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Negative work-to-family spillover stress and heightened cardiovascular risk biomarkers in midlife and older adults

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ABSTRACT

Objectives: The current study aimed to investigate the health implications of negative work-to-family spillover on cardiovascular risk biomarkers.

Methods: In a large-scale cross-sectional dataset of working or self-employed midlife and older adults in the United States ($N = 1179$), we examined five biomarkers linked to cardiovascular risk, including high-density lipoprotein (HDL) cholesterol, low-density lipoprotein cholesterol, triglyceride, interleukin-6, and C-reactive protein. Negative work-to-family spillover, measured using a four-item self-reported questionnaire, was included into our model to study its association with these cardiovascular risk biomarkers.

Results: Our findings indicate a significant association between negative work-to-family spillover and cardiovascular risk biomarkers – higher triglycerides ($\beta = 0.108$, $p < .001$), interleukin-6 ($\beta = 0.065$, $p = .026$), and C-reactive protein ($\beta = 0.067$, $p = .022$), and lower HDL cholesterol ($\beta = -0.104$, $p < .001$). The associations on triglycerides ($\beta = 0.094$, $p = .001$) and HDL cholesterol ($\beta = -0.098$, $p < .001$) remained significant even after controlling numerous control variables of demographics, medication, health-status, and health-related behaviors. The findings were also consistent against slight variations in the analytic method and adjustment for multiple comparisons.

Conclusions: The current study supports the premise that spillover of work-related tensions into family life is associated with objective physiological changes that contribute to cardiovascular risk.

1. Introduction

In today's fast-paced society, the concept of work-life balance has become increasingly prominent due to the tension between professional duties and individual personal commitment [1–4]. With increasing job demands, extended working hours, and blurring of boundaries between work and personal domains due to technology [5–8], there is an increasing propensity for challenges and stressors from the workplace to permeate personal and family life, a phenomenon often referred to as negative work-to-family spillover [9]. The rising prevalence of negative work-to-family spillover is of significant concern, especially given the consistent findings from past research that outline its mental health, familial, and organizational implications [10–13]. Specifically, negative work-to-family spillover has been linked to an increase in depressive symptoms, burnout, substance abuse, and strained family relationships [9,14,15]. Concurrently, there is evidence linking negative work-to-family spillover to a decrease in work productivity and job satisfaction [10,15–18].

Beyond the mental health, familial, and organizational implications, there are growing concerns on the implication of negative work-to-family spillover on physical health conditions [9,19]. Research has consistently shown that stress exposure, similar with the kind experienced in negative work-to-family, can have a direct influence on biomarkers such as interleukin-6 and C-reactive protein [20–24]. Additionally, studies in animal models have demonstrated that prolonged stress exposure can lead to dysregulation of lipid metabolism [25–27]. It is also posited that the persistent rumination on work-related stressors triggers a prolonged activation of various biological systems that accumulate toll on the body and precipitate downstream physical health issues [28,29]. This notion aligns with the perseverative cognition hypothesis, which posits that repetitive thoughts or ruminations about past or anticipated stressors can detrimentally impact

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physiological processes and, subsequently, long-term health outcomes [30,31]. In this context, the concept of allostatic load – the cumulative wear and tear on the body's systems owing to chronic stress exposure [32,33] – becomes particularly relevant. The framework helps to understand how continuous activation of stress response systems can lead to dysregulation and, ultimately, various health impairments, including cardiovascular conditions [34–36].

In line with this, studies have shown that negative work-to-family spillover is associated with a spectrum of health outcomes, such as psychosomatic health complaints, fatigue, poor subjective health, self-reported chronic illnesses, and perceived deviations in sleep quality [19,37–39]. However, the focus has largely been on subjective health outcomes, while invaluable for capturing an individual's perception of their health [40,41], has been shown to be influenced by various factors such as personal bias, mood at the time of assessment, and cultural and sociodemographic factors [42,43]. More importantly, subjective health may not be sensitive to some physiological changes within individuals that are in the context of silent or asymptomatic conditions [44,45]. This is especially relevant for cardiovascular health, the leading causes of mortality globally [46], which often manifest silently, with physiological changes developing unnoticed for years before discernible symptoms emerge [47].

Considering the global prominence of cardiovascular diseases [48] and their potential linkage with chronic stressors [49,50], the current study aimed to investigate the health implications of negative work-to-family spillover on cardiovascular risk biomarkers. In a large-scale dataset of working or self-employed adults ($N = 1179$), we examined five biomarkers linked to cardiovascular risk, including high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triglyceride, interleukin-6, and C-reactive protein [51–53]. Given that higher levels of HDL cholesterol are indicative of lower cardiovascular risk while higher levels of LDL cholesterol, triglycerides, interleukin-6, and C-reactive protein are indicative of higher cardiovascular risk, we hypothesized that the increase in negative work-to-family spillover would be positively associated with HDL cholesterol and negatively associated with LDL cholesterol, triglycerides, interleukin-6, and C-reactive protein.

2. Method

2.1. Participants

The current study involved a cross-sectional sample of 1179 working or self-employed adults from the National Survey of Midlife Development in the United States (MIDUS) II: Biomarker Project [54] and MIDUS Refresher: Biomarker Project [55]. MIDUS II Biomarker Project, which took place between 2004 and 2009, is a subset of a large-scale longitudinal project from the original MIDUS I survey that initiated in 1995, with 7108 noninstitutionalized adults recruited through random digit sampling across 48 contiguous states of the United States. MIDUS Refresher Biomarker Project, conducted from 2012 to 2016, is derived from the MIDUS Refresher baseline cohort that commenced in 2011. Similar to MIDUS I, participants in the MIDUS Refresher survey were also selected through random digit dialing from the 48 contiguous states, evenly distributed by age and gender. The MIDUS Refresher was specifically designed to recruit new participants to replenish the original MIDUS I cohort. The average number of work hours per week for the current participants was 41.14 ($SD = 15.88$) for MIDUS II and 40.85 ($SD = 16.60$) for MIDUS Refresher.

Similar data collection methodology and identical measures were employed in both MIDUS II Biomarker Project and MIDUS Refresher: Biomarker Project. In both projects, participants attended an overnight stay at one of the three general clinical research centers in the United States, which includes University of California, Los Angeles, Georgetown University, and University of Wisconsin-Madison. During their stay, participants underwent a physical exam that included the collection of a fasting blood sample before breakfast on the second day of the participant's hospital stay [56]. The data collection received approval from the Health Sciences Institutional Review Boards at the University of Wisconsin-Madison (H-2008-0060) and all data collection procedures adhered according to the approved guidelines and regulations. Before taking part in the study, all participants have signed a written informed consent. We followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline [57]. Table 1 presents the descriptive statistics for demographics and key variables of the sample.

Table 1
Participants' Characteristics.

	n	*M*	*SD*	Range
Demographics				
Age	1179	52.64	10.98	26–83
Sex (% male)	1179	50.30%		
Race (% white)	1179	88.96%		
Education Attainment	1176	8.32	2.34	1–12
Household Income (in thousands)	1155	93.86	65.29	0–300
Health Status and Behaviors				
Diabetes (%)	1173	6.14%		
Hypertension (%)	1173	20.46%		
Stroke (%)	1173	0.34%		
Smoking (% current smoker)	1177	9.18%		
Exercise (% exercise regularly)	1179	79.30%		
Alcohol consumption (drink per month)	1179	2.66	1.52	1–6
Antihyperlipidemic Medication	1178	24.19%		
Main Variable				
Negative work-to-family spillover	1106	10.47	2.76	4–20
Cardiovascular Risk Biomarkers				
Triglycerides (mg/dL)	1171	126.75	122.73	25–3299
HDL cholesterol (mg/dL)	1169	56.18	18.50	20–137
LDL cholesterol (mg/dL)	1169	104.83	33.88	16–283
Interleukin-6 (pg/mL)	1170	2.35	2.09	0.12–21.82
C- reactive protein (ug/mL)	1169	2.63	4.87	0.03–79.30

Note. Values are shown before imputation and winsorization. Education attainment was rated on a scale of 1 (No school) to 12 (PhD, EdD, MD, LLB, LLD, JD, or other professional degree). HDL = high-density lipoprotein, LDL = low-density lipoprotein.

3. Measures

3.1. Negative work-to-family spillover

Negative work-to-family spillover was measured using a four-items "Stress at work makes you irritable at home" ($M = 3.53$, $SD = 0.82$, range = 1–5), "Job worries or problems distract you when you are at home" ($M = 3.62$, $SD = 0.87$, range = 1–5), "Your job reduces the effort you can give to activities at home" ($M = 3.15$, $SD = 0.91$, range = 1–5), and "Your job makes you feel too tired to do the things that need attention at home" ($M = 3.24$, $SD = 0.86$, range = 1–5) in MIDUS II and MIDUS Refresher. Participants rated themselves on a 5-point scale (1 = All of the time, 5 = Never). The measure was constructed by calculating the sum of the values of the 4 items. The measure was developed for the MIDUS study (9) and has been widely used and psychometrically validated by existing studies using MIDUS dataset [16,58–60]. Higher scores were coded to indicate higher negative work-to-family spillover (Cronbach's $\alpha_{\text{MIDUS II}} = 0.805$; Cronbach's $\alpha_{\text{MIDUS Refresher}} = 0.806$).

3.2. Serum lipid

In both MIDUS II: Biomarker Project and MIDUS Refresher: Biomarker Project, HDL cholesterol, triglyceride, and total cholesterol levels were determined using enzymatic colorimetric assays. For HDL cholesterol, the inter-assay and intra-assay coefficients of variability were 6.52% and 1.1–1.4% respectively in MIDUS II, and 3.56% and 1.1–1.4% respectively in MIDUS Refresher. For triglycerides, the inter-

assay and intra-assay coefficients of variability were 1.01% and 1.6% respectively in MIDUS II and 2.51% and 1.6% respectively in MIDUS Refresher. For total cholesterol, the inter-assay and intra-assay coefficients of variability were 2.65% and 0.51–0.81% respectively in MIDUS II and 4.13% and 0.51–0.81% respectively in MIDUS Refresher. LDL cholesterol was estimated using the Friedewald formula [61] from direct measurements of total cholesterol, triglycerides, and HDL cholesterol, with a formula as follows: LDL = total cholesterol – HDL – triglycerides/5. Given that the Fridewald formula begins to be unreliable when triglycerides are elevated [62], when calculating LDL cholesterol, 400 mg/dL was used as the upper limit for triglycerides if the Subject's triglyceride level is >400 mg/dL. The inter-assay coefficients of variability for LDL cholesterol in MIDUS II and MIDUS Refresher were 10.11% and 4.7%, respectively. Higher levels of HDL cholesterol are indicative of lower cardiovascular risk while higher levels of LDL cholesterol and triglycerides are indicative of higher cardiovascular risk.

3.3. C-reactive protein

C-reactive protein was measured using the BNII nephelometer (N Antiserum to Human Fibrinogen; Dade Behring, Inc., Deerfield, IL) with a particle-enhanced immunonepholometric assay range of 0.175–1100 μg/mL (reference range < 3 μg/ml) for MIDUS II and 0.164–800 μg/mL (reference range < 3 μg/ml) for MIDUS Refresher. The inter-assay and intra-assay coefficients of variability were 2.1%–5.7% and 2.3%–4.4% respectively in MIDUS II and 1.08%–4.3% and 2.3%–4.4% respectively in MIDUS Refresher. If participants' measure using BNII nephelometer were below the assay range, samples were re-assayed by immunoelectrochemiluminescence using the Meso Scale Diagnostics #K151STG high-sensitivity kit. Higher levels of C-reactive protein are indicative of higher cardiovascular risk.

3.4. Interleukin-6

Interleukin-6 was measured using the Quantikine® High-sensitivity enzyme-linked immunosorbent assay (ELISA) kit #HS600B (R&D Systems, Minneapolis, MN), with an assay range of 0.156–10 pg/mL and reference range of 0.45 to 9.96 pg/mL in both MIDUS II and MIDUS Refresher. All samples were tested in duplicate. The laboratory inter-assay and intra-assay coefficients of variance for interleukin-6 were 12.31% and 3.25%, respectively in MIDUS II, and 15.66% and 3.73% respectively in MIDUS Refresher. Higher levels of interleukin-6 are indicative of higher cardiovascular risk.

3.5. Education attainment

Education attainment was measured by asking the participants "what is the highest grade of school or year of college you completed?" through a phone interview in MIDUS II and MIDUS Refresher. It was rated on a 12-points scale (1 = No school/some grade school (1st - 6th grade); 2 = Eight grade/junior high school (7th – 8th grade); 3 = Some high school (9th – 12th grade; no diploma/no GED); 4 = GED; 5 = Graduated from high school; 6 = 1 to 2 years of college, no degree yet; 7 = 3 or more years of college, no degree yet; 8 = Graduated from a two-year college or vocational school, or associate's degree; 9 = Graduated from a four- or five-year college, or bachelor's degree; 10 = Some graduate school; 11 = Master's degree; 12 = Ph.D., ED.D., MD, DDS, LLB, LLD, JD, or other professional degree).

3.6. Use of antihyperlipidemic agent medication

The use of cholesterol medication was recorded by requiring participants to bring all their medication in their original containers during the study to ensure accuracy in MIDUS II and MIDUS Refresher. Each medication was matched through the Lexicomp® Lexi-Data database to their generic names and drug IDs, and ultimately to their therapeutic and pharmacologic class codes. The use of any form of antihyperlipidemic agent medication (e.g., HMG-CoA reductase inhibitor, fibric acid derivatives, bile acid sequestrants, cholesterol absorption inhibitors) was dummy coded (1 = *yes*, 0 = *no*).

3.7. Data analysis

The current study aimed to examine the association between negative work-to-family spillover and cardiovascular risk biomarkers indexed by HDL cholesterol, LDL cholesterol, triglyceride, interleukin-6, and C-reactive protein. For each cardiovascular risk biomarker, ordinary least squares regression was performed with negative work-to-family spillover as the independent variable. Two separate models were estimated for each criterion. In the first model, we controlled for demographic variables, such as age, gender, education attainment, household income and race, that may be associated with cardiovascular risk [63–67]. In the second model, in order to ensure the robustness of our estimate, we controlled for comorbidities of cardiovascular disease including history of hypertension, diabetes, and stroke for the past 12 months [68–70]. We also controlled for health-related behaviors variables that are associated with cardiovascular health [71,72], including current smoking status, regular exercise, and the use of antihyperlipidemic agent medication. We winsorized all our outcome indices to 3 SDs to reduce the influence of extreme outliers. MIDUS II: Biomarker Project and MIDUS Refresher: Biomarker Project do not incorporate sampling weights. Data analysis was performed using SPSS Version 25. Missing values were imputed using the expectation-maximisation (EM) algorithm (see Table 1 for the exact sample size for each variable).

4. Results

For lipid profile, as shown in Table 2, after controlling for demographics in Model 1, we found that negative work-to-family spillover significantly associated with HDL cholesterol ($\beta = -0.104$, $b = -0.704$, $SE = 0.183$, 95% CI = [−1.062, −0.345], $p < .001$) and triglycerides ($\beta = 0.108$, $b = 2.897$, $SE = 0.775$, 95% CI = [1.376, 4.418], $p < .001$), but not LDL cholesterol ($\beta = 0.036$, $b = 0.436$, $SE = 0.368$, 95% CI = [−0.286, 1.158], $p = .236$). In our Model 2, after controlling for health status and health-related behaviors, the association between negative work-to-family spillover and HDL cholesterol remained significant ($\beta = -0.098$, $b = -0.665$, $SE = 0.173$, 95% CI = [−1.003, −0.326], $p < .001$) and triglycerides ($\beta = 0.094$, $b = 2.532$, $SE = 0.775$, 95% CI = [1.011, 4.054], $p = .001$). However, in Model 2, the association between negative work-to-family spillover and LDL cholesterol was no longer significant ($\beta = 0.039$, $b = 0.480$, $SE = 0.353$, 95% CI = [−0.213, 1.174], $p = .174$).

For inflammation biomarkers, after controlling for demographics in Model 1, negative work-to-family spillover was significantly associated with interleukin-6 ($\beta = 0.065$, $b = 0.042$, $SE = 0.019$, 95% CI = [0.005, 0.078], $p = .026$) and C-reactive protein ($\beta = 0.067$, $b = 0.080$, $SE = 0.035$, 95% CI = [0.011, 0.148], $p = .022$). However, after controlling for health-status and health-related behaviors in Model 2, the association between negative work-to-family spillover and interleukin-6 was no longer significant ($\beta = 0.045$, $b = 0.029$, $SE = 0.018$, 95% CI = [−0.007, 0.065], $p = .115$) and C-reactive protein ($\beta = 0.047$, $b = 0.056$, $SE = 0.034$, 95% CI = [−0.012, 0.123], $p = .107$; see Table 3).

In addition, we also conducted sensitivity analyses to further ensure the robustness of our results in triglycerides and HDL cholesterol to slight variations in the analytic method and adjustment for multiple comparisons, such as using multiple imputation, using listwise deletion, using pairwise deletion, correcting for multiple comparisons using Bonferroni procedure, winsorizing triglycerides and HDL cholesterol to 4 *SD*, log-transforming triglycerides and HDL cholesterol levels after winsorization, analyzing only participants not taking

Table 2
Standard coefficient estimates of the negative work-to-family spillover on the lipid profile.

	Triglycerides				HDL Cholesterol				LDL Cholesterol			
	Model 1		Model 2		Model 1		Model 2		Model 1		Model 2	
	β	*b* (*SE*)	*β*	*b* (*SE*)	*B*	*b* (*SE*)	*β*	*b* (*SE*)	*β*	*b* (*SE*)	*β*	*b* (*SE*)
Negative Work-to-Family Spillover	0.108	2.90 (0.78)**	0.094	2.53 (0.78)*	−0.104	−0.704 (0.183)**	−0.098	−0.665 (0.173)	** 0.036	0.436 (0.368)	0.039	0.480 (0.353)
Age	0.016	0.11 (0.19)	−0.020	−0.13 (0.20)*	0.056	.092 (0.045)**	0.077	0.127 (0.045)*	−0.19	−0.56 (0.090)	0.088	0.265 (0.093)*
Sex (% male)	0.263	37.96 (4.06)**	0.257	37.03 (4.10)**	−0.425	−15.4 (0.956)	−0.449	−16.3 (0.912)	0.040	2.62 (1.93)	0.074	4.87 (1.87)*
Race (% white)	0.064	14.75 (6.51)*	0.073	16.80 (6.48)*	−0.020	−1.18 (1.53)	−0.037	−2.14 (1.44)	0.032	3.40 (3.09)	0.035	3.69 (2.95)
Education Attainment	−0.042	−1.30 (0.90)	−0.023	−0.71 (0.91)	0.095	0.737 (0.213)*	0.053	0.410 (0.203)	−0.068	−0.959 (0.428)	−0.060	−0.841 (0.416)
Household Income	−0.064	−0.08 (0.03)	−0.057	−0.06 (0.03)	0.009	0.003 (0.008)	−0.025	−0.007 (0.007)	−0.050	−0.025 (0.016)	−0.010	−0.005 (0.015)
Diabetes (%)			0.024	7.32 (8.68)			−0.017	−1.32 (1.93)			−0.081	−11.1 (3.96)
Hypertension (%)			0.094	16.81 (5.29)*			−0.069	−3.13 (1.18)*			0.020	1.65 (2.41)
Stroke (%)			−0.031	−38.74 (34.43)			0.014	4.34 (7.66)			−0.017	−9.78 (15.7)
Smoking (% current smoker)			0.035	8.75 (7.06)			−0.059	−3.70 (1.57)			0.084	9.55 (3.22)*
Exercise (% exercise regularly)			−0.082	−14.56 (5.03)*			0.091	4.07 (1.12)**			0.006	0.484 (2.29)
Alcohol consumption			−0.018	−0.87 (1.37)			0.285	3.41 (0.304)**			−0.018	−3.98 (0.622)
Antihyperlipidemic Medication			0.033	5.62 (5.17)			−0.083	−3.50 (1.15)*			−0.296	−22.7 (2.36)**

β = standardized regression coefficient. * $p < .05$, ** $p < .001$.

antihyperlipidemic medication, using unadjusted model, and treating the items in the negative work-to-family spillover measure separately in the model. Overall, slight variations of the analysis method did not influence the positive associations between negative work-to-family spillover and triglycerides and the negative associations between negative work-to-family spillover and HDL cholesterol, with an exception for the correlation between the item "Your job reduces the effort you can give to activities at home" and triglycerides in Model 2 (see Table S1 in the Supplemental Materials).

5. Discussion

In the present study, we examined the relationship between negative work-to-family spillover and serum biomarkers in midlife and older adults to elucidate the cardiovascular risk implications of work-life imbalance. Our findings indicate a significant association between negative work-to-family spillover and two biomarkers: higher triglycerides and lower HDL cholesterol. More importantly, the positive association between negative work-to-family spillover and triglyceride as well as the negative association between negative work-to-family spillover and HDL cholesterol remained significant after controlling for numerous control variables of demographics, medication, health-status, and health-related behaviors. The findings were also consistent against slight variations in the analytic method and adjustment for multiple comparisons. The current study supports the premise that spillover of work-related tensions into family life may manifest in objective physiological changes that contribute to cardiovascular risk.

Perseverative cognition hypothesis offers a valuable framework for understanding the heightened triglycerides and lower HDL cholesterol in individuals with high negative work-to-family spillover [30,31]. The hypothesis suggests that prolonged mental engagement with stressors, like those originating from work, can ignite a cascade of physiological responses. Indeed. chronic activation of the stress system, primarily the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) system has been shown to lead to dysregulation in lipid metabolism [73]. Over time, this dysregulation can manifest as elevated triglycerides and reduced HDL cholesterol, which has been widely recognized as crucial indicators of cardiovascular health [51].

Interestingly, the current study also observed positive associations between negative work-to-family spillover with inflammation biomarkers such as interleukin-6 and C-reactive protein. However, the results were weakened after health-status and health-related behaviors were controlled in the analyses. The findings may imply distinct pathways between negative work-to-family spillover and unhealthy lipid profile and between negative work-to-family spillover and systemic inflammation. While the association between negative work-to-family spillover and unhealthy lipid profile is likely to be driven by dysregulation in lipid metabolism as predicted by perseverative cognition hypothesis [30,31], it is plausible that the relationship between negative work-to-family spillover is explained by the higher likelihood of those experience negative work-to-family spillover to engage in poor health behaviors such as sedentary lifestyle and smoking [9,74], resulting in chronic disease such as hypertension and diabetes.

The current study does present some limitations. First, the current findings were mainly grounded in a cross-sectional design, restricting any causal inferences in the current study. Although the current study systematically controlled for a wide array of confounding variables, the risk for potential third variables cannot be entirely ruled out. Second, due to the cross-sectional design, reverse causation is still plausible where individuals with healthier health status and lower cardiovascular risk may be better equipped to manage work-to-family spillover effectively. This underscores the need for future studies to employ longitudinal study to ascertain the directionality of the observed relationship. Lastly, our study exclusively focused on midlife and older adults in the United States. For a comprehensive understanding, it is necessary for future studies to examine the relationship between negative work-to-

Table 3
Standard coefficient estimates of the negative work-to-family spillover on systemic inflammation.

	Interleukin-6				C-reactive Protein			
	Model 1		Model 2		Model 1		Model 2	
	β	*b* (*SE*)	*β*	*b* (*SE*)	*β*	*b* (*SE*)	*β*	*b* (*SE*)
Negative Work-to-Family Spillover	0.065	0.042 (0.019)	0.045	0.029 (0.018)	0.067	0.080 (0.035)	0.047	0.056 (0.034)
Age	0.228	0.036 (0.005)**	0.173	0.027 (0.005)**	0.007	0.002 (0.009)	-0.023	0.007 (0.009)
Sex (% male)	−0.020	−0.067 (0.97)	-0.022	−0.077 (0.097)	−0.0160	−1.02 (0.183)	-0.154	−0.980 (0.182)**
Race (% white)	−0.074	−0.406 (0.156)	-0.059	−0.322 (0.153)	−0.011	−0.111(0.293)	0.003	0.035 (0.287)
Education Attainment	−0.076	−0.055 (−0.022)	-0.047	−0.034 (.-022)	−0.140	−0.189 (0.041)	-0.118	−0.161 (0.041)**
Household Income	−0.042	−0.001 (0.001)	-0.029	−0.001 (0.001)	0.017	0.001 (0.001)	0.031	0.002 (0.001)
Diabetes (%)			0.073	0.522 (0.205)			0.101	1.34 (0.285)*
Hypertension (%)			0.153	0.647 (0.125)**			0.120	0.946 (0.234)**
Stroke (%)			0.003	0.091 (0.812)			0.064	3.51 (1.53)
Smoking (% current smoker)			0.033	0.195 (0.167)			-0.011	−0.122 (0.313)
Exercise (% exercise regularly)			-0.104	−0.438 (0.119)**			-0.099	−0.773 (0.223)*
Alcohol consumption			-0.074	−0.083 (0.032)			-0.072	−0.150 (0.061)
Antihyperlipidemic Medication			0.039	0.155 (0.122)			-0.037	−0.275 (0.229)

β = standardized regression coefficient. * $p < .05$, ** $p < .001$.

family spillover and cardiovascular risk biomarkers across different age groups and in various cultures to ensure generalizability.

In conclusion, the current study reveals a significant association between negative work-to-family spillover and heightened cardiovascular risk biomarkers – specifically elevated triglycerides and reduced HDL cholesterols – in midlife and older adults. The findings may suggest a tangible physiological implication of work-life imbalances among midlife and older adults. Findings from the current study underscore the urgency to address work-life imbalances, not only for psychological well-being but also for cardiovascular health, which holds broader implications for workplace policies and health interventions.

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CRediT authorship contribution statement

Andree Hartanto: Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Formal analysis, Conceptualization. **K.T.A. Sandeeshwara Kasturiratna:** Writing – review & editing, Validation, Methodology. **Meilan Hu:** Writing – review & editing, Validation. **Shu Fen Diong:** Writing – review & editing, Validation. **Verity Y.Q. Lua:** Writing – review & editing, Visualization, Validation.

Declaration of competing interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Data availability

All MIDUS datasets and documentation are archived and publicly available at the ICPSR repository (http://www.icpsr.umich.edu/) at the University of Michigan.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2024.111594.

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