Received: 31 March 2023 | Revised: 23 September 2024 | Accepted: 23 September 2024

DOI: 10.1111/famp.13077

FAMILY PROCESS

ORIGINAL ARTICLE

Relationship quality and educational attainment links to development of cardiometabolic morbidity and multimorbidity across middle adulthood

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Abstract

The prevalence of cardiometabolic morbidity (e.g., high blood pressure, heart attack, stroke, type 2 diabetes) and multimorbidity development (2 or more cardiometabolic morbidities) are rapidly growing in the US. Cardiometabolic morbidity and multimorbidity are linked to poor well-being outcomes, high healthcare costs, and mortality. There is little known about cardiometabolic multimorbidity health disparities, particularly regarding mutable factors that might be targeted in future health interventions. In the present study, using a biopsychosocial framework (Biobehavioral Family Model), we examine whether cardiometabolic morbidity and multimorbidity development are linked to premorbid family and marital relationships and if it differs depending on socioeconomic status (i.e., educational attainment) using three waves of Midlife in the US ($N = 4951$). We assessed cardiometabolic development with three conceptualizations: *number of cardiometabolic morbidities* (i.e., count variable), *individual cardiometabolic morbidities* (i.e., diabetes, high blood pressure, stroke, heart attack), and *severity of cardiometabolic multimorbidity* (e.g., 3+ vs. zero morbidities). Family strain increased the number of cardiometabolic morbidities (OR = 1.17) and the severity of multimorbidity (e.g., 3+ morbidities: OR = 1.38). People with a high school education experienced family support as a buffer to the negative health impact of education level. Generally, marital quality appeared less impactful on cardiometabolic morbidity and multimorbidity development compared to family strain. Positive and negative family characteristics appear to function differently across educational attainment. These findings indicate that adults' non-intimate family relationships predict important outcomes such as

diabetes, heart attack, stroke, and cardiometabolic multimorbidity and should be considered targets for preventative health interventions.

KEYWORDS

cardiometabolic risk factors, diabetes, education, family relations, heart attack, heart disease, high blood pressure, marriage, stroke

Cardiometabolic morbidity (e.g., high blood pressure, heart attack, stroke, type 2 diabetes) has been growing in prevalence in the US, particularly cardiometabolic *multi*morbidity (i.e., 2 or more cardiometabolic morbidities; Cheng et al., 2022; Glynn, 2009). In 2018, an estimated 14.4% of adults in the US had cardiometabolic multimorbidity and this percentage appears to be increasing (Cheng et al., 2022). Cardiometabolic morbidity (and multimorbidity) is linked to depression (Huang et al., 2022), cognitive dysfunction (Lyall et al., 2017), and multiplicative mortality for each additional cardiometabolic morbidity diagnosis (Di Angelantonio et al., 2015). While there is growing evidence that cardiometabolic morbidity and multimorbidity are more common among men, older adults, and non-Hispanic Black adults (Cheng et al., 2022), there is still limited research to date about cardiometabolic health disparities in the United States including mutable factors that may slow the increasing trajectory of cardiometabolic morbidity and multimorbidity prevalence via targeted intervention.

Educational attainment is one factor linked to prominent health disparities, with lower educational attainment associated with poorer health outcomes including higher rates of cardiometabolic morbidity and multimorbidity (Cheng et al., 2022; Havranek et al., 2015; Jackson et al., 2018; Kubota et al., 2017; Steele et al., 2017). Educational attainment is a proxy for the structural inequities that perpetuate health disparities as racially, ethnically, and economically marginalized people are often not granted access to institutions of higher education (East & Friedson, 2020; Evans & Kim, 2010). Additionally, the quality of close relationships is a powerful predictor of health across adulthood and important factor of and individual's social determinants of health. Specifically, higher-quality marital relationships (i.e., spousal/cohabiting partners) and non-marital family relationships (i.e., parents, siblings, and children) are consistently linked to better health outcomes across adulthood compared to lower-quality relationships (Carr & Springer, 2010; Roberson, Shorter, et al., 2018; Robles et al., 2014; Woods, Priest, & Roberson, 2020). However, we know less about how close relationships (e.g., marital and non-marital family relationships) and educational attainment intersect in their influence on the development of cardiometabolic morbidity and multimorbidity. Biopsychosocial frameworks, like the Biobehavioral Family Model (Wood, 1993; Wood et al., 2021), hypothesize that both positive and negative aspects of close relationships can impact health across adulthood and buffer against the stress of structural inequities that perpetuate health disparities. Understanding how relationship quality and educational attainment intersect would provide greater contextualization of previous research on close relationships and health and potentially offer a clearer path for intervention (Roberson & Fincham, 2018; Woods, Priest, & Roberson, 2020). The present study investigates how the quality of close relationships (i.e., marital and non-marital family relationships) may buffer against (or exacerbate) the impact of educational attainment on the development of cardiometabolic morbidity and multimorbidity development across 20 years of middle adulthood.

Theoretical model: Biobehavioral Family Model

Biopsychosocial models for health emphasize that social, psychological, and biological factors interact and contribute to disease development. One biopsychosocial model, the Biobehavioral

Family Model (BBFM), assumes the family system (e.g., marital and non-marital relationships) can serve as a buffer against and method of coping with stress to determine health outcomes (Wood et al., 2021), with substantial empirical support for explaining adult health (Roberson, Shorter, et al., 2018; Woods & Denton, 2014; Woods, Priest, & Roberson, 2020). The BBFM's exogenous predictor—the family emotional climate (FEC)—is conceptualized to include positive (e.g., warmth, support) and negative (e.g., criticism, hostility, withdrawal) exchanges within a family (i.e., marital and non-marital family relationships), and emphasizes the intensity and balance (or imbalance) of these emotional exchanges to reflect family-level relationship quality. FEC has largely been operationalized as the present quality of family relationships (e.g., marital and non-marital). However, there is evidence that it may be important to account for the long reach of parent–child relationship quality experienced in childhood in conjunction with measuring concurrent FEC (Priest et al., 2019; Woods, Roberson, & Priest, 2020).

The BBFM posits that FEC impacts a family member's disease activity (e.g., disease development or symptom severity of a specific illness) via biobehavioral reactivity—individual family members' psychophysiological responses to stress. For example, the impact of a more negative FEC on worse disease activity is conveyed via greater biobehavioral reactivity (i.e., worse psychophysiological distress). Biobehavioral reactivity thus serves as a mediator, linking FEC and disease activity. However, this will not be directly tested here but has been tested previously with MIDUS (e.g., Priest et al., 2020). As such, this study examines, in part, the direct connection between FEC and the development of cardiometabolic conditions.

One aspect of the BBFM framework that has received only minimal empirical attention is stressors caused by structural inequities (e.g., race/ethnicity discrimination, poverty, educational attainment) on health (Priest et al., 2020). Wood et al. (2000) hypothesize that FEC can mitigate contextual stressors' effects on our health. This hypothesis can be applied in two ways: FEC serving as a moderator, or, FEC serving as a mediator. First, acting as a moderator whereby negative FEC leaves individuals unbuffered from these contextual stressors that directly impact health, or conversely, a positive FEC can buffer against the impact of contextual stress on health. Second, acting as a mediator, stress within the FEC can increase and serve to convey the negative effects of external stressors on worse health outcomes. There is one known test of each of these interpretations. Using FEC as a moderator, Roberson and Fincham (2018) found that positive FEC buffers against the stress of low income on diabetes management. Examining FEC as a mediator, Priest et al. (2020) found that the stress of discrimination conveys a negative effect on health through FEC for African Americans. While mediating tests are important to explain *why* contextual stressors impact health outcomes, moderating tests are more appropriate for explaining differences within a sample given exposure to an external stressor and can better prime findings for interventions. Therefore, given that the purpose of the study is to identify how FEC may differentially buffer or exacerbate the impact of educational attainment on cardiometabolic morbidity (and multimorbidity), we will test the BBFM using the moderation approach.

Close relationships and cardiometabolic morbidity and multimorbidity developments

Previous research examining close relationships and health outcomes has found that the quality of non-marital family relationships has a larger effect on long-term health outcomes compared to the quality of marital relationships (Roberson et al., 2023; Woods, Priest, & Roberson, 2020). Close relationships (i.e., marital and non-marital family relationships) may

be particularly relevant to cardiometabolic morbidities. Poor quality or strained close relationships contribute to increased risk of cardiovascular disease (De Vogli et al., 2007; Farrell & Simpson, 2017; Orth-Gomer et al., 2000) and type 2 diabetes (Roberson & Fincham, 2018; Troxel et al., 2005; Whisman et al., 2014), while positive close relationships decrease these risks (Roberson & Fincham, 2018; Troxel et al., 2005; Tulloch & Greenman, 2018). Poor relationship quality is also linked to increases in multiple cardiometabolic risk factors including increased daily blood pressure (Bennett-Britton et al., 2017; Grewen et al., 2005), carotid artery intima medial thickness (a subclinical marker of cardiovascular disease risk; Atallah et al., 2014), inflammation (Ford et al., 2019; Kiecolt-Glaser & Wilson, 2017), and greater body mass index (Skoyen et al., 2018).

There are several theories on pathophysiological mechanisms that underlie marital and family relationships influence on health and, specifically, cardiometabolic morbidity development. A primary theory is the activation of the hypothalamic–pituitary-adrenocortical (HPA) axis from stressors such as family stress and a lack of social support (Popovic et al., 2022). Stressors that activate the HPA axis negatively impact the cardiovascular system by promoting the escalation of the atherosclerotic process leading to cardiovascular disease development (Popovic et al., 2022). The activation of the HPA axis leads to the release of glucocorticoids (i.e., cortisol), catecholamines (i.e., epinephrine and norepinephrine), and adrenocorticotropic hormone (Popovic et al., 2022). Glucocorticoids are responsible for several physiological functions, including immunity, metabolism, and cardiovascular activity (Xia & Li, 2018). When chronic stress is applied through stressors such as lower family and marital quality, glucocorticoid resistance can occur, which leads to excessive inflammation and hyperactivity of corticotropic-releasing hormone as well as the sympathetic nervous system (SNS), which contributes to the subsequent development of disease (Xia & Li, 2018). Additionally, the hormones released from activation of the HPA axis and SNS (glucocorticoids and catecholamines) and higher levels of pro-inflammatory cytokines can promote endothelial dysfunction, leading to vascular inflammation and the development of atherosclerosis (Popovic et al., 2022).

Despite these pathophysiological theoretical underpinnings, less is known about how specific characteristics of close relationships are linked to the pattern of cardiometabolic morbidity and multimorbidity development including (1) the number of cardiometabolic morbidities (i.e., the number of cardiometabolic morbidities on a linear scale; Farrell & Stanton, 2019; Tulloch & Greenman, 2018), (2) individual cardiometabolic morbidities (e.g., diabetes development, heart disease), and (3) the severity of cardiometabolic multimorbidities (e.g., development of 3+ versus zero cardiometabolic morbidities).

Educational attainment and cardiometabolic morbidity and multimorbidity development

Education attainment has long been linked to health disparities, whereby individuals with lower educational attainment have increased morbidity and mortality (Cohen et al., 2010; Zajacova & Lawrence, 2018). For example, Blümel and colleagues (2020) found a 40% higher likelihood of multimorbidity in individuals with lower educational attainment. This health disparity is also seen in younger age groups, adults aged 30–64 (Johnson-Lawrence et al., 2017), and across gender and racial groups (Cohen et al., 2010; Zajacova & Lawrence, 2018). Specifically, lower educational attainment is linked with a higher prevalence of cardiovascular risk factors, higher incidence of cardiovascular events (Havranek et al., 2015; Kubota et al., 2017), increased stroke risk (Jackson et al., 2018), and greater risk of developing type 2 diabetes (Steele et al., 2017). However, it is not just educational attainment that directly impacts health

disparities. Educational attainment should be seen as a proxy measure for the structural factors that both prevent marginalized groups (e.g., racially, ethnically, and economically) from entering institutions of higher education and perpetuate inequities that limit access to health-promoting factors like preventative healthcare, healthier food options, higher wages, and safer work and home environments (East & Friedson, 2020; Evans & Kim, 2010). Examining the role of close relationships in the context of individuals' educational attainment may illuminate a critical path for targeted interventions to alleviate cardiometabolic morbidity disparities in the United States.

Present study

Marital and non-marital family relationships impact many indicators of health across adulthood. Given the pathways by which close relationships impact health, these relationships are likely linked to the development of cardiometabolic morbidity and multimorbidity. It is also important to understand how close relationships and educational attainment intersect to predict cardiometabolic morbidity development to contextualize the previous research and potentially offer a clearer path for intervention. Using the BBFM framework, this present study is led by three hypotheses (see Figure 1).

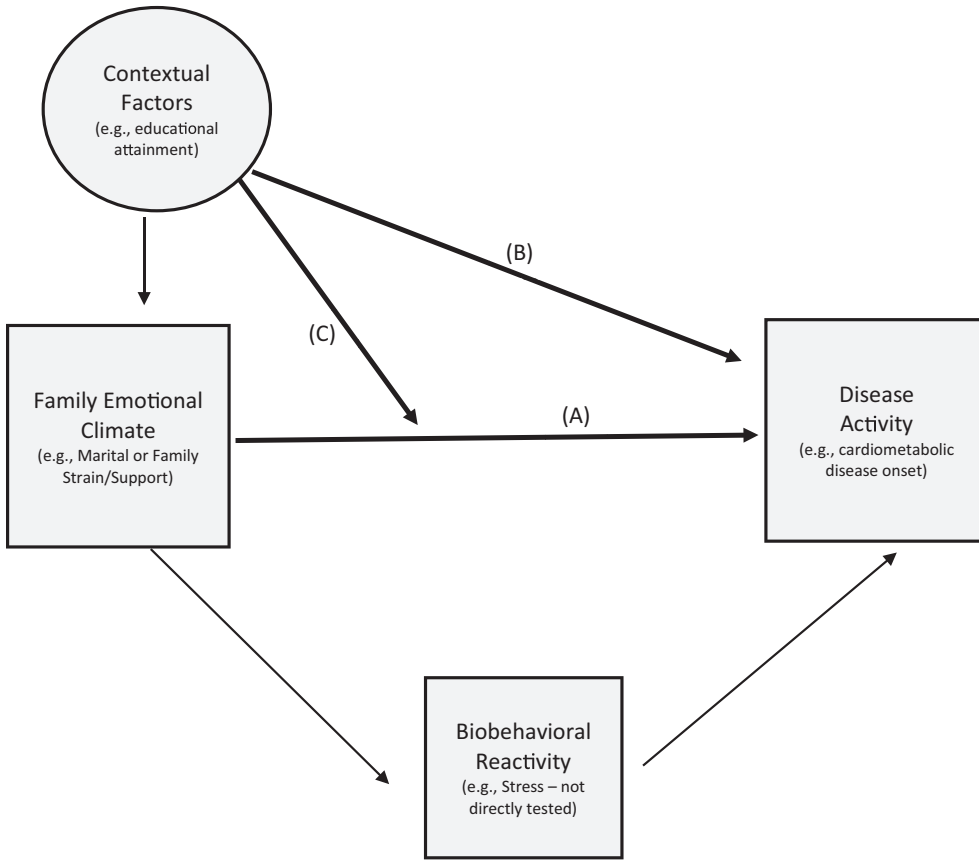

FIGURE 1 The biobehavioral family theoretical framework. Hypothesis 1 is a test of path A, Hypothesis 2 is a test of path B, and Hypothesis 3 is a test of path C.

H1. Family emotional climate (FEC) predicts the development of cardiometabolic morbidity and multimorbidity across 20 years in middle adulthood. Specifically, a positive FEC (greater marital and family support) predicts a decreased risk of cardiometabolic morbidity and multimorbidity, and a negative FEC (greater marital and family strain) predicts an increased risk of cardiometabolic morbidity and multimorbidity.

H2. Educational attainment predicts the development of cardiometabolic morbidity and multimorbidity across 20 years in middle adulthood. Specifically, having a college education will decrease the risk of cardiometabolic morbidity and multimorbidity compared to having a high school education or some college (e.g., an associate degree).

H3. Educational attainment interacts with FEC to predict the development of cardiometabolic morbidity and multimorbidity. Specifically, a positive FEC buffers against lower education's negative impact on cardiometabolic development. A negative FEC exacerbates the negative effects of lower educational attainment on cardiometabolic development.

We test these hypotheses across three conceptualizations of the development of cardiometabolic morbidity and multimorbidity. First, we examine the *number of cardiometabolic morbidities* by examining the additive effect on the total number of developed cardiometabolic morbidities on a linear scale. Second, we examine the *individual development of cardiometabolic morbidities* (e.g., high blood pressure, diabetes, stroke, and heart attack). Third, because we know cardiometabolic morbidities tend to cluster (i.e., people often develop more than one; Cheng et al., 2022; Reiter-Brennan et al., 2021) but it is unclear how relational and contextual factors are linked to the *severity* of these multimorbidities (i.e., 2 cardiometabolic morbidities, 3+ cardiometabolic morbidities), we examine the *severity of cardiometabolic multimorbidity development*. We repeated these hypotheses *first* with non-marital family relationship quality (family strain and support) and *second* with marital relationship quality (marital strain and support).

METHOD

Sample

Our sample includes adults who participated in all three waves of the Midlife Development in the US (MIDUS). MIDUS is a nationally representative survey of adults in the US to examine the contribution of psychosocial factors on aging and mental and physical health trajectories (Brim et al., 2004; Ryff et al., 2017). MIDUS 1, the initial collection of data (1995–1996; $N = 7108$, $M_{age} = 46.38$, $SD = 13.0$; 51.1% female), recruited participants using random-digit-dialing. MIDUS 2, the first follow-up wave (2004–2006), has a response rate of 75.4% ($N = 4963$, $M_{age} = 55.43$, $SD = 12.45$; 56.8%). MIDUS 3, the second follow-up (2013–2014), includes 66% of MIDUS 1 participants ($N = 3294$, $M_{age} = 63.64$, $SD = 11.35$; 54.9% female; Ryff et al., 2017).

To test the hypotheses using family strain and support, we include all individuals who completed MIDUS 1 and either MIDUS 2 or 3 ($n = 4951$) in order to predict the development of cardiometabolic morbidity and multimorbidities that occur in MIDUS 2 and 3. The sample used to test family strain and support will be referred to as the *family sample*. To test the hypotheses using marital strain and support, we use a subsample of the family sample. Specifically, we narrowed the family sample to include only individuals who report being married (or cohabitating)

at MIDUS 1 ($n = 3487$) to examine the effect of marital strain and support. The sample used to test marital strain and support will be referred to as the *marital sample*. Demographics for both samples are presented in Table 1.

For models that test the *number of cardiometabolic morbidities* (i.e., a count variable), we further reduce the family and marital samples depending on the individual cardiometabolic morbidities tested. Specifically, when testing the risk of heart attack development over 20 years (MIDUS 2 and 3 combined), individuals who report having had a heart attack at MIDUS 1 are *not* included in the sample (family sample: $n = 4512$; marital sample: $n = 3060$). Additionally, when we examine the *number of cardiometabolic morbidities* (i.e., count variables) and the *severity of cardiometabolic multimorbidities* (e.g., 3+ vs. 0 morbidities) over

TABLE 1 Distribution of key variables for the non-marital family dataset ($N = 4951$) and the Marital dataset ($N = 3487$).

Variable	Non-marital family dataset Mean (*SD*) or %	Marital dataset Mean (*SD*) or %
Age	46.44 (12.51)	46.87 (12.16)
Gender		
Female	53%	50%
Male	47%	50%
Race		
White	94%	95%
Non-White	6%	5%
Marital status		
Married/Cohabiting	70%	100%
Other (e.g., single, divorce, widowed)	30%	0%
Family emotional climate		
Family strain	2.19 (0.59)	2.07 (0.58)
Family support	3.45 (0.60)	3.48 (0.57)
Marital strain	2.22 (0.60)	2.07 (0.58)
Marital support	3.59 (0.54)	3.60 (0.53)
Education		
High school	6%	8%
Some college	41%	57%
College education	53%	35%
Risk of individual cardiometabolic 20 year onset		
Heart attack 20 year onset	3%	3%
High blood pressure 20 year onset	32%	32%
Diabetes 20 year onset	12%	11%
Stroke 20 year onset	5%	5%
Risk of total cardiometabolic 20 year onset (count)	0.54 (0.80) (Range: 0–4)	0.54 (0.79) (Range: 0–4)
Risk of cardiometabolic 20 year onset clustering		
3 or more cardiometabolic 20 year onsets	3%	3%
2 Cardiometabolic 20 year onsets	10%	10%
1 Cardiometabolic 20 year onset	26%	26%
0 Cardiometabolic 20 year onsets	62%	61%

20 years, individuals who report having any cardiometabolic morbidities at MIDUS 1 are *not* included in the samples (family sample: $n = 3732$; marital sample: $n = 2532$). This further reduction in the sample is to test the development of cardiometabolic morbidities and multimorbidity accurately in MIDUS 2 and 3 by removing individuals who report one or more diagnoses at MIDUS 1.

According to the University of Tennessee, Knoxville Institutional Review Board, the use of MIDUS is not classified as human subjects research.

Measures

We assess the predictor variables (i.e., FEC and educational attainment) and control variables during MIDUS 1. The original MIDUS researchers used mean imputation and assigned scale scores if respondents completed at least one item to accommodate missing data (Brim et al., 2004). For the outcome variables (i.e., the cardiometabolic morbidity and multimorbidity measures), we combine the cardiometabolic morbidity self-reports over the 20 years of MIDUS 2 and 3. Descriptive statistics are presented in Table 1.

Family Emotional Climate (FEC). For each of our family sample models, FEC is measured using the family strain and support scales found in MIDUS (Walen & Lachman, 2000), which have been substantiated in previous studies using the BBFM (Priest et al., 2020; Roberson, Woods, et al., 2018; Woods, Priest, & Roberson, 2020). The family strain and support measure includes four items, each on a scale ranging from 1 (*a lot*) to 4 (*not at all*), and both have good internal consistency ($\alpha = 0.78$ and $\alpha = 0.83$, respectively). In the second set of models, the marital sample models, FEC is measured as marital strain and marital support, both used in previous tests of the BBFM (Roberson, Shorter, et al., 2018; Woods, Priest, & Roberson, 2020). Marital strain and support each include six items, with responses ranging from 1 (*often*) to 4 (*never*); both are reliable ($\alpha = 0.83$ and $\alpha = 0.87$, respectively). These measures have been used to operationalize the positive and negative aspects of FEC (Priest et al., 2019, 2020; Roberson, Shorter, et al., 2018; Woods, Priest, & Roberson, 2020; Woods, Roberson, & Priest, 2020).

Educational attainment is operationalized as a categorical variable: (1) high school, GED, or less, (2) some college or technical degree, and (3) 4-year college degree or graduate degree that is dummy coded so that 4-year college degree/graduate degree serves as the reference category. A similar categorization of education has frequently been used with MIDUS data (Grzywacz et al., 2004; Grzywacz & Almeida, 2008) as educational attainment may not represent a linear association but a curvilinear association with health factors linked to cardiometabolic morbidity and multimorbidity (e.g., Cohen et al., 2013; Jeon et al., 2015). We grouped the GED and high school educational attainment categories together as these levels of education are not mutually exclusive, and each is similarly predictive of early mortality (compared to higher levels of educational attainment; Rogers et al., 2010). These educational attainment levels capture well-established structural inequities and socioeconomic differences (Adler et al., 1994; Marks & Shinberg, 1998; Marmot et al., 1997). Also, compared to other measures of socioeconomic status (e.g., income, occupation), education tends to have fewer missing data points.

Cardiometabolic Morbidity and Multimorbidity Development are measured with three conceptualizations of cardiometabolic morbidity and multimorbidity. Utilizing three different constructs of the same variables provides a greater understanding of how these conditions function independently and together in the context of FEC and educational attainment. First, to assess the accumulation of risk of developing cardiometabolic morbidities, we use a count variable of the total number of cardiometabolic morbidities. Cardiometabolic conditions are highly correlated, with a diagnosis of one condition being a risk factor for developing other cardiometabolic morbidities (Cheng et al., 2022; Reiter-Brennan et al., 2021). Therefore, this measure captures the potential risk of cardiometabolic development accumulation. To

compute the *number of cardiometabolic morbidities* variable, we summed the incidence of high blood pressure (HBP), diabetes, stroke, and heart attack development across the 20 years of MIDUS 2 and 3. The final count variable ranged from 0–4 cardiometabolic morbidities.

Second, we examine the *individual cardiometabolic morbidity* development with four dichotomous variables: HBP, diabetes, stroke, and heart attack development. MIDUS 2 and 3 asked participants if they had experienced this health incident in the last 12 months and if they had a history of this health incident. Participants were coded as "yes" if they reported having a history or experiencing a health incident in the last 12 months. The tested models did not include individuals who reported a history of the health incident at MIDUS 1. This process created 4 new variables: HBP Development (0 = none, 1 = new HBP); Diabetes Development (0 = none, 1 = new diabetes); Stroke Development (0 = none, 1 = new stroke); and, Heart Attack Development (0 = none, 1 = new heart attack).

Third, we examine the differences in the *severity of cardiometabolic multimorbidity* using a categorical variable of the number of cardiometabolic morbidity development. Though we know that cardiometabolic conditions tend to cluster, we wanted to test whether FEC and educational attainment increase the risk of a person experiencing a specific number of morbidities simultaneously, as a higher number of cardiometabolic morbidities indicates more severe multimorbidity. To compute the *severity of cardiometabolic multimorbidity*, we categorized the *number of cadiometabolic morbidities* into a four-level categorical variable. Specifically, categorical levels include: 0 = zero cardiometabolic developments, 1 = 1 cardiometabolic development, 2 = 2 cardiometabolic developments, 3+ = 3–4 cardiometabolic developments. The reference category is zero cardiometabolic morbidities.

Control Variables are assessed at MIDUS 1 (the same time as FEC and educational attainment). To account for the known influence of childhood family environment on health outcomes, *Childhood FEC* is accounted for by including measures of maternal and paternal affection—previously used in BBFM tests with adult populations (Priest et al., 2019; Woods, Roberson, & Priest, 2020). As the present hypotheses specify a longitudinal association between concurrent (baseline) FEC and long-term cardiometabolic morbidities, we sought to control for one aspect of FEC in childhood via the inclusion of maternal and paternal affection. Each are assessed using a 7-item scale with responses ranging from 1 (*a lot*) to 4 (*not at all*) which were reverse coded then averaged (Rossi, 2001). Additionally, for the marital models, family strain and support are included as additional control variables to isolate an independent effect of marital relationship quality.

Gender and age—factors not commonly linked to structural inequities but frequently linked to cardiometabolic morbidity and multimorbidity—were also included as control variables in every model. Gender is a dichotomous variable (0 = men, 1 = women), and age is a continuous variable (range: 20–75).

For models testing *individual cardiometabolic morbidity*, a total of all other *pre-existing cardiometabolic conditions* (range from 0 to 3) is included as a control variable to account for the known clustering of cardiometabolic morbidities (i.e., multimorbidity). For the models examining the *number of cardiometabolic morbidities* and *severity of cardiometabolic multimorbidities*, participants with 1 or more pre-existing cardiometabolic conditions at MIDUS 1 are removed from the samples; therefore, it is not necessary to include the pre-existing cardiometabolic morbidities as a control variable.

Analytic strategy

First, we conducted a priori power analyses to determine the sensitivity of each model. We used the following to compute sensitivity: (a) the distribution of cardiometabolic development, (b) the sample size for each test, (c) a two-tailed alpha of 0.05, and (d) power estimation

(1-beta) of 0.80. We conducted all sensitivity tests in Gpower (Erdfelder et al., 1996). The family and marital samples are powered to detect parameters with a small effect size ranging from OR = 1.06–1.33 and OR = 1.07–1.39, respectively.

All hypotheses are tested in Mplus 8 using full information maximum likelihood to handle missing data (Muthén & Muthén, 1998–2017). Across the individual cardiometabolic morbidity variables, the percentage of missing data varied: Stroke (4% missing), Diabetes (21% missing), Heart Problems (9% missing), High Blood Pressure (20% missing), and Heart Attack (1% missing). The pattern of missing data was linked to several control variables (e.g., gender, marital status), making the data Missing at Random (MAR). There were no missing data on the combined *number of cardiometabolic morbidities* and *severity of cardiometabolic multimorbidity* variables because we used all available information in the calculation.

First, hypotheses were tested with the larger family sample of three-wave MIDUS participants using family strain and support as FEC measures. Next, hypotheses were tested in the marital subsample, using marital strain and support as FEC measures. All hypotheses were tested simultaneously in each regression model. For H1, we examined the strain and support FEC parameter estimate. For H2, we dummy-coded the categorical education variable and interpreted the parameter estimates of high school education and some college education (college education is the reference category when interpreting these parameters). For H3, we interpret the parameter estimates of four interaction terms: (1) positive FEC*high school education, (2) positive FEC*some college education, (3) negative FEC*high school education, and (4) negative FEC*some college education. We probe significant interactions with simple slopes analysis to determine the differential effect of FEC depending on education.

Additionally, we examined bivariate correlations among the FEC variables to evaluate any concerns for multicollinearity. The results indicate family strain and support are not a concern for multicollinearity ($r = -0.39$, $p < 0.001$), and while marital strain and support are more strongly correlated ($r = -0.67$, $p < 0.001$), it does not rise to the threshold of concern for multicollinearity ($r = 0.80$; Berry & Feldman, 1985). Nevertheless, in addition to standardizing independent variables to reduce multicollinearity, linear ridge regression can be used to mitigate bias in parameter estimation caused by multicollinearity (Kim, 2020). Mplus calculations employ ridged regression automatically as needed (Muthen, March 14, 2006).

We use three regression methods to examine the three conceptualizations of cardiometabolic morbidity and multimorbidity development. First, we use Poisson regression to examine the *number of cardiometabolic morbidities*. A significant positive parameter estimate would indicate that a one-point increase in the FEC variable would be linked to an increased risk of developing additional cardiometabolic morbidities in an additive, or linear fashion, while a significant negative parameter estimate would indicate that a one-point increase in the FEC variable would be linked to a decreased risk of developing additional cardiometabolic morbidity. Second, to examine the *individual cardiometabolic morbidity* (i.e., HBP, diabetes, stroke, and heart attack), we ran a series of logistic regressions. A significant positive parameter estimate would indicate that higher FEC scores would be linked to an increased risk of developing the tested cardiometabolic morbidity, while a significant negative parameter estimate would indicate that higher FEC variable scores would be linked to a decreased risk of developing the tested cardiometabolic morbidity. Third, we used multinomial logistic regression to examine the *severity of cardiometabolic multimorbidity*. Multinomial logistic regression allows us to identify whether FEC's impact has a qualitatively different effect on numeric clusters of cardiometabolic developments. A significant positive parameter estimate would indicate that higher FEC scores would be linked to an increased risk of developing a specific level of multimorbidity severity (e.g., 2, 3+) versus developing zero cardiometabolic morbidities, while a significant negative parameter estimate would indicate that lower FEC scores would be linked to not developing zero cardiometabolic morbidities versus the specific level of multimorbidity. As explained above, for multinomial logistic and Poisson regression models, samples are

limited to individuals who reported having zero cardiometabolic developments at MIDUS 1. For logistic regression models, samples are limited to individuals who did not have the tested cardiometabolic development at MIDUS 1.

RESULTS

(Non-marital) family models: Number of cardiometabolic morbidities

We used a Poisson regression to test the *number of cardiometabolic morbidities* (Table 2). Family support is not significantly linked to the number of morbidities, whereas family strain is linked to a 17% increased risk of developing an additional cardiometabolic morbidity, as hypothesized. Also, individuals with some college education have a 10% *lower* risk of developing additional cardiometabolic morbidities than those with a college education. Individuals with only some college have a slightly *lower* risk of developing additional cardiometabolic morbidities compared to those with a college degree or more.

Lastly, there are two significant interaction effects in this Poisson regression model. First, there is a significant interaction between family support and high school education. For individuals with a high school education, family support is linked to a 30% *decrease* in the development of additional cardiometabolic morbidities ($B = -0.36$, $SE = 0.14$, OR = 0.70, $p < 0.05$), while there is no significant link between family support and number of cardiometabolic morbidities for individuals with a college education ($B = -0.003$, $SE = 0.04$, OR = 0.00, $p = 0.94$), a finding in support of H3. The second significant interaction is between family support and some college education. Interestingly, family support is not associated with the number of cardiometabolic morbidities for individuals with some college ($B = 0.17$, $SE = 0.09$, OR = 1.18, $p = 0.06$) nor individuals with a college degree ($B = 0.22$, $SE = 0.24$, OR = 1.25, $p = 0.64$). Meaning, while these two groups are significantly different from each other, neither effect is statistically different from zero. Therefore, though there is mathematical significance, these two parameters have no meaningful difference because they are not individually different from zero.

(Non-marital) family models: Individual cardiometabolic morbidity

Next, we sought to further understand the results of the total morbidity number and examined the individual cardiometabolic morbidities for the non-marital family models using logistic regressions (Table 2). First, for heart attack development, family strain was linked to an 80% increase in the risk of heart attack, while education was not directly linked to the risk of heart attack development. However, education interacts with family strain. Simple slope analysis revealed that for individuals with a high school education, family strain is not significantly linked to heart attack development ($B = -0.67$, $SE = 0.48$, OR = 0.51, $p = 0.16$). In contrast, for individuals with a college education, family strain increases the risk of heart attack development by 80% ($B = 0.59$, $SE = 0.20$, OR = 1.80, $p < 0.05$). There is also a significant interaction between family support and high school education. However, simple slopes analysis indicates that family support is not linked to heart attack development for individuals with a college education ($B = 0.23$, $SE = 0.20$, OR = 1.26, $p = 0.25$) nor with a high school education ($B = -0.64$, $SE = 0.40$, OR = 0.53, $p = 0.11$). Meaning, while individuals in these educational attainment groups were significantly different from one another, their individual effects are not statistically different from zero.

There is only one significant predictor among the key variables for HBP development. Contrary to our second hypothesis, individuals who reported having completed some college had a 24% lower risk of HBP development than individuals with a college education.

TABLE 2 Poisson regression (total onsets), logistic regression (individual disease onsets), and multinomial logistic regression (onset clustering) models examining the effect of marital strain, marital support, education, and their interactions on cardiometabolic onset over 20 years.

	Total onsets 20 years		**Heart attack onset 20 years**		**High blood pressure onset 20 years**		**Diabetes onset 20 years**		**Stroke onset 20 years**		**Onset clustering 20 years**					
	(*N* = 3732)		**(*N* = 4512)**		**(*N* = 2819)**		**(*N* = 4214)**		**(*N* = 4591)**		**(*N* = 3732)**					
	OR = 1.06		**OR = 1.24**		**OR = 1.10**		**OR = 1.12**		**OR = 1.18**		**OR = 1.33**		**OR = 1.17**		**OR = 1.10**	
											3+ vs. 0 Onsets		**2 vs. 0 Onsets**		**1 vs. 0 Onsets**	
A priori power analyses	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**
High school education	0.12 (0.10)	1.13	0.45 (0.35)	1.56	0.04 (0.18)	1.04	0.36 (0.21)[t]	*1.43*	0.36 (0.26)	1.43	0.34 (0.21)	1.40	0.20 (0.25)	1.22	−0.10 (0.20)	0.90
Some college education	**−0.10 (0.05)***	**0.90**	*−0.48 (0.27)*[t]	*0.62*	**−0.27 (0.10)***	**0.76**	**−0.23 (0.11)***	**0.79**	−0.14 (0.15)	0.87	−0.34 (0.21)	0.71	−0.19 (0.12)	0.83	−0.09 (0.08)	0.91
Family support	0.003 (0.04)	1.01	0.23 (0.20)	1.26	−0.16 (0.10)	0.85	−0.06 (0.10)	0.94	−0.03 (0.15)	0.97	0.10 (0.20)	1.10	−0.03 (0.11)	0.97	−0.005 (0.08)	1.01
Family strain	**0.16 (0.04)****	**1.17**	**0.59 (0.20)***	**1.80**	0.07 (0.10)	1.07	**0.39 (0.10)****	**1.48**	**0.29 (0.13)***	**1.34**	**0.32 (0.16)***	**1.38**	**0.32 (0.10)****	**1.38**	*0.14 (0.07)*[t]	*1.15*
Interaction: 1 Family strain and high school education	−0.21 (0.12)[t]	0.81	**−1.26 (0.51)***	**0.28**	−0.40 (0.29)	0.67	−0.34 (0.28)	0.71	−1.00 (0.30)	0.37	−0.31 (0.36)	0.73	−0.54 (0.49)	0.58	−0.23 (0.22)	0.79
Interaction 2: Family support and high school education	**−0.36 (0.14)***	**0.70**	**−0.86 (0.40)***	**0.42**	−0.13 (0.29)	0.87	0.13 (0.37)	1.14	−0.41 (0.31)	0.66	−0.99 (0.54)[t]	0.37	−0.56 (0.39)	0.57	*−0.62 (0.34)*[t]	*0.54*
Interaction 3: Family strain and some college education	0.09 (0.08)	1.09	−0.04 (0.41)	0.96	−0.26 (0.18)	0.77	0.23 (0.19)	1.26	0.40 (0.23)[t]	1.49	0.31 (0.27)	1.36	0.09 (0.25)	1.09	0.01 (0.13)	0.99
Interaction 4: Family support and some college education	**0.17 (0.08)***	**1.18**	0.53 (0.39)	1.70	0.05 (0.18)	1.05	**0.39 (0.19)***	**1.49**	0.33 (0.25)	1.39	**0.82 (0.34)***	**2.27**	0.12 (0.21)	1.13	0.12 (0.15)	1.13
Mother affection	−0.06 (0.04)	0.94	−0.23 (0.17)	0.79	0.01 (0.08)	1.01	−0.12 (0.10)	0.89	−0.09 (0.13)	0.91	−0.09 (0.19)	0.91	**−0.21 (0.10)***	**0.81**	−0.005 (0.07)	0.99

TABLE 2 (Continued)

	Total onsets 20 years		**Heart attack onset 20 years**		**High blood pressure onset 20 years**		**Diabetes onset 20 years**		**Stroke onset 20 years**		**Onset clustering 20 years**					
	(*N* = 3732)		**(*N* = 4512)**		**(*N* = 2819)**		**(*N* = 4214)**		**(*N* = 4591)**		**(*N* = 3732)**					
	OR = 1.06		**OR = 1.24**		**OR = 1.10**		**OR = 1.12**		**OR = 1.18**		**OR = 1.33**		**OR = 1.17**		**OR = 1.10**	
											3+ vs. 0 Onsets		**2 vs. 0 Onsets**		**1 vs. 0 Onsets**	
A priori power analyses	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**
Father affection	−0.01 (0.03)	0.99	0.01 (0.15)	0.99	−0.01 (0.07)	0.99	0.12 (0.08)	1.13	0.14 (0.11)	1.15	−0.13 (0.16)	0.88	0.08 (0.08)	1.08	−0.03 (0.06)	0.97
Marital status	0.06 (0.20)	1.06	**9.28 (2.01)****	**10k+**	−0.26 (0.32)	0.77	−0.05 (0.46)	0.95	−0.16 (0.55)	0.85	0.43 (1.12)	1.53	−0.03 (0.47)	0.97	0.09 (0.34)	1.09
Age	**0.03 (0.02)****	**1.03**	**0.06 (0.01)****	**1.06**	**0.04 (0.004)****	**1.04**	**0.02 (0.004)****	**1.02**	**0.06 (0.01)****	**1.06**	**0.07 (0.01)****	**1.07**	**0.06 (0.01)****	**1.06**	**0.05 (0.003)****	**1.05**
Gender	**−0.16 (0.05)****	**0.85**	**−1.19 (0.21)****	**0.30**	0.12 (0.09)	1.13	**−0.38 (0.11)****	**0.68**	−0.16 (0.14)	0.85	**−0.85 (0.21)****	**0.43**	**−0.25 (0.12)***	**0.78**	0.06 (0.08)	1.06
Pre-existing conditions	–	–	**0.49 (0.22)***	**1.63**	0.31 (0.22)	1.36	**0.70 (0.13)****	**2.01**	**0.39 (0.16)***	**1.48**	–	–	–	–	–	–

Note: All bolded parameters are statistically significant at $p < 0.05$. Italics are parameters trending toward significance < .10.

Abbreviations: OR, odds ratio; *SE*, standard error.

**$p < 0.001$; *$p < 0.05$;

$^{t}p < 0.10$.

Conversely, diabetes development is linked to baseline family relationship quality and education. Specifically, family strain is associated with a 48% increase in the risk of diabetes development. Also, participants who reported completing some college had a 21% decreased risk of diabetes development than individuals with a college education. There is also a significant interaction effect for participants who completed some college with family support. However, we see that the results of the simple slopes for participants with some college education ($B = 0.33$, $SE = 0.23$, OR = 1.39, $p = 0.16$) and participants with a college degree ($B = -0.06$, $SE = 0.10$, OR = 0.94, $p = 0.56$) are not significantly different from zero for either group. Finally, for stroke, baseline family strain is the sole variable significantly linked to development, specifically to a 34% increase in stroke development risk. Zero significant interaction effects indicate that family strain is problematically linked to stroke development risk across all educational categories.

In sum, higher family strain is associated with an *increased* risk of heart attack, diabetes, and stroke development. Completing some college education was associated with a *decreased* risk of high blood pressure and diabetes development (compared to participants who completed a college degree). Family support is not a significant predictor of any of the four cardiometabolic morbidity developments, and we discovered few significant interactions. As a result, we found mixed support for H1, evidence contrary to H2, and minimal support for H3 other than a family strain and education interaction effect linked to heart attack development, specifically for people with a college education.

(Non-marital) family models: Severity of cardiometabolic multimorbidities

Finally, we examine predictors of severity of cardiometabolic multimorbidity using multinomial logistic regression (Table 2). Family support was not significantly associated with cardiometabolic multimorbidity, reflecting the above results. However, family strain is linked to a 30% increase in the risk of developing 3 or more cardiometabolic multimorbidities compared to no cardiometabolic morbidities. For this comparison group (3+ vs. 0 developments), there is also a significant interaction between family support and some college education. Simple slopes analysis revealed that for participants who completed some college, family support was linked to a 144% *increase* in the risk of having 3+ morbidity developments compared to 0 developments ($B = 0.89$, $SE = 0.44$, OR = 2.44, $p = 0.04$), contrary to our second hypothesis. However, for individuals with a college education, family support did not significantly differentiate between these multimorbidity groups ($B = 0.11$, $SE = 0.19$, OR = 1.12, $p = 0.57$). Next, for individuals with 2 new cardiometabolic developments (versus 0 developments), family strain is linked to a 38% increased risk of having 2 new cardiometabolic developments, compared to zero new cardiometabolic developments ($B = 0.32$, $SE = 0.10$, OR = 1.38, $p < 0.05$), as hypothesized in H1.

Marital models: Number of cardiometabolic morbidities

For the marital models, we also tested the number of cardiometabolic morbidities with Poisson regression (Table 3). While neither marital support nor strain was directly linked to the number of morbidities, having completed some college is linked to a 13% reduction in the risk of developing each additional cardiometabolic morbidity. There was a significant interaction between marital strain and high school education. Specifically, for individuals with high school education, marital strain was linked to a 45% decreased risk of having additional cardiometabolic developments ($B = -0.60$, $SE = 0.25$, OR = 0.55, $p = 0.02$), contrary to our third hypothesis. In contrast, marital strain was not linked to cardiometabolic developments for individuals with a college education ($B = -0.03$, $SE = 0.07$, OR = 0.97, $p = 0.68$).

TABLE 3 Poisson regression (total onsets), logistic regression (individual disease onsets), and multinomial logistic regression (onset clustering) models examining the effect of marital strain, marital support, education, and their interactions on cardiometabolic onset over 20 years.

	Total onsets 20 years		**Heart attack onset 20 years**		**High blood pressure onset 20 years**		**Diabetes onset 20 years**		**Stroke onset 20 years**		**Total onsets 20 years**		**Onset clustering 20 years**					
	(*N* = 2532)		**(*N* = 3060)**		**(*N* = 2625)**		**(*N* = 2992)**		**(*N* = 3117)**		**(*N* = 2532)**		**(*N* = 2532)**					
	OR = 1.07		**OR = 1.34**		**OR = 1.12**		**OR = 1.18**		**OR = 1.26**		**OR = 1.07**		**OR = 1.39**		**OR = 1.24**		**OR = 1.14**	
													3+ vs. 0 Onsets		**2 vs. 0 Onsets**		**1 vs. 0 Onsets**	
A priori power analyses	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**
High school education	0.14 (0.11)	1.15	0.46 (0.36)	1.58	−0.06 (0.20)	0.94	**0.48 (0.22)***	1.62	0.34 (0.29)	1.40	0.14 (0.11)	1.15	0.27 (0.47)	1.31	0.30 (0.27)	1.34	−0.10 (0.23)	0.90
Some college education	**−0.14 (0.06)***	**0.87**	*−0.62 (0.32)*[t]	*0.54*	**−0.21 (0.10)***	**0.81**	**−0.43 (0.15)***	0.65	−0.26 (0.21)	0.77	**−0.14 (0.06)***	**0.87**	−0.55 (0.29)	0.58	−0.16 (0.15)	0.85	*−0.19 (0.10)*[t]	*0.83*
Marital support	−0.05 (0.07)	0.95	−0.14 (0.19)	0.87	−0.06 (0.13)	0.94	−0.01 (0.16)	0.99	0.22 (0.24)	1.24	−0.05 (0.07)	0.95	−0.12 (0.34)	0.89	−0.18 (0.19)	0.84	0.14 (0.13)	1.15
Marital strain	−0.03 (0.07)	0.97	**−0.85 (0.34)***	**0.43**	0.06 (0.12)	1.06	0.10 (0.16)	1.10	0.16 (0.24)	1.17	−0.03 (0.07)	0.97	−0.38 (0.35)	0.68	0.01 (0.18)	0.00	0.14 (0.12)	1.15
Interaction: 1 Marital strain and high school education	**−0.57 (0.24)****	**0.56**	−0.48 (0.79)	0.62	**−1.37 (0.46)****	**0.25**	−0.09 (0.45)	0.91	−0.65 (0.58)	0.52	**−0.57 (0.24)****	**0.56**	−1.05 (1.00)	0.34	−0.70 (0.54)	0.50	**−1.11 (0.77)***	**0.33**
Interaction 2: Marital support and high school education	−0.41 (0.26)	0.66	0.40 (1.07)	1.49	−0.81 (0.56)	0.44	−0.06 (0.49)	0.94	0.27 (0.81)	1.31	−0.41 (0.26)	0.66	−0.02 (1.16)	0.98	−0.92 (0.60)	0.41	−0.43 (0.66)	0.65
Interaction 3: Marital strain and some college education	−0.03 (0.14)	0.97	−0.24 (0.65)	0.79	−0.03 (0.23)	0.97	−0.12 (0.32)	0.89	0.20 (0.47)	1.22	−0.03 (0.14)	0.97	0.21 (0.67)	1.23	−0.09 (0.35)	0.91	−0.23 (0.23)	0.79
Interaction 4: Marital support and some college education	−0.16 (0.14)	0.85	0.43 (0.79)	1.54	−0.34 (0.25)	0.71	0.17 (0.35)	1.18	−0.35 (0.48)	0.70	−0.16 (0.14)	0.85	−0.28 (0.63)	0.76	−0.22 (0.36)	0.80	−0.38 (0.25)	0.68
Family support	−0.08 (0.06)	0.92	−0.14 (0.19)	0.87	−0.18 (0.10)[t]	0.84	−0.06 (0.13)	0.94	−0.22 (0.17)	0.80	−0.08 (0.06)	0.92	−0.23 (0.23)	0.79	−0.06 (0.14)	0.94	−0.13 (0.10)	0.88
Family strain	*0.10 (0.05)*[t]	*1.10*	*0.48 (0.25)*[t]	*1.62*	0.14 (0.09)	1.15	0.17 (0.13)	1.18	0.19 (0.17)	1.21	*0.10 (0.05)*[t]	*1.10*	0.19 (0.21)	1.21	0.19 (0.13)	1.21	0.12 (0.09)	1.13

(Continues)

TABLE 3 (Continued)

	Total onsets 20 years		**Heart attack onset 20 years**		**High blood pressure onset 20 years**		**Diabetes onset 20 years**		**Stroke onset 20 years**		**Total onsets 20 years**		**Onset clustering 20 years**					
	(*N* = 2532)		**(*N* = 3060)**		**(*N* = 2625)**		**(*N* = 2992)**		**(*N* = 3117)**		**(*N* = 2532)**		**(*N* = 2532)**					
	OR = 1.07		**OR = 1.34**		**OR = 1.12**		**OR = 1.18**		**OR = 1.26**		**OR = 1.07**		**OR = 1.39**		**OR = 1.24**		**OR = 1.14**	
													3+ vs. 0 Onsets		**2 vs. 0 Onsets**		**1 vs. 0 Onsets**	
A priori power analyses	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**	***B* (*SE*)**	**OR**
Mother affection	−0.05 (0.05)	0.95	−0.17 (0.21)	0.84	0.03 (0.09)	1.03	−0.09 (0.12)	0.91	−0.14 (0.16)	0.87	−0.05 (0.05)	0.95	−0.05 (0.22)	0.95	*−0.22 (0.12)*[t]	*0.80*	0.05 (0.09)	1.05
Father affection	*−0.07 (0.04)*[t]	*0.93*	−0.11 (0.18)	0.90	−0.02 (0.07)	0.98	−0.01 (0.10)	0.99	*0.25 (0.14)*[t]	*1.28*	*−0.07 (0.04)*[t]	*0.93*	**−0.38 (0.18)****	**0.68**	−0.05 (0.10)	0.95	−0.03 (0.08)	1.03
Age	**0.04 (0.002)****	**1.04**	**0.06 (0.01)****	**1.06**	**0.04 (0.004)****	**1.04**	**0.03 (0.005)****	**1.03**	**0.05 (0.01)****	**1.05**	**0.04 (0.002)****	**1.04**	**0.09 (0.01)****	**1.09**	**0.06 (0.01)****	**1.06**	**0.05 (0.01)****	**1.05**
Gender	**−0.17 (0.06)***	**0.84**	**−1.02 (0.26)***	**0.36**	0.16 (0.10)	1.17	**−0.46 (0.14)****	**0.63**	−0.12 (0.18)	0.89	**−0.17 (0.06)***	**0.84**	**−0.94 (0.28)****	**0.39**	**−0.32 (0.14)***	**0.73**	*0.17 (0.10)*[t]	*1.18*
Pre-existing conditions	–	–	0.40 (0.27)	1.49	*0.41 (0.24)*[t]	*1.51*	**0.74 (0.15)****	**2.10**	**0.44 (0.20)***	1.55	–	–	–	–	–	–	–	

Note: All bolded parameters are statistically significant at $p < 0.05$.

Abbreviations: OR, odds ratio; *SE*, standard error.

**$p < 0.001$; *$p < 0.05$;

[t]$p < 0.10$.

Marital models: Individual cardiometabolic morbidity

Next, we expand the Poisson regression (Total Morbidities) finding by examining individual cardiometabolic developments with logistic regressions (Table 3). First, only marital strain was linked to heart attack development. Specifically, for each 1-unit increase in marital strain, the risk of having a heart attack *decreases* by 57%, contrary to our first hypothesis. Next, for HBP development, neither marital support nor strain is associated with HBP development risk. However, education is significantly linked to the risk of new HBP development. Participants who reported completing some college had an 81% *lower* risk of HBP development than participants with a college education, contrary to our second hypothesis. Additionally, there was a significant interaction between marital strain and education. While marital strain is not directly linked to the risk of HBP development, simple slopes analysis revealed that it is associated with a 73% *decrease* in the risk of HBP development for participants with a high school education ($B = -1.30$, $SE = 0.48$, OR = 0.27, $p = 0.007$). For individuals with a college education, marital strain was not linked to the risk of HBP development ($B = 0.07$, $SE = 0.12$, OR = 1.07, $p = 0.57$). This result is also inconsistent with H2, as we hypothesized marital strain would potentiate the risk to cardiometabolic health posed by lower educational attainment.

For diabetes development, analyses did not reveal evidence for a link between marital support or strain and diabetes development. However, as hypothesized, a high school education was associated with a 62% greater risk of developing diabetes than a college degree. In contrast, participants with some college have a 35% *lower* risk of diabetes development than individuals with a college education. This latter finding contradicts our second hypothesis, although aligns with several of our other results described thus far. Finally, there are no significant results for stroke development.

In sum, completing some college education is associated with a lower risk of HBP and diabetes development (compared to participants who completed a college degree), like our nonmarital family models above. Marital support is not a significant predictor of the risk of novel occurrence of any of the four cardiometabolic developments. In total, we find support for H1 when measuring FEC as marital strain, mixed evidence in support of H2, and find evidence contrary to H3 in the context of HBP development.

Marital models: Severity of cardiometabolic multimorbidity

Finally, we test the multinomial logistic regression models to examine the severity of cardiometabolic multimorbidity developments (Table 3). Among the key variables, there is only one significant factor differentiating individuals who reported one cardiometabolic development from those who experienced zero new morbidities. For these individuals, there is an interaction between marital strain and having a high school education, whereby marital strain is linked to a 62% *decreased* risk of having a new cardiometabolic development, compared to participants with zero morbidities, for individuals with a high school degree ($B = -0.98$, $SE = 0.49$, OR = 0.38, $p = 0.04$). In comparison, marital strain does not differentiate between 1 morbidity development and zero new morbidities for individuals with a college degree ($B = 0.14$, $SE = 0.12$, OR = 1.15, $p = 0.25$). This final result is contrary to H3; we hypothesized that a high school education would increase the severity of multimorbidity and that the negative impact of marital strain would potentiate the risk.

DISCUSSION

Overall, we found mixed but intriguing findings regarding the impact of FEC on cardiometabolic morbidity and multimorbidity development among adults in the context of educational

attainment. First, while we found support for our first hypothesis when measuring FEC as family strain, we failed to find broad support for the potential benefits of family or marital support, or risk of marital strain, on long-term cardiometabolic morbidity and multimorbidity, with a few notable exceptions. Additionally, we found that more stressful family relationships (i.e., greater strain) significantly differentiated between participants who developed severe cardiometabolic multimorbidity (i.e., *two or more* new cardiometabolic morbidities) compared to zero developments. In other words, the results suggest higher family strain was not solely associated with the development of one new cardiometabolic development over 20 years of adulthood but is linked to the development of cardiometabolic *multi*morbidity, including acute severe cardiac events such as heart attack and stroke. This development of cardiometabolic multimorbidity was not evident with *marital* strain, only family strain. These findings align with prior BBFM-guided research, which has found significantly worse health outcomes tied to particularly negative FECs marked by high levels of family strain (e.g., Woods, Priest, & Roberson, 2020; Woods, Roberson, & Priest, 2020). We also found surprising results from tests of our second hypothesis; namely, educational attainment operated as a nonlinear predictor of cardiometabolic morbidity. In fact, this appears to be a curvilinear association—a pattern similar to one others have identified (Cohen et al., 2013; Jeon et al., 2015). Namely, participants who earned some college education demonstrated a lower risk for cardiometabolic morbidity development than participants who earned college degrees.

Finally, we found varying results for tests of our third hypothesis. While completing some college education was frequently health-protective, family support actually increased cardiometabolic morbidity development for these participants. In contrast, greater family support was associated with a *decreased* risk of heart attack and number of cardiometabolic morbidities for participants with a high school education. Similarly, contrary to our hypothesis, *greater marital strain was associated with a decreased risk of high blood pressure development, number of cardiometabolic morbidities, and significantly predicted differences in one* versus zero cardiometabolic morbidity developments for participants with a high school education (results that do not suggest the development of multimorbidity).

Findings for Hypothesis 1 suggest that family strain (versus family support, marital support, or marital strain) may be a stronger predictor of cardiometabolic health. This is not the first study to suggest the stronger influence of adult family relationships compared to marital relationships. Recently, research using MIDUS data discovered that the quality of non-marital family relationships (especially stressful family connections) was the primary relationship indicator of health appraisal and morbidity over 10 and 20 years (Woods, Priest, & Roberson, 2020). Whereas Woods and colleagues (2020) estimated the total developments of diseases across all chronic conditions assessed in MIDUS, the present study teases out links to specific cardiometabolic morbidity and multimorbidity posing the greatest threats to midlife health. Further, prior research testing the BBFM has found a stronger association between family strain and health than family support, marital strain, or marital support and health (Priest et al., 2019; Signs & Woods, 2020; Woods, Roberson, & Priest, 2020). Even in the presence of high levels of family support, high levels of family strain appear to be linked to long-range morbidity outcomes (Woods, Roberson, & Priest, 2020), and our present study adds to this literature by emphasizing that family strain is predictive of severe cardiometabolic multimorbidity, not simply the development of a single new health condition. Taken together, these results may indicate that the most impactful relationship for health is non-marital family relationships, and specifically, stress and strain occurring in these relationships. Conflictual, demanding, non-marital family relationships may be especially powerful influencers of health given the longitudinal nature of these relationships and the potential for chronic stress to cause long-term wear-and-tear on the human body (Kiecolt-Glaser & Wilson, 2017; Priest et al., 2019; Woods, Bridges, & Carpenter, 2020; Yang et al., 2014). Overall, the results of this study provide novel evidence of the negative effects of family strain on long-term cardiometabolic health.

Hypothesis 2 points to a curvilinear effect of educational attainment. However, previous research has shown a linear effect whereby greater educational attainment is linked to better health, and specifically cardiometabolic health (Havranek et al., 2015; Johnson-Lawrence et al., 2017; Kubota et al., 2017). Our results, however, do not suggest a linear relationship between educational attainment and health. In other words, more education might not always improve health outcomes, and in our study, people with some college had better health outcomes than people with a 4-year degree. These might be explained if we consider the structural inequities linked to health outcomes and educational attainment. Individuals who are at the greatest risk of experiencing structural inequities and are traditionally excluded from college education yet can attain some college (e.g., an associate's degree or trade school) may have better access to healthcare than their family of origin, thus bolstering their long-term cardiometabolic health. Additionally, attaining a college education may launch individuals into high-stress job opportunities with a double-edged sword of higher salary and higher daily stress. Examining these educational attainment differences within a generational lens could be critical for future intervention research as Boomers may experience the benefits of a 4-year college education differently than younger generations (e.g., Millennials, Gen Alpha).

The complex associations between education and cardiometabolic morbidity and multimorbidity development were also demonstrated in tests of interaction effects between education and FEC. While family support operated as hypothesized for participants with a high school education (compared to those with college degrees), the remainder of our interaction effects did not support our Hypothesis 3. Namely, we see that family support for the some college group was linked to a 144% *increase* in 3+ multimorbidity development while higher marital strain for the high school education group experienced a 62% *decreased* risk of development. Both of these findings were the opposite of what was hypothesized. While these are longitudinal models, there may be spillover with directionality of effect. For example, it may be that premorbid cardiometabolic symptoms may increase support experienced in family relationships, especially if there is a family history of cardiometabolic disease. Similarly, strain experienced in a marital relationship could be the result of spouses working to ensure their partners follow doctor-recommended health behaviors (e.g., diet, exercise, attending medical appointments). Though previous research has indicated that "nagging" or "stonewalling" to encourage diabetes-related self-management behavior is considered by patients to be negative and non-supportive (Bennich et al., 2017), these negative health-related interactions may be both effective at supporting spouses engaging in healthy behaviors (and thus protecting against worse health outcomes) while also creating marital strain in the present sample. An important next step for this line of research is to engage dyadic data to better understand this interaction between family relationship quality, educational attainment, and cardiometabolic morbidity and multimorbidity.

As noted below, one limitation of the MIDUS data is restricted racial, ethnic, and economic diversity (Radler & Ryff, 2010; Song et al., 2018; Woods, Priest, & Roberson, 2020). Prior research suggests race moderates the benefits of education for health (Churchwell et al., 2020; Havranek et al., 2015). Specifically, this line of research indicates that the educational attainment-health gradient is weaker for Black Americans than White Americans, meaning that equivalent levels of education do not have the same protective health benefit for Black Americans as for White Americans (Assari, 2018). The present findings may point to variations in the benefits of economic attainment that occur primarily for White adults and should be interpreted with caution. Future research should replicate our categorical approach to explore educational attainment's impact and understand the potentially unique health benefit of having only some education beyond a high school diploma. Specifically, it would be important to understand how educational attainment impacts health for racially, ethnically, and economically marginalized populations historically excluded from higher education institutions.

Overall, the current educational attainment findings suggest a potentially novel health-protective impact of earning some college education (versus earning a baccalaureate or higher

degree). As originally constituted (Wood, 1993; Wood et al., 2000), the BBFM cannot explain the current findings regarding educational attainment and cardiometabolic disease developments. Prior reviews of the BBFM suggest that the model could be expanded to incorporate key social determinants of health (Wood et al., 2021). The literature suggests that educational attainment is a powerful example. As described above, previous examinations of the BBFM testing extensions of the model have manifested associations between contextual variables and FEC. Specifically, Priest and Woods' (2015) investigation of the impact of nativity status on the direct FEC-disease activity pathway for Latino Americans, and Priest and colleagues' (2020) expansion of the model to test a discrimination-FEC link for African Americans. Similarly, economic hardship in adulthood has been linked to variation in couples' cardiometabolic health (i.e., body mass index), which was then linked to disease development in later life (Wickrama et al., 2020). These authors similarly propose socioeconomic trajectories (measured to include education) as a potential contextual factor impacting family members' health over time, in part via non-marital family relationship quality. In total, testing moderators of the BBFM's mediation pathway may inform how we understand for whom the model's FEC-biobehavioral reactivity-disease link is especially powerful and where we may be most able to intervene to improve health outcomes. Specifically, tests could tease out whether levels of educational attainment serve as a chronic stressor, promoting health disparities, or serve as health-promoting via proximally influencing socioeconomic sufficiency or distally influencing levels of family support or strain.

Implications for intervention

Given the present findings, which emphasize the (1) role of family strain in later cardiometabolic disease development and (2) the protective impact of completing some college, it may be important for interventions promoting cardiometabolic health to consider both. There is a proliferation of interventions targeting primary and secondary prevention of the diseases included in this study, focusing on improving patient self-management to mitigate disease risk and improve health outcomes over time. For example, interventions to improve hypertension outcomes have emphasized lifestyle management, focusing on improving modifiable risk factors such as a lack of exercise, high sodium diet, and smoking (Jonkman et al., 2016; Yang et al., 2019). Though there is repeated evidence demonstrating the impact of family on self-management behaviors (Birditt et al., 2016; Mayberry et al., 2021; Roberson, Shorter, et al., 2018; Woods et al., 2023), disease-specific interventions rarely address the impact of patients' closest relationships (Reid et al., 2013; Shields et al., 2012; Tulloch et al., 2021; Woods, Bridges, & Carpenter, 2020). An exception may be the emotionally focused therapy-informed Healing Hearts Together program for patients with CVD and their intimate partners, though, to date, tests of the intervention have emphasized relationship quality and mental health rather than CVD management or outcomes (Tulloch et al., 2021). Separately, there is evidence to support intervening in both the family relationship and socioeconomic needs simultaneously. For example, Coop Gordon et al. (2019) demonstrated relationship quality improvements for a primarily underserved sample of couples participating in a brief relationship checkup intervention; this intervention included the tailored provision of community resources to participants, including workforce development opportunities to simultaneously address couples' socioeconomic stressors. The Coop Gordon et al. (2019) adaptation of the Marriage Checkup intervention (Cordova et al., 2014) has also been supported for medical populations, mental health, and improving health behaviors (Cordova et al., 2017; Darling et al., 2022; Gray et al., 2020; Roberson et al., 2020).

It may also be important to pair family-centered self-management interventions with family-centered policy shifts to alleviate the burden of early disease impacts on close relationships. Examples include public awareness campaigns to promote the importance of healthy family relationships on long-term physical health, incorporating relationship skill-building programs

into education, and insurance reimbursement for the intentional assessment of social support (or loneliness) in primary care to address patients in greatest need of early relationship intervention (Umberson & Karas Montez, 2010). Given the impact family strain has on the severity of cardiometabolic multimorbidity, for adults who have already experienced a significant health event (such as a heart attack or stroke), mitigating additional impacts of these disease developments on relationship distress may also be addressed via policy initiatives. For example, compensation for caregiving efforts, coverage for at-home medical care, and paid FMLA are needed to intentionally support the variations in which families may be impacted by a family member's chronic health concern. Additionally, our results linking family relationship quality to cardiometabolic morbidity development may point to the importance of family-centered care. For example, medical providers who frequently approach disease self-management recommendations to patients with premorbid symptoms or one cardiometabolic diagnosis may benefit patients' outcomes by including family members' education on behavioral change implementation and problem-solving barriers. Additionally, including education on effective communication skills and intra-family problem-solving of disease management may be an innovative strategy to slow the increasing prevalence of cardiometabolic multimorbidity in the US.

The present study is guided by the BBFM and is the first study to establish direct links between the valence of relationship quality (i.e., support versus strain) occurring in specific types of relationships (i.e., non-marital family relationships versus marital relationships) to the developments of specific cardiometabolic diseases in middle adulthood. Further, it is the first to our knowledge to test links between categories of educational attainment and future cardiometabolic disease occurrence. Findings in both areas—and their intersections—provide unique, novel areas for future investigation. To best translate the results of these subsequent studies into meaningful areas for intervention, research should continue to be theoretically driven and intentionally test the expansion of the BBFM to incorporate contextual moderators.

Limitations and future research

This study represents an important step in teasing out associations between the quality of close relationships, intersections with educational attainment, and the incidence of specific cardiometabolic diseases. However, it is not without its limitations, and the project's results present opportunities for future research. While MIDUS represents a methodological advance in assessing midlife health longitudinally, a vital consideration is the project's demographic composition, which is limited by having surveyed mostly White participants with higher educational attainment. Additionally, with the limited number of data collection follow-up times (two), advanced statistical methods such as survival analysis and growth curve models are prohibited; this study should be replicated with other datasets with more frequent data captures (e.g., Health and Retirement Study; see Fisher & Ryan, 2018). While prior research has demonstrated similar links between relationship quality and health using the BBFM with underrepresented samples (Priest et al., 2020; Roberson et al., 2023; Woods & Denton, 2014), the use of the MIDUS core sample in the present study limits our ability to contribute in this regard and results may not generalize to racially, ethnically, and socioeconomically marginalized groups. Testing longitudinal links between relationship quality and health in the context of education with systematically marginalized samples will be a critical next step. Examining the moderation of educational attainment and non-marital family relationships may not be equivalent to examining the moderation of educational attainment and marital relationships because, temporally, a family relationship may precede education, while this may not be true for a marital relationship. Although we tested family strain and support and marital strain and support (as well as parental affection) to delineate each within our regression models' unique impacts, we did not assess the intersection or co-occurrence of strain and support within relationships.

Recent research suggests that relationship ambivalence—or the experience of simultaneously high levels of strain and high levels of support within a relationship—may be especially predictive of worse adult health outcomes (Woods, Roberson, & Priest, 2020).

In addition, this study did not account for genetics, an additional factor whereby non-marital family relationships are associated with health. Likely, genetics account for part of the results found here. Not only may genetic pathways directly increase the risk of cardiometabolic diseases (Erdmann et al., 2018), but they may also be associated with the quality of family relationships (e.g., via temperament, epigenetic effects; Woods, Bridges, & Carpenter, 2020). Future BBFM-guided studies may benefit from simultaneously examining family-level genetic, socioeconomic, and relationship pathways to cardiometabolic disease.

CONCLUSION

Though prior research has delineated links between relationship quality and morbidity in midlife, the present study reflects an important next step in documenting associations between positive and negative quality in marital and (non-marital) family relationships and specific cardiometabolic morbidity and multimorbidity developments. Further, this project incorporated direct and intersectional tests, links between education and cardiometabolic morbidities, a contextual variable not often intentionally included in the family-health literature. Specifically, our findings also point to how positive and negative family characteristics may function differently across classes as defined by educational attainment. Additionally, our findings demonstrate the relative strength of family strain as a baseline variable linked to later severe disease developments. These results signify the importance of considering non-marital family relationships for adults in predicting important outcomes such as diabetes, heart attack, and stroke. Research on adult health guided by the BBFM had not yet explored specific disease outcomes. Future research building on the present study should expand these tests to incorporate more complex longitudinal models, dyadic data, and mediating factors as hypothesized by the BBFM to explain the associations between family strain and cardiometabolic morbidity and multimorbidity development.

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How to cite this article: Roberson, P. N. E., Woods, S., Tasman, J., & Hiefner, A. (2024). Relationship quality and educational attainment links to development of cardiometabolic morbidity and multimorbidity across middle adulthood. *Family Process*, *00*, 1–26. https://doi.org/10.1111/famp.13077