Stress-to-CVD Pathways and Midlife Mortality among U.S. Non-Hispanic White Females

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Abstract:

Overlooked in the wave of attention for higher mortality among middle-aged whites in the U.S. is evidence for rising mortality among white females in midlife by cardiovascular disease (CVD). The selectivity of this risk by age and sex suggests the potential involvement of a period effect that reshaped the population risk profile due to psychosocial stress — namely, the burdensome coupling of motherhood and work outside the home, which originated with females born during the Baby Boom and may have provoked enough stress to result in CVD morbidity by middle age. Using IPUMS-NHIS data for the 1997–2009 period, we examine how these stressors fit in the stress–CVD pathway for this generation of women. Results show that motherhood and employment are large and significant, yet mediated, predictors of midlife mortality, indicating potentially complex causal mechanisms requiring additional analyses.

1. RESEARCH PROBLEM

In 2015, Case and Deaton reported a recent and disturbing population-level trend: increasing mortality among working-age non-Hispanic whites in the United States. Their study elicited a torrent of interest from national news media, not least because of the authors' suggestion that much of the heightened risk was attributable to increases in suicide, alcohol abuse, and drug overdose. The study also prompted academic speculation about alternative explanations, since the nature of this group-specific higher mortality risk was unclear and deserved a closer look (Diez Roux 2017). Given that cardiovascular disease (CVD) is the leading cause of death among women in the U.S. (Robbins et al. 2013; McSweeney et al. 2011; Levit et al. 2011) and considering evidence for sex selectivity in midlife mortality (Montez and Zajacova 2014; Acciai and Firebaugh 2017), conditions for CVD-related death among women merit a closer look as well. Indeed, the recently reported link between CVD and higher mortality among non-Hispanic white (NHW) females (Harper et al. 2017) underlies an important question whose answer may contribute to our understanding of Case and Deaton's findings: *among contemporary white females in midlife, has CVD-based mortality increased relative to similarly-aged white males, other similarly-aged females, or the overall downward trend in midlife mortality? And, if so, why?*

Data from the National Center for Health Statistics (NCHS) compressed mortality file for the period 1999–2016 can shed light on the first part of this question. Figures 1 and 2 illustrate all-cause and CVD-related NHW crude death rates (CDR) at midlife (ages 35 to 64) by age group and sex.



Fig. 1 — Pct change in *overall* annual CDR for U.S. non-Hispanic whites (ref = 1999)



Fig. 2 — Pct change in *CVD-related* annual CDR for U.S. non-Hispanic whites (ref = 1999)

Regarding all-cause mortality (Figure 1), all non-Hispanic whites aged 35–54 had higher annual death rates from 2000 on, relative to 1999, but only females aged 45–54 experienced an *upward* trend. For CVD-related mortality specifically (Figure 2), females aged 45–54 showed annual death rates mostly unchanged from 1999, in contrast with the declining trend in all other groups except (surprisingly) females a decade younger.

If it can be argued from Figure 2 that NHW females at midlife are experiencing slightly worse CVD-related mortality, the second element of the earlier question — why — becomes focal. To that end, this study examines how CVD, CVD behavioral and medical risk factors, and U.S. non-Hispanic white female mortality at midlife interrelate. We hypothesize that *stress* is a key CVD risk factor for contemporary middle-aged female mortality. Evidence from natural experiments and controlled studies strongly posits a causal link between stress and diseases such as CVD (Cohen et al. 2007, Steptoe and Kivimäki 2013). Middle-aged females may be susceptible to stress based on the following premises: (1) Since higher mortality has so far been seen only in working-age adults (Case & Deaton 2015), chronic conditions may be a distal cause of midlife mortality; (2) CVD is often a chronic condition, resulting from years or even decades of exposure (Levit et al. 2011); (3) if contemporary middle-aged women are experiencing higher rates of CVD-related mortality, it might be reasoned that a *period-specific effect* may have led to the chronic conditions that induced those CVD-related deaths decades later; and (4) for NHW females in their 40s or 50s in the first decade of the 21st century,

obvious candidates for a period effect include the sexual and cultural revolutions of the 1960s, which loosened the norms about women's work outside the home.

This last point goes to the heart of the stress–CVD pathway as well as our argument that the first generation of women to come of age at a time of burgeoning gender equity in the labor market may also have been the first generation to pay the price for that newfound freedom with their health. This is because the rate of progress in empowering women to strive academically and work outside the home outpaced the rate of success in deprecating socially reproduced gender roles that assigned primary responsibility for maintaining the family to women and the responsibility for earning a livelihood to men (Anderson and Leslie 1991). Consequently, women continued to be more likely to do most of the child-rearing and housework even if they worked outside the home (e.g., Cinamon et al. 2002). The stress of juggling the roles of wage-earner and parent within this "second shift" scenario could feasibly exacerbate stress responses in females to a degree not experienced by most men (Low et al. 2010).

Conceptually, we conceive stress as the linchpin to CVD, with its origin in workload (see Figure 3). Workload as a source of stress is explained by the *job demand–control model*, where different levels of job demand and job control affect the psychosocial stress response (Häusser et al. 2010). The key factor is job control; work that places high demands on the worker is easier to bear when the worker has autonomy (control) in how he or she responds to the demand. Jobs with high demands but low control can significantly increase the odds of early death (Amick et al. 2002).

Fig. 3 — Conceptual diagram of the stress-CVD-midlife mortality pathway



Working mothers, unfortunately, are often denied the level of job control required to procure the necessary flexibility to accommodate schedules relating to child-rearing (Reifman et al. 1991).

Working mothers born in the Baby Boom (and reaching middle age in the early 2000s) may have experienced greater amounts of second-shift stress that, over the course of many years, heightened CVD risk factors directly via the stress response or indirectly through stress-coping behaviors such as heavy alcohol use, smoking, or physical inactivity. Either way, the ultimate result might be higher CVD-related mortality.

2. DATA AND METHODS

Data and Sample

Data for this study come from the IPUMS-NHIS public-use database (Blewitt et al. 2016), which links National Health Interview Survey (NHIS) data to death certificate records in the National Death Index (NDI). Our analytic sample (n = 793,325) encompasses the period from 1997 to 2009 and is restricted to cases with NDI follow-up eligibility.¹ Case records were kept if respondents were female, non-Hispanic white, and between 35 and 64 years old in any of three periods: 1997–99, 2002–04, or 2007–09. We subset the sample into three age ranges (35-44, 45-54, and 55-64) standing for three stages of the life course: lead-in to middle age, conventional middle age, and "late" middle age. Grouping by age allows risks to be examined from a biological (time-independent) age perspective. Other subsets were formed by restricting membership by age range in each of the three-year periods (1997–99, 2002–04, 2007–09). From these latter subsets, two birth cohort subsamples were formed by pooling the members of sequential subsets separated by age and time (e.g., the 35–44 age group in 1997–99 and the 45–54 age group in 2007–09), to identify birth cohort effects. By way of these subsamples, we can account for age, period, and cohort effects. Table 1 summarizes the size of each subset sample and the number of reported deaths in each.

Variables

Four sets of independent variables were sequentially added into the models. The baseline model was defined by three CVD-risk stressor surrogates for work and child-rearing: (1) did the respondent ever raise children at home; (2) does the respondent live with a partner; and (3) type of occupation, classified as management/executive, professional, sales/service/administrative, or other. Marriage has been shown to have a protective effect on health (e.g., Liu 2009) and, in this study, living with a partner is used to proxy for the effect of sharing some of the stress-inducing burdens in the work-family dynamic. Occupations are disaggregated because job demand and control aspects vary by occupational type (von Bonsdorff et al. 2012).

The second set of predictors consisted of four binary *health status* indicators: (1) CVD risk, indicating if the respondent was ever informed by their physician of being at risk for stroke, hypertension, diabetes, angina, heart attack, or other heart conditions; (2) physical limitation; (3) self-reported health; and 4) body mass index. To dichotomize the latter two variables, self-reported health was recoded as excellent/very good vs. all other levels and BMI was recoded as normal vs. overweight/obese (underweight persons were recoded to missing due to their small number).

¹ For the purposes of the public-use data file, NHIS respondents were eligible for follow-up if they were 18 years or older at time of survey and had given enough data for later matching (Blewitt et al. 2016).

The third group of predictors included three *health behaviors*: (1) alcohol consumption, in the form of number of drinks per event; (2) three levels of smoking history (never, former, current); and (3) three levels of exercise (none, light/moderate, vigorous). These variables differ from the binary CVD risk variable in the set of health status indicators by their greater tangibility as activities or behaviors that researchers have associated with CVD. They have often also been associated or causally linked by researchers; e.g., the link between CVD and stressful workplace conditions, high job demands, and child-rearing. By adding them to the model after accounting for health status, we can evaluate the extent to which these behaviors *mediate* any observed health status effects — in particular, the CVD-risk indicator.

Sample subset	Period	Size	Deaths	Survival
Aged 35 to 44	1997-99	14,252	433	.9696
	2002-04	11,421	161	.9859
	2007-09	8,299	26	.9969
	Pooled	33,972	620	.9817
Aged 45 to 54	1997-99	12,036	795	.9339
	2002-04	11,520	353	.9694
	2007-09	9,805	119	.9879
	Pooled	33,361	1,267	.9620
Aged 55 to 64	1997-99	8,127	1,253	.8458
	2002-04	8,409	624	.9258
	2007-09	8,035	151	.9812
	Pooled	24,571	2,028	.9175
Aged 35 to 64	1997-99	31,934	2,481	.9223
	2002-04	30,212	1,138	.9623
	2007-09	25,843	296	.9885
Birth cohort A (<i>Aged 35 to 44 in</i>	born 1953-64) 1997-99	24,057	552	.9771
Birth cohort B (Aged 45 to 54 in	born 1943-54) 1997-99	20,071	946	.9529

Table 1 — Summary statistics for the analytical subsamples

Source: NHIS-LMF via IPUMS-NHIS.

Lastly, a series of four *socioeconomic* indicators were included as controls to form the fullspecification model. Socioeconomic conditions are shown to explain mortality outcomes, typically by structuring the context for proximal determinants of health (Link and Phelan 1995; Braveman et al. 2010; Bell et al. 2018). Educational attainment (aggregated at five levels) was the primary socioeconomic indicator, as researchers have revealed a strongly graded mortality response across attainment levels (e.g., Howard and Sparks 2015; Lawrence et al. 2016). The second indicator was household income, stratified at three levels (up to \$34,999, \$35,000–74,999, and \$75,000 or more). Third was homeownership status (indicating whether the respondent's home was rented or owned/being bought), and fourth was health insurance status (has insurance vs. has no insurance).

Methods

A two-part approach was used to estimate the mortality risk profile of the sample. First, agespecific all-cause mortality risks were derived from nested Cox proportional hazard regression models. Advantages of proportional hazard models in survival analysis include their flexibility (as assumptions about data distribution are relaxed in Cox regression) and their ability to account for binary outcome variables and the time to outcome event (Powers and Xie 1999). Although the data in this study are not in true event-history form, we can still use the Cox method because the NHIS survey data are linked to NDI death certificate data such that the latter constitutes a t + 1 data set in relation to the survey data collected at time t. In other words, every survey respondent in the NHIS-LMF experiences an interval of time that starts in the survey year (1997 or later) and continues until the survival event is experienced or the year 2009, whichever comes first. Individuals who died by 2009 were modeled as having "failed" to survive; all others were right-censored after 2009.

Each of the three categories of subset data were run through each stage of the nested Cox model and diagnosed for adherence to the proportionality assumption. (Of the 32 Cox models estimated, only five were minimally in violation.) The above steps were repeated to obtain CVD-specific mortality risks using logistic regression, owing to the difficulty in assuring proportional risk in the smaller sample sizes. We defined CVD-specific mortality as any death with an underlying cardiovascular cause of death per ICD-10 categorization and any other cause of death where hypertension was flagged as a contributing cause.

3. RESULTS

Below, we summarize findings from the first of the two phases in this study.

Age, Period, and Cohort Effects: All-Cause Mortality

We first examined the risks of all-cause mortality by age, period, and cohort to evaluate the direct effects of the two main-effect stressors (work and child-rearing). In short, motherhood was more strongly associated with all-cause mortality than occupation type whether the effect was age-specific, period-specific, or cohort-based, and this association was not greatly attenuated or mediated after controlling for health conditions, health behaviors, or socioeconomic conditions.

Effects for each of the three age groups are shown in Table 2, and the hazard ratios shown are interpreted in terms of relative risk. For females aged 35–44 (Models 1A–1D), type of occupation was the dominant hazard. Women working in sales, service, administrative, or related (SSAR) or professional positions were up to twice as likely to have died by 2009 as women working in other types of job, and this risk increased when moving from the least-specified to the most-specified model. The age-specific effect for females aged 45–54 (Models 2A–2D) was largely inconclusive, apart from heavy alcohol users who showed an increased risk of death that was both statistically significant and substantial in size. The first evidence of child-rearing as a hazard is seen among women aged 55–64 (Models 3A–3D). Like younger women in certain types of job, these older women

were almost twice as likely to have died by 2009 if they had raised children. They were also at higher risk if they had earned a bachelor's degree.

The mortality risk posed by child-rearing is shown clearly in the period effects. In all three periods (1997–99, 2002–04, and 2007–09), mothers aged 35–64 were two to three times as likely to die by 2009 than childless women of the same age. The size of the effect was highly resilient even after multiple attempts to mediate it. Also worth noting are the negative effects of heavy alcohol use, higher education, and living with a partner in the younger age groups, as well as the universally protective effect of being aware of CVD risk factors. Protective effects were also seen in all health indicators except BMI when the sample is organized by birth cohort. Both cohorts had a substantially lower risk of dying if they had ever been informed of CVD conditions, which is plausible if one assumes that these individuals took steps to improve their health after being informed of their risk. By contrast, the mechanisms that account for the fact that having a physical limitation or less than very good health was also protective might be harder to describe.

Apart from these protective effects (which were also strong for females with high incomes), the two cohorts show dissimilar vulnerability to mortality. Birth cohort A (Models 7A–7D, representing females born between the mid-1950s and mid-1960s) were less susceptible by exposure to motherhood than females born a decade earlier (cohort B, Models 8A–8D), while the reverse was true for heavy alcohol use. Females in the older cohort were at no greater risk of dying if they had not completed high school, while the risk was dramatically higher for females born just a decade or so later. In fact, only females in cohort A were at remarkably greater risk of dying if they had either the highest or lowest amounts of education.

Age, Period, and Cohort Effects: CVD-related Mortality

For mortality caused directly or indirectly by cardiovascular conditions, the risks of dying were more complex. In all but one case, statistically significant coefficients among the motherhood, partnership, and occupation main effects in the baseline models — all of which were protective — were completely attenuated once the full specification models were estimated. The exception — Model 12D, 1997–99 period effect for all females aged 35–64 — is also notable for being only one of two models where BMI was significant to .05 (the other being Model 12C).

Otherwise, the principal pattern in these model results is found in the increasing risk of dying by CVD-related cause as biological age progresses. Regarding age-specific effects (Table 5), females aged 35–44 who were not current smokers showed minimal levels of risk. (Current smoking was among the few risk factors that was positively and persistently associated with a dramatically higher risk of CVD-related death regardless of model scenario.) Females aged 45–54 showed vulnerability when self-reported health was less than very good (168% higher odds than females reporting excellent health) but also demonstrated lower risk if they exercised frequently (Model 10D). Only among those in the oldest age group did CVD risk factors consistently prove to be associated with CVD-related mortality, but vigorous exercise again significantly reduced the hazard. Notably, females aged 55–64 who earned an advanced degree were highly protected — at 94% lower odds than females of the same age with only a high school diploma, these women enjoyed the strongest protective effect of any predictor in any model specification.

Table 2 — Age effects on all-cause mortality for NHW females: Cox PH model results

	TIONOIN	A	Model 11	~	Model 1C	Z	Iodel 1D		Model 2	v	Model 2B		Model 2C		Model 21		Model	3A	Model	3B	Model	3C	Model	30
<i>u</i>)	n = 13,55	- 	(n = 12,05)	6	(n = 11, 895)	<i>u</i>)	= 10,734)		(n = 13,01)	2)	n = 11,515	5	(11,311)	÷	i = 10,04	0	(n = 8, 8)	42)	(n = 7, 9)	125)	(n = 7, 7)	38)	(n = 6, 6	62)
Predictors	HR	se	HR	se	HR s	e	HR se		HR	sc	HR	se	HR s	9	HR	se	HR	se	HR	sc	HR	se	HR	sc
Ever raised children $(ref = no)$ Yes 1.	1.35	21	1.26	22	1.23 .2	2	30 .21	~	1.18	EL.	1.26 *	15 1	1. 7 <u>2</u>	6 1	20	17	1.26 *	61.	1.61 **	.26	1.62 **	.27	1.88 **	* .35
Living with partner $(ref = no)$ Yes	.86	.13	.81	.14	.1. 08.	4	95 .2,		1.16	.12	1.23	14 1	L. * 29	5 1	.35 *	18	1.05	.10	1.08	Ц	1.21	.14	1.29	.18
Occupation type (<i>ref</i> = all types not listed below)		ç		ę		ŗ	1			ŗ					ŝ	\$	ż			3	20	2	5	2
Management/executive 1.	1.03	-23	1.12	-28	1.36 .3	1	56 .5.	2	1.01	.17	1.05	20	.94	6	.79	19	16.	.13	.95	.16	.95	.16	.83	.16
Professional 1.	1.35	.28	1.53	.36	1.82 * .4	8 2.	11 * .6	~	1.20	.17	1.31	20 1	.28 .2	0 1	.18	25	1.11	.15	1.18	.17	1.12	.17	.87	.19
Sales, service, admin, and related 1.	1.41	.26	1.59 *	32	1.78 ** .3	9 2.0	05 ** .5		1.02	.13	. 76.	13	1. 86.	4	.89	15	1.07	.12	1.09	.14	66	.14	.90	.14
CVD risk (<i>ref</i> = not informed of risk) Informed of risk			1.02	21	.91 .2	0	78 .2	_			. 87	10	1. 06.	-	.93	12			.91	.10	16'	II.	.87	.12
Physical limitation (<i>ref</i> = <i>no limitation</i>) Has limitation			1.04	22	1.03 .2	с. С	77 .20	0			.95	14	I. 86.	5 1	.02	16			1.06	.13	11.11	.14	1.08	.15
Self-reported health (ref = excellent/very good) Less than "very good"			.73	.13	г Б		50 *I.	3			.92	12	. SS	4	88	15			.84	.10	.83	.10	.84	.13
Body mass index (<i>ref = in normal range</i>) Overweight or obese			1.19	22	1.30 .2	5	24 .2'	-			.94	=	. 94 I.	5	.95	13			1.05	.12	1.05	.12	1.10	.14
Alcohol consumption (<i>ref = none</i>) Light to moderate (1-3 drinks at a time)					. 92	8 1.0	01 .20						.141	5 1	24	18					1.11	.13	1.08	.14
Heavy (4+ drinks at a time)					1.07 .3	3 1.	13 .4:	2				1	54 .3	7 2	** 90"	56					1.15	.39	1.38	.53
Smoking history (ref = never smoked) Former smoker					<i>с</i> уб	ý	20	~					* 15	·	52	2					98	2	04	15
Current smoker					1.09 2	1.0	06						85 .1	1 (1	.82	13					1.09	.13	1.15	.16
Exercise (ref = does not exercise)																								
1-3 times per week					1. 69.	9	76 .2(0				Г	20 2	0 1	.23	23					11.11	.16	.95	.15
4+ times per week						7 1.	14 .20	5				I	.08 .1	5 1	.15	17					1.01	.13	1.00	.14
Highest degree $(ref = HS diploma)$ Less than high school						1	56 .5							1	29	26							.76	.14
Some college						1.	66 .5(0						I	.23	24							1.21	.20
Bachelor's degree							91 .2	10							.24	24							1.56 *	.32
Advanced degree						ч.	98 .49	•						1	.03	30							1.03	.30
Household income (<i>ref</i> = <i>up to \$34,999</i>) \$35,000-74,999							16 91							-	07	21							50	14
\$75,000 and higher							62 22	6							.82	18							.83	.19
Homeownership (ref = owned or being bought) Renting						11	20 .3(0							.93	14							1.17	.17
Health insurance (ref = has insurance)						-	- -								00	ţ							V	0.
Model for indications						.	CT CT	0							.07								1.10	кт .
Model Jit Indicators			000																				ł	
Log Itkelinoou AIC	1673		1374		-000 1343		ccc-		3899		-1008		3052		2721		483	- 1	392	4 4	376		-1.74 313	+ 10

Significance level: * $p < 0.05, \, \ast \ast \, p < 0.01, \, \ast \ast \ast \, p < 0.001$

Table 3 — Period effects on all-cause mortality for NHW females: Cox PH model results

	Model 4A $(n = 11, 815)$	Model 4B $(n = 10, 676)$	Model $(n = 10, 5)$	4C 548)	Model 4D $(n = 9,256)$	Model 5A* $(n = 12, 198)$	Model 5B* $(n = 10,778)$	Model 50 $(n = 10,58)$	2)	Model SD $(n = 8,961)$	Mo (n = n)	del 6A 11,392)	Model 6 $(n = 10, 0)$	B 13)	Model 6C $(n = 9, 814)$	Mot	lel 6D 9,221)
Predictors	HR se	HR se	HR	se	HR se	HR se	HR se	HR	se	HR se	H	k se	HR	se	HR se	HI	se
Ever raised children <i>(ref = no)</i> Yes	2.17 *** .20	2.65 *** .28	2.65 ***	* .28	2.70 *** .33	2.12 *** .28	2.24 *** .33	2.28 ***	.35	2.53 *** .43	2.18	*** .43	2.53 ***	.60	3.06 *** .79	2.82	17. ***
Living with partner $(pef = no)$ Yes	1.35 *** .12	1.37 ** .13	1.62 **	.16	1.74 *** .21	1.13 113	1.1514	1.12	.15	1.27 .22	.95	.17	.95	21	1.25 .32	1.59	.49
Occupation type (<i>ref</i> = all types not listed below) Management/executive	.95 .12	1.02 .14	.93	.14	.85 .15	1.04 .19	. 95	.85	61.	.80 .21	.95	28	.75	30	.53 .69	1.27	99.
Professional	1.17 .14	1.22 .15	11.11	.15	.9818	1.32 21	1.23 .22	1.14	.22	1.06 .28	16.	21	89.	.20	.74 .24	.49	.20
Sales, service, admin, and related	.96 .10	11. 99.	.89	.11	.86 .12	1.25 .18	1.15 .18	1.05	.18	1.09 .21	1.18	.24	.97	.25	.95 .28	.78	.27
CVD risk (<i>ref = not informed of risk</i>) Informed of risk		.72 ** .07	.72 **	.07	80. ** 17.		.58 *** .08	.58 ***	80.	.59 ** .10			.55	.13	.42 ** .12	30	*** .10
Physical limitation (<i>ref</i> = <i>no limitation</i>) Has limitation		1.14 .14	1.17	.15	1.03 .15		1.09 .15	1.01	.15	71. 76.			1.02	23	1.24 .32	1.36	.46
Self-reported health (ref = excellent/very good) Less than "very good"		.94 .10	88.	60.	.97 .12		.80 .12	.85	.14	.15			88.	30	1.25 .53	1.01	.53
Body mass index (<i>ref</i> = <i>in normal range</i>) Overweight or obese		1.01	1.13	Ħ	1.19 .13		1.22 .17	1.20	17	91. 81.1			1.13	.25	1.09 .26	1.08	.28
Alcohol consumption (ref = none) Light to moderate (1-3 drinks at a time)			1.21	.13	1.27 *15			1.30	.19	1.32 .21					.95 .24	.75	.23
Heavy (4+ drinks at a time)			2.24 ***	* .46	2.97 *** .72			1.88 *	.58	1.88 * .63					4.32 ** 2.2	4.04	2.3
Smoking history (ref = never smoked) Former smoker			.72 *	.10	.8312			.78	.15	.84 .18					.94 .33	.84	.33
Current smoker			1.20	.13	1.30 * .16			.82	.13	.94 .16					1.43 .42	1.16	.38
Exercise (ref = does not exercise) 1-3 times per week			1.39 **	LL :	1.28 .17			96.	81.	el. 1e.					.83 .28	.76	.29
4+ times per week			1.25 *	.14	1.22 .16			1.06	.17	1.00 .18					1.27 .37	1.20	.39
Highest degree $(ref = HS diploma)$ Less than high school					1.02 .17					.49 ** .12						1.05	.54
Some college					1.10 .17					.93 .19						2.70	* 1.1
Bachelor's degree					1.42 * .23					1.52 .36 90 75						1.52	.56
Household income for $f = m to $ §3 d 000)					401					07. DO.						0	
\$35,000-74,999					1.04 .13					.84 .16						1.23	.38
\$75,000 and higher					.97 .19					.73 .21						1.09	.52
Homeownership (ref = owned or being bought) Renting					1.05 .13					1.54 .27						1.78	.55
Health insurance (ref = has insurance)					16 96 1					01						60	40
Model fit indicators					17 071					61. <i>61.</i>						76.	P.
Log likelihood	-3130	-2628	-253(0	-2100	-1506	-1253	-1171		-1000	E	-577	-385		-345	1	321
AIC	6273	5277	5091		4249	3024	2525	2374		2048		1167	788		721		685

Significance level: * p < 0.05, ** p < 0.01, *** p < 0.001

Table 4 — Cohort effects on all-cause mortality for NHW females: Cox PH model results

	Model (m = 0.4	7A 548)	Model 7	7 B * 93)	Model 7	50) 50)	Model 7	*Q.	Model (n = 7	18A	Model (n = 7]	8B 26)	Model $\begin{cases} m \\ m $	8C	Mode (n = 6	18D
Predictors	HR	se	HR	se	HR	se	HR	se	HR	se	HR	se	HR	se	HR	se
Ever raised children $(ref = no)$ Yes	1.45 *	.22	133	23	1.32	.24	1.38	.28	1.69 **	** .23	1.63 **	25	1.62 **	.25	1.56 *	.27
Living with partner (ref = no) Yes	1.33	20	1.23	.21	1.30	.24	1.47	.33	1.08	.13	86.	.13	1.09	.16	1.40 *	23
Occupation type (<i>ref</i> = all types not listed below) Management/executive	1.08	.24	17.	.19	.76	.22	.72	.24	1.15	23	1.12	.26	1.34	.34	1.18	34
Professional	1.07	21	88.	.20	16.	.23	.87	.27	1.13	.18	1.07	61.	1.13	.21	66.	.25
CVD risk (ref = not informed of risk) Informed of risk	Ķ.	/1.	** 0 9.	л. П.	.92 .62 *	.12			01.1	/1'	•90. * 69.	.17	• * 89.	07. II.	.00 .64 *	.12
Physical limitation (<i>ref</i> = <i>no limitation</i>) Has limitation			69.	.14	99.	.15	.64	.15			.62 **	Ţ	* 89.	.13	.63	.13
Self-reported health (ref = excellent/very good) Less than "very good"			.55 **	II.	.52 **	E.	.46 **	Ŧ			.72	.12	.64 *	.12	* 09"	.14
Body mass index (<i>ref</i> = <i>in normal range</i>) Overweight or obese			1.31	23	1.39	.26	1.33	.28			1.14	.16	1.15	.17	1.00	.17
Alcohol consumption (ref = none) Light to moderate (1-3 drinks at a time) Heavy (4+ drinks at a time)					89. 1.01	.19 32	1.05 1.43	24 53					.92 2.36 *	.14 .80	.98 3.99 *	.17
Smoking history $(ref = never smoked)$ Former smoker					.80	.23	.86	27					.70	.14	.75	7L.
Current smoker Exercise $(ref = does not exercise)$ 1.3 times nor vool					1.17	17.	1.44	رى 20					1.0/	8I. 18	1.0/	07 6
4+ times per week					1.06	52	1.23	28					83	.16 .14	ee. 68.	.17
Highest degree $(ref = HS diploma)$ Less than high school							2.53 **	.87							1.07	.27
Some college							1.00	.32							1.69 *	.39 26
Dauticion s degree Advanced degree							3.42 **	1.7							1.76	19
Household income (ref = up to \$34,999) \$35,000-74,999							.83	.21							86.	.18
\$75,000 and higher							.45 *	.15							.54 *	.15
Homeownership (ref = owned or being bought) Renting							.73	.17							1.14	.25
Health insurance (ref = has insurance) Does not have insurance							.82	.22							.81	91.
Model fit indicators Log likelihood AIC	-83 167	5 5	-66 134	4 8	-63 129	41	-55 114	- *	-130	52 53	-104	L 4	-987 2006	2	-8 17	53

Significance level: * $p < 0.05, \, \ast \ast \, p < 0.01, \, \ast \ast \ast \, p < 0.001$

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		Model : $(n = 13, 5)$	9A (13)	Model : $(n = 11, 0)$	9B 196)	Model $(n = 10, 1)$	9C	Model 9 $(n = 9, 15)$	D 2)	Model 10A $(n = 13,012)$	Model $(n = 11)$	110 B ,515)	Model 1 $(n = 11, 3)$	0C 11)	Model 10D $(n = 10,042)$		Model 11A $(n = 8,842)$	Mod	el 11B 7,925)	Mode $(n = 1)$	111C (,311)	Model 1 $(n = 10, 0)$	11D (42)
Transmission Advances	Predictors	OR	se	OR	se	OR	se	OR	se	OR se	OR	se	OR	se	OR s	e	OR se	OR	k se	OR	se	OR	se
Understand Underst	Ever raised children $(ref = no)$ Yes	.44 *	.16	44 *	81.	44.	20	.49	.27	.74 .21	.75	23	.76	24	.74 .2	6	1.18 32	1.04	33	1.01	.32	1.18	.42
Containing void	Living with partner $(ref = no)$ Yes	1.20	.46	1.23	.54	1.28	59	2.00	1.3	.46 ** .14	5.	.18	.57	.19	.63 .2	9	.44 *** .10	54	* .14	.66	.17	.65	.23
	Occupation type (ref = all types not listed below)	* 06	2	;	01	ŝ	5	36	ş	31 + 0L	0F	ç	6		50	0	c t	10	ţ	-	46	E -	F
Section M^{+} (i) M^{-} (i) M^{-} (i) M^{-} (i) M^{+} (i)	Professional	.20 **	c1. 21.	3 6	.10	ç 4	33	c 4	38	26 *** 10 26 *** 10	./.	07. 17	co. 89.	28 28	4. CC.	ç 7	./2 74 53 * .16	16.	24	1.10	37	1.95	86
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	Sales, service, admin, and related	.30 *	.15	.46	.23	99.	34	.72	.45	.37 ** .13	.55	.20	.70	.26	.62 .2	10	.53 * .15	69.	.21	.86	-26	89.	33
Protein limit of $\gamma^{}$ module ($\gamma^{}$ module	CVD risk (ref = not informed of risk) Informed of risk			1.92	.85	2.22	1.0	2.15	1.0		1.69 *	44.	1.53	.41	1.35 .4	0		3.01	*** .82	3.03	** .83	3.15 **	1.0
Strength of manufactory of a control form	Physical limitation (<i>ref = no limitation</i>) Has limitation			1.00	ĩ	1.00	,	1.00			2.45 *	* .73	1.89 *	-59	1.73 .6	2		1.35	.35	1.19	.33	1.11	.33
Operation (q_1) q_1 q_2 q_1 q_1 q_1 q_1 q_2 q_2 q_2 q_2 q_2 q_2 q_2 <td>Self-reported health (ref = excellent/very good) Less than "very good"</td> <td></td> <td></td> <td>1.77</td> <td>77.</td> <td>1.38</td> <td>.65</td> <td>2.16</td> <td>П</td> <td></td> <td>3.32 *</td> <td>** 1.0</td> <td>2.18 *</td> <td>.70</td> <td>2.68 * 1.</td> <td>0</td> <td></td> <td>2.12</td> <td>** .58</td> <td>1.72</td> <td>47</td> <td>1.52</td> <td>.47</td>	Self-reported health (ref = excellent/very good) Less than "very good"			1.77	77.	1.38	.65	2.16	П		3.32 *	** 1.0	2.18 *	.70	2.68 * 1.	0		2.12	** .58	1.72	47	1.52	.47
Anoton formation ($\gamma'' = mod)$ large to make at time) γ'' γ''' γ'' γ''' γ''' γ'''' γ'''' γ''''' γ''''' $\gamma''''''''''''''''''''''''''''''''''''$	Body mass index (<i>ref</i> = <i>in normal range</i>) Overweight or obese			1.47	.60	1.48	.60	2.18	86.		86.	.28	1.06	.32	.93 .2	6		16.	.21	1.06	.26	1.47	.41
	Alcohol consumption $(ref = none)$ Light to moderate (1-3 drinks at a time)					.57	.27	.71	.36				.54 *	.15	. 18	S				76.	.26	1.17	.34
Solution (ref) 13	Heavy (4+ drinks at a time)					1.00	,	1.00					.94	.48	.85 .4	18				.85	.54	.71	.54
	Smoking history (ref = never smoked) Former smoker					1.99	1.3	2.70	2.0				1.35	.60	1.00 .5	9				1.68	.61	1.34	.58
Corrected (1 - does not certric) 3 <t< td=""><td>Current smoker</td><td></td><td></td><td></td><td></td><td>4.12 *</td><td>2.4</td><td>5.21 *</td><td>3.4</td><td></td><td></td><td></td><td>4.26 **1</td><td>* 1.5</td><td>4.56 *** 1.</td><td>7</td><td></td><td></td><td></td><td>4.17</td><td>** 1.4</td><td>3.91 ***</td><td>* 1.5</td></t<>	Current smoker					4.12 *	2.4	5.21 *	3.4				4.26 **1	* 1.5	4.56 *** 1.	7				4.17	** 1.4	3.91 ***	* 1.5
4 +times per week 24 11 24* 11 24* 14 24* 14 24* 14 24* 14 24* 14 24* 14 24* 14 24* 14 24* 14 24* 14 24* 24 17 24* 10 24* 24* 14 24*	Exercise (ref = does not exercise) 1-3 times per week					.42	.29	.46	.32				.67	.24	.80 .3	0				.75	.24	69.	.25
Higher degree ($r_f - IS diplow)$ Les timing) school 10 24 19 20	4+ times per week		and the office of the second second			.82	.45	<i>TT.</i>	.42				.24	.11	.28 ** .ì	4				.57	.17	.49 *	.18
Some college 50 <td>Highest degree $(ref = HS diploma)$ Less than high school</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>2.49</td> <td>1.9</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>.40 .2</td> <td>1</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>1.00</td> <td>.41</td>	Highest degree $(ref = HS diploma)$ Less than high school							2.49	1.9						.40 .2	1						1.00	.41
Backledwick degree 2.87 2.3 3.4	Some college							1.07	.89							36						LL.	.29
Avances agree 2.10 2.0 2.0 2.0 2.0 2.0 2.0 0.0	Bachelor's degree							2.87	2.3						.46	4						17.	.30
	Advanced degree							7.10	0.2							9						.00	90.
	Household Income $(ref = up to 334, 999)$ \$35,000-74,999							89.	.60						 90. 	0						.64	.21
Honeownership (rf = owned or being bough) 1.8 45 1.18 45 1.11 36 Rening Rening 1.3 38 3 3 23 23 23 23 23 23 23 23 24 93 1032 1605 367 304 304 367 1376 59 29 23 50 1376 505 13376 1377 1376 1376 <td>\$75,000 and higher</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>1.44</td> <td>1.4</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>.29 .2</td> <td>9</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>.62</td> <td>.34</td>	\$75,000 and higher							1.44	1.4						.29 .2	9						.62	.34
Health is insurance 33 25 33 25 85 36 69 29 Does not have ($rg = has insurance$) 33 25 33 25 69 29 Does not have ($rg = has insurance$) 33 25 85 36 69 29 Model (hi indicators 0357 0037 1032 1605 2067 0221 0635 076 1376 AlC 105.4 102.1 98.3 89.0 266.1 248.0 223.7 206.7 021 0655 1376	Homeownership (ref = owned or being bought) Renting							1.83	86						1.18	5						E1	36
Does not have meanine	Health insurance $(ref = has insurance)$																						
	Does not have insurance							.33	25						.85	90						69	.29
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	Model fit indicators																						
	Pseudo R ² ATC	.0435		.0532	0 -	960. 2.86	- ~	.1207 89.0		.0397 266.1	.10	32	.160:	K F	206.5		.0221 433.9	Зб	635 80.4	0. %	7.2	319.3	9.00

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	Model 12A	Model 12B	Model 12C	Model 12D	Model 13A	Model 13B	Model 13C	Model 13D	Model 14A	Model 14B	Model 14C	Model 14D
Duralistan	(C16,11 = N)	(n = 10, 0/0)	(n = 10, 248)	(0C2, V = N)	(n = 12, 198)	(n = 10, 1/8)	(760,01 = 0)	$(1cc,\delta = n)$	(n = 11, 392)	(n = 10,04.5)	(n = 9, 814)	$(1 < C, \delta = n)$
L'reactors	UK se	UK se	OIK Se	UK se	OK Se	UK SC	OK Se	OK SC	UK se	UK se	UK se	UK se
Ever raised children $(ref = no)$ Yes	.48 ** .11	.47 ** .11	.43 ** .11	.50 ** .13	. 19	.82 .26	.73 .25	.62 .25	.31 * .16	.42 .26	.46 .28	.37 .24
Living with partner $(ref = no)$ Yes	.59 ** .12	.62 * .14	. 68 .16	.73 .23	.44 ** .13	.51 *15	.65 .20	.54 .24	.44 .22	.82 .48	.74 .44	.70 .54
Occupation type (<i>ref</i> = all types not listed below) Management/executive	.44 **[3	.60 .20	.75 .25	.84 .34	.45 .20	.62 .32	.78 .45	.92 .64	.28 * .18	<i>19</i> . 86.	.90 .65	.88
Professional	.22 *** .07	.32 ** .11	.42 * .16	.62 .32	6I. IS	.90 .36	1.49 .65	1.92 1.0	.36 * .19	.55 .37	.82 .52	.92 .82
Sales, service, admin, and related	.40 *** .10	.53 * .14	.64 .17	.58 .19	.40 * .14	.50 .20	.69 .29	.88	.42 .22	.80 .50	.96 .58	.64 .41
CVD risk (ref = not informed of risk) Informed of risk		2.19 *** .48	2.39 *** .53	2.19 ** .54		3.23 *** 1.0	3.05 *** .97	2.94 ** 1.1		4.70 ** 2.7	4.13 * 2.5	3.52 * 2.1
Physical limitation $(ref = no \ limitation)$ Has limitation		1.37 .39	1.26 .37	1.41 .45		2.18 * .69	1.69 .58	1.5058		3.52 ** 1.7	2.65 * 1.3	2.55 1.4
Self-reported health (ref = excellent/very good) Less than "very good"		2.63 *** .60	2.08 ** .50	2.18 ** .61		1.97 .73	1.46 .54	1.90 .83		9.88 ** 7.4	6.27 * 4.7	13.27 * 13.
Body mass index (<i>ref</i> = <i>in normal range</i>) Overweight or obese		1.34 .29	1.57 * .35	1.73 * .42		1.18 .34	1.20 .35	1.56 .51		.66 .33	.75 .39	.78 .40
Alcohol consumption (ref = none) Light to moderate (1-3 drinks at a time)			.84 .21	.89 .24			.54 * .17	.68 .22			.58 .27	.36 .21
Heavy (4+ drinks at a time)			.83 .36	.64 .31			1.00 -	1.00 -			.76 .78	.76 .84
Smoking history (ref = never smoked) Former smoker			1.96 .70	1.90 .78			1.11 53	1.04 .58			2.13 1.4	.90 79
Current smoker			5.15 *** 1.6	5.10 *** 1.8			3.28 ** 1.1	4.19 *** 1.6			4.01 * 2.5	3.89 * 2.3
Exercise (ref = does not exercise) 1-3 times per week			1.01 .28	1.23 .35			38 .19	.16			.61 .38	.41 .35
4+ times per week			.48 .15	.50 * .17			.70 .25	.53 .21			.24 .19	.29 .22
Highest degree $(ref = HS diploma)$ Less than high school				.91 .32				2.13 1.3				.09 *
Some college				.67 .24				1.78 .86				.31 .21
Bachelor's degree Advanced degree				./3 .29				16. 10.1 .42 .35				.65 .56 1.00 -
Household income (ref = up to \$34,999)				Ę								
\$75,000 and higher				.0/ .23 .95 .50				1.28 .79				c/. 11.1 10. <i>2</i> 7.
Homeownership (ref = owned or being bought) Renting				1.48 .38				.65 .32				1.29 .90
Health insurance (<i>ref</i> = <i>has insurance</i>) Does not have insurance				.88				II. * II.				1.48 .87
Model fit indicators												
Pseudo R ² AIC	.0446 341.1	.0938 312.4	.1418 292.0	.1626 267.3	.0303 211.2	.0929 197.1	.1163 189.9	.1650 166.6	.0530 182.8	.1876 129.7	.2358 119.8	.2936 117.7

Significance level: * p < 0.05, *
* p < 0.01, *** p < 0.001

Table 7 — Cohort effects on CVD-related mortality for NHW females: Logistic regression results

	Model 15A $(n = 7,963)$	Model $(n = 7,, n)$	15B (26)	Model 1 $(n = 6,9)$	5C 98)	Model 1 $(n = 6,2)$	5D 90)	Model $(n = 9,5)$	16A 48)	Model 1 $(n = 8, 4)$	6B 93)	Model 1 $(n = 8,3)$	50)	Model $(n = 6)$	16D 308)
Predictors	OR se	OR	se	OR	sc	OR	se	OR	sc	OR	se	OR	se	OR	se
Ever raised children $(ref = no)$ Yes	.95 31	.82	30	<i>LL:</i>	.28	.87	36	.53	23	89.	29	.76	32	.76	30
Living with partner $(ref = no)$ Yes	.37 *** .10	.49 *	.15	.57	.18	.79	.30	.73	.33	77.	41	.68	.35	.81	.59
Occupation type (<i>ref</i> = all types not listed below) Management/executive	56 27	03	30	1 30	85	1 94	00	31 **	12	35	74	55	26	96	36
Professional	36 ** 14	49	51	81	37	1.38	82	19 **	1 =	.22 *	15	-24	19	21	20
Sales, service, admin, and related	.41 * .15	.56	22	.72	29	.75	.35	.39 *	.19	.62	.32	.81	4	.62	36
CVD risk (ref = not informed of risk) Informed of risk		1.13	.36	1.16	.38	.92	.32			2.33 *	89	2.36 *	76.	2.03	.87
Physical limitation (ref = no limitation) Has limitation		1.13	37	86.	35	90	.34			2.12	66.	1.86	.83	1.88	88.
Self-reported health (ref = excellent/very good) Less than "very good"		2.63 **	88.	1.96 *	.64	2.38 *	.93			3.10 **	1.3	2.02	.92	2.67	1.4
Body mass index <i>(ref = in normal range)</i> Overweight or obese		1.12	35	1.26	.40	1.03	.36			88.	.40	1.01	.50	1.28	.63
Alcohol consumption (<i>ref</i> = <i>none</i>) Light to moderate (1-3 drinks at a time)				.82	.28	.82	.30					.54	21	.44	.20
Heavy (4+ drinks at a time)				2.06	1.2	1.66	1.0					.38	.38	.41	.42
Smoking history (<i>ref</i> = <i>never smoked</i>) Former smoker				1.58	.73	1.15	.64					2.56	2.0	2.95	2.8
Current smoker				4.43 **	* 1.8	3.75 **	1.6					8.65 **	5.8	13.91 **	
Exercise (<i>ref</i> = <i>does not exercise</i>) 1-3 times per week				16	.34	1.16	.46					.81	.45	<i>TT.</i>	.50
4+ times per week				.27 **	.14	.31 *	.17					.59	.30	.67	.32
Highest degree $(ref = HS \ diploma)$ Less than high school						.43	.26							.15 *	.14
Some conege						c 4 .	77							co.	5
bacnetor s degree Advanced degree						.40 .61	-25 -49							1.00	
Household income (<i>ref</i> = up to \$34,999) \$35,000-74,999						49	21							1.30	.95
\$75,000 and higher						* 80.	60.							2.53	2.2
Homeownership (ref = owned or being bought) Renting						1.36	.50							2.18	1.4
Health insurance $(ref = has insurance)$ Does not have insurance						1.08	.45							88.	.60
Model fit indicators															
Pseudo R ² AIC	.0375 316.7	.047	8	.110 269.	0.4	.167 238.		.046 199.	5	.118	0.0	.192	8 0	.229	32
Significance level: * $p < 0.05,$ ** $p < 0.01,$ *** $p < 0.001$															

Across periods and by birth cohort, the lack of significant effect in the stressor indicators continued to hold. Professional women in birth cohort B had 79% lower odds of a CVD-related death, but this was significant only to .1. Instead, the most notable results involved health indicators (CVD risk, physical limitation, and self-reported health). With increasing age and with the passage of time, these variables became more acutely linked to the likelihood of a CVD-related death. Two striking examples: Women born in the mid-1940s to mid-1950s (birth cohort B) were almost 14 times more likely to die a CVD-related death if they were current smokers (Model 16D), and all women in the sample in the 2007–09 period were over 13 times as likely to experience the same if they reported anything less than very good health (Model 14D). Overall, examples of statistically significant protective effects to counter the rising risk grew fewer as time passed.

4. DISCUSSION AND NEXT STEPS

The recent trend in rising mortality within the U.S. non-Hispanic white population has attracted attention because of the disquieting nature of causes attributed to it. But if Case and Deaton can be attributed with popularizing the notion of "deaths of despair", it is the subsequent work of researchers such as Masters et al. (2017), Acciai and Firebaugh (2017), and others that has shown that the mechanisms of the underlying causes are more complex. In that spirit, our study aims to find out if the higher relative incidence of CVD-related mortality among contemporary NHW females in middle age is linked to long-term stressors — specifically, motherhood and work outside the home — whose burdensome "second shift" coupling originated with their generation. The transition to adulthood and early career years of women who reached middle age at the turn of the millennium were coterminous with the culturally revolutionary postwar era, when the social structures of gender roles and gender norms began to crumble, opening the door for new life opportunities. These women were the first to attempt the feat of "having it all" and set the example for all women who came after (Erwin 2010). From this perspective alone, the notion that these pioneers endured unforeseen levels of stress as they strove to balance work and family is merited (Rayburn 1986).

The findings from the first of two phases of our study, presented in this paper, provide a firstpass assessment of the link between CVD-related midlife mortality and surrogate measures for motherhood or working outside the home. While the results do not automatically make a strong case for either a direct or indirect link, they aid in framing the second phase of analysis by identifying key patterns. These include:

- 1. *Stress related to both motherhood and work outside the home substantially increases the risk of death overall, but not CVD-related death specifically.* This suggests that the risk posed by these stressors may be related with other causes of death, which will be examined in phase two of this study via causal models.
- 2. The protective effect of being aware of CVD risk factors on overall mortality risk does not extend to CVD-related mortality. This suggests that the mechanisms that lead to a lower overall risk of dying following a medical diagnosis of a CVD risk factor (for example, changing eating habits or starting to exercise) may have little effect on preventing an eventual cardiovascular failure. Put another way, a CVD risk factor, once identified, may be a condition that is not ever truly *cured*, but simply *managed*. This could easily account

for events where CVD is only listed as a *contributing* cause of death instead of the immediate cause.

- 3. Current smoking is a consistently strong predictor of CVD-related death but not of allcause mortality, suggesting that its etiological pathway is narrowly defined. In other words, most of the mortality hazard of smoking might work through cardiovascular mechanisms. This is, of course, strongly defended in the literature. The correlation between current smoking, the CVD-risk health indicator, and CVD mortality among middle-aged white females will be evaluated in phase two of the study, particularly since CVD-risk does not appear to mediate the current-smoker effect.
- 4. The types of occupations women work in until they reach middle age appear to affect allcause mortality risk only in the short-term. This may point to as-yet-unrecognized challenges in revealing the CVD-mortality pathway conceptualized here (at least from the data publicly available). It may alternately point to important age-specific — and possibly gendered — effects that represent heightened difficulties among workers in the early stages of their careers.
- 5. The protective effects of motherhood and working outside of the home on CVD-related mortality are quickly lost once other variables are controlled for. An explanation for this may reside in the encoding of artifacts like "motherhood" and "work". Absent other information, child-rearing and gainful employment imply the ability to marshal resources and be responsible factors that could certainly correlate with improved health and lower odds of early death. As soon as other variables are added, the ability for motherhood or work to account for any effect on their own simply disappears. We aim to identify the mechanism for subsuming these effects in phase two.

Phase two of the study will involve tests for motherhood and work interactions, additional hypothesis testing to rule out other causes of death through the pathways argued above, and exploration of race/ethnicity differences for women on color.

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