Stress, Longevity and Cardiovascular Outcomes among African American Families in the Jackson Heart Study

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Objective: Little is known about the relationship between family longevity, stress, and CVD in African Americans.

Methodology: Data consisted of 3274 participants aged ≥50 years with information on parental living status, the three indicators of stress, and cardiovascular health from the Jackson Heart Study (JHS).

Results: Those with both parents dead had significantly fewer life events than those with mother alive but father dead and those with both parents alive. Controlling for age, sex, and education, there were significant main effects for the number of life events, as well as the three parental living status variables in comparison to the both dead category. There is evidence for mediation with life events and weekly stress events, but not with global stress.

Discussion: The results show that there is a pattern on longevity in African American families that intersects with their stress experiences. Further examination of how generational patterns of stress are passed down is warranted. (*Ethn Dis.* 2014;24[4]:456–461)

Key Words: Stress, Longevity, Cardiovascular Outcomes, Families

Introduction

The difference in remaining life expectancy between African Americans and Caucasians at 65 is about two years and some estimates suggest that by age 85, African Americans have a longevity advantage.[1] One possible explanation for this convergence or cross-over effect is that African Americans who make it to the oldest ages represent "exceptional survivors" who have developed or possess physiological and psychological survival advantages to counteract the impediments to healthy active living.[2] One of the most formidable and well-recognized bio-behavioral variables related to physiological damage and premature death is perceived stress. However, few studies have examined the intersection between perceived stress, poor health and longevity among African Americans. We explore the degree to which perceived stress is associated with longevity and cardiovascular (CV) conditions among middle-age to older African Americans.

The relationship between health and stress, particularly perceived stress (the degree to which situations are appraised as stressful), is a central issue in the study of health disparities experienced by African Americans.[3] Perceived stress among African Americans is linked to factors such as racial discrimination, social inequality, disadvantaged neighborhoods, and limited health care system access.[3–5] Persistent poor health among African Americans may be a function of an equally "unhealthy" social environment through which attributions made about ambiguous and or negative circumstances perceived as stressful may be transmitted across generations to continue to have implications for health outcomes. There is preliminary evidence that suggests perceived stress and coping style have both a heritable component and a family environment component from studies of twins.[6] Although, it is not clear how parental attributions made about ambiguous/negative circumstances (eg, attribution bias) might be transmitted across generations to continue to exert negative persistent influence on health outcomes and increase health disparities among African Americans.[7]

The goal of our study was to examine the relationships between perceived psychological stress, CV health conditions, and patterns of family longevity.

The goal of our study was to examine the relationships between perceived psychological stress, CV health conditions, and patterns of family longevity. We hypothesized that: 1) Stress will be greater in those who report short-lived parents; 2) the relationship between family type (ie, parental living status) and CV conditions will depend on stress; and 3) family type will mediate the relationship between stress and CV conditions.

Methods

The data are from the baseline examination of the Jackson Heart Study (JHS), a single-site, longitudinal, population-based, cohort study prospectively investigating the determinants of CV conditions among African Americans living in the tri-county area (Hinds, Madison, and Rankin counties) of the Jackson, Mississippi metropolitan area. Baseline data collection occurred between September 2000 and March 2004. Recruitment, sampling, and data collection methods have been described previously.[8–11] Participant recruitment was limited to individuals aged 35 to 84 years, but allowed relatives <35 and

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>84 to enroll in order to increase the sample power of the family component of the study.[12] The total cohort consists of 5,301 African-American men and women aged 21–94. The final sample (*n* = 3274) was composed of participants aged ≥50 years with information on parental living status, the three indicators of stress, and cardiovascular health.

The Institutional Review Boards of the following participating institutions approved the study: the University of Mississippi Medical Center, Jackson State University, and Tougaloo College. All of the participants provided written informed consent.

Measures

We examined three measures of psychosocial stress: 1) the Global Perceived Stress Scale (GPSS), which was given in the baseline clinic examination; 2) the Weekly Stress Inventory (WSI or hassles), which was given to participants at the baseline examination to complete and mail back to the JHS; and 3) Major Life Events (MLE), which was collected through telephone interview in the annual follow-up survey.

The Global Perceived Stress Scale is an 8-item, self-report measure of perceived chronic stress. It assesses perceived stress across eight broad domains. Participants were asked, "Over the past 12 months how much stress did you experience?… 1) In your job?, 2) in your relationships with others?, related to medical problems?…" Responses for this scale ranged from A (not stressful) to D (very stressful). In the analysis, they were recoded to numeric values (eg, from not stressful=1 to very stressful=4). Scores were summed across the eight responses to create a total score with an observed range from 0 to 24.

The WSI is an 87-item questionnaire that assessed specific experiences of minor stress across a broad range of life domains over a 7-day period, including but not limited to, work tasks, relationships, finances, transportation, household responsibilities, and leisure activities.[13] Participants were instructed to assess the level of severity of these stressors during this timeframe on a 7-point scale defined as follows: did not happen; not stressful; slightly stressful; mildly stressful; moderately stressful; stressful; very stressful; extremely stressful. Scores were summed to create a total score with an observed range from 0 to 14.

The survey of MLE, based on Holmes and Rahe's Life Events Scale,[14] included 11 items in which participants were asked to answer yes or no if they had experienced the following situations within the last 12 months: 1) serious personal illness, 2) being a victim of physical assault, 3) being a victim of a robbery or home burglary, 4) losing a relative/close friend due to violence, 5) gunfire at home/neighborhood, 6) death of a relative or close friend, 7) major illness or injury of a close friend/relative, 8) moving to a lower socioeconomic residence/ neighborhood, 9) job loss, 10) forced retirement, or 11) experiencing divorce/separation from spouse. These items were summed to create a total observed score ranging from 0 to 9.

Select demographic factors including age, education, and sex were based on self-report during the baseline interview. The dependent variable was the sum of the number of CV conditions (prevalent high blood pressure, stroke, and heart disease). Prevalent heart disease was derived from a history of heart disease, medication use and indication of a silent myocardial infarction from electrocardiogram (ECG) readings. Stroke was self-reported. Prevalent hypertension status was defined as a measured blood pressure ≥ 140/90 mm Hg and/or use of antihypertensive medications.[15]

RESULTS

Descriptive characteristics of the study sample are presented in Table 1.

Hypothesis 1

Stress will be greater in those who report shorter-lived parents relative to those with longer-lived parents. This hypothesis was tested by conducting an ANOVA for each of the three indicators of stress (Table 2). Weekly stress events, $F_{(3, 2019)} = 3.75$, $P=.011$, $\eta^2=.006$; global stress, $F_{(3, 3242)} = 26.58$, $P<.001$, $\eta^2=.024$; and major life events, $F_{(3, 3138)} = 10.76$, $P<.001$, $\eta^2 =.01$ each revealed a significant effect of parental living status. Those with both parents dead had significantly fewer life events than those with mother alive but father dead and those with both parents alive.

Hypothesis 2

The relationship between family type (ie, parental living status) and CV conditions will depend on stress. We

Table 1. Baseline characteristics

Variable	Mean	SD	Range
Sex, 0=men, 1=women	.3	.4	0–1
Baseline age	62.5	8.1	50–95
Education	13.9	4.3	0–19
Parental living status, 0=no, 1=yes			
Both parents alive	.1	.25	0–1
Mother alive, father dead	.2	.4	0–1
Father alive, mother dead	0	.2	0–1
Both parents dead	.6	.4	0–1
Stress measures			
Weekly stress events	1.7	2.4	0–13
Life events	2.8	2.1	0–9
Global stress	4.3	4.0	0–23
Number of cardiovascular conditions	.7	.6	0–3

Table 2. Stress by parental living status

Type of Stress	Both Alive (*n* = 223)	Mother Alive/Father Dead (*n* = 744)	Father Alive/Mother Dead (*n* = 135)	Both Dead (*n* = 2172)
Weekly stress events	1.80 (2.39)	1.97[a] (2.64)	2.21 (2.87)	1.62[b] (2.33)
Global stress	5.32[a] (4.33)	5.19[a] (4.29)	4.99[a] (3.98)	3.87[b] (3.84)
Life events	3.24[a] (1.98)	3.04[a] (2.16)	3.08 (2.18)	2.65[b] (2.10)

Data are means (SD) unless indicated otherwise.
[a] *P*<.01. Reflects significant differences based on Tukey's HSD posthoc comparisons.
[b] *P*<.05. Reflects significant differences based on Tukey's HSD posthoc comparisons.

conducted separate multiple regression analyses for each stress type. The parental living status variable was dummy-coded so that we could compare three groups with a referent group. The dependent variable was the sum of the number of CV conditions, which ranged from 0 to 3. Results of these regression models can be found in Table 3.

Life Events

Controlling for age group (young-old: 50–64 and old-old: ≥65), sex, and education, there were significant main effects for the number of life events, as well as the three parental living status variables in comparison to the both dead category: both parents alive, mother alive but father dead, and mother dead but father alive. Importantly, there

was a significant interaction of *both parents alive × life events*, indicating that people with both parents dead are less reactive to life event stressors compared to people with both parents alive. This model accounted for 11% of the variance in CV conditions.

When age category was included as a factor in the interaction terms, there was a significant *mother alive × age* group interaction (β=.05, *P*=.047). However, none of the 3-way interactions (*parental status × life events × age group*) were significant. This model accounted for 11% of the variance in CV conditions. The significant 2-way interaction was decomposed by conducting separate models for each age group. For the young-old, having a mother alive (β=−.14, *P*<.001) was associated with

having fewer CV conditions, but it was not a significant predictor in the old-old group (β=.01, *P*=.863). We were also interested in the potential differences between parental living status and CV conditions with distinct levels of life event stressors. Therefore, we conducted a 2 (life event stressors: median split) × 4 (parental status) factorial ANOVA where we also controlled for age group, sex, and education. There were main effects for age group (F [1, 3116] = 19.05, *P*<.001), sex F [1, 3116] = 5.06, *P*=.024, education F [1, 3116] = 47.46, *P*<.001, and parental status (F [3, 3116] = 8.50, *P*<.001), and there was also a *significant life event × parental living status* interaction (F [3, 3116] = 2.97, *P*=.031). The interaction revealed that the participants with both parents alive and few life event

Table 3. Parental living status as a moderator of the relationship between stress and cardiovascular conditions

Predictors	Model 1: Life Events B (SE) [β]	Model 2: Weekly Stress Events B (SE) [β]	Model 3: Global Stress B (SE) [β]
Intercept (both dead)	.61[c] (.05)	.98[c] (.05)	.96[c] (.04)
Sex	−.05[b] (.02) [−.04]	−.05[a] (.02) [−.04]	−.04[a] (.02) [−03]
Education	−.02[c] (.01) [−.12]	−.02[c] (.01) [−.12]	−.02[c] (.01) [−12]
Age group	.19[c] (.02) [.13]	.17[c] (.03) [.11]	.18[c] (.03) [.12]
Stress	.01 (.01) [.01]	−.01 (.01) [−.01]	.01 (.01) [.04]
Both alive	−.38[c] (.04) [−.24]	−.27[c] (.04) [−.18]	−.32[c] (.04) [−.20]
Mother alive	−.16[c] (.04) [−.11]	−.15[c] (.03) [−.11]	−.13[c] (.04) [−.10]
Father alive	−.12 (.07) [−.04]	−.16[b] (.06) [−.06]	−.09 (.07) [−.03]
Both alive × stress	.03[a] (.01) [.06]	−.01 (.01) [−.02]	.01 (.01) [.01]
Mother alive × stress	.01 (.01) [.01]	.01 (.01) [.01]	−.01 (.01) [−.02]
Father alive × stress	.01 (.02) [.01]	.01 (.02) [.01]	−.01 (.01) [−.02]
Stress × age group		.01 (.01) [.01]	.01 (.01) [.01]
Both alive × stress × age group		.02 (.06) [.01]	.03 (.02) [.02]
Mother alive × stress × age group		.06[a] (.03) [.04]	.03[a] (.01) [.03]
Father alive × stress × age group		−.05 (.06) [−.02]	.01 (.03) [.01]

[a] *P*< .05.
[b] *P*<.01.
[c] *P*<.001.
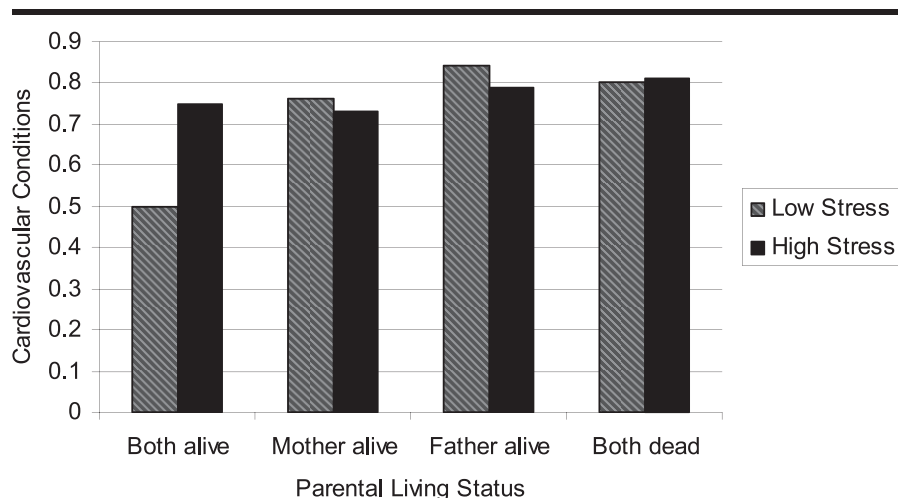
Fig 1. Life events (low, high) X parental living status interaction

stressors had the fewest CV conditions (see Figure 1). There were no differences between the parental living status groups for greater life event stressors.

Weekly Stress Events

A similar model was applied for WSI. Although there was no main effect of weekly stress events, younger age, being female, having more education, and having a mother, father, or both parents alive, was associated with fewer total CV conditions. None of the 2-way interactions were significant ($Ps>.5$), but there was a significant 3-way interaction of *mother alive* × *WSI* × *age group*. This model accounted for 10% of the variance in CV conditions. The interaction was decomposed by conducting separate models for the two age groups. For the young-old group, more education ($\beta=-.10$, $P<.001$), and having a mother ($\beta=-.14$, $P<.001$), father ($\beta=-.08$, $P=.002$), or both parents alive ($\beta=-.22$, $P<.001$) was associated with fewer CV conditions. In contrast, more education ($\beta=-.17$, $P<.001$) was the only significant predictor of CV conditions in the old-old group. Although the simple slopes were not significant, more weekly stress tended to be associated with fewer CV conditions in the young-old group ($\beta=-.02$, $P=.496$), but they were associated with more CV conditions in the old-old group ($\beta=.03$, $P=.483$).

Global Stress Total Score

Similar models were applied for the global stress total score. In a multiple regression with both parents dead as the reference group, there were no significant effects of global stress or 2-way interactions between global stress and parental living status. However, when age group was included in the interaction terms, there was a significant 3-way interaction of *mother alive* × *global stress* × *age group*. This model accounted for 11% of the variance in CV conditions. This interaction was decomposed by conducting separate models for the two age groups. In the young-old group, those with a mother alive had a negative association between global stress and CV conditions ($\beta=-.01$, $P=.859$), however, those in the old-old group with a mother alive had a positive association between global stress and CV conditions ($\beta=.01$, $P=.812$) though not significant. It is important to note that these slopes are not significantly different from zero, but they are different from each other (as reflected in the significant 3-way interaction).

Hypothesis 3

Parental living status will mediate the relationship between stress and CV conditions. We followed the steps for mediation outlined by Baron and Kenny.[16] Results from the mediation analyses are presented in Table 4. The

first step was to establish a relationship between the predictor (stress) and the outcome (CV conditions). Life event stressors ($\beta=-.05$, $P<.001$), weekly stress events ($\beta=-.04$, $P=.011$) and global stress ($\beta=-.04$, $P=.002$) were each related to CV conditions, so testing for mediation with these stress variables was possible. Next, we correlated the number of life events with each parental living status category (both dead, both alive, mother alive, father alive) in Step 2. Life events were significantly associated with both parents dead, both parents alive, and mother alive, but not with father alive. Weekly stress events were significantly associated with both parents dead and mother alive but not both parents alive or father alive. Global stress was significantly associated with all parental living status variables: both dead, both alive, mother alive, and father alive.

In the final step, we conducted a separate multiple regression for each stress type controlling for age group, sex, and education and included the parental living status categories that were significant in Step 2. We found that life events were no longer a significant predictor of CV conditions, but that having both parents dead or both parents alive were significant. In addition, we found that weekly stress events were no longer significantly related to CV conditions, but that having both parents dead or mother alive were significant. Finally, we found that global stress was still significantly related to cardiovascular conditions but that having both parents alive, father alive, or mother alive were also significant predictors of CV conditions. Note that all four parental living status categories were significant in Step 2 for global stress but we chose to use the category of both parents dead as the comparison group to keep consistent with previous models. In sum, there is evidence for mediation with life events and weekly stress events, but not with global stress.

DISCUSSION

Our findings suggest that for each measure of stress, there was a significant

Table 4. Parental living status as a mediator of the relationship between stress and cardiovascular conditions

	Life Events	Weekly Stress Events	Global Stress
Step 1: IV→ DV[d]	−.05[c]	−.04[a]	−.04[b]
Step 2: IV → Mediator			
Both dead	−.10[c]	−.07[b]	−.15[c]
Both alive	.06[b]	.01	.07[c]
Mother alive	.07[c]	.05[a]	.12[c]
Father alive	.03	.04	.04[a]
Step 3: IV + Mediator → DV			
Sex	−.04[b]	−.04[c]	−.03[a]
Age group	.13[c]	.13[c]	.14[c]
Education	−.12[c]	−.12[c]	−.12[c]
Stress	.03	−.04	.04[b]
Both dead	.09[b]	.21[c]	—
Both alive	−.12[c]	—	−.19[c]
Mother alive	−.02	.09[c]	−.10[c]
Father alive	—	—	−.04[b]

[a] $P < .05$.
[b] $P < .01$.
[c] $P < .001$.
[d] Coefficients in the first step reflect standardized regression coefficients; the second step reflects correlation coefficients; the third step reflects standardized regression coefficients.

effect for parental living status and that lower levels of stress were reported for participants for whom both parents were deceased. These findings suggest that once parents have passed on, there may be less to worry about the parent and thus produce lower reported levels of stress and weekly stress events. This is in part supported by the results that indicated that having one parent alive had an impact of reports of global perceived stress and weekly stress events. It appears caring for elderly parents may be a significant source

Our findings suggest that for each measure of stress, there was a significant effect for parental living status and that lower levels of stress were reported for participants for whom both parents were deceased.

of stress in late life. This is consistent with caregiving literature.[17,18]

Alternatively, perhaps those in the later part of life are just not as stressed as younger people.[19] Outliving one's parents makes them the senior person in the family and place them in the position of the sage, wise and calm one. In addition, sources of stress typically associated with young and/or middle-adulthood are no longer salient for most older adults (eg, child-rearing, and being concerned with career/employment goals). There may be a higher level of satisfaction with completing most of one's life goals at this stage and more emphasis on leisure activities for pleasure.

Stress, Family Type and CVD

For life events, not only are people with both parents dead less reactive to life event stressors compared to people with both parents alive, but this relationship had an impact on the number of CV conditions. The link between stress and CVD has been well studied and suggests stress as a significant risk factor for experiencing CVD and related

risk factors.[20] As risk factors accumulate with age, the risk for CV conditions increases.[21–23] To this end, we compared age groups to see if there were differences in the relationship between stress, parental living status and CV conditions. Our findings suggest that there is a life course pattern. That earlier in late life, having one or both parents alive was associated with better health or fewer CV conditions, but this was not true for those in later life. Education was found to have a protective effect (higher education was associated with fewer CV conditions) in later life.

While there was no main effect of life events on CV conditions, there was an interaction that showed differences between the parental living status groups and life event stressors. Figure 1 shows that in the group where both parents are alive, there is a difference in CV conditions between the low life events group and the high life events group.

Limitations

There are study limitations that are important to consider. First, one important variable we did not have available is ratings of early family environment. Various studies[23,24] have all discussed how early parental style can influence factors across the life course that impact biological mechanisms and stress exposure. Early life harsh parenting can lead individuals to avoid stressors later in life. Taylor et al[11] suggest people from harsh environments somewhat overreacted to stressful situations and that this was thought to arise from the impact harsh parenting had on patterning of neural structures.

The JHS is restricted to a single site, which limits generalizability beyond the southeastern region. Given the sampling strategy, JHS is not a representative sample of the African American population. However, the JHS is the largest study of CVD in middle-age and elderly African American men and women. The JHS also offers a comprehensive array of perceived stress instruments that tap

into global events, weekly hassles and major life events. This allows us to highlight the heterogeneous nature of the JHS sample, as perceived reports of psychosocial stress and selected longevity profiles may influence CVD and related risk factors differently across our sample of African Americans.

CONCLUSION

The findings show a definite familial pattern of health. It is still not completely clear why there is familial pattern to health and the psychosocial factors related to CVD health. However, given the striking pattern of differentials when both parents are alive vs dead, it is quite possible that a shared environmental component to these effects exists. What is not clear is how attributions made about ambiguous/negative circumstances may be transmitted across generations to continue to exert negative persistent influence on health outcomes and increase health disparities among African Americans. The transmission may occur passively (by sharing the same living space) or actively (parental messages to the children). More research on familial environmental effects that account for genetic effects are needed to further parse out the assumptions presented here.

REFERENCES
1. Dupre ME, Franzese AT, Parrado EA. Religious attendance and mortality: implications for the black-white mortality crossover. *Demography*. 2006;43(1):141–164.
2. Whitfield KE. Biobehavioral perspectives on health in late life. In: Whitfield KE ed. *Annual Review of Gerontology and Geriatrics, Volume 30, 2010, Focus on Biobehavioral Perspectives on Health in Late Life*. New York, NY: Springer Publishing Company, 2010;1–4.
3. Cohen S, Janicki-deverts D, Miller GE. Psychological stress and disease. *JAMA*. 2007;298(14):1685–1687.
4. Bruce MA, Beech BM, Sims M, et al. Social environmental stressors, psychological factors, and kidney disease. *J Investig Med*. 2009;57(4):583–589.
5. Sims M, Diez-Roux AV, Dudley A, et al. Perceived discrimination and hypertension among African Americans in the Jackson Heart Study. *Am J Public Health*. 2012;102 Suppl 2:S258–65.
6. Whitfield KE, Brandon DT, Robinson E, Bennett G, Merritt M, Edwards C. Sources of variability in John Henryism. *J Natl Med Assoc*. 2006;98(4):641–647.
7. Whitfield KE, Wiggins SA, Brandon DT. Genetics and health disparities: fears and realities. *J Natl Med Assoc*. 2003;95(7):539–543.
8. Carpenter MA, Crow R, Steffes M, et al. Laboratory, reading center, and coordinating center data management methods in the Jackson Heart Study. *Am J Med Sci*. 2004;328(3):131–144.
9. Fuqua SR, Wyatt SB, Andrew ME, et al. Recruiting African-American research participation in the Jackson Heart Study: methods, response rates, and sample description. *Ethn Dis*. 2005;15(4 Suppl 6):S6–18–29.
10. Payne TJ, Wyatt SB, Mosley TH, et al. Sociocultural methods in the Jackson Heart Study: conceptual and descriptive overview. *Ethn Dis*. 2005;15(4 Suppl 6):S6–38–48.
11. Taylor HA, Wilson JG, Jones DW, et al. Toward resolution of cardiovascular health disparities in African Americans: design and methods of the Jackson Heart Study. *Ethn Dis*. 2005;15(4 Suppl 6):S6–4–17.
12. Wilson JG, Rotimi CN, Ekunwe L, et al. Study design for genetic analysis in the Jackson Heart Study. *Ethn Dis*. 2005;15(4 Suppl 6):S6–30–37.
13. Brantley PJ, Jones GN. *Daily Stress Inventory: Professional Manual*. Odessa, FL: Psychological Assessment Resources; 1989.
14. Holmes TH, Rahe RH. The Social Readjustment Rating Scale. *J Psychosom Res*. 1967;11(2):213–218.
15. Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289(19):2560–2572.
16. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol*. 1986;51(6):1173–1182.
17. Hareven TK., ed. *Aging and Generational Relations over the Life Course: A Historical and Cross-Cultural Perspective*. New York: Walter de Gruyter; 1996.
18. Pearlin LI, Mullan JT, Semple SJ, Skaff MM. Caregiving and the stress process: an overview of concepts and their measures. *Gerontologist*. 1990;30(5):583–594.
19. Almeida DM, Horn MC. Is daily life more stressful during middle adulthood? In: Brim OG, Ryff CD, Kessler RC, eds. *How Healthy Are We? A National Study of Well-being at Midlife*. Chicago, IL: University of Chicago Press, 2005;425–451.
20. Whitfield KE, Weidner G, Thorpe RJ, Edwards CL. Cultural aspects of health psychology. In: Weiner IB, Nezu AM, Nezu CM, et al. *Handbook of Psychology, Health Psychology* 2nd Edition John Wiley & Sons, 2012;538–563.
21. Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, Zhu S, Markovitz JH. Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation*, 2004;(1):74–78.
22. Scuteri A, Manolio TA, Marino EK, Arnold AM, Lakatta EG. Prevalence of specific variant carotid geometric patterns and incidence of cardiovascular events in older persons. The Cardiovascular Health Study (CHS E-131). *J Am Coll Cardiol*. 2004;21;43(2):187–193.
23. Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. *Psychol Bull*. 2002;128(2):330–366.
24. Telch MJ, Ilai D, Valentiner D, Craske MG. Match-mismatch of fear, panic and performance. *Behav Res Ther*. 1994;32(7):691–700.

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