Cardiometabolic Risks Associated with Work-to-Family Conflict: Findings from the Work Family Health Network

Abstract:

Introduction: Work and family conflict is increasingly pervasive in today's workforce and is associated with worse cardiovascular health. However, the extent to which this combined stress relates to individual risk factors of cardiovascular disease overtime is unclear. This study leverages a randomized field experiment and investigates the observational associations of work and family stressors with five cardiovascular risk factors (blood pressure, glycosylated hemoglobin, cholesterol, body mass index (BMI) and cigarette consumption) used to establish a recently developed cardiometabolic risk score (CRS) over an 18 month study period. We hypothesize that work-to-family conflict (WTFC) will be associated with worse markers of cardiometabolic risk at baseline and that high WTFC will be associated with a faster rate of increase in cardiometabolic risk over time, compared to low WTFC.

Methods: The current analyses utilized four waves of data (baseline, 6 months, 12 months and 18 months) among 1,524, predominantly female employees working in an extended care setting. Employees provided biological markers through dried blood spots as well as self-reported data on a variety of sociodemographic, health and work and family variables. We estimated multilevel linear models that accounted for multiple measures per employee as well as nesting of employees within worksites to test whether work-to-family conflict (WTFC) at baseline was associated with worse cardiometabolic outcomes (CRS and individual risk factors) over an 18 month study period. Secondarily, we tested the effects of family-to-work conflict (FTWC) at baseline on these outcomes as well.

Results: WTFC was positively associated with BMI at baseline (β=0.53, p=0.02, CI=(0.08, 0.98)) and in pooled outcome analyses across all four study waves (β=0.59, p=0.01, CI=(0.12, 1.04)). WTFC was associated with greater increases in BMI over time (β=0.08, p=0.0007, CI=(0.03, 0.15)) as well. Higher levels of WTFC were associated with lower HDL cholesterol averaged across waves (β=-0.32, p=0.01, CI=(-0.57, -0.08)) but not with the individual factors of glycosylated hemoglobin, total cholesterol, blood pressure or cigarette smoking at baseline or over the course of the study nor with CRS in pooled or longitudinal analyses.

Conclusion: Our data suggest that WTFC is consistently associated with BMI over the 18 month study period. We speculate that BMI, which is linked to potentially malleable behaviors, are more closely linked to interrole conflict than biological markers. We recommend that future research continue to clarify the effects of work and family stressors on individual risk factors for CVD in a variety of occupational settings.

Introduction:

Employees report that dueling demands both at work and home are increasingly common [1], and research indicates that this interrole conflict is associated with poorer employee health, including worse cardiovascular outcomes [2-4]. However, the extent to which stress at home and at work relate to individual risk factors of cardiovascular disease over time warrants additional investigation. The current study examined associations between work and family conflict (WTFC), a measure intended to reflect perceived stress arising due to conflicting demands in these two realms of life, and a variety of behavioral and biological markers related to risk of cardiovascular disease (CVD). We test these relationships with an observational design based on a study assessing a randomized field experiment. This research largely draws on job strain theory

[5, 6], specifically the Demand-Control-Support model [7]. The model considers the combination of job demands and job control believed to produce a sense of strain and incorporates workplace social support hypothesized to combat these strains. The current study focuses on pressures both at work and in the home. A larger body of research has examined work strain and CVD outcomes specifically, and this literature focuses predominantly on Caucasian, male samples. We extended this work and investigated these relationships in a young, predominantly female and racially diverse occupational cohort of extended care employees. Because cardiovascular disease often develops gradually, biological markers of CVD risk serve as sensitive and meaningful pre-disease pathways for otherwise latent illness. They provide a useful assessment approach by which the effects of psychosocial stress on cardiovascular outcomes may be understood in a relatively healthy study population. In doing so, we built upon existing research that has examined the effects of work and family demands on "objective" cardiovascular measures [8, 9] and offer a longitudinal perspective on the link between WTFC and CVD link in a unique study population.

Changes in labor dynamics and related transformations in the home have prompted an increasing number of Americans to experience work and family strains simultaneously. Female labor force participation has risen significantly in recent years (42% to 57% from 1950 to 2007), with the most pronounced increases among working mothers (47% to 71% from 1975 to 2007). National data also indicate that, while men and women are less likely to endorse traditional gender roles today compared to forty years ago, increases in self-reported work and life strains have been reported by all employed parents [1]. The incompatibility of home and professional life is often referred to as "work-family conflict," a term which Greenhaus and Beutell

introduced almost thirty years ago to describe "a form of interrole conflict in which the role pressures from the work and family domains are mutually incompatible in some respect" [10]. Frone and colleagues further posited that work-family conflict is a bidirectional phenomenon (operating work-to-family and family-to-work), presenting itself when efforts to fulfill responsibilities in one realm interfere with the ability to succeed in another [11, 12]. Though this study acknowledges the relevance of measures of family-to-work conflict and examines its effects, WTFC is the exposure of emphasis here given that an occupational cohort comprises the sample.

The current study focuses on work and family stress and its effects on risk of CVD, which currently contributes to one in four deaths in the U.S. [13]. Numerous studies suggest that a variety of work and family stressors are associated with overall CVD risk, proxies for and actual measures of cardiovascular disorders as well as individual risk factors of CVD, including blood pressure, cholesterol, smoking and diet, exercise and obesity. Yet, much of this work is cross-sectional and among small samples. For example, research suggests that higher levels of support from managers for work and family issues was associated with lower CVD risk as measured by the presence of two or more of five modifiable risk factors among a cohort of 400 extended-care employees interviewed at one time point [2]. Prior work with our study sample also confirmed that higher demands related to work and family life may be associated with increased cardiometabolic risk as measured by a newly developed and validated cardiometabolic risk score (CRS) [14]. Berkman found that low family supportive supervisor behaviors at the level of the nursing home facility and high work-to-family conflict (WTFC) at the individual level were associated with increased cardiometabolic risk in a cross-sectional, baseline analysis

[3]. In these studies, supervisor support is hypothesized to buffer the impact of work-family conflict on a number of outcomes.

Most other research considering risk of disease per se has been conducted in high risk populations. For example, a longitudinal study of 80 female patients with atherosclerosis indicated that women with high self-reported stress from family or work experienced significant disease progression over a 3-year period, as measured by increased mean coronary luminal diameter. Women who did not report stress in one of these life realms experienced slower progression of atherosclerosis suggesting that satisfaction with work and home may be protective among female patients [4]. As part of the Stockholm Female Coronary Risk Study, Orth-Gomer and colleagues examined the effects of work stress and, separately, marital stress on risk of a recurrent cardiovascular event, such as death and myocardial infarction, among fewer than 300 women with existing coronary heart disease. They found that higher marital stress was associated with increased odds of recurrent event among partnered women but that work stress did not predict subsequent events over the course of nearly five years [15]. A follow-up study in the same sample found that higher exposure to work *and* marital stress resulted in the most heightened risk of recurrent coronary event, compared to no stress or one form of stress only [16].

Other work has considered work and family stress in relation to conditions and behaviors known to increase risk of cardiovascular disease. Twenty five years ago, Frankenhauser assessed blood pressure for 60 male and female Swedish white-collar employees. She noted that blood pressure increased during work hours and subsequently decreased after the workday in men only [9]. These results suggested that "total workload" (that is, the strain at work and home) affected biological functioning but that these processes are different for men and women. More recently,

Frone and colleagues identified a significant, increased risk of developing hypertension with higher levels of WTFC (but not in the direction of family-to-work) among employed parents (half men, half women, n=267) over a four year period [17]. Similarly, a cross sectional study of white-collar women found that reports of a high stress job coupled with caregiving responsibilities for children was associated with higher systolic and diastolic blood pressures (n=199) [18]. Among a predominantly female cohort of 398 health care employees with children at home, Thomas and Ganster conducted a series of path analyses and found that flexible scheduling and supportive supervisors positively impacted employee perceptions of control over work and family matters and reduced WTFC. WTFC was then significantly and associated with higher blood cholesterol but not blood pressure [19]. A similarly sized sample revealed that higher WTFC was associated with increased cigarette use indirectly through negative affect among a simple random sample of predominantly white, working mothers of adolescents [20]. Lallukka and colleagues utilized data from the British Whitehall II Study, the Finnish Helsinki Health Study, and the Japanese Civil Servants Study and tested the cross-sectional effects of WTFC on coronary risk related health behaviors in all three groups. They found that measures of WTFC were positively and significantly associated with current smoking among men but not women in the Finnish cohort; no associations with other behaviors in the Finnish cohort were evident. They also found no association with smoking, alcohol use, physical activity or diet or among the British or Japanese cohorts [21]. A longitudinal study of a workplace intervention to reduce WTFC among white-collar, retail employees (as part of pilot work for the current study, n=550), showed that the program increased the odds of quitting smoking and decreased smoking frequency [22]. This pilot study also found the intervention was associated with improvements in exercising behavior and promoting perceptions of adequate time for healthy meals [22, 23].

Multiple cross-sectional studies have also concluded that WTFC was associated with lower physical activity and poorer diet (i.e.: eating more high-fat foods and fewer healthy foods) [24-28] though, again, most of these studies employed small sample sizes. Similarly, increased WTFC related to significantly increased odds of being obese in the cross-sectional MIDUS sample (n=1547) [29].

Despite evidence suggesting links between strain at home and work and a variety of outcomes related to cardiovascular disease risk, this scientific literature presents a number of shortcomings and challenges. Investigators have examined biological outcome measures such as blood pressure, cholesterol and progression or development of CVD events. However, studies on the same outcome are limited in number and, thus, the literature lacks consistency of evidence for a given risk factor. Further, some of this work focuses on patient populations [4, 16] for whom pathways between stress and disease might be different than those in healthy populations. With a few exceptions [21, 27, 29], most of the aforementioned analyses were conducted among small samples, which may result in chance findings or lack of power to detect significant effects. Participants within studies also tended to be racially homogeneous. Additionally, although we do reference a few longitudinal analyses [17, 30] (Moen, 2013) supporting these associations, many studies are cross-sectional. A recent review indicated that 89% of work-family research utilized only one exposure and outcome measure at a single point in time [31], a design which greatly constrains researcher's abilities to draw strong causal inferences about the relationship between work and family stressors and health. Cross-sectional designs are particularly troublesome because health limitations could very plausibly pose challenges to the successful management of work and family demands. Additionally, understanding the effects of work and family stressors on CVD is constrained by the fact that the outcome takes years, often decades, to emerge, and

longitudinal research that anticipates the development of disease with long latency can be costly and logistically demanding.

We seek to address some of the limitations of this earlier work by conducting a longitudinal study among a predominantly female and racially and ethnically diverse sample and with a focus on behavioral as well as biological markers representing CVD risk. Biomarkers serve as a useful alternative in epidemiologic research to self-reported outcome measures. Typically collected by means of blood, saliva or urine, they serve as underlying risk factors for as well as potential intermediate variables along the pathway to disease, which is particularly useful for conditions that develop slowly over time. Biomarkers provide direct information on physiological processes in the body and are thus more reliable than subjective measures of health [32, 33], which reduces concerns of reverse causation in research. In addition, we examine behavioral outcomes associated with CVD risk such as smoking, which may be more susceptible to work and family stressors and more malleable over time compared to biological markers. Similarlym BMI is closely linked with behaviors that may also be more malleable and change more quickly than other biomarkers.

Generally, we hypothesize that WTFC will be associated with worse markers of cardiometabolic risk and that these associations will vary by level of WTFC over time. First, we pool outcome data over four study waves to increase the statistical power to identify these relationships, and then explicitly examine changes over the study period. Additionally, we control for treatment status in the randomized field experiment to disentangle the effects of the WFHN intervention on our outcomes of interest. Other analyses forthcoming assess the impact of the intervention directly on the cardiometabolic outcomes. To our knowledge, no longitudinal study of the effects of work and family stressors on a composite CVD risk score has been

conducted, nor have a series of risk factors been examined over time. Specifically, we address the following aims and hypotheses:

Aim 1: Assess the effect of WTFC *on individual cardiovascular risk factors at baseline*.

Hypothesis 1: WTFC will be associated with less healthy markers of cardiometabolic risk at baseline.

Aim 2: Assess the effect of WTFC at baseline on *markers of cardiometabolic risk (CRS and individual risk factors) pooled across multiple study waves.*

Hypothesis 2: WTFC at baseline will be associated with less healthy markers of cardiometabolic risk averaged across baseline, 6 months, 12 months and 18 months.

Aim 3: Examine whether *the rate of change in markers of cardiometabolic risk (CRS and individual risk factors) from baseline to 18 months varies by levels of WTFC at baseline.*

Hypothesis 3: High WTFC at baseline will be associated with a faster rate of increase in cardiometablic risk over time compared to low WTFC at baseline.

Methods:

Sample:

This study is part of Phase II of the Work Family Health Network (WFHN) project, a joint research endeavor sponsored by the National Institutes of Health and the Centers for Disease Control and Prevention, among others. This phase of the WFHN involved data collection from employees (as well as their managers, spouses and their children) over the course of 18 months as part of an employer-supported workplace intervention in a group randomized field experiment. The WFHN identified a New England company with numerous nursing home

facilities, which we will refer to as "LEEF" and included thirty worksites that were distributed across Massachusetts, Maine, New Hampshire, Vermont, Connecticut, and Rhode Island.

Each of the 1,723 eligible employees within these worksites who worked more than 22 hours each week during the day or evening was invited to complete a computer-assisted personal interview (CAPI) [34]. A total of 1,524 LEEF employees participated at baseline, resulting in response rate of over 88%. Data were collected at four waves (baseline, 6 months, 12 months and 18 months) using the same procedures as baseline. The exposure, WTFC, was unrelated to dropout over the course of the study and, thus, we utilize all available employee data for subsequent waves and do not employ a complete case analysis (please see Sample Characteristics below for more details on missing data and dropout). We do not have data on non-participants. We also do not explicitly test intervention effects in this study but control for and examine the role of treatment status in a variety of ways (see Measures and Analysis below). Employees who provided all components for the larger WFHN study, including blood samples, received $60 for their participation.

Measures:

Trained field interviewers administered computer-assisted personal interviews and on-site health assessments at all waves as described elsewhere [35], which addressed employee demographics, socioeconomic status, family demographics, respondent's work environment, physical health, mental health, and family relationships. After obtaining written consent from all respondents, interviews and health assessments lasted approximately 50 and 20 minutes, respectively, and were on occasion collected on different days.

Exposure Variable

Work-to-family conflict is thought to reflect the extent to which responsibilities in the domains of work and family are incompatible [10]. The current study incorporated a widely-used measure of this inter-role conflict developed and validated by Netermeyer and colleagues among employed individuals in various industries [36]. The survey included five questions to address work-to-family conflict that asked whether the demands of work interfere with family or personal time, the employee's job produces strain that makes it difficult to fulfill family or personal duties and things employees want to do at home do not get done because of the demands work puts on them. Individual item responses were coded 1-5 (strongly disagree to strongly agree) and averaged to generate a continuous measure (internal consistency reliability of the scale was high; alpha=0.9). For the purposes of the current study, only baseline measures of WTFC were considered.

As mentioned, work-family conflict is thought to be bidirectional in nature and believed to operate from work-to-home as well as home-to-work. We explored inter-role conflict from home to work as well. Similar to the WTFC measure, employees were asked five questions to address family-to-work conflict (FTWC) (whether the demands of family interfere with work, employees have to put off doing things at work because of demands on time at home and family-related strain interfere with employee's ability to perform job-related duties). Individual item responses were coded 1-5 (strongly disagree to strongly agree) and averaged to generate a continuous measure (alpha=0.8). Again, only baseline measures of FTWC were examined in this analysis.

Outcome Variables:

Prior research established and validated a measure of cardiometabolic risk based on modifiable risk factors in the Framingham risk score [37], including blood pressure, cholesterol, HbA1c, BMI and cigarette smoking status. The score used here was calculated based on age- and sex-based means in our particular sample and was validated independently among Framingham offspring data to predict risk of a cardiovascular event [14]. We build upon a recent paper examining baseline, cross-sectional effects of WTFC on the CRS [3] and focused on the individual components of this score (at baseline and overtime), all of which were measured continuously.

Employees were asked to provide dried blood spots (DBS) by a finger stick. Interviewers wearing appropriate personal protective equipment disinfected the employee's middle or ring finger with an alcohol swab and proceeded to prick the finger with a sterile, disposable micro-lancet. As previously described [38], as blood spots were collected, air-dried, and sealed in a plastic bag for room-temperature shipment with desiccant for storage at −86°C until assayed for cholesterol by means of a protocol specifically validated for this study from serum to DBS equivalents [39]. At the time of the blood draw, study staff also collected a 1 microliter blood droplet to measure HbA1c levels (DCA Vantage Analyzer, Siemens Healthcare Diagnostics, Frimley, Camberley, UK). Prior to blood sampling, three seated blood pressure readings were collected at least 5 minutes apart during the interview with wrist blood pressure monitors (HEM-637, Omron Healthcare, Bannockburn, IL). These three readings were averaged to create a continuous measure. Body mass index was calculated as height/weight^2 (height measured by Seca213/214 stadiometers, Seca North America, Hanover, MD; weight measured by Health-O-Meter 800KL, Jarden Corporation, Rye, NY). Height and weight measurements were taken at the

same time as other physical health assessments. Cigarette consumption was assessed by respondent self-report. Employees were asked if they smoke cigarettes every day, some days or not at all, how many days they smoke cigarettes on average in a week, and how many tobacco cigarettes they smoke on an average day. Responses were multiplied to produce a measure of cigarettes per week. Non-smokers received a score of zero cigarettes per week. Based on the aforementioned components, we calculated a cardiometabolic risk score for each subject (age- and sex-specific strata use different score calculations) [3, 14]. For additional information on specific measures, refer to Bray, 2013.

Covariates:

A number of sociodemographic variables and covariates relevant to the association between WTFC and cardiometabolic risk were also assessed: occupation (employee's official job title, coded nurse or other), marital status (currently married or do you have a permanent romantic partner that lives with you?), employee gender (male/female), income (assessed in $5,000 increments and categorized as greater than 300% of the national poverty threshold or less), age in years, total number of work hours (how many hours employees worked in a typical week at any job) and number of children less than or equal to 18 years old living in the household for 4 or more days/week (none/one or more). Race/ethnicity was coded as White, Black, Hispanic or other race. Dummy variables for each racial/ethnic group were generated, and the reference group was assigned to White race. Foreign-born status was coded yes/no depending on whether an employee was born in this country or not. Because this observational analysis is embedded within an existing randomized control trial in which a workplace program sought to reduce work-to-family conflict and improve health outcomes, we controlled for whether the

employee worked within a workgroup assigned to control or intervention status (blinded and labeled treatment 1 and treatment 2) in pooled and longitudinal analyses.

Similar to Berkman et al [3], we included a number of work environment measures (at the individual- and workgroup-levels) assessed at baseline as covariates to assess the independent effects of WTFC on cardiometabolic risk, above and beyond these factors. Job strain, a measure of employee stress that does not explicitly incorporate family context, was assessed through questions pertaining to psychological job demands and job control or decision authority. According to the work of Karasek and colleagues, high job demands paired with low control are hypothesized to be detrimental to physical and psychological wellbeing. In response to questions pertaining to physical activity, heavy lifting and awkward body positions (*job demands*) as well as degree of skill, task variability and autonomy (*job control*) at work, subjects strongly disagreed, disagreed, neither, agreed or strongly agreed (valued 1 - 5, respectively and measured continuously) that these elements were part of their jobs [5, 6, 40] (Cronbach's alpha=0.6 for psychological job demands and Cronbach's alpha=0.6 for job control). A measure of managerial support, family supportive supervisory behavior (FSSB), tapped into employee appraisals of supervisor's behavior relating specifically to work and family. Research indicates that FSSB is negatively associated with employee reports of WTFC and turnover intentions and positively associated with positive work-to-family and family-to-work spillover as well as job satisfaction [41, 42]. The scale asked employees about four domains related to family-related supervisory support, including emotional support (supervisor makes you feel comfortable talking to him/her about conflicts between work and non-work), instrumental support (supervisor works effectively with employees to creatively solve conflicts between work and non-work), role modeling (supervisor demonstrates effective behaviors in how to juggle work and non-work

issues) and creative management (supervisor organizes departmental work to jointly benefit employees and the company). The current study used a short form of FSSB derived from employee responses to four items, categorized 1-5 (strongly agree to strongly disagree) and averaged to generate an overall score, with higher scores reflecting greater FSSB [43] (Cronbach's alpha=0.9). Similarly, we utilized a modified version of Thomas and Ganster's schedule control scale [44]. Employees were asked how much choice they had over when they took vacation, when they can take off a few hours, when workdays begin and end, working at another location, the number of personal phone calls they can make or receive during work, how much they take work home and about shifting to part time work if full time (and vice versa). Responses ranged from very little to very much (1-5) and an overall score of schedule control was obtained by calculating the average score of these 8 items (Cronbach's alpha=0.7).

As appropriate, we also controlled for baseline medication use (i.e.; insulin for the outcome HbA1c, cholesterol medication for total cholesterol and HDL cholesterol and blood pressure medication for systolic and diastolic blood pressure). Because those individuals who were not diagnosed with a specific condition (i.e.: diabetes) were not asked about medication use, missing data for these questions were coded as non-use to provide a full sample of responses. Finally, for longitudinal analyses that sought to capitalize on multiple measures, wave of data collection (baseline, 6 months, 12 months and 18 months) was also included as a covariate and operationalized as a continuous measure.

Analysis:

To test whether WTFC was related to individual cardiometabolic risk factors at baseline only (Aim 1), we estimated multilevel linear regression models that accounted for the nesting of

employees within worksites by modeling random effects for the site level. To test whether WTFC was associated with the CRS and various cardiometabolic risk factors pooled across waves (Aim 2) and whether the rate of change in these outcomes varied over levels of WTFC (Aim 3), we estimated multilevel linear regression models that also accounted for multiple measures per employee by modeling random effects for the employee level. In these models, we used outcome data from four times points (baseline, six months, twelve months and eighteen months), a method which improves statistical power of cross-sectional analyses. We utilized baseline exposure and covariate data, which was not time updated with the goal of reducing reverse causality. A time*exposure interaction term was included separately in models to address Aim 3 specifically.[*]

Due to differences in our longitudinal CRS results compared to a previous baseline analysis [3], we conducted a post hoc analysis to verify the likelihood of reverse causation and tested the effects of the CRS at baseline on work and family stressors pooled across the fours study waves. Because this observational study is embedded within a randomized field

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Aim 1: $\text{Cardiomet}_{ij} = \beta_0 + \beta_1 (\text{WTFC}) + \beta_2 (\text{Covariates}) + e_{0ij} + u_{0j}$
Where: i= employee; and j = workgroup
And:
$[e_{0ij}] \sim N(0, \sigma^2_{e0})$
$[u_{0j}] \sim N(0, \sigma^2_{u0})$

Aim 2: $\text{Cardiomet}_{tij} = \beta_0 + \beta_1 (\text{WTFC}) + \beta_2 (\text{Time}_{tij}) + \beta_3 (\text{Treatment}_j) + \beta_4 (\text{Covariates}) + t_{0tij} + e_{0ij} + u_{0j}$
Where: t= time; i= employee; and j = workgroup
And:
$[t_{0tij}] \sim N(0, \sigma^2_{t0})$
$[e_{0ij}] \sim N(0, \sigma^2_{e0})$
$[u_{0j}] \sim N(0, \sigma^2_{u0})$

Aim 3: $\text{Cardiomet}_{tij} = \beta_0 + \beta_1 (\text{WTFC}) + \beta_2 (\text{Time}_{tij}) + \beta_3 (\text{WTFC}) (\text{Time}_{tij}) + \beta_4 (\text{Treatment}_j) + \beta_5 (\text{Covariates}) + e_{0ij} + u_{0j}$
Where: t= time; i= employee; and j = workgroup
And:
$[t_{0tij}] \sim N(0, \sigma^2_{t0})$
$[e_{0ij}] \sim N(0, \sigma^2_{e0})$
$[u_{0j}] \sim N(0, \sigma^2_{u0})$

experiment, we conducted an additional post hoc analysis of pooled outcome models stratified by treatment status as well.

Models controlled for treatment status (in pooled and longitudinal analyses), sociodemographic variables (race, income, sex, occupation, age, foreign born status), possible antecedents of WTFC (marital status, number of children and work hours) and relevant work environment factors like FSSB, schedule control and job strain, all measured at baseline. Model results do not differ with and without the inclusion of antecedents of WTFC nor work environment variables and, thus, we adjust for them in-line with the work of Berkman and colleagues. Where appropriate, we adjusted for baseline medication use as well (blood pressure and cholesterol medication as well as insulin use). We did not control for medication use in models with CRS as the outcome because multiple medication use measures would be warranted, and other model results did not change meaningfully with the inclusion of medication use. All outcomes were modeled continuously and analyses conducted using the mixed procedure in SAS.

Results:

Sample Characteristics:

At baseline, on a scale of 1 to 5, the mean work-to-family conflict score was 2.79 (sd = 0.91) (see Table 1.1 for descriptive statistics at baseline). The mean 10-year cardiometabolic risk score represents a 7.75% 10-year CVD risk (sd=8.15%), meaning that fewer than 8 out of 100 with the average level of risk will have a cardiovascular event in the next 10 years.[†] Mean BMI in our sample was 29.45, considered obese, and roughly 30% of employees smoked (an average

[†] Over 20% is considered high global risk, according to some researchers (Dagostino 2008). Thus, the mean risk in our sample is fairly low.

Table 1.1: Descriptive Statistics

N=1524 baseline	N	Mean (sd) / %
Cardiometabolic Risk Score	1412	7.75 (8.15)
BMI	1501	29.45 (7.03)
Total Cholesterol	1464	190.79 (28.78)
HDL Cholesterol	1473	63.56 (5.54)
Systolic Blood Pressure	1511	114.79 (13.09)
Diastolic Blood Pressure	1511	72.36 (9.40)
HbA1c	1453	5.51 (0.61)
Cigarettes/week	1522	23.45 (45.87)
WTFC	1520	2.79 (0.91)
FTWC	1522	2.07 (0.58)
Work Hours	1520	39.95 (10.63)
FSSB	1510	3.69 (0.88)
Job Control	1511	3.45 (0.76)
Job Demands	1523	3.82 (0.75)
Schedule Control	1509	2.65 (0.73)
Age	1522	38.52 (12.48)
RN/LPN		
Yes	428	28.12
No	1094	71.88
Sex		
Male	118	7.74
Female	1406	92.26
Married/partnered		
Not married/partnered	566	37.14
Married/partnered	958	62.86
Race		
White	987	64.81
Black	200	13.13
Hispanic	204	13.39
Other race	132	8.67
Foreign Born		
Yes	405	26.57
No	1119	73.43
Income		
>300% poverty threshold	569	38.39
<300% poverty threshold	913	61.61
Children ≤ 18 years old		
None	710	46.59
One or more	814	53.41
Diabetes Medication (general)		
No	1510	99.08
Yes	14	0.92
Insulin Use		
No	1508	98.95
Yes	16	1.05
Blood Pressure Medication		
No	1308	85.83
Yes	216	14.17
Cholesterol Medication		
No	1510	99.08
Yes	14	0.92

of 23.45 cigarettes each week). Average total and HDL cholesterol, HbA1c and systolic and diastolic blood pressures were in-line with national levels [CDC]. We analyzed a total of 1,524 subjects enrolled at baseline, many of whom provided data at subsequent waves. Data were missing for roughly 16%, 29% and 34% of employees at 6 months, 12 months and 18 months, respectively. Related, roughly 40% of the sample dropped out of the study for at least one wave, whereas 27% and 13% dropped out of the study for two or three waves, respectively. General patterns for missing data (i.e.: non-participation in a survey) and dropout (i.e.: non-participation from one survey to the next) appear closely aligned. Older age and foreign-born status significantly predicted higher odds of missing survey data and dropout from the sample. We also find that higher job demands were associated with dropout for certain outcome variables, such as CRS and HDL cholesterol. WTFC at baseline was not associated with either missing data or dropout from the study. Accordingly, we do not employ a complete case analysis; we use all available data (thus, we can examine specific dropout patterns by outcome). Outcomes were highly correlated overtime; WTFC and related covariates were also highly correlated at baseline (see Appendices 1.1 and 1.2). Trends in outcome data over time are presented by treatment group in Appendix 1.3.

Results of statistical analyses:

While we did replicate baseline findings that suggest WTFC is associated with CRS at baseline, our primary exposure was not associated with CRS pooled across waves or longitudinally. Increased WTFC was associated with higher BMI at baseline and over the course of the study. Increased WTFC was also associated with lower HDL cholesterol in pooled analyses but not in baseline or longitudinal analyses. WTFC was not associated with total cholesterol, blood pressure, HbA1c or cigarette smoking in any analysis. While

not our primary research question of interest, we also conducted similar analyses with FTWC as the predictor variable and find that this exposure is also associated with higher BMI and lower HDL in pooled analyses but not any outcome in baseline or longitudinal analyses (see Table 1.2 and Appendix 1.4).

Baseline Associations:

At baseline, we found that one-point higher WTFC was associated with a half-point higher BMI (β=0.53, p=0.02, CI=(0.08, 0.98)). Baseline WTFC was not associated with baseline HDL cholesterol, total cholesterol, blood pressure, HbA1c or cigarette smoking. Baseline FTWC was not associated with any cardiometabolic risk factors at baseline.

Pooled Outcome Associations:

WTFC was not associated with CRS averaged across time. Pooled outcome analyses reflected a similar main effect between WTFC and BMI as baseline analyses (β=0.59, p=0.01, CI=(0.12, 1.04)). Higher WTFC at baseline was associated with lower HDL cholesterol averaged at all waves (β=-0.32, p=0.01, CI=(-0.57, -0.08)) but not other outcomes pooled over time. Higher FTWC at baseline was similarly associated with higher BMI in pooled analyses (β=0.62, p=0.05, CI=(-0.01, 1.25)) and lower HDL cholesterol (β=-0.41, p=0.02, CI=-0.76, -0.06)) but not other outcomes pooled across waves.

Table 1.2: Associations between baseline WTFC and markers of cardiometabolic risk

	Baseline			Pooled Outcome			Longitudinal		
	CRS								
	N=1334			**N= 1438 Obs=4281**			**N= 1438 Obs=4281**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	3.81	0.97	0.001	3.14	0.94	0.003	3.14	0.94	0.00
WTFC	**0.37**	**0.16**	**0.02**	0.27	0.16	0.09	0.24	0.17	0.15
Time				0.04	0.03	0.14	0.04	0.03	0.14
WTFC*Time							0.01	0.03	0.73
	BMI								
	N=1417			**N=1438 Obs=4581**			**N=1438 Obs=4581**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	27.48	1.34	<.0001	26.95	1.31	<.0001	26.94	1.31	<.0001
WTFC	**0.53**	**0.23**	**0.02**	**0.59**	**0.22**	**0.01**	0.42	0.23	0.07
Time				**0.06**	**0.02**	**0.004**	**0.06**	**0.02**	**0.002**
WTFC*Time							**0.08**	**0.02**	**0.0007**
	HDL Cholesterol								
	N=1397			**N= 1438 Obs= 4484**			**N= 1438 Obs= 4484**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	58.35	1.79	<.0001	58.70	1.38	<.0001	58.70	1.38	<.0001
WTFC	-0.23	0.17	0.18	**-0.32**	**0.12**	**0.01**	-0.32	0.20	0.11
Time				**-0.29**	**0.06**	**<.0001**	**-0.29**	**0.06**	**<.0001**
WTFC*Time							-0.001	0.07	0.99
	Total Cholesterol								
	N=1388			**N=1438 Obs=4481**			**N=1438 Obs=4481**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	148.25	8.70	<.0001	147.45	7.19	<.0001	147.44	7.19	<.0001
WTFC	0.23	0.81	0.78	-0.21	0.67	0.75	-0.31	1.03	0.77
Time				**2.89**	**0.32**	**<.0001**	**2.89**	**0.32**	**<.0001**
WTFC*Time							0.40	0.35	0.90

Table 1.2: Associations between baseline WTFC and markers of cardiometabolic risk (continued)

	Systolic Blood Pressure								
	N= 1425			N= 1438 Obs =4600			N= 1438 Obs =4600		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	119.83	2.45	<0.001	119.43	2.28	<.0001	119.41	2.28	<.0001
WTFC	-0.12	0.39	0.77	-0.05	0.35	0.89	-0.33	0.41	0.43
Time				**-0.93**	**0.09**	**<.0001**	**-0.93**	**0.09**	**<.0001**
WTFC*Time							0.13	0.10	0.20

	Diastolic Blood Pressure								
	N= 1425			N= 1438 Obs=4600			N= 1438 Obs=4600		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	77.18	1.87	<.0001	76.27	1.70	<0.001	76.27	1.70	<.0001
WTFC	-0.08	0.29	0.78	-0.21	0.26	0.40	-0.19	0.31	0.54
Time				**-0.60**	**0.07**	**<0.001**	**-0.60**	**0.08**	**<.0001**
WTFC*Time							-0.01	0.08	0.90

	HbA1c								
	N=1377			N= 1438 Obs=4402			N= 1438 Obs=4402		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	7.60	0.18	<.0001	8.46	0.18	<.0001	8.46	0.18	<.0001
WTFC	0.02	0.02	0.39	0.01	0.02	0.47	0.02	0.02	0.29
Time				**-0.04**	**0.00**	**<.0001**	**-0.04**	**0.00**	**<.0001**
WTFC*Time							-0.004	0.005	0.39

	Cigarettes/week								
	N=1437			N= 1438 Obs=4648			N= 1438 Obs=4648		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	-22.13	8.83	0.02	-23.88	8.34	0.01	-23.86	8.34	0.01
WTFC	1.84	1.45	0.21	0.78	1.33	0.56	1.43	1.45	0.32
Time				**-0.94**	**0.24**	**<.0001**	**-0.95**	**0.24**	**<.0001**
WTFC*Time							-0.30	0.26	0.25

All models control for treatment status, race, income, age, sex, marital status, foreign born status, occupation, work hours and number of children. Models also account for FSSB, job strain and schedule control at the individual- and group-levels, WTFC at the group level and, where appropriate, medication

Longitudinal Associations:

We found that the average rate of change of BMI was 0.06 units per each six month study wave (p=0.002). The rate of change in BMI from baseline to 18 months also increased with higher levels of WTFC (β=0.08, p=0.01, CI=(0.03, 0.15)) (see Figure 1.1). No other longitudinal effects were observed in our data.

Post-hoc analysis to assess reverse causation:

We did not observe that WTFC was associated with the CRS in pooled or longitudinal analyses, however, previous findings from the same sample have suggested WTFC was associated with CRS in the baseline, cross-sectional setting [3]. To understand why we find a cross sectional association but not significant relationships in pooled or longitudinal analyses, we examined the effect of CRS at baseline on pooled WTFC as part of a post hoc analysis (β=-0.005, p=0.06, CI=(-0.001, 0.002)). These results suggest that CRS may marginally predict WTFC pooled across waves (see Appendix 1.5). We conducted the same analysis with FTWC at baseline and found that CRS did not predict this stressor in pooled analyses (β=-0.0005, p=0.79, CI=(-0.004, 0.003)).

Post-hoc analysis to examine treatment effects:

This observational study is embedded within a randomized field experiment. Thus, we examined pooled outcome models stratified by treatment status to verify residual effects of the intervention. In pooled outcome models stratified by treatment status, we observed that higher levels of WTFC at baseline are significantly associated with higher pooled CRS but only in the treatment group (β=0.47, p=0.03, CI=(0.05, 0.90); however, the effects of WTFC on CRS in the

control group were in the same direction (β=0.12, p=0.59, CI=(-0.32, 0.56)). Higher WTFC was associated with higher BMI but this was evident only in the control group (β=0.92, p=0.004, CI=(0.29, 1.55) and not the treatment group (β=0.23, p=0.46, CI=(-0.38, 0.84)). Associations of higher WTFC with lower HDL cholesterol observed in both the treatment (β=-0.31, p=0.08, CI=(-0.65, 0.04)) and control groups (β=-0.30, p=0.10, CI=(-0.65, 0.05)) were of borderline statistical significance. The effects of WTFC on other cardiometabolic outcomes were similar to non-stratified analysis. Further, there were no differences of WTFC on the rate of change in outcomes in models stratified and not stratified by treatment status (Data not presented).

Discussion:

The current study examined whether WTFC was associated with individual risk factors as well as a composite score for CVD in a longitudinal cohort of nursing home employees. Utilizing a range of behavioral and biological outcome measures, we found that WTFC was positively associated with BMI at baseline and in pooled analyses. The rate of change in BMI over time also increased with higher levels of WTFC at baseline. Higher WTFC was associated with lower HDL cholesterol in pooled analyses only. WTFC was not associated with CRS pooled across waves or longitudinally nor was this work and homelife stressor associated with total cholesterol, blood pressure, HbA1c or cigarette smoking in any analysis. Below we will first discuss our results for BMI, placing them in the context of theoretical perspectives and the scientific literature, before offering explanations for these findings in our sample. Then, we will proceed to discuss the findings on the association between WTFC and other outcomes in our study and review limitations and strengths of this research.

WTFC and BMI:

The most consistent and significant findings in this study pertain to the relationship between WTFC and BMI. Several theories help to explain why BMI, which has proximate behavioral risks for heart disease, might be associated with this form of stress. From a coping and mood-management perspective, individuals are believed to pursue activities and behaviors that result in a positive emotional and affective experience [45]. Thus scholars argue that inter-role conflict prompts individuals to seek comfort through food and inactivity, or other ultimately risk-related health behaviors, as a method for maximizing immediate and short term pleasure in the face of stress [46, 47]. In line with the role strain and conservation of resources perspectives, time and physical and mental energy are believed to be finite resources that impact health behaviors [48]. In terms of BMI, when faced with demands from work and home, competition for resources and priority setting in according with social roles (i.e.: being a "good" spouse, parent or employee) results in less time and energy for activities such as making a nutritious meal or exercising [49, 50].

The scientific literature offers evidence that work and/or family strains are positively associated with BMI and related outcomes, although this relationship may not be the same for men and women. Kivimaki and colleagues prospectively examined the effects of the work stress on BMI and found that job demands at baseline were associated with BMI in women after five years, adjusting for baseline BMI. Among men, job strain increased the likelihood of weight gain among those with the highest BMIs but predicted weight loss among individuals with the lowest BMIs, suggesting that some may eat more due to stress whereas others eat less [51]. In a nationally-representative study with the MIDUS sample, high job demands were associated with weight gain among men and women but more perceived constraints in life and strain in relations

with family were associated with weight gain only in women over the nine year study period [52]. While no studies to date have examined the combined effects of work and family stressors on BMI per se, higher WTFC has also been cross-sectionally linked with lower physical activity and poorer diet (i.e.: eating more high-fat foods and fewer healthy foods) and obesity [24-28]. In the information technology arm of our WFHN study, work-family strain, operationalized by spousal work hours, related to choices around exercise and diet, all measured at baseline. Specifically, among men, having an employed partner was associated with higher odds of infrequent exercise, and longer spousal work hours predicted fast food consumption among women [53]. Taken together, stressors related to work and home may be associated with increased weight and BMI, but the types of stressors that affect men and women could differ. The current study adds to this literature in examining the effects of combined work and family stressors on BMI over time in a predominantly female sample of lower and middle wage earners in health care. We encourage replication of this research question to confirm and clarify these relationships both in men and women as we were underpowered to examine gender differences here.

Our longitudinal findings may also reflect that BMI is more susceptible to changes in the social environment than biological markers representing CVD risk over the 18-month study period. Theoretical frameworks linking stress to health, such as the Integrated Model of Stress, suggest that behavioral pathways chronologically precede and subsequently relate to disease [54]. Both diet and physical activity may have a relatively quick impact on BMI. In a pilot workplace intervention related to the current WFHN study, a program to address work and family stress improved employee health-related behaviors but not general measures of health. Specifically, over a six month follow-up period, treatment workgroups exhibited improvements

in exercising behavior and perceptions of time to prepare healthy meals but no direct changes due to treatment in non-behavioral measures of well-being such as self-reported health and psychological distress were evident [22, 23]. The authors stated that they first focused on changing health-promoting behaviors, such as diet and physical activity, which are more likely to change over a relatively short period of time. In the current study, we similarly find that WTFC is associated with BMI but not many biological measures, perhaps because health behaviors are more easily and quickly changed than physiological indicators of disease. Our findings do not suggest any associations between WTFC and smoking behaviors measured by cigarette consumption.

WTFC and CRS and other risk factors:

WTFC was inversely associated with HDL cholesterol in pooled outcome analyses in our data. Unlike BMI, we did not detect any baseline or longitudinal associations between WTFC and this outcome, though effect estimates and confidence intervals for the effects of WTFC on BMI are not substantially different from analysis to analysis (B=-0.23, CI=(-0.56, 0.10); B=-0.32, CI=(-0.57, -0.08); B=0.32, CI=(-0.72, 0.07) in baseline, pooled and longitudinal analyses, respectively). Selection bias may explain why significant effects are observed for HDL cholesterol in pooled analyses only. Roughly 36% of the sample with any measure of HDL at baseline left the study by 18 months. Our data does not allow us to specifically examine whether those employees with the worst HDL cholesterol were the same ones dropping out of the study; only a small percentage (3.4%) of the sample had "risky" levels of HDL cholesterol (< 40 mg/dL), and none of these individuals left the study. Still, predictors of HDL attrition from baseline to 18 months do suggest that certain characteristics associated with dropout may be

resulting in a less healthy sample in which effects of WTFC on HDL cholesterol are detected across waves but not at baseline. For example, older age, higher job demands and foreign-born status are significantly associated with higher odds of dropout for this outcome over the course of the study. Data also suggests that older age and higher job demands are associated with significantly higher (i.e.: better) HDL at every time point. Thus, if older individuals and employees with the highest job demands are systematically leaving the study over time, those remaining in the sample will be less healthy and we are more likely to detect the potentially deleterious effects of WTFC on HDL in the pooled analyses compared to baseline. There were no discernable differences in HDL levels by foreign-born status at any time point and, thus, differential dropout by this variable is unlikely to explain our findings.

We anticipated that WTFC would be associated with the CRS over the course of the study's 18 months due to previous findings that suggested WTFC was positively and significantly associated with CRS at baseline in this same sample. For every 1 point increase in the WTFC scale, Berkman and colleagues found that cardiometabolic risk over a 10 year period increased almost 0.40 percentage points, CI= (0.04-0.74) [3]. While we do not find support that WTFC is associated with CRS in these longitudinal analyses, we note that previous baseline findings are consistent with the baseline trends we detect between WTFC and individual risk factors and speculate that, perhaps, BMI singlehandedly drove the baseline effects of WTFC on baseline CRS. We also find that 39% of the sample with CRS at baseline had left the study by 18 months and that older age predicted CRS values among employees that dropout over time. Like HDL cholesterol, older age is associated with significantly higher CRS at all timepoints. If older employees are leaving the study, the remaining sample is likely to be younger and healthier and, thus, it will be challenging to detect effects of WTFC on CRS over time. Another explanation for

failure to find effects on WTFC on CRS beyond baseline may reflect challenges in detecting changes in biological phenomena (vs. behaviors) over an 18 month study period. Reverse causation serves as another explanation for the discrepant results at baseline and across waves and worse cardiometabolic health could actually cause higher WTFC (and not the other way around). In fact, our post hoc analysis revealed that the effect of CRS at baseline on pooled WTFC across waves was borderline significant ($p=0.06$). These findings suggest that lower CRS could be associated with higher WTFC across waves. Therefore, the causal relationship between these variables may not be in the hypothesized direction because, for example, individuals with higher cardiovascular risk become more strained by work and family demands due to their poor health status. Finally, it is also possible that WTFC is not associated with CRS, and the baseline associations were found due to chance.

WTFC was not associated with HbA1c, total cholesterol, blood pressure or cigarette consumption at any point in time. Research on job strain and cardiovascular outcomes serves to support the plausibility of these relationships and motivates the current study. However, our null findings may be attributed to the unique composition of this sample as well as the fact that the combination of work and family stressors may not affect health the same way as job strain alone. As mentioned previously, the scientific literature suggests a strong link between work stress [6, 30, 55, 56], home life stress [15, 30, 57] and the combination of the two [2] with poorer cardiovascular outcomes. The most extensive work in this area concerns how workplace stress specifically is associated with incident heart disease. Kivimaki and colleagues report in a meta-analysis of prospective cohort studies that effect estimates vary from almost 40% increased risk to 2- or even 4-fold increases in risk of CVD (including incident coronary heart disease and ischemic heart disease as well as CVD death) due to some form of job strain (job demands and

decision authority, or job control). A recent systematic review also suggests that work stress may be related more consistently with the development of cardiovascular disease in men than in women [58]. Most of the samples referenced in job strain literature are predominantly male, Caucasian employees, whereas the WFHN occupational cohort is racially and ethnically diverse and overwhelmingly comprised of women. Further, multiple studies suggest that the dual demands of work and family affect men and women differently and, unlike the job strain literature, *women* may experience interrole strain and subsequent poor health more acutely [1, 59-62]. For example, a hallmark study examining blood pressure, heart rate and catecholamine excretion among Swedish white-collar workers indicated that biological markers were elevated among all employees during work hours but that differential stress responses were evident among men and women after work hours. After leaving work adrenaline, blood pressure and heart rate declined among men but not among women, suggesting perhaps that men are privileged to "unwind" at home while women's stress persists [9]. After noting the dearth of research on female-only samples, Orth-Gomer and colleagues studied the role of work and family stressors on recurrent cardiac event among women only and found that strain from both realms predicted worse health outcomes [15, 16]. Subsequently, these researchers examined the effects of behavioral interventions on stress attenuation among patients with acute coronary syndrome. Psychological assessments indicated that men and women exhibited unique discussion styles and preferences regarding group composition.

In light of these literatures, two plausible explanations for our predominantly null findings remain. It may be that work and family stress, like job strain, affects the CVD risk of men more than women, and we can attribute the lack of association between WTFC and most of our outcomes of interest to a predominantly female sample. Alternatively, if per the work and

family scholarship, these dual demands are indeed more pronounced in women, our results may indicate that work and family stress does not actually predict most individual CVD risk factors, at least not in a racially and ethnically diverse sample such as that used here. While disentangling these possibilities is beyond the scope of the current research, the linkage between work-family conflict and CVD risk factors warrants further scrutiny, and we encourage future research to focus on both male and female study populations.

Limitations and Strengths:

We acknowledge some limitations in the current study. WTFC is reported by employees, and this perception of work and family stress could be potentially misclassified. For example, sicker employees may report more conflict than their healthy counterparts, resulting in a possible overestimation of the effects of work-family stress on health. Similarly, employees consented to participate in the study and, though response rates were reasonably high, there is also a possibility that either healthier or sicker employees selected into the study, which could bias results in either direction. This observational study is embedded within a randomized field experiment in which an intervention was administered to employees in certain workgroups in an effort to improve health. Despite thoroughly examining the effects treatment status on our outcomes, it is plausible that the observed effects of WTFC on cardiometabolic risk factors such as BMI and HDL cholesterol across waves could be due to the intervention itself because randomization did not occur at the individual level. We found little evidence for this as part of a post hoc analysis, however. In models stratified by treatment status, the only outcome for which stronger, significant effects were observed in the intervention group was CRS, which was not significantly associated with WTFC in non-stratified pooled analyses, and it is worth noting that

the effects of WTFC on CRS were in the same direction in both treatment groups. We also note substantial attrition from baseline to 18 months. While we replicated our findings using outcome data at baseline, 6 months and 12 months only (thus, remedying some concern of attrition throughout the study period), failure of employees to continue in the study remains a limitation of our analysis. Additionally, while the WTFC measure employed here is widely-used and validated among workers in many settings [36], the scale may not be appropriate for this particular study population. Additionally, the etiologic period for changing biological indicators of CVD risk (such a HbA1c, blood pressure and total cholesterol) due to WTFC is unclear, and the 18 month study may not be sufficient to detect these changes, assuming a causal relationship, particularly given that the few existing longitudinal studies using these variables have generally been of a longer duration. This may not be a large concern given that WTFC was measured at only one time point, however. Finally, although multiple worksites were involved in the study, the data also represents the experiences of a single industry that was willing to participate in a workplace intervention, which limits the generalizability of our findings to other industries. We suggest that the research pertaining to work and family demands and conditions continue to take place in a variety of settings.

This work also exhibits a number of substantive and methodological strengths. This is the first study to longitudinally examine work and family conflict and variety of biomarkers representing cardiovascular disease risk in an occupational cohort. Prior studies examining the effects of job strain and cardiovascular health are somewhat limited in scope, comprising of study populations that are predominantly male, Caucasian employees. Similarly, roughly three-quarters of the work-family literature utilizes predominantly Caucasian samples [31]. Our study represents racially and ethnically diverse, predominantly low wage cohort of healthcare workers,

offering a broader perspective on both the job strain and work-family literatures (though, admittedly, the heterogeneity of the sample could have resulted in a loss of statistical power and contributed to our null results as well).

Additionally, this study utilized predominantly objective outcome measures, including assessed measures of blood pressure, blood draws to ascertain HbA1c and cholesterol and validated methods for measuring BMI. The use of these biological markers and other objective measures offer meaningful improvement to the validity of work-family research. Cigarette consumption, work-to-family conflict and many covariates of interest were self-reported in our sample, however. Further, the majority of work-family scholarship is cross-sectional in nature [63], although a few exceptions do exist [64-68]. The use of multiple outcome measures strengthens our study's internal validity and helps to ensure that exposures precede outcomes. We also employ multilevel methods to appropriately account for the clustering of employees within worksites (and multiple time points per employee), a method which yields accurate standard errors and confidence intervals.

Conclusion:

A longitudinal study of nursing home employees indicates that WTFC is consistently associated with BMI but not many other biological and self-reported measures representing cardiovascular disease risk. We speculate that outcomes associated with behaviors are more closely linked to interrole conflict than biological markers, particularly with a relatively short study period of 18 months. We recommend that future research continue to clarify the effects of work and family stressors on individual risk factors for CVD in a variety of occupational settings.

Appendix 1.1: Correlations of individual outcomes across waves*

	Outcome at 6 months	Outcome at 12 months	Outcome at 18 months
CRS baseline	0.96	0.95	0.92
HBA1c baseline	0.83	0.75	0.73
Systolic Blood Pressure baseline	0.75	0.76	0.71
Diastolic Blood Pressure baseline	0.69	0.66	0.62
Total Cholesterol baseline	0.38	0.40	0.34
HDL Cholesterol baseline	0.35	0.42	0.30
BMI baseline	0.96	0.95	0.93
Cigarettes/week baseline	0.87	0.85	0.84

*All correlations $p<0.0001$

Appendix 1.2: Correlations between WTFC and related covariates

	FTWC	FSSB	Schedule Control	Job Demands	Job Control
Pearson Correlation Coefficients	0.41	-0.22	-0.20	0.32	-0.22
P-Value	<.0001	<.0001	<.0001	<.0001	<.0001
Sample Size	1519	1508	1505	1520	1508

Appendix 1.3 Outcomes across waves

	Control					Treatment				
	Baseline		18 months			Baseline		18 months		
	N	Mean	N	Mean	p-value*	N	Mean	N	Mean	p-value*
WTFC	797	2.75	553	2.67	0.08	723	2.84	454	2.66	**0.01**
CRS	733	7.56	465	7.93	0.42	679	6.97	385	7.44	0.36
BMI	786	29.51	541	29.65	0.71	715	29.38	446	29.65	0.51
Total Chol	767	190.77	514	199.04	**<0.001**	697	190.82	417	201.36	**<0.001**
HDL	775	63.23	514	62.59	**0.03**	698	63.91	417	63.36	0.11
HbA1c	759	5.53	481	5.45	0.05	694	5.50	397	5.42	0.05
SBP	792	114.96	545	113.03	**0.01**	719	114.60	450	111.74	**0.0002**
DBP	792	72.64	545	71.27	**0.01**	719	72.06	450	70.25	**0.0009**
Cigarettes	799	24.05	553	21.34	0.26	723	22.78	454	18.24	0.09

* p-values tests whether values of a given variable are statistically different at baseline and at 18 months, within treatment arms

Appendix 1.4 Associations between baseline FTWC and markers of cardiometabolic risk

	Baseline			Pooled			Longitudinal		
	CRS								
	N=1339			**N=1439 Obs=4284**			**N=1439 Obs=4284**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	3.49	0.94	0.001	3.13	0.94	0.003	3.13	0.94	0.003
FTWC	0.19	0.23	0.41	0.13	0.22	0.56	0.20	0.25	0.43
Time				0.04	0.03	0.14	0.04	0.03	0.15
FTWC*Time							-0.03	0.05	0.52
	BMI								
	N=1418			**N=1439 Obs=4584**			**N=1439 Obs=4584**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	27.53	1.34	<.0001	26.99	1.32	<.0001	26.99	1.32	<.0001
FTWC	0.63	0.33	0.06	0.62	0.32	0.05	0.54	0.33	0.10
Time				**0.06**	**0.02**	**0.003**	**0.06**	**0.02**	**0.003**
FTWC*Time							0.04	0.03	0.26
	Total Cholesterol								
	N= 1389			**N= 1439 Obs=4484**			**N= 1439 Obs=4484**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	148.36	8.67	<.0001	176.12	4.67	<.0001	176.09	4.67	<.0001
FTWC	-1.02	1.17	0.38	-0.85	0.97	0.38	-2.52	1.56	0.10
Time				**2.89**	**0.32**	**<.0001**	**2.91**	**0.32**	**<.0001**
FTWC*Time							0.76	0.55	0.17
	HDL Cholesterol								
	N=1398			**N=1439 Obs=4487**			**N=1439 Obs=4487**		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	58.57	1.79	<.0001	62.15	0.94	<.0001	62.15	0.94	<.0001
FTWC	-0.38	0.24	0.12	-0.41	0.18	0.02	-0.53	0.30	0.08
Time				**-0.28**	**0.06**	**<.0001**	**-0.28**	**0.06**	**<.0001**
FTWC*Time							0.05	0.11	0.62

Appendix 1.4 Associations between baseline FTWC and markers of cardiometabolic risk (continued)

Systolic Blood Pressure

	N= 1426			N=1439 Obs=4603			N=1439 Obs=4603		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	120.12	2.47	<.0001	113.95	2.20	<.0001	113.93	2.20	<.0001
FTWC	-1.01	0.56	0.07	-0.60	0.52	0.25	-1.29	0.62	0.04
Time				**-0.94**	**0.09**	**<.0001**	**-0.93**	**0.09**	**<.0001**
FTWC*Time							**0.32**	**0.16**	**0.04**

Diastolic Blood Pressure

	N=1426			N=1439 Obs=4603			N=1439 Obs=4603		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	77.27	1.88	<.0001	73.61	1.63	<.0001	73.60	1.63	<.0001
FTWC	-0.75	0.42	0.08	-0.53	0.37	0.15	-0.86	0.47	0.07
Time				**-0.60**	**0.08**	**<.0001**	**-0.60**	**0.08**	**<.0001**
FTWC*Time							0.15	0.13	0.25

HbA1c

	N=1378			N=1439 Obs=4405			N=1439 Obs=4405		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	7.60	0.18	<.0001	5.75	0.12	<.0001	5.75	0.12	<.0001
FTWC	0.01	0.03	0.68	0.00	0.03	0.95	0.02	0.03	0.63
Time				**-0.04**	**0.00**	**<.0001**	**-0.04**	**0.00**	**<.0001**
FTWC*Time							-0.01	0.01	0.27

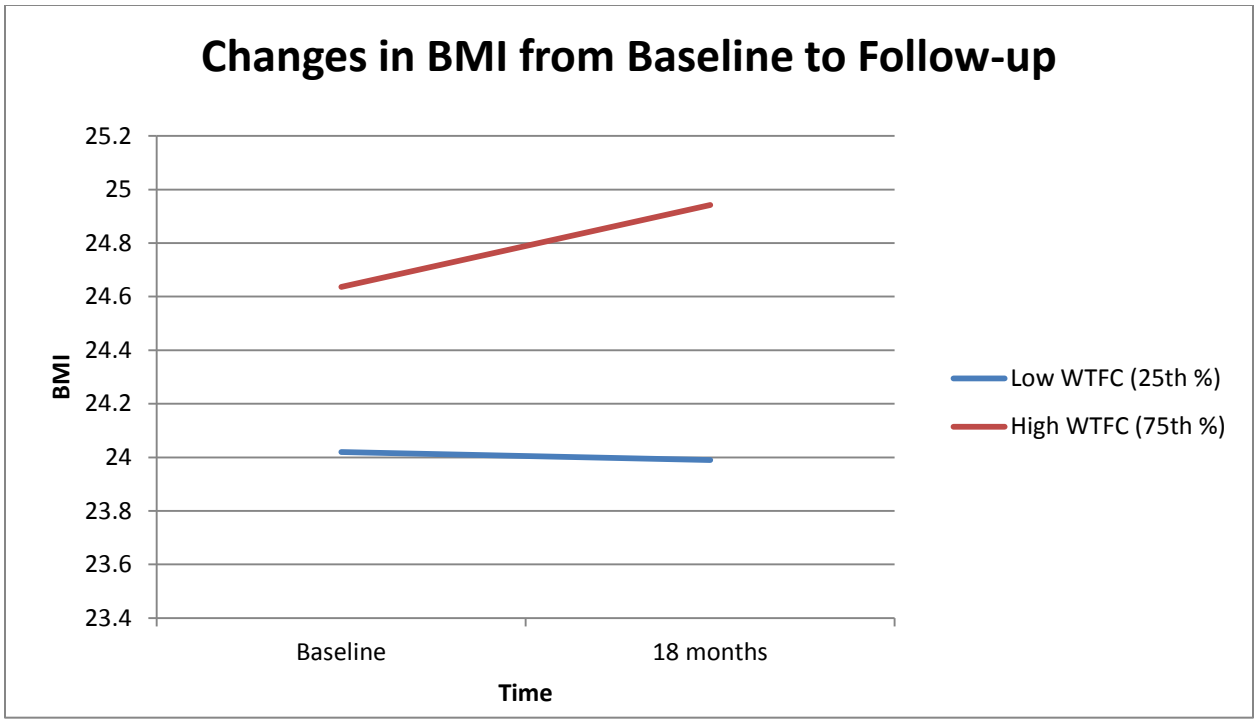
Cigarettes/week

	N=1438			N=1439 Obs=4651			N=1439 Obs=4651		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	-20.52	8.78	0.03	-21.96	8.30	0.01	-21.96	8.30	0.01
FTWC	-1.51	2.09	0.47	-1.81	1.92	0.34	-1.82	2.10	0.39
Time				**-0.94**	**0.24**	**<.0001**	**-0.94**	**0.24**	**<.0001**
FTWC*Time							0.004	0.41	0.99

All models control for treatment status, race, education, age, sex, marital status, foreign born status, occupation, work hours, number of children, FSSB, job strain and schedule

Appendix 1.5: Tests of reverse causation (Pooled)

	WTFC N=1338 obs=4333			FTWC N=1339 obs=4337		
	Estimate	**Standard Error**	**p-value**	**Estimate**	**Standard Error**	**p-value**
Intercept	2.94	0.09	<.0001	2.14	0.06	<.0001
CRS at baseline	-0.005	0.00	0.06	-0.0005	0.002	0.79
Time	-0.03	0.01	<.0001	0.00	0.01	0.48



* Figure generated using the following regression equation and inputting values for time and WTFC:
$BMI_{tij} = 26.94 + 0.42(WTFC) + 0.06(Time_{ij}) + 0.08(WTFC)(Time_{ij})$

Figure 1.1: Changes in BMI from Baseline to Follow-up by Level of WTFC*