the small sample size necessitated combining gay/lesbian with bisexual respondents, which may have obscured heterogeneity across these groups. However, when we disaggregated these groups, the direction and magnitude of the relationships remained unchanged for women (we were unable to run separate models for the men, as there were only 18 bisexual men in the analytic sample). Further, the sample size was large enough to stratify results by sex. We were unable to examine how intersectional identities (i.e., individuals with multiple stigmatized categories, such as sexual minority women who were also members of a racial/ethnic minority) may have influenced the results, which remains an important area for future study. Although we controlled for multiple established risk factors for CVD outcomes, there is the possibility of unmeasured confounding. Future studies with a more complete list of covariates are needed to further minimize confounding. We also note that longitudinal studies are subject to attrition bias. Although we demonstrated that there were minimal differences between those respondents who were excluded from our complete cases analysis (relative to those who were included), it remains unclear how differential loss-to-follow-up might have biased observed relationships between stressful life events and CVD risk in this study. Finally, stressful life events checklists are the most widely used instruments for assessing stress in large community samples in which stressor interviews are prohibitive in time and cost (Grant et al., 2003). Nevertheless, stressor interviews, which are the gold-standard measures of stressful life events (Monroe, 2008), should be used in future studies.

The study also had several noteworthy methodological strengths. The data on cardiometabolic biomarkers were measured rather than based on self-report, improving validity of study measures. Add Health is a nationally representative probability-based survey; results are therefore generalizable to LGB young adults in the United States. Add Health is also a prospective cohort study, which afforded the opportunity to capture exposure to stressful life events across adolescence and early young adulthood, rather than at a single point in time. In addition to these methodological advantages, the current study also provided novel information regarding potential determinants of CVD risk among LGB popu-

lations. Indeed, to our knowledge, this is the first study to document stressful life events as a risk factor for cumulative cardiometabolic risk among LGB young adults.

At present, no evidence-based interventions exist to prevent CVD within LGB populations. Although more research is needed, the current study has provided some preliminary evidence that contributes to the future development of preventions and interventions that seek to reduce CVD outcomes in LGB populations. In particular, our results suggest that augmenting coping skills and improving social support to reduce vulnerability to stress might be effective in lowering CVD risk among LGB populations, a hypothesis that warrants additional study.

## References

- Adair, L., & Dahly, D. (2005). Developmental determinants of BP in adults. *Annual Review of Nutrition*, 25, 407–434. doi:10.1146/annurev.nutr.25.050304.092538
- Adkins, D. E., Wang, V., & Elder, G. H., Jr. (2009). Structure and stress: Trajectories of depressive symptoms across adolescence and young adulthood. *Social Forces*, 88, 31–60. doi:10.1353/sof.0.0238
- American Diabetes Association. (2011). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 34, S62–S69. doi:10.2337/dc11-S062
- Bauer, G. R. (2014). Incorporating intersectionality theory into population health research methodology: Challenges and the potential to advance health equity. *Social Science & Medicine*, 110, 10–17. doi:10.1016/j.socscimed.2014.03.022
- Bontempo, D. E., & D'Augelli, A. R. (2002). Effects of at-school victimization and sexual orientation on lesbian, gay, or bisexual youths' health risk behavior. *Journal of Adolescent Health*, 30, 364–374. doi:10.1016/S1054-139X(01)00415-3
- Bosma, H., Peter, R., Siegrist, J., & Marmot, M. (1998). Two alternative job stress models and the risk of coronary heart disease. *American Journal of Public Health*, 88, 68–74. doi:10.2105/AJPH.88.1.68
- Bostwick, W. B., Boyd, C. J., Hughes, T. L., & McCabe, S. E. (2010). Dimensions of sexual orientation and the prevalence of mood and anxiety disorders. *American Journal of Public Health*, 100, 468–475. doi:10.2105/AJPH.2008.152942
- Brummett, B. H., Babyak, M. A., Singh, A., Jiang, R., Williams, R. B., Harris, K. M., & Siegler, I. C. (2013). Socioeconomic indices as independent correlates of C-reactive protein in the National Longitudinal Study of Adolescent Health. *Psychosomatic Medicine*, 75, 882–893. doi:10.1097/PSY.0000000000000005
- Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr., . . . Roccella, E. J. (2003). Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP. *Hypertension*, 42, 1206–1252. doi:10.1161/01.HYP.0000107251.49515.c2
- Cochran, S. D., & Mays, V. M. (2007). Physical health complaints among lesbians, gay men, and bisexual and homosexually experienced heterosexual individuals: Results from the California Quality of Life Survey. *American Journal of Public Health*, 97, 2048–2055. doi:10.2105/AJPH.2006.087254
- Conron, K. J., Mimiaga, M. J., & Landers, S. J. (2010). A population-based study of sexual orientation identity and gender differences in adult health. *American Journal of Public Health*, 100, 1953–1960.
- Everett, B., & Mollborn, S. (2013). Differences in hypertension by sexual orientation among U.S. young adults. *Journal of Community Health*, 38, 588–596.
- Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health*, 26, 469–500. doi:10.1146/annurev.publhealth.26.021304.144542
- Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent

- psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin*, 129, 447–466. doi:10.1037/0033-2909.129.3.447
- Guagnano, M. T., Ballone, E., Colagrande, V., Della Vecchia, R., Manigrasso, M. R., Merlitti, D., . . . Sensi, S. (2001). Large waist circumference and risk of hypertension. *International Journal of Obesity and Related Metabolic Disorders*, 25, 1360–1364.
- Hammen, C., Henry, R., & Daley, S. E. (2000). Depression and sensitization to stressors among young women as a function of childhood adversity. *Journal of Consulting and Clinical Psychology*, 68, 782–787.
- Hatzenbuehler, M. L. (2009). How does sexual minority stigma “get under the skin”? A psychological mediation framework. *Psychological Bulletin*, 135, 707–730. doi:10.1037/a0016441
- Hatzenbuehler, M. L., & McLaughlin, K. A. (2014). Structural stigma and hypothalamic-pituitary-adrenocortical axis reactivity in lesbian, gay, and bisexual young adults. *Annals of Behavioral Medicine*, 47, 39–47. doi:10.1007/s12160-013-9556-9
- Hatzenbuehler, M. L., McLaughlin, K. A., & Slopen, N. (2013). Sexual orientation disparities in cardiovascular biomarkers among young adults. *American Journal of Preventive Medicine*, 44, 612–621. doi:10.1016/j.amepre.2013.01.027
- Hubert, H. B., Feinleib, M., McNamara, P. M., & Castelli, W. P. (1983). Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham Heart Study. *Circulation*, 67, 968–977. doi:10.1161/01.CIR.67.5.968
- Inzlicht, M., McKay, L., & Aronson, J. (2006). Stigma as ego depletion: How being the target of prejudice affects self-control. *Psychological Science*, 17, 262–269. doi:10.1111/j.1467-9280.2006.01695.x
- Iso, H., Date, C., Yamamoto, A., Toyoshima, H., Tanabe, N., Kikuchi, S., . . . Ohno, Y. (2002). Perceived mental stress and mortality from cardiovascular disease among Japanese men and women: The Japan Collaborative Cohort Study for Evaluation of Cancer Risk Sponsored by Monbusho (JACC Study). *Circulation*, 106, 1229–1236. doi:10.1161/01.CIR.0000028145.58654.41
- Kaprio, J., Koskenvuo, M., & Rita, H. (1987). Mortality after bereavement: A prospective study of 95,647 widowed persons. *American Journal of Public Health*, 77, 283–287. doi:10.2105/AJPH.77.3.283
- Kark, J. D., Goldman, S., & Epstein, L. (1995). Iraqi missile attacks on Israel: The association of mortality with a life-threatening stressor. *Journal of the American Medical Association*, 273, 1208–1210. doi:10.1001/jama.1995.03520390068035
- Karlamangla, A. S., Singer, B. H., & Seeman, T. E. (2006). Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur Studies of Successful Aging. *Psychosomatic Medicine*, 68, 500–507. doi:10.1097/01.psy.0000221270.93985.82
- King, K. E., Morenoff, J. D., & House, J. S. (2011). Neighborhood context and social disparities in cumulative biological risk factors. *Psychosomatic Medicine*, 73, 572–579. doi:10.1097/PSY.0b013e318227b062
- Kubzansky, L. D., Koenen, K. C., Jones, C., & Eaton, W. W. (2009). A prospective study of posttraumatic stress disorder symptoms and coronary heart disease in women. *Health Psychology*, 28, 125–130. doi:10.1037/0278-6133.28.1.125
- Kubzansky, L. D., Koenen, K. C., Spiro, A., 3rd, Vokonas, P. S., & Sparrow, D. (2007). Prospective study of posttraumatic stress disorder symptoms and coronary heart disease in the Normative Aging Study. *Archives of General Psychiatry*, 64, 109–116. doi:10.1001/archpsyc.64.1.109
- Lee, S., Colditz, G. A., Berkman, L. F., & Kawachi, I. (2003). Caregiving and risk of coronary heart disease in U.S. women: A prospective study. *American Journal of Preventive Medicine*, 24, 113–119. doi:10.1016/S0749-3797(02)00582-2
- Major, B., & O'Brien, L. T. (2005). The social psychology of stigma. *Annual Review of Psychology*, 56, 393–421. doi:10.1146/annurev.psych.56.091103.070137
- McDade, T. W., Burhop, J., & Dohnal, J. (2004). High-sensitivity enzyme immunoassay for C-reactive protein in dried blood spots. *Clinical Chemistry*, 50, 652–654. doi:10.1373/clinchem.2003.029488
- McLaughlin, K. A., Conron, K. J., Koenen, K. C., & Gilman, S. E. (2010). Childhood adversity, adult stressful life events, and risk of past-year psychiatric disorder: A test of the stress sensitization hypothesis in a population-based sample of adults. *Psychological Medicine*, 40, 1647–1658. doi:10.1017/S0033291709992121
- McLaughlin, K. A., Hatzenbuehler, M. L., Xuan, Z., & Conron, K. J. (2012). Disproportionate exposure to early-life adversity and sexual orientation disparities in psychiatric morbidity. *Child Abuse & Neglect*, 36, 645–655. doi:10.1016/j.chiabu.2012.07.004
- Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, 60, 501–524. doi:10.1146/annurev.psych.60.110707.163551
- Monroe, S. M. (2008). Modern approaches to conceptualizing and measuring human life stress. *Annual Review of Clinical Psychology*, 4, 33–52. doi:10.1146/annurev.clinpsy.4.022007.141207
- Neaton, J. D., & Wentworth, D. (1992). Serum cholesterol, BP, cigarette smoking, and death from coronary heart disease: Overall findings and differences by age for 316,099 White men. Multiple Risk Factor Intervention Trial Research Group. *Archives of Internal Medicine*, 152, 56–64. doi:10.1001/archinte.1992.00400130082009
- Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin*, 115, 424–443. doi:10.1037/0033-2909.115.3.424
- Ornelas, I. J. K. M., Perreira, K. M., & Ayala, G. X. (2007). Parental influences on adolescent physical activity: A longitudinal study. *International Journal of Behavioral and Nutritional Physical Activity*, 4, 3–12. doi:10.1186/1479-5868-4-3
- Orth-Gomér, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *JAMA: Journal of the American Medical Association*, 284, 3008–3014. doi:10.1001/jama.284.23.3008
- Osei, K., Rhinesmith, S., Gaillard, T., & Schuster, D. (2003). Is glycosylated hemoglobin A1c a surrogate for metabolic syndrome in nondiabetic, first-degree relatives of African-American patients with Type 2 diabetes? *The Journal of Clinical Endocrinology & Metabolism: Clinical and Experimental*, 88, 4596–4601. doi:10.1210/jc.2003-030686
- Poulter, N. (2003). Global risk of cardiovascular disease. *Heart*, 89, ii2–ii5. doi:10.1136/heart.89.suppl\_2.ii2
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, 128, 330–366. doi:10.1037/0033-2909.128.2.330
- Ridker, P. M. (2003). Cardiology patient page: C-reactive protein. A simple test to help predict risk of heart attack and stroke. *Circulation*, 108, e81–e85. doi:10.1161/01.CIR.0000093381.57779.67
- Roercke, M., & Rehm, L. (2010). Irregular heavy drinking occasions and risk of ischemic heart disease: A systematic review and meta-analysis. *American Journal of Epidemiology*, 171, 633–644. doi:10.1093/aje/kwp451
- Rohlfing, C. L., Wiedmeyer, H. M., Little, R. R., England, J. D., Tennill, A., & Goldstein, D. E. (2002). Defining the relationship between plasma glucose and HbA(1c): Analysis of glucose profiles and HbA(1c) in the Diabetes Control and Complications Trial. *Diabetes Care*, 25, 275–278. doi:10.2337/diacare.25.2.275
- Sandfort, T. G., de Graaf, R., Bijl, R. V., & Schnabel, P. (2001). Same-sex sexual behavior and psychiatric disorders. *Archives of General Psychiatry*, 58, 85–91. doi:10.1001/archpsyc.58.1.85
- Seeman, T. E., Crimmins, E., Huang, M. H., Singer, B., Bucur, A., Gruenewald, T., . . . Reuben, D. B. (2004). Cumulative biological risk

- and socio-economic differences in mortality: MacArthur studies of successful aging. *Social Science & Medicine*, 58, 1985–1997. doi:10.1016/S0277-9536(03)00402-7
- Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: Cumulative allostatic load. *Annals of the New York Academy of Sciences*, 1186, 223–239. doi:10.1111/j.1749-6632.2009.05341.x
- Shin, S. H., Edwards, E. M., & Heeren, T. (2009). Child abuse and neglect: Relations to adolescent binge drinking in the National Longitudinal Study of Adolescent Health (Add Health). *Addictive Behaviors*, 34, 277–280. doi:10.1016/j.addbeh.2008.10.023
- Slopen, N., Kubzansky, L. D., McLaughlin, K. A., & Koenen, K. C. (2013). Childhood adversity and inflammatory processes in youth: A prospective study. *Psychoneuroendocrinology*, 38, 188–200. doi:10.1016/j.psychneuen.2012.05.013
- Slopen, N., Lewis, T. T., Gruenewald, T. L., Mujahid, M. S., Ryff, C. D., Albert, M. A., & Williams, D. R. (2010). Early life adversity and inflammation in African Americans and whites in the midlife in the United States survey. *Psychosomatic Medicine*, 72, 694–701.
- Thompson, P. D. (2003). Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 23, 1319–1321.
- Troxel, W. M., Matthews, K. A., Bromberger, J. T., & Sutton-Tyrrell, K. (2003). Chronic stress burden, discrimination, and subclinical carotid artery disease in African American and Caucasian women. *Health Psychology*, 22, 300–309. doi:10.1037/0278-6133.22.3.300

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