




In Our Lives and Under Our Skin: An Investigation of Specific Psychobiological Mediators Linking Family Relationships and Health Using the Biobehavioral Family Model

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The objective of this study was to use the Biobehavioral Family Model (BBFM) to delineate which psychophysiological variables link romantic and family relationship satisfaction variables to health outcomes. Data from individuals who reported being partnered from the second wave of the National Survey of Midlife Development in the United States (MIDUS II), Project 4 (n = 812) were used to test a structural equation model which explored which psychophysiological variables potentially mediated associations between positive and negative family emotional climate variables and disease activity. This model found that current and past family variables had larger associations with the psychophysiological variables than romantic partner variables; depressive symptoms, anxiety, and inflammation partially mediated associations between family relationships and health; and, contrary to the hypotheses, romantic partner and family support were linked to worse health outcomes. However, the findings should be viewed with regard to the cross-sectional design of the study. Overall, the findings support the use of the BBFM as a model that can guide clinical interventions.

Keywords: Families and Health; Biopsychosocial; Mental Health; Chronic Health Conditions; Biobehavioral Family Model; Biomarker

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INTRODUCTION

Research has repeatedly shown that romantic and family relationships contribute to adult mental and physical health (e.g., Carr & Springer, 2010; D’Onofrio & Lahey, 2010; Hartmann, Bazner, Wild, Eisler, & Herzog, 2010; Martire, Lustig, Schulz, Miller, & Helgeson, 2004). This research has primarily focused on three ways in which family relationships affect health: (1) direct biological pathways (e.g., genetics, infection, toxic environments), (2) learned and shared health behaviors (e.g., smoking, diet, exercise), and (3) psychophysiological pathways (e.g., psychological and physiological stress reactions related to family distress) (Campbell, 2003; Gruenewald & Seeman, 2010; Hartmann et al., 2010). Though the three pathways likely mutually influence one another over time and across the lifespan, psychophysiological mechanisms have garnered increasing focus

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in the literature (Gruenewald & Seeman, 2010; Robles, Slatcher, Trombello, & McGinn, 2014). Despite increasing evidence of psychophysiological mediators linking families and health, relationship researchers often ignore the connections between biological and romantic and family relationship processes (D'Onofrio & Lahey, 2010). Understanding psychophysiological stress reactivity is critical to understanding how relationship experiences impact health outcomes in order to determine the most effective prevention and intervention efforts to improve health outcomes for family members across the life course (Farrell & Simpson, 2016).

The purpose of this study was to expand the understanding of how psychophysiological variables link close relationships to health by addressing relevant gaps in the literature. Specifically, these limitations include that most research on close relationships and health focuses primarily on romantic relationships (i.e., marriage) while ignoring other family relationships. Additionally, the assessment of family relationships is often a broad construct that does not explore positive and negative aspects of relationships separately. Moreover, research on close relationships and health often examines only one psychophysiological variable or a composite variable, thereby ignoring the possibility that specific psychophysiological variables may overlap or serve as intervening mechanisms while others may not (Smith, Baron, & Grove, 2014). Lastly, families and health research often lacks a theoretical foundation, which may limit the applicability of findings for intervention.

An example of recent research that faces these limitations includes a recent study conducted by Donoho, Crimmins, and Seeman (2013). Using the same dataset as the present study, the researchers investigated the associations between marital support and strain and two markers of inflammation. Though the purpose of this research was to investigate one pathway by which marriage affects health, the authors only loosely draw from theory, examine solely the effects of marital relationships (for those married at least 10 years), and include only two biomarkers of inflammation, despite the seven biomarkers included in the data. Further investigation using this comprehensive dataset is possible and warranted (Donoho et al., 2013).

Overall, the purpose of addressing the gaps outlined here was twofold. The first goal was to expand the measurement and assessment of family and psychophysiological variables in order to delineate specific pathways that link close relationships to health. We did this by including both positive and negative measures of both romantic partner and family relationships, and by examining links between these measures, nine psychophysiological variables, and health outcomes. The second goal was to couch these findings in the hypotheses of the Biobehavioral Family Model (BBFM; Wood, 1993) and, in so doing, be able to demonstrate how the specific pathways found in this dataset could further inform the development of this theoretical model, as well as family interventions.

Current Limitations in Biopsychosocial Research

Family relationships are ignored

Though many studies have linked lower *romantic* relationship satisfaction to worse mental and physical health (e.g., Kiecolt-Glaser & Newton, 2001; Robles et al., 2014; Whisman, 2007), *family* relationships, those close relationships beyond the committed romantic relationship (e.g., children, siblings, parents, in-laws, etc.), are often ignored when examining adult mental health and chronic disease (Carr & Springer, 2010; Hartmann et al., 2010; Robles et al., 2014). Only slightly more than half (51.4%) of adults in the United States report living with a spouse. There is also a continual decrease in the number of married adults and increase in those who report never having married (U.S. Census Bureau, 2015). These changing demographics suggest that ignoring family relationships in examinations of adult health could exclude many unmarried adults.

Moreover, when links between adult family relationships and health are examined, findings suggest that family relationships may have stronger associations with psychophysiological stress and mental and physical health than romantic relationships (e.g., Priest & Woods, 2015; Priest et al., 2015). As evidence continues to support the powerful impacts family relationships have on health, including adult children's influences on parents' well-being (Umberson, Pudrovska, & Reczek, 2010), the importance of sibling relationships in caregiving (Namkung, Greenberg, & Mailick, 2016), and the impact of grandparents providing childcare without support (Lee, Clarkson-Hendrix, & Lee, 2016), it becomes increasingly evident that studying nonromantic relationships in adult health research is critical (Thomas, Liu, & Umberson, 2017).

Additionally, it may be important to examine both current family relationships as well as experiences with families while growing up. Research on adverse childhood experiences (ACEs) has repeatedly found links between the experience of more ACEs and worse mental and physical health problems in adulthood (e.g., Bellis, Lowey, Leckenby, Hughes, & Harrison, 2014; Mersky, Topitzes, & Reynolds, 2013). Thus, it would be important for research on families, stress, and health to examine not only how *both* romantic and family relationships are linked to health, but how past and current family relationships affect health.

Narrow operationalization

Although broad measures of romantic and family relationship satisfaction have been linked to stress and health, the measures used often provide a limited overview of solely relationship satisfaction. Many have argued for the need to examine positive and negative aspects of relationships separately, as it may provide more valid measurement and a more comprehensive view of relationship quality (e.g., Donoho et al., 2013; Kiecolt-Glaser & Newton, 2001; Mattson, Rogge, Johnson, Davidson, & Fincham, 2013). Similarly, in their study of adult children's relationships with aging parents and in-laws, Willson, Shuey, and Elder (2003) examined health as a predictor of intergenerational relationships, and state, "Feeling close to and supported by a parent does not preclude one from also harboring negative feelings toward that parent" (p. 1067–1068). It may be that positive and negative aspects of romantic and family relationships have unique associations with psychophysiological variables and/or health.

Similar to measures of relationship satisfaction, measures of psychophysiological variables in families and health research have also been limited. Use of psychophysiological measures has been more common when examining romantic relationship satisfaction and health (e.g., Robles et al., 2014), but less common when used to study family relationships and health (e.g., Priest et al., 2015). Moreover, when including psychophysiological variables in research, typically single indicators are used, such as blood pressure, or composite measures, such as allostatic load (Brooks et al., 2014; Priest et al., 2015). When measured as a composite, associations between romantic and family relationships are sometimes small or nonexistent (e.g., Brooks et al., 2014; Priest et al., 2015).

Individual stress responses to family processes are driven by several physiological systems, with individual variability and large changes over time (D'Onofrio & Lahey, 2010). Researchers may overlook specific psychophysiological mechanisms that link romantic and family relationship processes to health by using single physiological measures or composite measures of psychophysiological variables. Therefore, it would be important to examine multiple psychophysiological mechanisms individually and simultaneously, to clarify which specific objective and comparative mechanisms may link family relationships to physical health.

Theory and intervention

Researchers have been called upon to focus more on biological considerations in their examinations of family process, and this biopsychosocial research is further recommended

to be theory-driven and comprehensive (D'Onofrio & Lahey, 2010). Although the field includes multiple complex, biopsychosocial, conceptual models of family-health pathways (e.g., Berg & Upchurch, 2007; Kiecolt-Glaser & Newton, 2001; Luecken & Lemery, 2004; Uchino, 2009; Walsh, 2016), these models remain untested (Robles et al., 2014) or are infrequently used in research testing their assumptions (Priest et al., 2015; Roberson, Shorter, Woods, & Priest, 2018). When tested, research using these theoretical models focuses solely on the health of young adult (e.g., Berg et al., 2009; Fabricius & Leucken, 2007) or married adult populations or, alternatively, fails to garner evidence in support of the models' hypotheses (e.g., Uchino et al., 2016). In addition, although much research has linked romantic and family relationships to psychophysiological stressors and to health, few couple and family-based interventions have been successfully adapted for chronic health problems in adults (Campbell, 2003; Shields, Finley, Chawla, & Meadors, 2012). When adapted, they are frequently atheoretical and focus mostly on psychoeducation, rather than relationship-oriented interventions (Hartmann et al., 2010) or family therapy (Shields et al., 2012).

The Biobehavioral Family Model

One way to address these limitations is through conducting research guided by the Biobehavioral Family Model (BBFM). The BBFM is a biopsychosocial approach to health that highlights the mutually influencing roles of family, psychological, and biological processes on individual family members' experiences of health and illness (Wood, 1993; Wood & Miller, 2002). The BBFM incorporates three constructs: family emotional climate, biobehavioral reactivity, and disease activity (Wood, 1993). The construct of family emotional climate describes the intensity and positivity or negativity of ongoing family processes (Wood, 1993; Wood et al., 2008). The relative balance of positive (e.g., warm, affirming, supportive) and negative (e.g., conflictual, critical, hostile) interactions in families contributes to individual family member experiences of relational stress (Wood, Miller, & Lehman, 2015).

Biobehavioral reactivity is proposed as the psychophysiological intensity with which an individual family member reacts to the family emotional climate, representing arousal and stress regulation or dysregulation (Wood et al., 2008, 2015). Biobehavioral reactivity is the mediating construct of the BBFM that ties family processes to health outcomes (Wood, 1993). Previous research has used measures of depressive symptoms and anxiety, as well as composite measures of allostatic load, to operationalize this construct (Priest et al., 2015; Wood et al., 2008; Woods & Denton, 2014). Although distinguished as unique subconstructs (i.e., depressive and anxiety symptoms as psychological, allostatic load as physiological biobehavioral reactivity), depression and anxiety both have physiological components. These physiological symptoms (e.g., insomnia, psychomotor agitation, fatigue, nausea, pain) produce the mediating effect of depression and anxiety, such that physical manifestations of stress contribute, in turn, to illness processes. Lastly, the construct of disease activity is frequently operationalized as self-reported health and the presence of illness (Priest & Woods, 2015; Priest et al., 2015; Woods & Denton, 2014).

The BBFM predicts that a negative family emotional climate will contribute to individual family members experiencing greater biobehavioral reactivity, which in turn contributes to increased disease activity or worsened physical health (Wood, 1993; Wood et al., 2008). In other words, the model posits a mediation effect of biobehavioral reactivity, such that the influences of stressful family environments on physical health are through individual family members' experiences of psychophysiological dysregulation.

Although the hypotheses of this theoretical model have been examined many times with different samples (e.g., Priest & Woods, 2015; Priest et al., 2015; Wood & Miller, 2002;

Wood et al., 2015; Woods & Denton, 2014), research using the BBFM shares the same limitations described earlier. Specifically, when using the BBFM to examine adult health, romantic and family relationships are often examined in separate models (e.g., Priest et al., 2015), while positive and negative components of family emotional climate are examined conjointly (e.g., Woods, Priest, & Roush, 2014). Further, measures of biobehavioral reactivity have solely included depressive symptoms, anxiety, or a composite measure of psychophysiological stress (e.g., Priest et al., 2015), failing to delineate the relative effects of individual biological systems in the model. Lastly, BBFM research with adult family members has, to date, failed to incorporate considerations of adverse childhood experiences, thereby limiting operationalizations of family emotional climate to current understandings of family process.

Present Study

Using data from the National Survey of Midlife Development in the United States (MIDUS; Ryff et al., 2012) and using the framework of the BBFM, this study examined whether positive and negative aspects of romantic and family emotional climate and adverse childhood experiences (ACEs) were linked to nine individual measures of psychophysiological stress markers (two mental health measures and seven biomarkers). Additionally, in this study, psychophysiological stress markers were examined as variables that mediated the association between the family emotional climate variables and disease activity variables. The present study extends the authors' prior research examining pathways between family and intimate partner relationships, a composite measure of allostatic load, and physical health outcomes using MIDUS II data (Priest et al., 2015). Therefore, in accordance with the guiding theoretical model, the following mediation hypotheses are tested in this study:

- (1) A direct relationship between family emotional climate (i.e., adverse childhood experiences, family support and strain, and partner support and strain) and biobehavioral reactivity (i.e., depressive symptoms, anxiety, and individual allostatic load systems);
- (2) A direct relationship between biobehavioral reactivity and disease activity (i.e., number of chronic conditions, number of prescription medications, overall health quality, and quality of health compared to peers); and,
- (3) An indirect relationship between family emotional climate and disease activity, such that the effects of family emotional climate on disease activity outcomes are through the mediating construct of biobehavioral reactivity.

METHOD

Sample

Data for the present study are from the second wave of the National Survey of Midlife Development in the United States (MIDUS II), Project 4 ($N = 1,255$; Love, Seeman, Weinstein, & Ryff, 2010). MIDUS II is a follow up and extension of the original MIDUS study; Project 4 is specifically the Biomarker Project of MIDUS II. The goal of the MIDUS studies is to "delineate the biopsychosocial pathways through which converging process contribute to diverse health outcomes" (Singer & Ryff, 1999, p. 18). Project 4 was specifically designed to obtain biomarkers of health for a subsample of MIDUS II participants. These participants completed a two-day clinical visit where samples of saliva, blood, and urine were drawn, where participants' blood pressure, medication usage, and heart rate variability were assessed, and a comprehensive physical exam was given.

Participants also completed a self-report health exam. Data for this project were gathered between 2004 and 2009. A complete description of the protocol is described in Love et al. (2010).

Demographic Characteristics

The present project used those from Project 4 who reported currently being in a committed intimate partnership (i.e., married or cohabiting; $n = 812$). This constitutes 64.7% of the full Project 4 sample. In this sample, 50.7% are identified as male and 49.3% identified as female; the average age was 54.71 years old. In addition, 3.3% reported not having completed high school, 20.5% reported having a high school degree or GED, 27.9% had completed some college or graduated from a two-year college or vocational school, 24% had a Bachelor's degree, and 24% had completed some graduate school or completed a graduate degree. Median household income for the sample was \$72,500. The vast majority of the sample identified white as their primary racial origin (88.4%), with 7.1% identifying as black or African American, 1.5% identified as Native American or Alaska Native, and 2.7% identified as others. The vast majority also reported being married (97.2%) and 2.8% reported cohabiting with a partner.

Measures

Family emotional climate variables

The family emotional climate was conceptualized to consist of five constructs: family strain, family support, partner strain, partner support, and adverse childhood experiences (ACEs).

Strain

The *family strain* measure was comprised of four items that asked questions regarding how often respondents' family members (*other* than their spouse/intimate partner) make too many demands, criticize them, let them down, and get on their nerves. The *partner strain* measure was comprised of the same four items (reworded to ask about an intimate partner) but also included one additional item that asked if their partner made them feel tense. For each of these strain measures, participants responded using a Likert scale ranging from 1 (*often*) to 4 (*never*). The internal consistency was adequate for both *family strain* (Cronbach's $\alpha = .79$) and *partner strain* (Cronbach's $\alpha = .87$).

Support

The *family support* measure was also comprised of six items that asked respondents how much their family members (*other* than their spouse/intimate partner) care about them and how much the respondents care about family members, how much their family understand them, how much their family can be relied on, how much the family can be opened up to, and how much the family understand the way they feel. The *partner support* measure included six items that asked about how much their partner cares about them, understands them, appreciates them, can be relied on for help, can open up to a partner about worries, and how much the respondents can relax around their partner. For each of these support measures, participants responded using a Likert scale ranging from 1 (*a lot*) to 4 (*not at all*). The internal consistency was adequate for both *family support* (Cronbach's $\alpha = .84$) and *partner support* (Cronbach's $\alpha = .90$).

The four family emotional climate measures specific to strain and support were adapted from Schuster, Kessler, and Aseltine (1990) for the MIDUS study in order to study family and intimate partner relationship dynamics. Each of the four scales was scored by calculating the mean values of each of the items in the scale. Higher scores on each measure

suggest higher levels of the trait. Each of these scales demonstrated reliability with the MIDUS II respondents (Ryff et al., 2004).

Adverse childhood experiences

The ACEs construct was determined by five subscales of the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003). These subscales ask about the adverse childhood experiences of emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Each question within the subscale had a range from 1 (never true) to 5 (very often true) and each subscale was comprised of 5 items. Items were added together to create a composite score for each of the subscales, with scores ranging from 5 to 25; higher scores reflected greater abuse. Each of the subscale scores was then used as an indicator of a latent ACEs variable. The internal consistency across all of the subscales was adequate ranging from Cronbach's alpha .70 to .94.

Biobehavioral reactivity variables

Biobehavioral reactivity was initially measured using two constructs for psychological biobehavioral reactivity (i.e., anxiety and depressive symptoms) and seven biological systems for physiological biobehavioral reactivity (i.e., allostatic load). As discussed above, while depression and anxiety are psychological, they include physiological symptoms that contribute to the mediating effect of biobehavioral reactivity. Therefore, the depressive and anxiety symptom subscales used presently reflect the nature of these psychological subconstructs of biobehavioral reactivity, and are inclusive of physiological concomitants, such that anxiety and depression are both measured as psychophysiological mediators.

To measure anxiety and depressive symptoms, the Anxious and Depressive Symptoms subscales of the Mood and Anxiety Symptom Questionnaire (Clark & Watson, 1991) were used. The Anxious Symptoms subscale included 11 items that asked about things such as how often during the past week the respondent was startled easily, felt nervous, felt tired, was unable to relax, hands were cold and sweaty, and felt dizzy. Responses to these questions were ranged from 1—"not at all" to 5—"extremely." These items were added together, with a higher score suggesting greater levels of anxiety. This measure has shown to be reliable for MIDUS respondents (Ryff et al., 2007). Internal consistency for anxiety was adequate (Cronbach's alpha = .82).

The Depressive Symptoms subscale asked respondents 12 questions such as how often during the past week they felt depressed, thought their life was a failure, and felt sluggish or tired. Responses to these questions were ranged from 1—"not at all" to 5—"extremely." These items were added together, with a higher score suggesting greater levels depressive symptoms. This measure has also shown to be reliable for MIDUS respondents (Ryff et al., 2007). Internal consistency for depressive symptoms was adequate (Cronbach's alpha = .90).

Allostatic load

The construct of biobehavioral reactivity in the BBFM reflects the manner and intensity with which individual family members respond psychophysiological to stress and emotional challenge, particularly in the family environment. Individuals may experience emotion regulation and modulated physiological response, or conversely experience exacerbated distress and systemic dysregulation that contributes to disease activity (Wood et al., 2015). Similarly, allostasis reflects the many ways in which we respond to stress, reflecting adaptive physiological processes that respond to stress in order to promote self-protection and flexibility, especially over the life course (McEwen, 2017). However, when chronically overstimulated and distressed, the imbalanced and cumulative effects of our neural and hormonal stress responses wear on our physiological systems, promoting disease activity; this is allostatic load (McEwen, 2008).

In order to determine physiological biobehavioral reactivity we examined seven separate biological systems comprising allostatic load (Brooks et al., 2014; Gruenewald et al., 2012): sympathetic nervous system (SNS) activity, parasympathetic nervous system (PNS) activity, cardiovascular functioning, glucose metabolism, lipid/fat metabolism, hypothalamic–pituitary–adrenal axis (HPAA) activity, and inflammation. The use of seven systems is more thorough than most research studies constructing an allostatic load composite score (Buckwalter et al., 2011). Dysregulation within each of these physiological systems is calculated through multiple indicators, each presented in Table 1. Under stress, one would expect to see a coordinated, and circular, cascade of effects in each of these systems, such that they respond to and impact one another, and promote disease over time. Specifically, distress/response to stress could be reflected in elevated SNS activity, and conversely a decrease in PNS activity, or an imbalance of the two (e.g., as seen in the body’s fight-or-flight response, including increased levels of epinephrine, decreased heart rate variability), which in turn may be connected to increased cardiovascular activity (i.e., higher heart rate and blood pressure), increased immune response (i.e., inflammation, indicated by, e.g., higher levels of fibrinogen, which promotes coagulation) and HPAA activity (e.g., higher cortisol production), and impaired glucose metabolism (i.e., higher levels of hemoglobin A1c, indicative of plasma glucose concentration) and lipid/fat metabolism (e.g., indicated by elevated LDL cholesterol levels) (Gruenewald et al., 2012; McEwen, 2008, 2017).

Risk scores for each of the seven systems were calculated using the method outlined by Brooks et al. (2014), representative of high-risk ranges of allostatic dysregulation in each (Buckwalter et al., 2011). These scores could range from 0 to 1, with scores closer to 1 suggesting a higher percentage of risk. These scores were computed by using high-risk cut

TABLE 1

Variables Used to Create Risk Scores for Each of the Physiological Systems Comprising Allostatic Load

Physiological System	Indicators of System Dysregulation
Sympathetic nervous system	Norepinephrine Epinephrine
Parasympathetic nervous system	Differences in heart rate Low frequency heart rate variability High frequency heart rate variability Heart rate variability: Standard deviation of R-R Root mean squared successive
Cardiovascular	Pulse Systolic blood pressure Diastolic blood pressure
Glucose metabolism	Glucose Insulin
Lipid/Fat metabolism	Hemoglobin HDL LDL Body mass index Weight–height ratio
Hypothalamic–pituitary–adrenal axis	Triglycerides Blood DHEA-S Cortisol level
Inflammation	Serum soluble E-selectin Serum soluble ICAM-1 Blood C-reactive protein Serum IL 6 Fibrinogen

points reflective of an individual biomarker's conveyance of increased risk for worse health outcomes (e.g., the risk for cardiovascular disease), as outlined by Gruenewald et al. (2012). For example, glucose metabolism was measured by creating risk scores of three indicators— hemoglobin, glucose, and insulin. If a participant's score exceeded the cutoff for risk for the individual marker, the participant was given a score of 1. These scores were added together and then divided by the number of indicators. Therefore, if an individual had a glucose metabolism score of .33, this would suggest they had one variable which exceeded the risk value. If they had a score of .67, they had two; and a score of 1 would suggest that the respondent exceeded the risk value on each of the glucose metabolism indicators. This process replicates previous measurements of allostatic load in research using the current dataset (Brooks et al., 2014; Gruenewald et al., 2012; Priest et al., 2015).

Disease activity

Disease activity was examined using four questions. The first two items asked the respondents to report the quality of their overall health and the quality of their health compared to others their same age. The latter two items ask the respondent to report the number of symptoms and chronic conditions they had and the second question asked about the number of prescription medications each participant was currently taking. The first two items were used as indicators for a latent variable named *comparative health* and the latter two items were used as indicators for a latent variable named *morbidity*.

Analytic Plan

Structural equation modeling (SEM) was used to test the hypotheses of the study. Prior to testing the hypotheses of the study, measurement models were run to evaluate the best ways to use the family emotional climate, biobehavioral reactivity, and the disease activity variables of the study in the SEM that was used to test the studies hypotheses. Specifically, two measurement models were tested. The first measurement model had six latent factors. Specially, this model had an ACEs latent factor which applied each of the ACE variables as indicators; a family emotional climate latent factor which applied the family support and strain variables and the partner support and strain variables as indicators; a biomarker latent factor which applied all of the seven biomarker variables as indicators; a psychological distress latent factor that applied the anxiety and depressive symptoms variables as indicators; a comparative health latent factor that applied the self-rate health and comparative health question as indicators; and a morbidity latent factor that applied the chronic symptoms and prescription medication variables as indicators.

The second measurement model tested also included the ACEs latent factor, and the comparative health and morbidity latent factors; however, in this model the family support and strain, partner support and strain, biomarker, depressive symptoms, and anxiety variables were measured as observed variables. This was done in order to examine specific associations between variables. In this model, the family support and strain, the partner support and strain, and the ACE latent factor were correlated, as were the biomarker and depressive symptoms and anxiety variables.

After the measurement models identified the best fitting measurement model, the full structural model was run. This SEM model tested the hypotheses of the study. Specifically, this model estimated the direct associations between the family emotional climate variables and the biobehavioral reactivity variables, the direct association between the biobehavioral reactivity variables and the disease activity, and the indirect associations between the family emotional climate variables and the disease activity variables through the biobehavioral reactivity variables. In other words, this model examined whether the nine biobehavioral reactivity variables (cardiovascular functioning, metabolic lipids,

metabolic glucose, inflammation, sympathetic nervous system, hypothalamic–pituitary–adrenal axis, parasympathetic nervous system, anxiety, and depression) mediated the association between the five family emotional climate variables (partner strain, partner support, family strain, family support, and the latent adverse childhood experiences variable) and the two disease activity latent variables (comparative health and morbidity).

Mplus 7 was used to conduct the SEM (Muthén & Muthén, 2014) using maximum likelihood estimation with robust standard errors (MLR). Multiple linear regression (MLR) has been shown to be robust to nonnormality and nonindependence of observations (Asparouhov, 2005). Missing data were handled within Mplus with full information maximum likelihood. Mediation analyses were conducted on the full structural model using the delta method (Olkin & Finn, 1995) in Mplus. This method has been recommended in order to produce accurate standard errors and is frequently used in path analysis (MacKinnon & Luecken, 2008; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002).

To assess model fit, five fit indices were used. Specifically, the χ^2 test, the root mean square error approximation (RMSEA), the comparative fit index (CFI), the Tucker–Lewis index (TLI), and the standardized root mean square residual (SRMR) were used to evaluate model fit. Models that fit the data well typically have small nonsignificant χ^2 statistics, RMSEA, and SRMR values less than .10, and CFI and TLI values greater than .90 (Kline, 2011). The χ^2 statistic tends to perform poorly when models are being estimated with large samples and variables that are not normally distributed, as is the case in these analyses. However, the other fit statistics do not share the same limitation (Kline, 2011).

RESULTS

The mean, standard deviation, median, and range of all variables used in the models are reported in Table 2. Correlations between all variables are reported in Table 3.

Measurement Models—Latent Variables

The fit statistic for the first measurement model (the model with six latent variables) were $\chi^2 (194) = 720.030$, $p < .001$; RMSEA = .058 (90% CI .053–.062); CFI = .865; TLI = .839; SRMR = .059. Of the five statistics, only the SRMR statistic indicated good fit. The fit statistics for the second measurement model (with three latent variables and 13 observed variables) were $\chi^2 (169) = 638.782$, $p < .001$; RMSEA = .059 (90% CI .054–.063); CFI = .879; TLI = .835; SRMR = .090. This model had a similar fit to the first model; only the SRMR indicated good fit. To determine if one model had better fit for the data, a Satorra Bentler chi-square difference test was conducted. The results suggested that the second model had significantly better fit ($\chi^2 = 80.267$, $p < .001$). Because the goal of this study was to examine specific variables that link the family emotional climate to disease activity, and because the second model had significantly better fit, we used the variables from the second measurement model in the subsequent analysis. Unstandardized and standardized factor loadings for the three factor measurement model are reported in Table 4.

We also examined the correlations from the confirmatory factor analysis (CFA) model to assess potential problems with multicollinearity (Table 2). Among the predictor variables, absolute values of the correlations ranged from .22 to .64. $R = .64$ is the only correlation in the high range and the next highest correlation $r = .49$. To ensure that the one higher correlation of $r = .64$ was not problematic for multicollinearity, we calculated the variance inflation factor (VIF). The VIF is a measure of how the variance of an estimated parameter is inflated by the correlation between the variables. For this one concerning correlation, the score (VIF = 1.69) fell under the acceptable cutoff of 2.50 (Allison, 2012). Therefore, there are likely no concerns with multicollinearity.

TABLE 2
Descriptive and Distribution Statistics for all Measures Used in the Structural Equation Model

	<i>M (SD)</i>	<i>Median</i>	<i>Observed Range</i>
<i>Family emotional climate variables</i>			
Partner strain	2.15 (.60)	2.00	1.00–4.00
Partner support	3.65 (.50)	3.83	1.17–4.00
Family strain	1.99 (.56)	2.00	1.00–4.00
Family support	3.56 (.57)	3.75	1.00–4.00
ACE: emotional abuse	7.70 (3.95)	6.00	5.00–25.00
ACE: physical abuse	6.86 (2.85)	6.00	5.00–25.00
ACE: sexual abuse	6.39 (3.60)	5.00	5.00–25.00
ACE: emotional neglect	9.43 (4.27)	8.00	5.00–25.00
ACE: physical neglect	6.71 (2.61)	5.00	5.00–25.00
<i>Biobehavioral reactivity variables</i>			
Cardiovascular	.25 (.28)	.33	.00–1.00
Lipid	.25 (.26)	.25	.00–1.00
Metabolic glucose	.47 (.28)	.33	.00–1.00
Inflammation	.25 (.25)	.20	.00–1.00
Sympathetic nervous system	.22 (.35)	.00	.00–1.00
Hypothalamic–pituitary–adrenal axis	.22 (.30)	.00	.00–1.00
Parasympathetic nervous system	.22 (.36)	.00	.00–1.00
Anxiety	16.43 (4.49)	15.00	11.00–42.00
Depression	17.92 (6.27)	16.00	12.00–60.00
<i>Disease activity variables</i>			
Number of chronic illnesses	3.94 (2.82)	3.00	.00–18.00
Number of prescription medications	2.79 (2.95)	2.00	.00–22.00
Self-reported physical health	2.32 (.93)	2.00	1.00–5.00
Physical health compared to other	2.17 (.94)	2.00	1.00–5.00
<i>Demographic variables</i>			
Age	54.93 (11.47)	54.00	34.00–83.00
Gender	.49 (.50)	.00	.00–1.00
Education	7.79 (2.46)	8.00	1.00–12.00

Full Hypothesized Model

The model fit statistics for the full structural model, representing our hypotheses reflective of the BBFM, indicated a good fit to the data, with the exception of the χ^2 index (χ^2 (98) = 218.761, $p < .001$; RMSEA = .039 (90% CI .032–.046); CFI = .964; TLI = .918; SRMR = .023).

The results of the final model indicated that family emotional climate variables were significantly linked to six of the biobehavioral reactivity variables (see Figure 1). Specifically, partner strain was associated with anxiety; family strain was linked to greater anxiety and depressive symptoms; family support was linked to greater inflammation and HPAA scores and to lower depressive symptoms; and the ACEs variable was linked to greater cardiovascular system, lipid/fat metabolism, and inflammation risk, and to worse anxiety and depressive symptoms.

Of the seven biobehavioral reactivity variables, three were linked to comparative health. Specifically, greater glucose and inflammation risk scores were linked to worse comparative health, as was increased reports of depressive symptoms. Four of the biobehavioral reactivity variables were linked to morbidity. Specifically, greater inflammation, HPAA, and PNS risk scores were linked to worse morbidity, as were greater reports of anxiety.

The results of the mediation analyses (Table 5) showed that only three pathways between the family emotional climate variables and the disease activity variables had

TABLE 3
Correlations Between the Manifest and Latent Variables

	ACE	Family Support	Family Strain	Spousal Support	Spousal Strain	Objective Health	Subjective Health	Depression	Anxiety	Cardio	Lipid	Glucose	Inflam.	SNS	HPAA
Family Support	-.40 (.04)*														
Family	.27 (.04)*	-.32 (.04)*													
Strain	-.25 (.04)*	.30 (.04)*	-.17 (.04)*												
Spousal Support	.22 (.04)*	-.24 (.04)*	.29 (.04)*	-.64 (.02)*											
Spousal Strain	.11 (.04)*	-.04 (.04)	-.03 (.04)	.09 (.04)*	-.08 (.04)										
Objective Health	.16 (.04)*	-.14 (.04)*	.09 (.04)*	-.01 (.04)	.02 (.04)	.49 (.04)*									
Subjective Health	.20 (.05)*	-.25 (.05)*	.25 (.04)*	-.05 (.03)	.83 (.17)*	.18 (.06)*	.28 (.05)*								
Depression	.21 (.04)*	-.16 (.05)*	.25 (.04)*	-.005 (.03)	.58 (.13)*	.20 (.06)*	.18 (.04)*	-.68 (.03)*							
Anxiety	.10 (.04)*	.02 (.04)	-.01 (.04)	-.03 (.03)	-.002 (.01)	.06 (.04)	.11 (.04)*	.03 (.04)	.07 (.04)*						
Cardio	.15 (.04)*	-.07 (.04)*	.05 (.04)	-.01 (.03)	.01 (.01)	.05 (.04)	.19 (.04)*	.08 (.04)*	.04 (.04)	.20 (.04)*					
Lipid	-.001 (.03)	-.04 (.04)	-.01 (.04)	-.01 (.03)	-.01 (.01)	.20 (.05)*	.18 (.04)*	.01 (.03)*	-.02 (.04)	.15 (.04)*	.26 (.03)*				
Glucose	.10 (.04)*	.02 (.04)	.04 (.04)	-.03 (.03)	-.005 (.01)	.26 (.04)*	.24 (.04)*	.06 (.04)*	-.02 (.04)	.20 (.04)*	.28 (.03)*	.23 (.03)*			
Inflam.	.001 (.03)	.01 (.04)	.003 (.03)	.01 (.02)	-.003 (.01)	.12 (.04)*	.03 (.04)	.02 (.04)	.02 (.03)	.14 (.04)*	-.08 (.03)*	.03 (.04)	.10 (.04)*		
SNS	-.04 (.03)	.06 (.03)*	-.04 (.03)	-.02 (.03)	-.005 (.01)	.23 (.04)*	-.003 (.04)	-.02 (.04)	.01 (.03)	-.03 (.04)	-.13 (.04)*	.003 (.04)	.003 (.04)	.14 (.04)*	
HPAA	.02 (.04)	.01 (.04)	.005 (.04)	.01 (.03)	-.005 (.01)	.32 (.04)*	.12 (.04)*	.02 (.04)	-.03 (.04)	.17 (.04)*	.11 (.04)*	.23 (.04)*	.20 (.04)*	.12 (.04)*	.07 (.04)
PNS															

Note. HPAA = Hypothalamic-pituitary-adrenal axis; Inflam. = Inflammation; PNS = Parasympathetic nervous system; SNS = Sympathetic nervous system.

* $p < .05$.

TABLE 4
Unstandardized and Standardized Factor Loadings for the Three Factor Measurement Model

Item	Factor loadings	
	<i>B (SE)</i>	<i>β (SE)</i>
Adverse childhood experiences		
Emotional abuse	1.000 (-)	.879 (.043)
Physical abuse	.571 (.046)	.696 (.040)
Sexual abuse	.418 (.062)	.423 (.052)
Emotional neglect	.997 (.070)	.796 (.033)
Physical neglect	.547 (.051)	.742 (.051)
Comparative health		
Self-reported health	1.000 (-)	.868 (.043)
Health compared to others	.726 (.080)	.622 (.042)
Morbidity		
Symptoms and chronic conditions	1.000 (-)	.765 (.043)
Number of prescriptions	1.065 (.124)	.774 (.043)

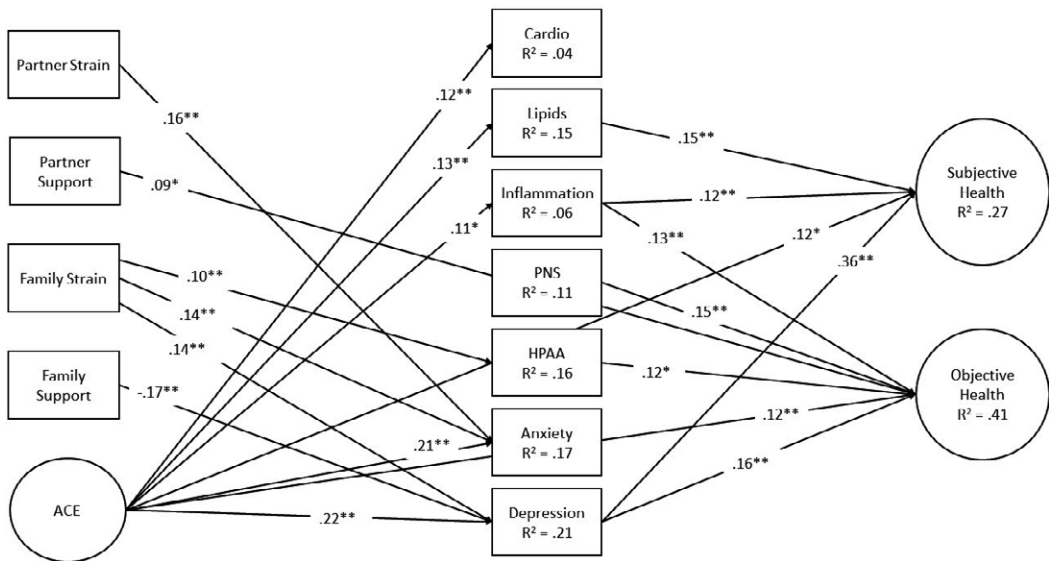


FIGURE 1. Final model, $\chi^2(98) = 218.761, p < .001$; RMSEA = .039 (90% CI .032-.046); CFI = .964; TLI = .918; SRMR = .023; non-significant ($p > .05$) paths are removed to ease interpretation. ACE = Adverse Childhood Experiences, SNS = Sympathetic nervous system, PNS = Parasympathetic Nervous System, HPAA = Hypothalamic Pituitary Adrenal Axis.

a significant total effect and at least one significant mediator. The total effect for the pathway between ACEs on comparative health was significant. Inflammation risk and depressive symptoms were both significant mediators of this association; however, the direct pathway remained significant, suggesting only partial mediation.

The total affect of the ACEs variable on morbidity was also significant. The only significant mediators of this association were inflammation risk and anxiety symptoms. With these mediators, the direct effect of this pathway was no longer significant.

TABLE 5

Results of the Mediation Analysis for Those Pathways With a Significant Total Effect and at Least One Significant Mediator

ACE → Comparative Health	Estimate	Standard Error
Total	.230**	.055
Indirect		
ACE → Cardio → Comparative Health	.002	.005
ACE → Lipids → Comparative Health	.012	.080
ACE → Glucose → Comparative Health	.000	.005
ACE → Inflam → Comparative Health	.018*	.009
ACE → SNS → Comparative Health	.000	.000
ACE → HPAA → Comparative Health	-.001	.002
ACE → PNS → Comparative Health	.001	.002
ACE → Depress → Comparative Health	.049**	.016
ACE → Anxiety → Comparative Health	-.002	.012
Direct		
ACE → Comparative Health	.149**	.054
ACE → Morbidity	Estimate	Standard Error
Total	.152**	.050
Indirect		
ACE → Cardio → Morbidity	-.006	.006
ACE → Lipids → Morbidity	-.008	.008
ACE → Glucose → Morbidity	.000	.005
ACE → Inflam → Morbidity	.023*	.010
ACE → SNS → Morbidity	.000	.002
ACE → HPAA → Morbidity	-.009	.008
ACE → PNS → Morbidity	.008	.012
ACE → Depress → Morbidity	.008	.012
ACE → Anxiety → Morbidity	.041**	.015
Direct		
ACE → Morbidity	.095	.049
Family Support → Comparative Health	Estimate	Standard Error
Total	-.122*	.051
Indirect		
Sup → Cardio → Comparative Health	.001	.003
Sup → Lipids → Comparative Health	-.001	.004
Sup → Glucose → Comparative Health	-.005	.005
Sup → Inflam → Comparative Health	.013	.007
Sup → SNS → Morbidity	.000	.000
Sup → HPAA → Comparative Health	.001	.003
Sup → PNS → Comparative Health	.001	.002
Sup → Depress → Comparative Health	-.030*	.013
Sup → Anxiety → Comparative Health	.000	.001
Direct		
Sup → Comparative Health	-.101*	.048

** $p < .01$; * $p < .05$.

The total effect of family support on comparative health was also significant. The depressive symptoms variable was the sole significant mediator, rendering the direct pathway insignificant. Additionally, there was a significant total effect of partner support on morbidity; however, none of the biobehavioral reactivity variables significantly mediated this pathway. Pathways between the family strain and partner strain variables and disease activity did not produce significant total effects.

DISCUSSION

The purpose of this study was to address previous limitations of the close relationships and health literature using the BBFM as a theoretical guide. The first limitation we addressed was that of research focusing solely on the role of marriage/romantic relationships on health outcomes, ignoring family relationship more broadly. Contrary to previous findings that linked romantic partner relationship quality to physiological stress (e.g., Donoho et al., 2013), the present findings suggest that when romantic and family relationships were included in the same model, only family relationship variables were linked to biomarker measures of allostatic load.

For example, greater reports of ACEs were associated with higher-risk cardiovascular functioning (i.e., elevated resting blood pressure and heart rate), lipid/fat metabolism (e.g., a body mass index greater than 32, elevated LDL cholesterol), and inflammation (e.g., high serum levels of interleukin-6), in the present study. This reflects previous findings by Danese et al. (2009), who found that children exposed to ACEs were at greater risk of developing elevated inflammation levels, a cluster of metabolic risk markers (similar to the present study, e.g., high blood pressure, greater BMI, low HDL cholesterol), and depression in adulthood.

Additionally, family support was significantly associated with greater inflammation and HPA axis risk; specifically, greater family support was linked to markers of inflammation and HPA axis activity. This association was in the opposite direction than expected. As noted by Keicolt-Glaser, Gouin, and Hantsoo (2010) and Stephens, Mahon, McCaul, and Wand (2016), psychosocial stressors can provoke markers of both inflammation and HPA axis. What is unique about the findings of this study is that family support, but not family strain, was linked to greater levels of risk.

One possible explanation for this finding is the role of enmeshment in families. Enmeshed families are demarcated by intense contact that may heighten support and dependency within the family but this comes at the expense of independence and exploration outside of the family (Minuchin, 1988; Olson, 2000). There is some evidence to suggest that adolescents with highly enmeshed families are at greater risk of poor emotion regulation (e.g., Kivisto, Welsh, Darling, & Culpepper, 2015). It may be that the family support measure used in this study is capturing some individuals with highly enmeshed families. This enmeshment may lead to high inflammation and HPA axis risk. Future research would benefit from exploring concepts, such as enmeshment, as factors of the family emotional climate that might influence biobehavioral reactivity and disease activity.

Another unique finding of this study was the lack of association between partner support and strain and depression. Though many previous studies have found associations between romantic partner relationship quality and depression (e.g., Whisman, 2007), when romantic and family relationship quality were included in the same model in the present study, only family relationship variables (family support, family strain, and ACEs) were linked to depressive symptoms. Additionally, the family emotional climate variables were significantly associated with many biobehavioral reactivity variables; however, partner support was not linked to any biobehavioral reactivity variables and partner strain was only significantly associated with anxiety. These findings suggest that if researchers are only studying romantic relationships when examining adult health, they may be ignoring additional relational mechanisms with potentially important effects on mental and physical health for adults.

The second limitation addressed was the narrow operationalization of romantic and family relationships satisfaction variables and psychophysiological variables in families and health research. In this study, we examined the differential impacts of positive and

negative aspects of romantic and family relationship quality on health separately. The results of the present study support the recommendation of considering the valence of relational quality in health research, as positive and negative aspects of close relationships showed different and unique associations with biobehavioral reactivity and disease activity (e.g., Donoho et al., 2013; Kiecolt-Glaser & Newton, 2001; Mattson et al., 2013; Willson et al., 2003).

For example, examining positive and negative aspects of close relationships separately resulted in another potentially unique finding. Specifically, reports of greater partner support and connection were linked to worse disease activity, or, an increased likelihood of having a chronic illness and/or using prescription medications (even when accounting for the mediating biobehavioral reactivity variables). Previous research has suggested that social support may, at times, promote problematic health behaviors, which may be contributing to these findings. Specifically, individuals with supportive partners may be sharing risky health habits including a lack of exercise or problematic drinking (Christakis & Fowler, 2007; Uchino, 2006; Wills, Yaeger, & Sandy, 2003). However, other research has suggested that chronic diseases may lead an individual to seek more support (Berg & Upchurch, 2007). In other words, given the cross-sectional nature of the data, it may be that those with chronic illness might report greater partner support because their illness requires greater support from their partner. Future research is needed to tease out the directionality behind this association.

To further address the second limitation of family-health research, this study also examined multiple, individual biobehavioral reactivity pathways of the BBFM using distinct biomarker and mental health variables. Overall, there were direct pathways between several measures of biobehavioral reactivity and physical health exogenous variables. However, only inflammation, depression, and anxiety served as partially mediating mechanisms linking family emotional climate to disease activity. These findings support the work of Kiecolt-Glaser, Gouin, and Hantsoo (2010) and Kiecolt-Glaser, Derry, and Fagundes (2015), who have also suggested depression and inflammation as the linking mechanism between social relationships and health.

It may be that psychological subconstructs of biobehavioral reactivity provide a stronger family-health pathway than allostatic stress responses. This may be in part due to allostatic biomarker measures reflecting a longitudinal process, whereby the impacts of chronic relational stress affects physiological biobehavioral reactivity over time (e.g., Danese & McEwen, 2012). It also may be that the present set of biomarkers does not fully capture the range of psychobiological mediators due to limitations of measurement, and the complicated interdependent effects of each of the body's physiological systems. Therefore, impacts on allostatic load may not be adequately represented in a cross-sectional sample. Indeed, participants' ACEs scores produced significant associations with three physiological systems: cardiovascular functioning, lipid/fat metabolism, and inflammation risk scores.

Additionally, it may be that measures assessing strain in family relationships and anxiety or depressive symptoms capture similar subjective experiences, and therefore overrepresent distress in these constructs, as opposed to more objective and physiological measures of the stress response (e.g., through measuring heart rate variability and cortisol). This effect may also be found in using a subjective self-report measure of physical health, which was captured in the present study using items assessing individual thoughts about overall health, and health compared to peers. What is interesting, however, is that the depressive symptoms measure was linked only to comparative health while anxiety symptoms measure was linked only to morbidity.

The third limitation this study sought to address was the lack of theoretical foundation that is often present in families and health research. Although the families and health

field includes multiple complex, biopsychosocial, conceptual models of family-health pathways (e.g., Berg & Upchurch, 2007; Kiecolt-Glaser & Newton, 2001; Luecken & Lemery, 2004; Uchino, 2009; Walsh, 2016), many of these models remain untested (Robles et al., 2014). When these models are tested, they have often focused solely on the health of young adult (e.g., Berg et al., 2009; Fabricius & Leucken, 2007) or married adult populations or, alternatively, these studies do not garner evidence in support of the models' hypotheses (e.g., Uchino et al., 2016).

To address this limitation, the hypotheses of this study were based on the theoretical tenets of the BBFM (Wood, 1993). Many of the BBFM's hypotheses were supported in this study. Links between the family emotional climate variables and the biobehavioral reactivity variables were found, supporting the first hypothesis of this study; links between the biobehavioral reactivity variables and the disease activity variables were found, supporting the second hypothesis of this study; and three of the biobehavioral activity variables mediated the association between some of the family emotional climate variables and the disease activity variables.

Overall, the results found here, together with previous tests of the BBFM (e.g., Priest & Woods, 2015; Priest et al., 2015; Woods & Denton, 2011), suggest strong support for the model's hypotheses. The support for these hypotheses suggests that the tenets of the BBFM may prove useful in developing theory-driven, relationship-oriented interventions aimed at reducing family stress and symptoms of mental and physical illness. Some researchers have begun to incorporate the tenets of the BBFM in family therapy approaches (e.g., Priest, 2017; Theodoratou-Bekou, Andreopoulou, Andriopoulou, & Wood, 2012). Given the continued support of the model's hypotheses, the continued clinical application of the BBFM to relational and health problems is warranted.

Limitations and Future Research

Though the aims and findings of the present study address several limitations found in the family and health literature at large, this research has limitations of its own. First, the sample used in the analysis is not representative of the US population. The sample used here was primarily white, married, with higher than average income. Given this, it would be important for future research to explore similar pathways with more diverse samples as this may change the results found in this study. This may be especially true for populations at greater risk of health disparities and worse health outcomes (e.g., minority samples, individuals with less access to socioeconomic resources, etc.). In addition, a majority of the MIDUS II, Project 4 sample (almost two-thirds) reported their marital status as currently married or cohabiting. Therefore, despite a goal of the present study to expand families and health research to include a specific consideration of family members other than intimate partners, the current sample reports having both a committed intimate relationship as well as other family members. In other words, a limitation of the present study is that we do not account for differing or unique effects of family relationships that may be found for unpartnered, single adults.

Second, as described above, the present investigation uses cross-sectional self-report data. While a cross-sectional examination of these mechanisms is important to establish associations worthy of further study, the findings (especially regarding mediation) can be interpreted in either direction. The BBFM posits reciprocal pathways, and it may be equally likely that negative close relationships predict worse health outcomes, and that worse physical health predicts greater stress in romantic and family relationships. Moreover, as Maxwell, Cole, and Mitchell (2011) suggest, in some cases cross-sectional data can produce indirect effects that might not be found in longitudinal data. Future research should use longitudinal data and autoregressive models to examine the longitudinal effects of the mediators tested in this study (Maxwell et al., 2011).

Third, the use of self-report data specific to depression, anxiety, and disease activity may unintentionally conflate these associations. And, although depression and anxiety are frequently comorbid and increasingly suggested as potential facets of a broader psychopathological construct, research has yet to tease out a full understanding of why these two sets of symptoms so frequently co-occur (Ruscio & Khazanov, 2017). Therefore, although we forced their statistical independence to meet the present aims of the study, it may be that doing so misrepresents the role of depressive symptoms and anxiety as mediators connecting family relationships with health outcomes.

Fourth, the use of “number of prescription medications” as an indicator of the morbidity latent variable might influence the associations found here. For example, individuals may take prescription medications that are used to lower inflammation or cholesterol levels. This effect might, for example, be the reason why the cardiovascular functioning variable was not related to the morbidity variable.

Finally, in conjunction with constraints due to cross-sectional data, it is likely that our operationalization of the BBFM constructs is limited by failing to capture other important mechanisms connected to health. Examples include specific health behaviors, broader contextual factors that determine access to health care, environmental stress, biomarkers measured under stressful conditions, and health indicators defined objectively through medical provider assessment, as opposed to self-report. In addition, as this is the first test of the BBFM to incorporate ACEs scores, there may be additional research necessary to tease out the differential effects of types of adverse experiences. For example, Schneider, Loveland Cook, Cleveland, and Burge (2017) found differences among classes of ACEs and associations with stressful or supportive social networks and health outcomes, whereas we included ACEs as one latent variable. The BBFM has the ability to guide further research in expanding considerations of these variables for adult health and provides a thorough, well-tested, multilevel, systemic, biopsychosocial theoretical approach to assist with generating hypotheses, conducting analyses, and developing relational interventions (Wood et al., 2015) for use with adult health. The findings of the present study support its continued use in research in this area, as well as multiple unique pathways that require further investigation in future iterations of the theoretical model.

Although the present study has many strengths and aims to build upon prior research in the field using an empirically supported theoretical model, the above limitations affect the generalizability of the current results. The findings, and specific pathways, in our final model require replication and extension over time in order to more fully support the found effects on physical health outcomes for adults.

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