2019 PAA Submission
Updated: 09-18-2018
*Please don't circulate or cite this draft with the authors' permission

Early-Life Adversities and Recalcitrant Smoking in Midlife: An Examination of Gender and Life-Course Pathways

Chioun Lee and Lexi Harari

Department of Sociology, University of California-Riverside, USA

Abstract (150 words or less)

Though smoking cessation is the best way to reduce premature death among individuals who have cardiovascular diseases (CVD), a substantial percentage of individuals with these conditions continue to smoke. Given that stressful environments are well-known predictors of smoking, we investigate life-course pathways from early-life adversities (low childhood SES, family instability, and abuse) to risk of recalcitrant smoking in midlife (smoking with CVD). Using longitudinal data from Midlife in the U.S., we found that for women, both low childhood SES and family instability independently increase the risk of recalcitrant smoking. By including midlife mediators, the effect of childhood SES reduced dramatically but the effect of family instability remained. For men, the effect of childhood SES remained even after adding mediators and the effect is stronger than adult SES. Our findings expand the current understanding of childhood as a critical period that contributes to the persistence of health-risk behaviors in later life.

Keywords: Adverse Childhood Experience, Gender, Smoking, Premature Death, Cardiovascular Disease.

***Corresponding author:**

Chioun Lee

Department of Sociology, University of California at Riverside

1207 Watkins Hall, Riverside, CA 92521

Phone: (951) 827-4411; Fax: (951) 827-3330

chiounl@ucr.edu

INTRODUCTION

Despite the consistent decline in the prevalence of smoking over the past half century, smoking has persisted as the number one cause of preventable deaths in the United States (U.S. Department of Health and Human Services 2014). Much of smoking's link to premature mortality may be due to its association with the largest overall causes of death in the United States, cardiovascular disease (CVD) and cancer (McCarthy, 2014). Research has indicated that smoking cessation is the best remedy for reducing the risk of death among individuals who have CVD (Critchley & Capewell, 2003), yet a substantial percentage of individuals with smoking-related conditions continue to smoke (Rallidis, Hamodraka, Foulidis, & Pavlakis, 2005). To reduce morbidity and increase longevity, it is crucial to understand the life-course factors that predict such smoking recalcitrance.

Low socioeconomic position and stressful environments are well-known predictors of the initiation, frequency, and cessation of smoking (Slopen et al., 2013). Smoking is concentrated among lower socioeconomic groups, which are less likely to quit smoking compared to their more affluent counterparts (Hiscock, Bauld, Amos, Fidler, & Munafo, 2012). Individuals who are exposed to early-life adversities (ELAs), such as low SES and harsh family environment, are more likely to initiate smoking at younger ages and are less likely to quit smoking (Kristjansson, Sigfusdottir, Allegrante, & Helgason, 2009; Soteriades & DiFranza, 2003). Some studies have found that such smoking habits among victims of ELAs persist even beyond young adulthood (Taha, Galea, Hien, & Goodwin, 2014). During midlife, due to the increased risk of both cardiovascular dysregulation, expressed by elevated blood pressure (Yoon, Fryar, & Carroll, 2015), and the onset of cardiovascular diseases, such as heart attack and stroke (Roger et al., 2011), smoking may be especially harmful.

Given that ELAs have an adverse impact on life-course resources relevant to smoking cessation, we can expect that ELAs might lead to more harmful smoking behaviors, such as smoking despite having medical conditions (e.g., CVD) that may be complicated by the smoking habit. Yet, we are aware of only one study that has tested such a hypothesis (Edwards, Anda, Gu, Dube, & Felitti, 2007). Furthermore, the

effect of ELAs on smoking status in midlife may differ by gender. Cumulative inequality theory suggests that gender differences in the accumulation of inequality may produce differential vulnerability to ELAs (Ferraro & Shippee, 2009) through gender-specific life-course pathways (Lee & Ryff, 2016). That is, women and men may respond differently to different types of adversities, which may translate to different likelihoods of smoking throughout the life course. However, we are not aware of any studies that have explicitly investigated gender differences in smoking recalcitrance. Therefore, the primary goal of the current study is to identify early and midlife factors that differentiate individuals who quit smoking vs. those who continue smoking even though they have serious medical conditions. The present study examines the following questions:

1. What is the most influential ELA that affects the likelihood of being a recalcitrant smoker in midlife?
2. To what extent do adult SES and psychosocial factors mediate the association between ELAs and recalcitrant smoking?
3. Does the effect of ELAs on recalcitrant smoking differ for men and women?

DATA AND METHODS

Sample

Data for this study come from the MIDUS (Midlife Development in the U.S.) study, a national survey designed to assess the role of social, psychological, and behavioral factors in understanding differences in mental and physical health (n = 7,108; 52% women). MIDUS began in 1995/1996 (Wave [W] 1) with non-institutionalized, English speaking adults aged 25–74 in the 48 contiguous states (Brim, Ryff, & Kessler, 2004). MIDUS consists of a two-stage survey: a telephone interview and a self-administered questionnaire (SAQ). Approximately 89% of the sample completed both the telephone interview and SAQ at W1 (n = 6,325). Follow-up interviews with MIDUS respondents were completed in 2004–2006 (n

= 4,963). The longitudinal retention rate for W2 was 75% after adjusting for mortality. The present analysis uses data from the 4,963 individuals who participated in both the initial and follow-up surveys. Compared to individuals who died or were lost to follow-up [LFU], those who participated in both waves were more likely to be White, female, married, more highly educated, and to report having better health. Additional information about sampling, enrollment, and longitudinal retention is documented elsewhere (Radler & Ryff, 2010).

Measures

Early-life adversities (W1) includes the three most common domains of adversity in childhood: socioeconomic disadvantage, family instability, and parental abuse. The index of **low childhood SES** (Cronbach's $\alpha = .74$) captures both objective and subjective social status in early life. We used six indicators: mother's and father's education (1 = no school/some grade school to 12 = PhD, MD, or other professional degree), mother's and father's occupational prestige score, measured by Duncan's Socioeconomic Index (Hauser & Warren, 1997), welfare status (0 = never on welfare, 1 = ever on welfare), and financial level growing up (1 = a lot better off than the average family to 7 = a lot worse off). **Family instability** is a binary indicator based on the question, "Did you live with both of your biological parents up until you were 16?" Possible reasons for a negative response include parental death, separation or divorce, parents not living together, and never knowing a biological parent. For **parental abuse** (Cronbach's $\alpha = .84$), respondents were presented with a battery of items from the modified version of the Conflict Tactics Inventory (Straus, Hamby, Boney-McCoy, & Sugarman, 1996). Respondents were asked how often they had endured each of the following three domains of abuse: moderate physical abuse (prompts for this domain: pushed, grabbed, or shoved; slapped; object thrown at respondent), severe physical abuse (kicked, bit, or hit with a fist; hit with an object [or attempted]; beat up; choked; burned or scalded), and emotional abuse (made insulting remarks; sulked or refused to talk; stomped away; did something out of spite; made threats; kicked/smashed something in anger). The response options included never, rarely, sometimes, and often. Using the six items (the three domains of abuse with respect to each parent), we created the parental abuse index.

Smoking Status in Midlife (W2). We conducted two stages of analysis to identify midlife smoking status: never-smoker, ex-smoker, smoker without cardiovascular disease (CVD), and smoker with CVD ("recalcitrant smokers"). First, participants were classified as having never smoked, former smokers, or current smokers based on their responses to two questions: "Have you ever smoked cigarettes regularly—that is, at least a few cigarettes every day?" and "Do you smoke cigarettes regularly NOW?" Current smokers, which comprised 15% of the sample, were included in the CVD group if they met at least one of the following five conditions: (1) ever had heart problems suspected or confirmed by a doctor, (2) ever had a heart attack, (3) ever experienced or been treated for stroke in the past twelve months, (4) ever experienced or been treated for hypertension in the past twelve months, or (5) thought that their risk of heart attack was higher than other people their age. Individuals who had none of the above conditions were categorized as midlife smokers without CVD.

Midlife mediators (W1). Based on prior studies, we included five life-course mediators linking ELAs to smoking status in midlife. Because low SES individuals show higher rates of cigarette smoking in the U.S., we created an index of ***adult SES*** (α = .70), which was comprised of four indicators of objective SES: (a) educational degree (1 = no school/some grade school to 12 = PhD, MD, or other professional degree), (b) household income ($0–$300,000 or more), (c) wage/salary income ($0–$100,000 or more), and (d) current or previous occupation (1 = never employed or manual labor, 2 = service/sales/administrative, 3 = management/business/financial, 4 = professional). We also included two psychological factors: positive affect and purpose in life. For ***positive affect*** (α = .91), participants rated how frequently they had felt each of 10 positive emotions during the past 30 days using a 5-point scale: 1 = none of the time to 5 = all the time. The items were "cheerful," "in good spirits," "extremely happy," "calm and peaceful," "satisfied," "full of life," "enthusiastic," "attentive," "active," and "proud." Positive-affect scores represent the respondent's average rating of these items (range: 1 through 5). Prior studies have indicated that positive affect does not imply the absence of negative affect (e.g., depressive symptoms), but rather is a separate construct that is independently associated with lower morbidity and

increased longevity (Pressman & Cohen, 2005). Given that prior studies have shown that individuals who have higher purpose in life are more likely to engage in preventive health practices (Kim, Strecher, & Ryff, 2014), we created a ***purpose in life*** index, measured by a three-item version of Ryff's Scale of Psychological Wellbeing (Ryff, 1989). On a scale from 1 = strongly disagree to 7 = strongly agree, participants responded to three statements: "I live life one day at a time and don't really think about the future"; "Some people wander aimlessly through life, but I am not one of them"; and "I sometimes feel as if I've done all there is to do in life." Purpose in life was the average of these three items ($\alpha = .70$). Based on prior work (Glei, Goldman, Ryff, Lin, & Weinstein, 2012; Loucks et al., 2006), we created a ***social interaction*** index as the sum of the following four binary components: (a) whether R was married, (b) whether R had at least weekly contact with family members or friends, (c) whether R attended religious or spiritual services at least monthly, and (d) whether R participated in some other social activity (e.g., professional groups, sports, or social groups) at least monthly. We created an index of ***family support*** (Walen & Lachman, 2000) using four questions reflecting positive relations with family members, not including spouse or partner: (a) "How much do members of your family really care about you?" (b) "How much do they understand the way you feel about things?" (c) "How much can you rely on them for help if you have a serious problem?" and (d) "How much can you open up to them if you need to talk about your worries?" We included four demographic confounders from W1 which are associated with smoking status: age, race/ethnicity, number of children, and gender.

Analytic strategies. Using Chi-square tests, we compared how baseline characteristics vary by smoking status in midlife for men and women (Table 1). We then used ordinal least square (OLS) regression models to test (a) the effect of ELAs on each mediator and (b) gender differences in the association (Table 2). Finally, we estimated nested multinomial logistic regression models (Table 3) to estimate the log odds of each smoking status compared (reference category: ex-smokers). We limited our analyses to those who have ever smoked regularly (53% of men and 45% of women). We first tested the independent effect of each ELA on recalcitrant smoking (Model 1) and then examined the intervening role of life-course

mediators (Model 2). Using Stata's *ice* command (Royston & White, 2011), we implemented 10 imputations to predict missing variables. Given that the likelihoods of death and health-promoting resources differ by gender (Rogers, Everett, Saint Onge, & Krueger, 2010), the analysis was stratified by gender. Gender differences were tested by pooling data from both genders and testing gender interaction terms. The contribution of the mediators was evaluated by the percentage of total effects explained by each mediator.

RESULTS

Baseline Characteristics by Smoking Status

Table 1 displays the results from bivariate analyses that tested whether all variables used in the analyses varied by smoking status. We found no gender differences in being midlife smokers (16% for both genders), yet women showed a slightly higher prevalence of being recalcitrant smokers than men (9% vs. 7%). For both genders, midlife smoking status was significantly related to ELAs and midlife resources. Compared to other smoking status groups, midlife smokers with CVD were more likely to have experienced low childhood SES, parental abuse, and family instability. They also showed lower levels of adult SES, positive affect, and purpose of life, as well as fewer social interactions. Participants who were younger and non-white at baseline were more likely to be midlife smokers with CVD.

Early-Life Adversities and Midlife Mediators

Table 2 shows the inverse associations between ELAs and life-course mediators, which are statistically significant for both men and women (p-value trends < .001), albeit generally stronger for women. Specifically, the effects of parental abuse were stronger for women than men in terms of positive affect in midlife (β = -.21 vs. β = -.13, p < .05 for gender difference) and family support (β = -.26 vs. β = -.19, p < .05). Similarly, family instability harms women's social interaction more than men's (β = -.18 vs. β = -.07, p < .05). However, there appears to be relatively little gender difference in the associations between ELAs and adult SES or purpose in life.

Early-Life Adversities and Recalcitrant Smoking in Midlife

In a series of adjusted models (Table 3) that only include smokers, we first investigated the independent effect of each ELA on smoking status (Model 1). We then examined the extent to which the association between ELAs and smoking status is explained by midlife mediators (Model 2). Two types of ELAs independently affect likelihood of being recalcitrant smokers, but the effects differ by gender. For women, a one SD decrease in childhood SES increases the risk of being midlife smokers with CVD (compared to ex-smokers) by 23%, and growing up with family instability increases the risk of being midlife smokers with CVD (compared to ex-smokers) by 108%. For men, low childhood SES is a single and significant indicator that increases the likelihood of being midlife smokers with CVD (Relative Risk Ratio [RRR] = 1.57, $p < .001$). In the tests of gender interactions between ELAs and smoking status, we found that childhood SES is a stronger predictor for men than women, while family instability is stronger for women than men. We plotted these interactions in Figure 1.The childhood SES panel shows that the probability of being a recalcitrant smoker is lower for men than women when they have average or higher childhood SES, but the probability dramatically increases for men only when they have lower than average levels of childhood SES. The family instability panel illustrates that, among those who did not experience family instability, there is no gender difference in the probability of being a recalcitrant smoker; however, among those who experienced family instability, women are more likely to be recalcitrant smokers than men.

In Model 2 of Table 3, we found that three indicators (adult SES, positive affect, and purpose in life) significantly reduce the risk of being recalcitrant smokers (compared to ex-smokers). Female smokers who had higher adult SES and higher positive affect show reduced risk of being recalcitrant smokers. For male smokers, having higher positive affect and higher purpose in life reduced the risk of being recalcitrant smokers. Taking into account potential mediators reduced the coefficient of childhood SES by 45% for women, making it no longer statistically significant, but the coefficient of family instability did not change substantially between Model 1 and Model 3 (less than 10%) and remained

significant. For men, adding potential mediators had little impact on the effect of childhood SES on recalcitrant smoking. The effect of childhood SES is stronger than adult SES ($p < .05$), indicating the early-life origins of men's risk of being recalcitrant smokers in later life.

Table 1. Characteristics at baseline of full sample by Gender and Smoking Status at Wave 2

	Women (n=2648)					**Men** (n= 2315)				
	Never-smoker (55%)	**Ex-smoker (29%)**	**Smoker without CVD (7%)**	**Smoker with CVD (9%)**	Group differences	**Never-smoker (47%)**	**Ex-smoker (39%)**	**Smoker without CVD (8%)**	**Smoker with CVD (7%)**	Group differences
Early-life adversities at W1										
Low childhood SES	.00	-.01	.21	.24	**	-.22	.08	.07	.38	***
Parental abuse	-.20	.05	.02	.22	***	-.01	.12	.29	.29	***
Family instability	.18	.22	.25	.40	***	.14	.19	.30	.30	***
Midlife mediators at W1										
Adult SES	-.11	-.24	-.43	-.50	***	.45	.07	-.03	-.01	***
Positive affect	.10	-.12	-.20	-.47	***	.10	.01	.13	-.35	***
Purpose in life	.02	-.04	-.15	-.21	***	.16	.01	-.17	-.28	***
Family support	.13	.09	.09	-.11	**	-.08	-.11	-.21	-.21	ns
Social interaction	.13	-.05	-.44	-.30	***	.10	-.02	-.26	-.25	***
Demographic controls at W1										
Age	.01	.14	-.34	-.20	***	-.20	.34	-.38	-.21	***
White	.91	.94	.93	.91	*	.93	.95	.95	.92	ns
Number of children	.04	.08	-.02	.08	ns	-.16	.08	-.22	-.05	***

Notes: *$p < .05$, **$p < .01$, ***$p < .001$
ns = non-significant.
Chi-square tests were used to compare characteristics of baseline by smoking status for men and women.
CVD = cardiovascular disease

Table 2. Ordinal Least Square (OLS) Regression Predicting Midlife Mediators by Gender

	Adult SES	Positive affect	Purpose in life	Family support	Social interaction
Panel 1: Women (n=2648)					
Early-life adversities					
Low childhood SES	-.31***	-.01	-.11***	-.04*	-.08***
Parental abuse	.04	**-.21*** *a***	-.07***	**-.26*** *a***	-.08***
Family instability in childhood	.08	-.12*	-.04	-.11*	-.18*** ***a***
Panel 2: Men (n= 2315)					
Early-life adversities					
Low childhood SES	-.31***	.03	-.08**	-.03	-.06**
Parental abuse	-.03	**-.13*** *a***	-.13***	**-.19*** *a***	-.08***
Family instability in childhood	-.16**	-.17***	-.03	-.20***	-.07 ***a***

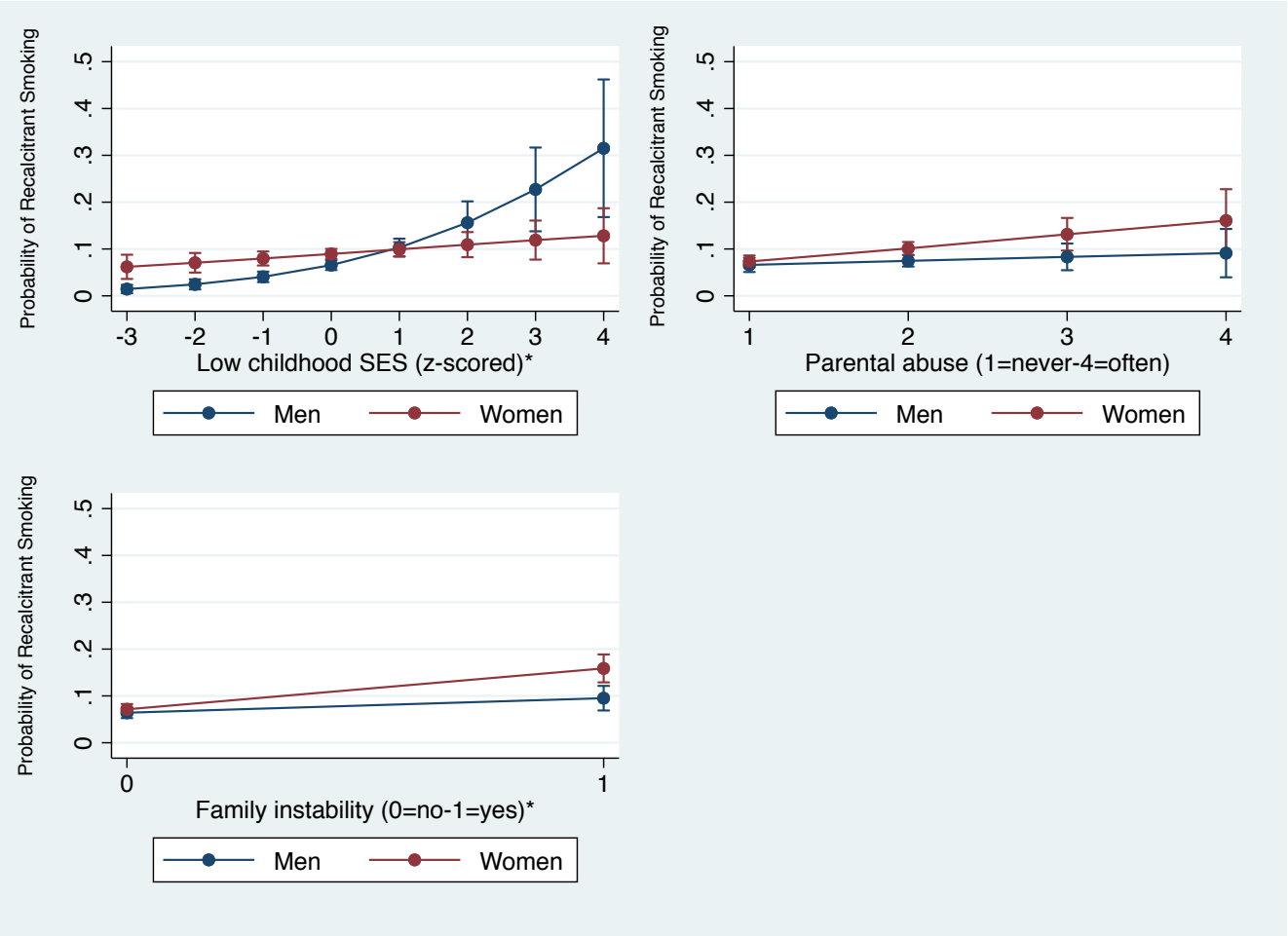
Note. All models included demographic controls at W
a refers to gender differences being statistically different
*$p < .05$, **$p < .01$, ***$p < .001$

Table 3. Multinomial Regression Estimates (Relative Risk Ratio [95%CI]) Predicting Midlife Smoking Status among Ever-Smokers (Ref. Ex-smokers) by Gender

	Model 1		Model 2	
	Smokers without CVD	**Smokers with CVD**	**Smokers without CVD**	**Smokers with CVD**
Panel 1: Female smokers (n=1183)				
Early Life Adversities				
Low childhood SES	**1.33 (1.12-1.58)****	**1.23 (1.15-1.58)****	**1.22 (1.10-1.58)***	**1.12 (.96-1.33)**
Parental abuse	0.86 (.78-1.07)	1.04 (.96-1.25)	0.87 (0.73-1.03)	0.99 (0.84-1.12)
Family instability	1.02 (.78-1.66)	**2.08 (1.66-3.12)*****	0.95 (0.63-1.39)	**1.99 (1.43-2.76)*****
Midlife Mediators				
Adult SES			**0.80 (0.63-.98)***	**0.76 (0.62-0.92)***
Positive affect			0.95 (0.77-1.18)	**0.82 (0.70-0.95)****
Purpose in life			1.07 (0.87-1.32)	1.06 (0.88-1.28)
Family support			1.13 (0.92-1.38)	1.01 (0.85-1.20)
Social interaction			**0.67 (0.55-0.80)*****	0.88 (0.74-1.03)
Panel 2: Male smokers (n= 1231)				
Early Life Adversities				
Low childhood SES	1.18 (1.03-1.48)	**1.57 (1.35-2.00)*****	1.09(0.90-1.32)	**1.56 (1.26-1.93)*****
Childhood abuse	1.05 (.93-1.29)	1.10 (.93-1.30)	1.06 (0.89-1.27)	0.97 (0.81-1.16)
Family instability	1.47 (1.11-2.33)	1.34 (.90-2.01)	**1.52 (1.03-2.23)***	1.33 (0.88-1.98)
Midlife Mediators				
Adult SES			0.81 (0.65-0.99)*	0.93 (0.75-1.15)
Positive affect			1.38 (1.08-1.75)**	**0.76 (0.63-0.99)****
Purpose in life			0.94 (0.74-1.17)	**0.77 (0.61-0.96)***
Family support			1.02 (0.85-1.22)	1.16 (.97-1.49)
Social interaction			0.85 (0.71-1.02)	0.93 (0.76-1.12)

Note. All models are adjusted for age, race-ethnicity, and number of children
SES= socioeconomic status
*$p < .05$, **$p < .01$, ***$p < .00$.

Figure 1 Probability of Recalcitrant Smoking (Smoking with Cardiovascular Disease) by Early-Life Adversity and Gender



Note. * refers to gender differences that are statistically different ($p < .05$).

REFERENCES

Brim, O. G., Ryff, C. D., & Kessler, R. C. (2004). *How healthy are we?: A national study of well-being at midlife*: University of Chicago Press.

Critchley, J. A., & Capewell, S. (2003). Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. *JAMA, 290*(1), 86-97.

Edwards, V. J., Anda, R. F., Gu, D., Dube, S. R., & Felitti, V. J. (2007). Adverse Childhood Experiences and Smoking Persistence in Adults with Smoking-Related Symptoms and Illness. *Perm J, 11*(2), 5-13.

Ferraro, K. F., & Shippee, T. P. (2009). Aging and cumulative inequality: How does inequality get under the skin? *The Gerontologist, 49*(3), 333-343.

Glei, D. A., Goldman, N., Ryff, C. D., Lin, Y.-H., & Weinstein, M. (2012). Social relationships and inflammatory markers: An analysis of Taiwan and the US. *Social Science and Medicine, 74*(12), 1891-1899.

Hauser, R. M., & Warren, J. R. (1997). Socioeconomic indexes for occupations: A review, update, and critique. *Sociological Methodology, 27*(1), 177-298.

Hiscock, R., Bauld, L., Amos, A., Fidler, J. A., & Munafo, M. (2012). Socioeconomic status and smoking: a review. *Annals of the New York Academy of Sciences, 1248*, 107-123. doi:10.1111/j.1749-6632.2011.06202.x

Kim, E. S., Strecher, V. J., & Ryff, C. D. (2014). Purpose in life and use of preventive health care services. *Proceedings of the National Academy of Sciences of the United States of America, 111*(46), 16331-16336. doi:10.1073/pnas.1414826111

Kristjansson, A. L., Sigfusdottir, I. D., Allegrante, J. P., & Helgason, A. R. (2009). Parental divorce and adolescent cigarette smoking and alcohol use: assessing the importance of family conflict. *Acta Paediatrica, 98*(3), 537-542. doi:10.1111/j.1651-2227.2008.01133.x

Lee, C., & Ryff, C. D. (2016). Early parenthood as a link between childhood disadvantage and adult heart problems: A gender-based approach. *Social Science and Medicine, 171*, 58-66. doi:http://dx.doi.org/10.1016/j.socscimed.2016.10.028

Loucks, E. B., Sullivan, L. M., D'Agostino, R. B., Sr., Larson, M. G., Berkman, L. F., & Benjamin, E. J. (2006). Social networks and inflammatory markers in the Framingham Heart Study. *Journal of Biosocial Science, 38*(6), 835-842. doi:S0021932005001203 [pii]

10.1017/S0021932005001203

McCarthy, M. (2014). Smoking remains leading cause of premature death in US. *BMJ: British Medical Journal (Online), 348*.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*(6), 925-971. doi:10.1037/0033-2909.131.6.925

Radler, B. T., & Ryff, C. D. (2010). Who participates? Accounting for longitudinal retention in the MIDUS national study of health and well-being. *Journal of Aging and Health, 22*(3), 307-331.

Rallidis, L. S., Hamodraka, E. S., Foulidis, V. O., & Pavlakis, G. P. (2005). Persistent smokers after myocardial infarction: a group that requires special attention. *International Journal of Cardiology, 100*(2), 241-245. doi:10.1016/j.ijcard.2004.08.040

Roger, V. L., Go, A. S., Lloyd-Jones, D. M., Adams, R. J., Berry, J. D., Brown, T. M., . . . Ford, E. S. (2011). Heart disease and stroke statistics—2011 update: a report from the American Heart Association. *Circulation, 123*(4), e18-e209.

Rogers, R. G., Everett, B. G., Saint Onge, J. M., & Krueger, P. M. (2010). Social, Behavioral, and Biological Factors, and Sex Differences in Mortality. *Demography, 47*(3), 555-578.

Royston, P., & White, I. R. (2011). Multiple imputation by chained equations (MICE): implementation in Stata. *J Stat Softw, 45*(4), 1-20.

Ryff, C. D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology, 57*(6), 1069.

Slopen, N., Kontos, E. Z., Ryff, C. D., Ayanian, J. Z., Albert, M. A., & Williams, D. R. (2013). Psychosocial stress and cigarette smoking persistence, cessation, and relapse over 9–10 years: a prospective study of middle-aged adults in the United States. *Cancer Causes & Control, 24*(10), 1849-1863.

Soteriades, E. S., & DiFranza, J. R. (2003). Parent's Socioeconomic Status, Adolescents' Disposable Income, and Adolescents' Smoking Status in Massachusetts. *American Journal of Public Health, 93*(7), 1155-1160.

Straus, M. A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The revised conflict tactics scales (CTS2) development and preliminary psychometric data. *Journal of family issues, 17*(3), 283-316.

Taha, F., Galea, S., Hien, D., & Goodwin, R. D. (2014). Childhood maltreatment and the persistence of smoking: A longitudinal study among adults in the US. *Child Abuse & Neglect, 38*(12), 1995-2006. doi:10.1016/j.chiabu.2014.10.022

Walen, H. R., & Lachman, M. E. (2000). Social support and strain from partner, family, and friends: Costs and benefits for men and women in adulthood. *Journal of Social and Personal Relationships, 17*(1), 5-30.

Yoon, S. S., Fryar, C. D., & Carroll, M. D. (2015). *Hypertension prevalence and control among adults: United States, 2011-2014*: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.