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# Work–family spillover and metabolic syndrome indicators: Findings from a national sample

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## Abstract

This study examines the link between negative work–family spillover and metabolic risk factors over a 9-year period. Data from two waves of the Midlife in the United States Survey were used to explore relationships between negative work–family spillover and four indicators of metabolic syndrome—blood pressure, triglycerides, body mass index, and glucose levels. In a sample of full-time working men and women ($N = 630$), increased negative spillover at baseline significantly predicted higher body mass index nearly a decade later, with a marginally significant effect for triglyceride levels. Increases in spillover also body mass index and glucose levels at follow-up. This study extends research tying work–life spillover to health and suggests that further investigation is needed to fully understand the long-term effects of work stress.

## Keywords

body mass index, longitudinal, metabolic syndrome, negative spillover, work–family conflict

The labor force has changed significantly in recent decades, and research suggests that work–family balance is a concern for working families (Allen and Martin, 2017; Clark, 2000; Michel et al., 2011). Although some studies indicate that firm boundaries between work and personal life can be beneficial, it is often the case that these boundaries are crossed, potentially creating conflict and strain between the two domains (Clark, 2000; Grandey and Cropanzano, 1999). Such interference may include job demands that interfere with household responsibilities or the inability to devote sufficient time and energy to family and friends outside of work (Greenhaus and Beutell, 1985; Kahn et al., 1964).

A number of studies have found that job demands are less important than the degree of role spillover in predicting stress and negative health outcomes (Kossek and Ozeki, 1998; Voydanoff, 2004). Negative spillover is defined as the bidirectional tension between roles and obligations of being a caregiver, spouse and an employee (Frone et al., 1997; Kossek and Ozeki, 1998). Negative work–family spillover (WFS) occurs when job responsibilities influence workers' attitudes, capabilities, or energies toward family, creating difficultly in meeting obligations and expectations (Kanter, 1977). Since both men and women manage dual and often competing demands, negative spillover has become increasingly common, leading to distress, feeling overwhelmed, and burned out

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(Allen et al., 2000; Barnett and Baruch, 1985; Dettmers et al., 2016; Kelly et al., 2014).

## Negative spillover, role strain, and health

Previous research finds that working conditions that induce stress (including those that interfere with personal life) are associated with poorer health outcomes (Beehr and Newman, 1978; Ganster and Rosen, 2013; Greenhaus and Allen, 2011; Rosenthal and Alter, 2012). Rooted in an interrole conflict framework, negative spillover is considered a chronic stressor (or strain) that depletes resources through competing demands on time and energy (Allen et al., 2000; Dich et al., 2015). A perceived or real depletion of resources gives rise to several negative psychological states (e.g. dissatisfaction, distress, depression, and anxiety), which may have long-term consequences (Grandey and Cropanzano, 1999; Greenhaus et al., 2006; Martire et al., 2000; Woods-Giscombé, 2010). In the absence of changes in role stress or environment, health can be compromised (Halbesleben et al., 2014). We draw upon role-strain theory in addition to a broad ecosocial paradigm (Krieger, 1994, 2001) to better understand the effect negative spillover may have on metabolic risk factors over time.

### *Metabolic syndrome and work–family relationships*

The metabolic syndrome (or MetS) is comprised of multiple risk factors including high blood pressure, impaired glucose control, weight, and lipid dysregulation (Alberti et al., 2006; Grundy et al., 2004). Together, these indicators increase the risk for cardiovascular disease (CVD) and type 2 diabetes mellitus, two chronic conditions reflective of health decline and disparities in the United States (Haffner, 2006; Kaur, 2014). In general, research has only begun to link environmental and workplace stressors with physiological outcomes. The current project examines the effect of negative spillover on metabolic syndrome indicators.

A growing body of research links role strain to negative health (Fransson et al., 2015; Frone, 2000; Frone et al., 1992; McMunn et al., 2015; Nyberg et al., 2014; Spruill, 2010; Van Hedel et al., 2016). The mechanism linking role strain to metabolic risk factors is believed to operate through a stress response system, in which hormones are released in response to stressful stimuli, resulting in increased heart rate, cardiac activity, and blood pressure (McEwen, 2012; McEwen and Seeman, 1999; Terrill et al., 2012). While sympathetic responses to acute stress are well documented, whether chronic stress exposure (e.g. WFS) contributes to the elevation of risk factors over time is not clear (Hamer and Malan, 2010; Rozanski et al., 1999; Spruill, 2010). Some studies have shown that individuals who exhibit significant (or "exaggerated") metabolic responses to acute stressors are at greater risk for CVD than those who show less reactivity (Low et al., 2009; Matthews et al., 1993; Treiber et al., 2003).

While useful in illuminating a potential pathway between stress and health, work–family studies to date have largely focused on acute, rather than chronic exposure to negative spillover (Berkman et al., 2015; Shockley and Allen, 2013). Advancing research in this area requires examining the effects of negative spillover over time. Understanding whether a delayed response exists, that is, whether exposure to negative spillover over the course of several years compromises health, is important in evaluating the relationship between the chronicity of spillover effects and negative health risk (Boylan and Ryff, 2015; Mottillo et al., 2010).

### *WFS and health over time*

In considering previous research on work–family interference, 94 percent of these studies have taken a cross-sectional approach (Casper et al., 2007). Yet research indicates that trajectories of negative WFS may remain relatively stable over time (or increase for certain subgroups) before decreasing in later life (McMunn et al., 2015; Rantanen et al., 2012). No study to our knowledge has examined the effects

of work–family conflict (WFC) on multiple metabolic syndrome risk factors over a nearly 10-year period. This is a critical gap in the literature, given that work–family tensions are likely to be ongoing. Therefore, the extent to which chronic stress from work–home responsibilities impact individual risk factors over time warrants additional investigation as several questions remain. First, does negative spillover, an indicator of WFC, carry the same negative effects over an extended period of time that has been indicated by cross-sectional studies? Second, what is the relationship between negative spillover over time and the metabolic syndrome, beyond those studied to date examining only one indicator? The focus of this study examines the effect of negative spillover over a 9-year span on four primary indicators of metabolic syndrome—body mass index (BMI), triglycerides, blood glucose, and systolic blood pressure.

### Current study

This article contributes to the existing literature in several ways. First, we examine the change in negative spillover over nearly a decade. The majority of work–family studies tied to health have been cross-sectional, which constrains the researchers' abilities to draw inferences about the relationship between work and family stressors and health considerably (Matthews et al., 2014). Second, we focus on primary indicators that are likely to reflect an increased risk in chronic conditions, such as CVD and type 2 diabetes mellitus, controlling for prior history of CVD-related conditions.

This study focuses on negative WFS as the exposure of emphasis, given the potential of this body of research to influence organizational policy broadly at the workplace level. Specifically, we hypothesize that higher levels of negative spillover at baseline predict higher levels of blood pressure, BMI, triglycerides, and glucose at time two, controlling for covariates (Hypothesis 1). Second, we hypothesize a positive relationship between change in negative spillover and biomarkers at time two, such that *increases* in WFS will be associated with *higher* levels of blood pressure, BMI, triglycerides, and glucose (Hypothesis 2). Because separate analyses did not find significant interactions by sex and marital status, we do not include these variables as potential moderators. The non-significant effect of marital status and sex on negative spillover has been documented in previous studies (Grzywacz, 2000; Panisoara and Serban, 2013), suggesting that work–family strain can present burdens for both men and women and is not necessarily limited by marital conventions. To account for any unintended cross-level effects, we control for both variables. Small numbers of racial subgroups were inadequate for longitudinal analysis or drawing conclusions regarding the effect of race. We present findings for a national sample of working adults with complete data at baseline and follow-up ($N = 630$).

## Method

### Participants and procedures

Data from the National Survey of Midlife Development in the United States (MIDUS) survey were used, which includes non-institutionalized, English-speaking adults. In the first wave of data collection (1995–1996), respondents were recruited via random digit dialing (RDD) from the 48 contiguous states. MIDUS I data collection (hereafter referred to as T1) occurred between January 1995 and September 1996. The second wave (MIDUS II) began in 2004 (hereafter referred to as T2) and continued until August 2005, with 75 percent of T1 respondents participating. Detailed information on the MIDUS assessments and longitudinal retention has been previously reported (Brim et al., 2004; Radler and Ryff, 2010). The MIDUS T2 survey added several new components (named projects) to collect additional data related to physiological characteristics and sample diversity. Projects 1–5 included a variety of assessments collected during T2 only. Biological data collected from a subset of T2 respondents comprised the Biomarker Project (P4), which aimed to

investigate the long-term consequences of behavioral and psychosocial factors on health and illness. The Biomarker Project contained data from 1255 respondents. Excluding participants who did not complete survey data at MIDUS T1, data from the Biomarker Project at T2 was attained from 633 participants in the longitudinal sample. Participants with stroke ($n=3$) at either of the two time points were removed; leaving 630 participants in the final analyses. Compared with the full longitudinal MIDUS sample, the subsample was younger ($M=42.7$ in subsample, $M=46.8$, in the original sample) and had a higher educational level. Approximately 93.2 percent of the subsample identified as White, 2.5 percent as African-American/Black, 0.3 percent as Asian, 0.6 percent as Native American, 0.5 percent as multiracial, and 2.1 percent as other. Women (49.8%; $N=314$) and men (50.2%; $N=316$) were nearly equally represented in the sample.

## Measures

*Negative spillover.* Negative spillover, assessed at T1 and T2 by a 4-item scale created for the MIDUS study (Grzywacz and Marks, 2000), measures perceptions that work interferes with home functions and responsibilities (Grzywacz, 2000). Sample items include "Your job reduces the effort you can give to activities at home" and "Job worries or problems distract you when you are at home." Responses were rated on a 5-point Likert-type scale. Appropriate items were reverse coded so that higher scores indicated a greater degree of spillover. A composite score summed the scores of all four individual items and was used to represent negative spillover in the analysis (see Table 1 for means and standard deviations). Internal consistencies at each time point was T1 $\alpha = .82$ and T2 $\alpha = .81$, respectively.

Biomarker data were collected from three General Clinical Research Centers (at the University of California, Los Angeles (UCLA), the University of Wisconsin, and Georgetown University) by trained staff members or clinicians. Participants' height, weight, and blood pressure were measured as part of a physical exam. BMI was assessed directly using height (cm) and weight (kg) measurements. Triglycerides, glucose levels, and blood pressure were assessed from lipid assay panels collected during the clinical encounter. Full details about protocol and procedures have been previously detailed (Dienberg Love et al., 2010).

*Covariates.* Control variables were age (years), sex, educational level (scores range from 1 = no school/some grade school to 12 = professional degree), T1 financial situation, T1 marital status, T1 use of blood pressure medication, heart conditions at T1 and T2, smoking and drinking history, as well as physical activity at T1 and T2. T1 blood pressure medication was measured by a question asking participants to indicate whether they were taking prescribed medication for high blood pressure (1 = Yes, 0 = No). Heart trouble was identified by asking the participants "Have you ever had heart trouble suspected or confirmed by a doctor?" (1 = Yes, 0 = No) at both T1 and T2.

## Data analysis

Analyses involved two steps to address primary hypotheses. First, T1 indicators were used to predict T2 biomarker outcomes (H1). A series of logistic regression models were constructed with covariates and T1 baseline levels of spillover, predicting systolic blood pressure, BMI, blood glucose, and triglycerides at T2. In a second analysis, covariates and baseline levels of negative spillover (T1) were entered with degree of change in negative spillover between T1 and T2 modeled against outcomes of interest (H2). In both models, outcomes were entered according to "high-risk" cutoffs of clinical significance (Alberti et al., 2006; Grundy et al., 2004). Since the metabolic syndrome confers increased risk of disease dependent upon higher levels of indicators, outcome variables were dichotomized to reflect increased risk using cutoff levels consistent with conventions from the American Heart Association, such as levels of BMI over 25 to confer overweight status and fasting plasma glu-

**Table 1.** Mean, standard deviations, and correlations of study variables.

| | *M* (*SD*) | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Systolic BP T2 | 123.56 (17.47) | – | | | | | | | | | | | | | | | | | | | | |
| 2. BMI T2 | 29.29 (5.93) | .24** | – | | | | | | | | | | | | | | | | | | | |
| 3. Triglycerides T2 | 136.89 (151.0) | .07 | .17** | – | | | | | | | | | | | | | | | | | | |
| 4. Glucose T2 | 99.86 (25.61) | .06 | .17** | .45** | – | | | | | | | | | | | 1 | | | | | | |
| 5. WFC T1 | 10.64 (2.64) | −.05 | .11* | .05 | .002 | – | | | | | | | | | | | | | | | | |
| 6. WFC change | −0.30 (2.83) | −.10* | −.04 | .06 | .03 | −.53** | – | | | | | | | | | | | | | | | |
| 7. Age T1 | 43.73 (9.77) | .27** | −.02 | 0 | .11** | −.08 | −.16** | – | | | | | | | | | | | | | | |
| 8. Sex (male) | 0.50 (0.50) | .12** | .07 | .16** | .17** | .02 | −.02 | .04 | – | | | | | | | | | | | | | |
| 9. Separated/divorced T1 | 0.16 (0.36) | .01 | 0 | −.03 | −.05 | −.06 | −.03 | .13** | −.17** | – | | | | | | | | | | | | |
| 10. Not married T2 | 0.14 (0.35) | −.07 | .02 | −.01 | −.09* | .03 | .06 | −.30** | .04 | −.17** | – | | | | | | | | | | | |
| 11. Education level T1 | 7.83 (2.34) | −.11** | −.11** | −.06 | −.04 | .08* | −.05 | .02 | .03 | −.04 | .13** | – | | | | | | | | | | |
| 12. Financial situation T1 | 6.18 (2.06) | .03 | −.10* | −.04 | −.01 | −.17** | −.01 | .19** | .07 | −.11** | −.08* | .09* | – | | | | | | | | | |
| 13. Smoking history T1 | 0.59 (0.49) | −.01 | 0 | .06 | .06 | −.04 | −.02 | .16** | .01 | .09 | −.08 | −.19** | 0 | – | | | | | | | | |
| 14. Smoking history T2 | 0.58 (0.49) | .01 | .01 | .07 | .05 | −.04 | −.03 | .13** | .03 | .14** | −.09 | −.22** | −.02 | .91** | – | | | | | | | |
| 15. Drinking history T1 | 0.41 (0.49) | .01 | .05 | .07 | .12** | .08 | .02 | −.07 | .26** | −.04 | .08 | .02 | −.08 | .20** | .17** | – | | | | | | |
| 16. Drinking history T2 | 0.71 (0.46) | −.09* | −.13** | .02 | −.03 | .05 | −.01 | −.01 | −.03 | −.04 | .01 | .10* | .14** | .01 | −.01 | .13** | – | | | | | |
| 17. Heart trouble T1 | 0.07 (0.26) | .11** | .04 | .03 | −.01 | .13** | −.02 | .05 | .03 | .05 | −.08 | −.01 | −.04 | −.03 | .03 | 0 | .03 | – | | | | |
| 18. Heart trouble T2 | 0.11 (0.32) | .10* | .06 | .10* | .10* | .08 | −.05 | .25** | .05 | .01 | −.09* | −.04 | .01 | .05 | .07 | .02 | .03 | .27** | – | | | |
| 19. BP medication | 0.07 (0.26) | .11** | .06 | −.03 | .07 | −.06 | −.07 | .23** | .01 | −.03 | −.09* | .00 | .06 | 0 | −.01 | .06 | .01 | .07 | .09* | – | | |
| 20. Physical activity T1 | 4.53 (1.48) | .02 | −.07 | .01 | −.05 | −.08* | .10* | −.21** | .21** | .00 | .10* | .05 | −.03 | −.05 | −.04 | .11** | .04 | −.08 | −.11** | −.07 | – | |
| 21. Physical activity T2 | 3.04 (1.36) | .04 | −.06 | .04 | −.01 | .02 | .04 | −.12** | .19** | −.09* | .06 | −.04 | −.03 | .01 | .02 | .07 | .091* | .01 | .00 | −.02 | .36** | – |
| *N* total = 630 | | | | | | | | | | | | | | | | | | | | | | |

BP: blood pressure; BMI: body mass index; SD: standard deviation; WFC: work–family conflict; T1: Time 1 (Year 1995); T2: Time 2 (Year 2003).
*$p < .05$; **$p < .001$.
Biomarker data.

cose levels higher than 110 to indicate pre-diabetes (1 = higher than 110 mg/dL, 0 = otherwise).

Change scores were used in the second step, as they often are, when only two time points are available (Peter et al., 1993; Rogosa, 1995; Turiano et al., 2012). Change scores were calculated using negative spillover at T1 subtracted from T2 scores. Individuals with positive change scores were those for whom the T2 score was higher than the T1 score (i.e. spillover increased over time). Conversely, negative change scores indicate a T2 score lower than the T1 evaluation of spillover (i.e. spillover decreased over time).

## Results

Overall, there was a positive change (increase over time) in spillover between T1 and T2 in this study. Correlations, means, and standard deviations for key study variables are noted in Table 1.

### *Systolic blood pressure at T2*

After adjusting for covariates, negative spillover at T1 did not significantly predict higher blood pressure at T2, nor did increases in spillover predict elevated blood pressure (see Table 2).

### *BMI at T2*

In this model, negative spillover at T1 predicted higher BMI at T2, $B$ ($SE$) = 0.19 (0.06), $p < .001$, adjusting for covariates. Increase in spillover (indicated by positive change score) from T1 to T2 also predicted higher BMI at T2 ($p < .05$).

### *Triglycerides at T2*

Negative spillover at T1 marginally increased the likelihood for raised triglycerides at T2. Increased spillover over time was not significantly associated with higher triglyceride levels at T2.

### *Blood glucose at T2*

Negative spillover at T1 was not significantly associated with higher blood glucose levels at T2; however, increase in spillover did predict higher blood glucose at T2, $B$ ($SE$) = 0.14 (0.07), $p < .05$.

Taken together, baseline levels of negative spillover at T1 predicted higher levels of BMI and were marginally associated with elevated triglycerides at T2 while increases in negative spillover between T1 and T2 predicted higher levels of BMI and blood glucose levels at T2.

## Discussion

In a national study of midlife working adults, we examined the relationship between negative WFS and metabolic syndrome biomarkers at a 9-year follow-up. Results from this study extend research on WFC and aid in better understanding the impact of negative spillover over time on indicators of health. Higher levels of spillover at baseline significantly predict increased BMI nearly a decade later, and increases in spillover predict increased BMI and high blood glucose levels at follow-up. Interestingly, negative spillover did have a significant effect on blood pressure. Overall, this study indicates the potential of negative spillover from work to family life to have an impact on metabolic risk factors years later, particularly for BMI. This research has the potential to broaden the scope of investigations of negative spillover and chronic disease biomarkers.

Although modest effects were observed, they should be expected given that weaker associations between self-report measures (e.g. perceived negative spillover) and objective measures have been well documented (Herbert and Cohen, 1993). Compared with self-reported measures, objective measures are more strongly associated with physiological and anthropometric biomarkers (Atienza et al., 2011; Lynch et al., 2010). Therefore, the ability to track these associations using subjective perceptions of negative spillover 9 years apart suggests that the relationships between perceived work–family spillover and physiological outcomes are moderately strong. Regarding the clinical significance of our findings, even modest changes in BMI and blood glucose levels may confer

**Table 2.** Predicting biomarkers at T2 from negative spillover (negWF) at T1 and change in negative spillover from T1 to T2 (negWF change).

| | Systolic blood pressure | | | BMI | | | Triglycerides | | | Glucose | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *B* | *SE* | $e^B$ | *B* | *SE* | $e^B$ | *B* | *SE* | $e^B$ | *B* | *SE* | $e^B$ |
| Model 1 | Nagelkerke $R^2$=.15 | Nagelkerke $R^2$=.10 | Nagelkerke $R^2$=.19 | Nagelkerke $R^2$=.13 | | | | | | | | |
| Age | 0.05** | 0.01 | 1.05 | −0.02 | 0.01 | 0.98 | −0.01 | 0.02 | 0.99 | 0 | 0.02 | 1 |
| Male | 0.37 | 0.25 | 1.44 | 0.42† | 0.25 | 1.52 | 1.60* | 0.42 | 4.97 | 0.63 | 0.38 | 1.88 |
| Separated/divorced/widowed T1 | 0.08 | 0.34 | 1.08 | 0.24 | 0.33 | 1.27 | −1.23† | 0.66 | 0.29 | −0.08 | 0.48 | 0.93 |
| Not married T2 | 0.38 | 0.34 | 1.47 | −0.15 | 0.34 | 0.86 | 0.22 | 0.47 | 1.24 | −1.04 | 0.65 | 0.35 |
| Education T1 | −0.15* | 0.05 | 0.86 | −0.03 | 0.05 | 0.97 | −0.24* | 0.08 | 0.79 | 0.02 | 0.07 | 1.02 |
| Financial T1 | 0 | 0.06 | 1.00 | −0.12* | 0.06 | 0.88 | 0 | 0.09 | 1.00 | 0.09 | 0.09 | 1.09 |
| Smoking history T1 | −0.28 | 0.56 | 0.76 | −0.55 | 0.57 | 0.58 | 0.67 | 0.81 | 1.96 | 0.5 | 0.83 | 1.65 |
| Smoking history T2 | −0.09 | 0.55 | 0.92 | 0.62 | 0.57 | 1.85 | −0.24 | 0.82 | 0.79 | 0.25 | 0.82 | 1.28 |
| Drinking history T1 | 0.22 | 0.25 | 1.24 | 0.19 | 0.24 | 1.21 | 0.02 | 0.34 | 1.02 | 0.17 | 0.34 | 1.18 |
| Drinking history T2 | −0.26 | 0.27 | 0.77 | −0.62* | 0.26 | 0.54 | 0.09 | 0.37 | 1.10 | −0.51 | 0.36 | 0.6 |
| Heart trouble T1 | 1.55* | 0.60 | 4.72 | 0.42 | 0.49 | 1.52 | 0.95 | 0.57 | 2.58 | −0.44 | 0.65 | 0.64 |
| Heart trouble T2 | −0.06 | 0.39 | 0.94 | 0.36 | 0.38 | 1.43 | 0.06 | 0.49 | 1.06 | 1.18* | 0.44 | 3.26 |
| BP medication T1 | 0.65 | 0.45 | 1.92 | 0.74† | 0.42 | 2.09 | −0.93 | 0.73 | 0.39 | 0.15 | 0.54 | 1.17 |
| Physical activity T1 | 0.10 | 0.09 | 1.11 | −0.10 | .008 | .090 | −0.08 | 0.12 | 0.92 | 0.01 | 0.12 | 1.01 |
| Physical activity T2 | −0.03 | 0.09 | 0.97 | −0.05 | 0.09 | 0.95 | −0.17 | 0.14 | 0.84 | −0.01 | 0.13 | 0.99 |
| Model 2 | Nagelkerke $R^2$=.17 | | | Nagelkerke $R^2$=.14 | | | Nagelkerke $R^2$=.21 | | | Nagelkerke $R^2$=.15 | | |
| Age | 0.05** | 0.05 | 1.05 | −0.01 | 0.01 | 0.99 | −0.01 | 0.02 | 0.99 | 0.01 | 0.02 | 1.01 |
| Male | 0.34 | 0.34 | 1.40 | 0.46† | 0.26 | 1.58 | 1.58* | 0.43 | 4.85 | 0.68† | 0.39 | 1.97 |
| Separated/divorced/widowed T1 | 0.02 | 0.02 | 1.02 | 0.32 | 0.34 | 1.38 | −1.19† | 0.67 | 0.30 | 0.04 | 0.49 | 1.04 |
| Not married T2 | 0.40 | 0.40 | 1.50 | −0.15 | 0.34 | 0.86 | 0.19 | 0.47 | 1.21 | −1.06 | 0.65 | 0.35 |
| Education T1 | −0.16 ** | −0.16 | 0.86 | −0.04 | 0.05 | 0.96 | −0.25* | 0.08 | 0.78 | 0.02 | 0.07 | 1.02 |
| Financial T1 | 0.01 | 0.01 | 1.01 | −0.09 | 0.06 | 0.92 | 0.03 | 0.09 | 1.03 | 0.1 | 0.1 | 1.1 |
| Smoking history T1 | −0.31 | −0.31 | 0.73 | −0.63 | 0.59 | 0.53 | 0.66 | 0.81 | 1.94 | 0.5 | 0.83 | 1.65 |
| Smoking history T2 | −0.07 | −0.07 | 0.94 | 0.77 | 0.59 | 2.17 | −0.17 | 0.81 | 0.84 | 0.3 | 0.83 | 1.35 |
| Drinking history T1 | 0.24 | 0.24 | 1.28 | 0.10 | 0.24 | 1.10 | −0.06 | 0.35 | 0.94 | 0.1 | 0.35 | 1.1 |
| Drinking history T2 | −0.25 | −0.25 | 0.78 | −0.70* | 0.27 | 0.50 | 0.08 | 0.37 | 1.08 | −0.55 | 0.36 | 0.58 |
| Heart trouble T1 | 1.61* | 1.61 | 5.03 | 0.26 | 0.50 | 1.30 | 0.82 | 0.59 | 2.27 | −0.59 | 0.67 | 0.56 |
| Heart trouble T2 | −0.04 | −0.04 | 0.96 | 0.19 | 0.38 | 1.21 | −0.08 | 0.50 | 0.92 | 1.14* | 0.45 | 3.13 |
| BP medication T1 | 0.61 | 0.61 | 1.84 | 0.95* | 0.43 | 2.58 | −0.76 | 0.75 | 0.47 | 0.33 | 0.55 | 1.39 |
| Physical activity T1 | 0.11 | 0.11 | 1.11 | −0.07 | 0.09 | 0.93 | −0.05 | 0.13 | 0.96 | 0.02 | 0.12 | 1.02 |
| Physical activity T2 | −0.03 | −0.03 | 0.97 | −0.08 | 0.09 | 0.92 | −0.19 | 0.14 | 0.83 | −0.02 | 0.13 | 0.98 |
| WFC T1 | 0.02 | −0.02 | 0.98 | 0.19** | 0.06 | 1.21 | 0.15† | 0.08 | 1.16 | 0.10 | 0.08 | 1.1 |
| WFC change | 0.09† | −0.09 | 0.92 | 0.12* | 0.05 | 1.13 | 0.09 | 0.07 | 1.09 | 0.14* | 0.07 | 1.16 |

T1: Time 1 (Year 1995); T2: Time 2 (Year 2003); $e^B$: exponentiated *B*; BMI: body mass index; SE: standard error; WFC: work–family conflict.
†.05 < *p* < .08; *$p$ < .05; **$p$ < .001.

increased risk of diabetes and vascular-related disease in later life, especially when considered with other risk factors (Alberti et al., 2006; Brotman et al., 2007; Everson et al., 1996). Additional longitudinal investigations on the effects of negative spillover are needed to clarify the role of occupational stressors in the etiology of disease risk.

Considerable research has been devoted to the study of work spillover effects, yet little of this research has examined the effect of spillover over time. The effects of WFS are likely to develop over years, often decades, and cross-sectional studies can be limited in discerning latent risk and lag effects. More importantly, this study suggests lasting effects of WFS in that baseline measures are related to key indices of health in a national sample nearly a decade later. This research supports similar findings from smaller samples conducted in occupation-specific studies (Jacobsen et al., 2014). Overall, our results emphasize the importance of exploring more complicated dimensions of the work-family interface, including how it impacts multiple indicators of health over time and to what extent these effects might be mediated by other protective or risk factors.

Several limitations warrant mention. Of primary concern is the lack of biomarker assessments at MIDUS T1, precluding testing of longitudinal relationships in physiological outcomes. Therefore, causality cannot be determined. Second, given the selected sampling of participants with both longitudinal and biomarker data, there was limited representation of individuals from racial and ethnic minority groups. Third, self-report measures were used for assessing WFS. While it is important to consider potential bias associated with using self-report measures, this limitation represents a broad challenge within the work–family literature (Allen, 2013). Furthermore, this investigation was focused on understanding the perceptions of spillover and therefore self-report is likely appropriate in distilling the effect of *perceived* stress from work and family. Despite some limitations, the observed associations support models of work-related strain, contributing to theoretical frames that link occupational stressors to health.

## Future directions

Given these findings, one direction for future research is a continuation of studies linking occupational stressors, particularly stress related to interferences between work and family, to health and risk factors for health. There is an especially critical need to highlight potential mechanisms through which WFC is consequential for health, expanding the depth of existing theoretical frameworks. Therefore, future studies may benefit from attending to both physiological and psychological manifestations of WFS over time and exploring psychosocial processes.

In particular, both baseline levels and increases in negative spillover over time predict elevated BMI, indicating that weight fluctuations may be associated with work–life balance, as recent research suggests (Lacey et al., 2017). Additional considerations, such as age of transition into the labor force, organizational and family support, consistency of ties to employment, and behavioral or stress-related pathways associated with these transitions may be important. Integrating this research requires a contextual approach that frames what has been described as "conflict," "spillover," and "interference" as a cumulative stressor, all of which are likely to indicate a chronic burden yielding wide-ranging effects (Edwards and Rothbard, 2000). Linking occupational stress with biomedical and psychological research represents an important step in advancing the implications for occupational research on health generally.

At the same time, additional attention is needed to understand preventive pathways that may stem the effects of WFC. In recent years, researchers have developed interventions focused on training supervisors in family support strategies, yielding benefits for employee physical health, job satisfaction, and reduced turnover (Hammer et al., 2011; Odle-Dusseau et al., 2016). Other studies have found that alternative work schedule initiatives and in-house childcare programs were beneficial to

workers' job quality and overall well-being (Kelly et al., 2014; Moen et al., 2011a, 2011b). Therefore, in spite of its complex nature, negative WFS can be intervened upon. In addition to framing organizational change as a benefit for employers and to some extent, job quality, a primary aim should measure how these changes impact employee health (Hwang and Hong, 2012; Kivimäki and Kawachi, 2015). To what extent modifications to workplace policies, degree of autonomy (e.g. schedule control), social support, or integration between home and family results in reducing conflict long term will be important considerations in future research.

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## Appendix 1

Authors used the publicly available MIDUS database for this study. A description of the MIDUS study can be found at http://midus.wisc.edu/. A bibliography of journal articles, working papers, conference presentations, and dissertations using these data is available at http://midus.wisc.edu/findings/index.php. All variables and relationships examined in this article have not been examined in any previous or current articles, or to the best of our knowledge, in any papers that will be under review soon.

| Variables in the complete dataset | MS1 (Status–Published) | MS2 (Status–Current) | MS3 (Status–Planned) |
|---|---|---|---|
| Negative work–family conflict T1 | | X | X |
| Negative work–family conflict T2 | X | X | X |
| Systolic blood pressure T2 | | X | |
| BMI T2 | | X | |
| Blood triglycerides T2 | | X | |
| Control: Age | X | X | X |
| Control: Gender | X | X | X |
| Control: Marital status | X | X | X |
| Control: Educational level | X | X | X |
| Control: Financial situation | | X | X |
| Control: Smoking history | X | X | X |
| Control: Drinking history | X | X | X |
| Control: History of heart problems | | X | |
| Control: History of stroke | | X | |
| Secondary control | X | | |
| Psychological well-being | X | | |
| Depression | | | X |
| Anxiety | | | X |
| Cognitive decline | | | X |
| Positive reappraisal | X | | |

BMI: body mass index.