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'My family is making me sick' – but, for both him and her?: examining the effect of gender on the association between close relationships and health

Patricia N. E. Roberson [a], Sarah B. Woods[b], Jacob B. Priest[c] and Melanie Miller[a]

[a]Department of Human Ecology, University of California, Davis, CA, USA; [b]Department of Behavioral Health Family and Community Medicine, University of Texas Medical Center, Houston, TX, USA; [c]Department of Rehabilitation and Counselor Education, The University of Iowa, Iowa, IA, USA

ABSTRACT

Using a biopsychosocial health approach, we examine the role of close relationships on health for men and women. With a cross-sectional US sample ($N = 872$), we examine a structural model to determine how close relationships (family and romantic) influence number of chronic disease and number of prescription medication through physiological (allostatic load) and psychological (depression/anxiety symptoms) biobehavioural reactivity differently by gender. For both men and women, family/couple relationships impact health through depression/anxiety symptoms more so than allostatic load. However, for women, family relationships can both positively and negatively influenced health outcomes when considering both indirect and direct associations. Findings indicate that there may be unmeasured coping mechanisms (eg exercise, alcohol consumption) that can differentially impact health for men compared to women. Also, when examining family and couple dimensions of relationships simultaneously, couple relationships appear to have less of an impact on health.

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There is overwhelming evidence of the impact of romantic and family relationships on physical health for adults (Carr & Springer, 2010; Robles, Slatcher, Trombello, & McGinn, 2014). Mechanisms through which close relationship influence health is limited but includes access to social support, individual mental health and physiological reactivity, healthcare utilization, and shared health behaviours (eg Carr & Springer, 2010). Recommendations for research in this area have repeatedly called for more thorough investigations of the mediators and moderators contributing to these pathways within a comprehensive biopsychosocial model (eg Carr & Springer, 2010; Robles et al., 2014). Therefore, research examining conditions under which marriages and families affect physical health must consider how contextual and demographic markers (eg gender) affect this association. There have been mixed findings regarding the role of gender on the association between relationship and health outcomes (eg Finkel et al., 2016; Williams & Umberson, 2004), possibly due to the lack of comprehensive model

CONTACT Patricia N. E. Roberson proberson@ucdavis.edu

testing, among other weaknesses of the literature (eg small sample sizes, differing foci on specific health outcomes, and varying methodologies specific to assessing close relationships). The present study seeks to move this line of research forward by testing in full a biopsychosocial framework to health, the Biobehavioral Family Model (BBFM), in order to examine gender differences in how close relationships may impact health outcomes through psychological and physiological reactivity.

Gender as a relationships-health moderator

Although there are consistently gender differences in individual variables (eg women are twice as likely as men to experience depression during their lifetime, Kessler, 2003; women live longer and have more health complaints across their lifetimes; Finkel et al., 2016), the influence of close relationships on various mental and physical health outcomes have provided inconclusive findings regarding gender difference (Carr & Springer, 2010; Robles et al., 2014). For example, in their meta-analysis, Robles et al. (2014) found no significant gender differences in the associations between marital quality and cardiovascular reactivity. Whisman, Uebelacker, Tolejko, Chatav, and McKelvie (2006) failed to find gender differences for the strength of associations between marital discord and depression and anxiety. Similarly, Davila, Karney, Hall, and Bradbury (2003), in a longitudinal study of newlywed couples' marital satisfaction and depression, found limited support for strong gender differences in within- or between-subject associations.

In contrast, some studies have found gender differences regarding the influence of close relationships on various mental and physical health outcomes. For example, Seeman, Singer, Ryff, Love, and Levy-Storms (2002) found gender differences on associations between social support and allostatic load. Specifically, using an older sample, they found that older men who reported greater support in their close relationships had lower levels of allostatic load, whereas this association was not significant for older women. Choi and Ha (2011) found low perceived partner support was associated with greater depressive symptoms for women only, whereas men and women both experienced less depression if they perceived high partner support. Similarly, found that relationship satisfaction predicted later depression for women yet predicted a different dimension of mental health (life satisfaction) for men.

Though these studies have mixed results, each of these studies only examined gender differences in the association between close relationships and mental health *or* close relationships and physical health. In order to better understand the influence of gender on these pathways, it would be important to examine the effects of close relationship on mental and physical health. Doing so may help better clarify the role, if any, that gender plays, on close relationships and health.

Allostatic load

Although an important concept specific to understanding health trajectories, especially for adults, the construct of allostatic load is relatively new and increasingly tied to research on systemic stress and disease activity (eg McEwen, 2002). Specifically, allostatic load highlights the effects of chronic stress and accompanying systemic physiological dysregulation

on long-term disease activity and individual health outcomes. As McEwen (2002) highlights, understanding allostatic load, and the resulting hormonal and structural changes for individuals' physiology, supports a focus on social support as ameliorative for maintaining or promoting positive health outcomes.

Specific to the present study's focus on analysing gender as a potential moderator of family-health pathways, McEwen (2002) has previously outlined potential sex differences in regard to the role of the hippocampus (and related neurotransmitters) in allostatic load. Specifically, differences in sex hormones (eg oestrogen) and their role in hippocampal plasticity may contribute to outcomes specific to the brain's resilience to stress. Prior research investigating pathways between relational health and health outcomes has discovered only minimal differences in allostatic load specific to gender. As noted above, while older men demonstrate a significant pathway between emotional support and lower allostatic load, similar but nonsignificant associations are found for older women (Seeman et al., 2002). Additionally, Geronimus, Hicken, Keene, and Bound (2006) found gender differences solely for African American adults and their health outcomes, unlike White samples, such that racial disparities in health and allostatic load scores are larger among women than men. Lastly, more recent findings highlight that gender may moderate allostatic load's contribution to the stress impacts of residence in an environmentally risky region on cardiovascular health risk (Maira, Cutchinb, & Peek, 2011).

Directly applicable for the present research, Priest et al. (2015) have recently utilized the concept of allostatic load (albeit, as a latent construct) to demonstrate the utility of the construct within the BBFM (Wood, 1993; Wood et al., 2008), and the theoretical model's ability to explain health outcomes for adults as a result of close family and intimate partner relationships. The present study therefore represents a replication and extension of this work, with a broad focus on model building and testing gender differences that may occur for each of the model's hypothesized pathways.

The BBFM: theoretical framework

Despite the proven interactions and comorbidities between mental and physical health (Prince et al., 2007), families and health researchers continue to investigate the effects of relationships either on one or the other. In addition, health outcomes examined in the literature continue to focus on broad, general health outcomes (eg mortality, self-rated global health) rather than specific health outcomes, measures of health risk, or physiological indicators of health, despite recommendations to move in this direction (Carr & Springer, 2010). Therefore, a biopsychosocial approach (Engel, 1977) to researching the specific mediators and moderators of pathways connecting close relationships and health is required such as the BBFM (Wood, 1993).

The BBFM is a multilevel, systemic, biopsychosocial model (Wood, Miller, & Lehman, 2015) and has been used to investigate the specific pathways by which family relationships affect both child and adult health outcomes for several years. This theoretical model describes the interdependence of relational and psychophysiological processes contributing to physical health outcomes for individual family members. Specifically, the BBFM posits that family emotional climate, which is defined as the quality (eg positivity/negativity, intensity) of family relationships, influences individual family members' biobehavioural reactivity (or, individuals' behavioural, emotional, and physiological responses

to stress; Wood et al., 2008). In other words, biobehavioural reactivity is conceptualized as individual family members' responsiveness to emotional stimuli, and represents emotion regulation (or dysregulation) and accompanying physiological regulation (or, dysregulation) (Wood et al., 2008). Biobehavioural reactivity, specific to individual family members' dysregulation, transmits the effects of contextual (ie family) stress to individuals' processes of disease, both in-the-moment and over time. Therefore, the construct of biobehavioural reactivity is the mediator through which family emotional climate contributes to physical health outcomes for individual family members. This mediating construct is typically measured as depression and anxiety symptoms, representative of disorders of emotion regulation (eg Wood et al., 2008; Woods & Denton, 2014), and more recently as allostatic load, representative of chronic physiological reactivity to stress and strain which is promoting disease activity (eg Priest et al., 2015).

Research has repetitively demonstrated the validity of the BBFM for adult populations, including for a general epidemiological sample (Woods, Priest, & Roush, 2014), an underserved primary care sample (Woods & Denton, 2014), for Latino Americans (Priest & Woods, 2015), and in incorporating allostatic load as a measure of biobehavioural reactivity using the present sample (Priest et al., 2015). In addition, the model has been tested with families with some found gender differences in relational security between mothers and fathers and their children with asthma (Wood et al., 2008).

Present study

The current study uses the BBFM as a theoretical framework to test the associations between close relationships (assessed as both romantic and family relationships) and health for adults through biobehavioural reactivity, and to examine potential gender differences in the model's pathways. As a theoretical model that has been tested in full, across multiple populations, and consistently substantiated, the BBFM provides advantages over earlier theory used to highlight connections between close relationships and physical health for adults as previously discussed. However, while the BBFM has been validated with paediatric and adult populations, and in examining social support (friends and relatives; Woods et al., 2014), family emotional climate, and intimate partner emotional climate (eg Priest et al., 2015), the model has yet to be examined with gender as a moderator. Given the many convoluted findings regarding the effects of gender on pathways connecting close relationships and health, examining this moderator with the BBFM is critical.

The present study represents an extension and replication of earlier BBFM research testing the application of the theoretical model for exploring adult health. Specifically, Priest et al. (2015) use the present dataset to test the inclusion of allostatic load in the model, and determined the construct is a meaningful addition to conceptualizing the model's construct of biobehavioural reactivity. Therefore, replicating hypotheses in this and other applications of the BBFM (eg Woods & Denton, 2014), we hypothesize the following mediation relationship:

1) A significant, direct pathway between family emotional climate (for both family and romantic relationships) and biobehavioural reactivity (measured as both depression and anxiety symptoms, and allostatic load);

2) A significant, direct pathway between biobehavioural reactivity and disease activity (measured using two distinct, objective measures of health);
3) Full mediation of the pathway between family emotional climate and disease activity, such that there is a significant, indirect mediation pathway of family emotional climate on disease activity through biobehavioural reactivity, and no significant direct pathway from family emotional climate to disease activity.

Lastly, given the mixed findings in the literature regarding the effects of gender on associations between close relationships and health, we explored the role of gender on each of the BBFM's pathways tested in the Structural Equation Model (SEM) depicted in Figure 1.

Method

Sample

Data for this study come from the National Survey of Midlife Development in the United States (MIDUS; Ryff et al., 2012). MIDUS is a national, longitudinal study which investigated the behavioural, psychological, and social factors accounting for health and well-being in a sample of over 7000 American adults. This proposal will use data from wave two of MIDUS, MIDUS II. MIDUS II was designed to examine patterns, predictors, and consequences of mental and physical health in midlife development and included the Biomarker Project. This project used a subset of respondents from the overall MIDUS II sample and asked them to participate in a clinical visit. This clinical visit included an assessment of seven different physiological systems: the cardiovascular, metabolic lipids, metabolic glucose, inflammation, the hypothalamic pituitary adrenal axis, and

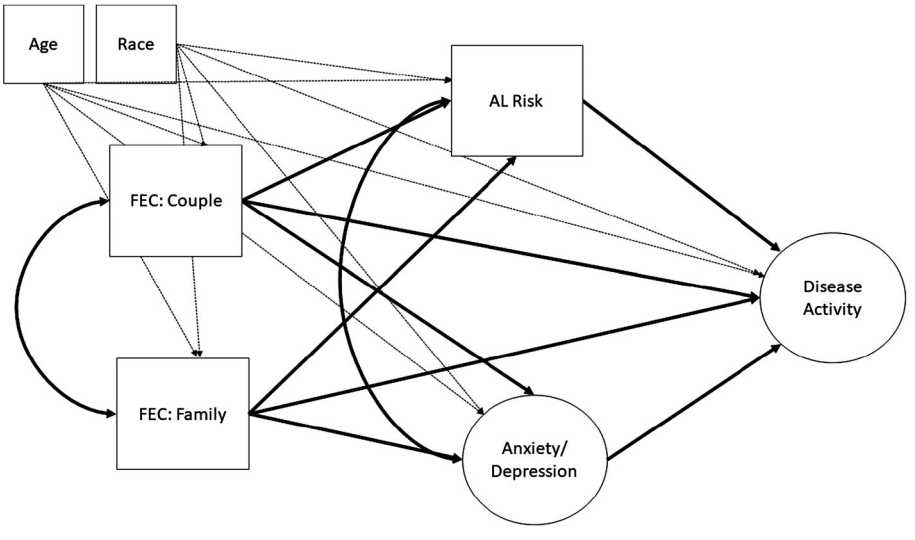

Figure 1. Tested Structural Equation Model of the BBFM for family emotional climate–romantic relationship and family relationships.
Note. Dotted lines indicate control variables.

the sympathetic and parasympathetic nervous systems. Additionally, there was comprehensive assessment given regarding levels of depression, anxiety, and physical health. A subsample of $n = 1255$ participated in the biomarkers project which included $n = 1054$ who participated in the original MIDUS study and an additional 201 sample recruited specifically for MIDUS II. A complete description of the protocol for the Biomarker Project is found in Love, Seeman, Weinstein, and Ryff (2010). In this study, we limit participants further by including only those in romantic relationships ($n = 872$). While the study had breadth and depth in terms of questionnaire items, the sample is predominantly White and middle to upper income.

Demographic characteristics

The present sample includes 57% women and were an average age of 54.54 (SD = 11.71). In terms of education, 3.2% reported no completing high school, 20.7% had a high school degree or GED; 28.7% had completed some college or graduated from a 2-year college or vocational school, 24% had a Bachelor's degree, and 23.4% had completed some graduate school or completed a graduate degree. This sample is 78% White with a mean personal income of $41,538. When compared to the original MIDUS study, those from MIDUS II Project 4 were more likely to have completed college, and less likely to smoke or have completed only high school or some college (Love et al., 2010).

Measures

Averages and distribution (eg range, skewness) for all variables are reported in Table 1.

Family emotional climate–family relationships

We measure family emotional climate by the observed variable of family strain for models 1 and 3. Family strain included four questions, for which participants were prompted to evaluate their family relationships *other* than with their spouse or intimate partner (ie 'How often do your family members make too many demands?,' 'How often do your family members criticize you?,' 'How often to your family members let you down?,' and 'How often do you family members get on your nerves?'). Response options range from 1 'not at all' to 4 'a lot' and are averaged so that higher scores meant more strain.

Family emotional climate–romantic relationships

We measure this variable by the observed variable of partner strain for models 2 and 3. The partner strain composite included six questions ('How often do you argue with your spouse/partner?,' 'How often does your spouse/partner make too many demands on you?,' 'How often does spouse/partner make you feel tense?,' 'How often does your spouse/partner criticize you?,' 'How often does your spouse/partner let you down?,' and 'How often does your spouse/partner get on your nerves?'). Response options range from 1 'not at all' to 4 'a lot' and are averaged so that higher scores meant more strain.

Biobehavioural reactivity

Biobehavioural reactivity includes measures of anxiety symptoms, depression symptoms, and allostatic load risk in all three models. The Anxious Symptoms and Depressive

Table 1. Descriptive statistics for all manifest variables for the entire sample ($N = 1255$).

	Mean (*SD*)	Median	Observed range	Skewness (*S.E.*)	Kurtosis (*S.E.*)
Number of prescription drugs	Men: 2.62 (2.78)	Men: 2.00	Men: 0–15	Men: 1.33 (.12)	Men: 1.75 (.24)
	Women: 2.98 (3.12)	Women: 2.00	Women: 0–22	Women: 2.10 (.12)	Women: 7.27 (.24)
Number of symptoms & chronic conditions	Men: 3.72 (2.55)	Men: 3.00	Men: 0–15	Men: 0.95 (.12)	Men: 1.52 (.24)
	Women: 4.13 (3.07)	Women: 4.00	Women: 0–18	Women: 1.09 (.12)	Women: 1.44 (.24)
AL risk	Men: 1.94 (0.95)	Men: 1.87	Men:.33–4.50	Men:.53 (.13)	Men: −0.18 (.26)
	Women: 1.86 (1.04)	Women: 1.67	Women: 0–5.07	Women: 0.64 (.13)	Women: −0.04 (.26)
Depressive Symptoms	Men: 7.27 (7.20)	Men: 5.00	Men: 0–45	Men: 1.84 (.12)	Men: 4.19 (.24)
	Women: 7.70 (7.67)	Women: 6.00	Women: 0–49	Women: 1.98 (.12)	Women: 5.17 (.24)
Anxious Symptoms	Men: 15.85(4.05)	Men: 15.00	Men: 11–36	Men: 1.59 (.12)	Men: 3.63 (.24)
	Women: 16.89 (4.57)	Women: 16.00	Women: 11–42	Women: 1.53 (.12)	Women: 4.80 (.24)
Support from Family	Men: 3.51 (0.57)	Men: 3.75	Men: 1–4	Men: −1.38 (.12)	Men: 1.53 (.24)
	Women: 3.62 (0.56)	Women: 3.75	Women: 1–4	Women: −2.32 (.12)	Women: 5.73 (.24)
Strain from family	Men: 1.93 (0.55)	Men: 2.00	Men: 1–3.75	Men: .44 (.12)	Men: 0.34 (.24)
	Women: 2.03 (0.57)	Women: 2.00	Women: 1–4	Women: .46 (.12)	Women: 0.18 (.24)
Support from Partner/ Spouse	Men: 5.75 (2.01)	Men: 6.00	Men: 3–12	Men: .84 (.12)	Men: 0.62 (.24)
	Women: 5.85 (2.19)	Women: 5.00	Women: 3–12	Women: .61 (.12)	Women: −0.34 (.24)
Strain from Partner/ Spouse	Men: 3.68 (0.47)	Men: 3.83	Men: 1.67–4	Men: −2.08 (.12)	Men: 4.29 (.24)
	Women: 3.55 (0.58)	Women: 3.67	Women: 1–4	Women: −1.88 (.12)	Women: 3.60 (.24)

Symptoms subscales of the Mood and Anxiety Symptom Questionnaire (Clark & Watson, 1991) are used to measure anxiety and depression symptoms. The Anxious Symptoms subscale was composed of four items (eg 'How often during the past week were you startled easily?'), which are averaged so that a higher score represents more anxiety symptoms. The Depressive Symptoms subscale is composed of three items (eg 'How often in the past week did you feel sad?'), which are averaged so that higher scores represent more depressive symptoms. Responses for both subscales range from 'not at all' to 'extremely.' Anxiety and depression symptoms are used as latent indicators for subjective biobehavioural reactivity.

Allostatic load

We use allostatic load as an additional measure of biobehavioural reactivity. Replicating previous studies that use an allostatic load measure (Gruenewald et al., 2012; Seeman et al., 2002), seven biological systems are examined: the cardiovascular system, metabolic lipids, metabolic glucose, inflammation, the hypothalamic pituitary adrenal axis, and the sympathetic and parasympathetic nervous systems. Contrary to Priest et al. (2015), which used five system indicators to develop a latent construct of allostatic load, the present study includes all seven biological systems and calculates allostatic load risk as a manifest variable.

For each of the seven systems, we calculate an allostatic load risk score. Scores are computed by coding each of the indicators as either 0 or 1; a 1 meaning that their score on the indicator is high risk (see Gruenewald et al., 2012). Then all indicators in each system are summed and divided by the number of indicators in each system. For example, cardiovascular functioning has three indicators: (1) resting systolic blood pressure, (2) diastolic blood pressure, and (3) pulse rate. This method is repeated for each system. Then, systems' scores are added together and divided by seven (the number of systems) to

create a composite allostatic load score with higher scores suggesting higher allostatic load risk. In the present a priori model (Figure 1), the allostatic load composite score is used as an observed variable of objective biobehavioural reactivity.

Disease activity

We measure disease activity with two questions in all three models as latent indicators. The first question asked about the number symptoms and chronic conditions of each respondent. The number of present conditions for each respondent ranges from 0 to 18. The second question asked about the number of prescription medications each participant is currently taking. Responses to this question range from 0 to 22. These questions have been used in the previous test of the BBFM (Priest et al., 2015; Priest & Woods, 2015; Woods et al., 2014).

Results

Preliminary analyses

We first examine the correlation matrix among all of the observed variables for men and women in the SEM models (Table 2). There was a wide range of correlations among the variables (absolute value range for $r = .02–.63$). We next examine the distribution of variables for men and women. Allostatic load risk, anxiety symptoms, depression symptoms, and chronic conditions, and prescription medications were not normally distributed (see Table 1) so we use the maximum likelihood robust (MLR) estimator in Mplus to reduce parameter biases estimation due to the non-normally distributed residual estimates. Finally, we ran confirmatory factory analysis of the latent constructs to test the measurement model for the subsequent models ($\chi^2(1) = 1.14$, $p = .28$, RMSEA = .013 [95% CI: .000, .095], CFI = 1.00, TLI = .99; standardized factor loadings: .67–.87). The measurement model fits the data well according to Kline's (2015) recommended cutoffs (CFI > .90, TLI > .90, and RMSEA < .08).

Hypotheses tests

To test the Hypotheses 1–3, we use SEM to test the a priori model (Figure 1). We use both family and romantic relationship strain as part of the family emotional climate in order to examine the effect of each while controlling for the other. Test of the full model (depicted in Figure 1) indicates adequate fit ($\chi^2(11) = 10.36$, $p = .50$, RMSEA = .000 [90% CI: .000–.036], CFI = 1.00, TLI = 1.002) according to Kline's recommendations (2015). Unstandardized estimates, standard errors, effect sizes, and latent factor loadings are in Table 3. Results indicate that A/D and AlRisk mediate the association between family relationships and health but only A/D mediates the association between romantic relationship and health. Using the MODEL INDIRECT function in Mplus, this finding is confirmed in the indirect analysis (Family → A/D → Health: $B = .32$, $S.E. = .11$ $p = .00$; Family → AlRisk → Health: $B = .11$, $S.E. = .05$ $p = .02$; RR → A/D → Health: $B = .22$, $S.E. = .07$ $p = .00$; RR → AlRisk → Health: $B = -.002$, $S.E. = .04$ $p = .95$).

Gender Multigroup. With the multigroup analysis function in Mplus, we examine if gender differences exist for this final model. The multigroup analysis had an adequate fit to the data ($\chi^2(26) = 31.13$, $p = .22$, RMSEA = .022 [90% CI: .000, .048], CFI = .99

Table 2. Correlation matrix among observed variable (men correlations are in the bottom of the matrix. women correlations are in the top of the matrix).

	Symptoms and chronic conditions	Prescription meds	AL risk	Depressive symptoms	Anxious symptoms	Family support	Family strain	Partner support	Partner strain
Symptoms and chronic conditions	–	.56**	.34**	.26**	.23**	−.12*	.16**	.001	.04
Number of prescription drugs	.63**	–	.30**	.19**	.17**	−.12*	.09	.08	.02
AL risk	.28**	.28**	–	.07	.01	−.004	.07	−.01	.04
Depressive symptoms	.18**	.17**	−.03	–	.63**	−.29**	.34**	−.19**	.21**
Anxious symptoms	.07	.04	−.04	.59**	–	−.24**	.31**	−.17**	.26**
Support from family	−.02	.02	.05	−.33**	−.17**	–	−.35**	.28**	−.22**
Strain from Family	−.06	−.004	−.04	.28**	.28**	−.28**	–	−.22**	.33**
Support from partner/spouse	.02	.11*	.05	−.19**	−.08	.35**	−.13**	–	−.69**
Strain from partner/spouse	−.04	−.13*	−.11*	.22**	.19**	−.27**	.32**	−.62**	–

**$p < .001$; *$p < .05$.

Table 3. The unstandardized estimates, standard errors, effect sizes and latent factor loadings for the test of the BBFM ($N = 783$) and multigroup test (men $n = 386$; women $n = 397$) with family emotional climate (FEC): romantic relationships (RRs) and family relationships.

Structural paths	*B* (*S.E.*)	*B*	*p*-value	Effect size
FEC: RR → Anxiety/Depression symptoms	1.03 (.28)	.18	.000	.13
Female: FEC: RR → Anxiety/Depression	1.16 (.42)	.20	.006	.13
Male: FEC: RR → Anxiety/Depression	0.82 (.36)	.15	.024	.12
FEC: RR → Allostatic load risk	−0.004 (.06)	−.002	.947	.002
Female: FEC: RR → Allostatic load risk	0.02 (.09)	.02	.780	.01
Male: FEC: RR → Allostatic load risk	−0.04 (.08)	−.02	.617	.02
FEC: Family → Anxiety/Depression symptoms	1.68 (.36)	.26	.000	.17
Female: FEC: Family → Anxiety/Depression	1.95 (.57)	.29	.001	.17
Male: FEC: Family → Anxiety/Depression	1.36 (.46)	.23	.003	.15
FEC: Family → Allostatic load risk	0.19 (.07)	.11	.008	.10
Female: FEC: Family → Allostatic load risk	0.27 (.12)	.14	.023	.11
Male: FEC: Family → Allostatic load risk	0.11 (.09)	.06	.221	.06
Anxiety/Depression Symptoms → Disease activity	0.20 (.04)	.28	.000	.18
Female: Anxiety/Depression → Disease activity	0.22 (.06)	.33	.000	.18
Male: Anxiety/Depression → Disease activity	0.12 (.04)	.18	.004	.15
Allostatic Load Risk → Disease activity	0.59 (.11)	.24	.000	.19
Female: Allostatic Load Risk → Disease activity	0.72 (.16)	.30	.000	.22
Male: Allostatic Load Risk → Disease activity	0.43 (.12)	.18	.000	.18
FEC: RR → Disease activity	−0.26 (.14)	−.07	.070	.06
Female: FEC: RR → Disease activity	−0.44 (.22)	−.11	.040	.10
Male: FEC: RR → Disease activity	−0.02 (.19)	−.005	.917	.005
FEC: FEC: Family → Disease activity	0.39 (.17)	.09	.022	.08
Female: FEC: Family → Disease activity	0.63 (.26)	.14	.017	.12
Male: FEC: Family → Disease activity	0.02 (.22)	.01	.915	.004
Allostatic Load corr. Anxiety/Depression symptoms	0.22 (.14)	.07	.123	.06
Female: Allostatic Load corr. Anxiety/Depression	0.17 (.23)	.05	.474	.04
Male Allostatic Load corr. Anxiety/Depression	0.24 (.16)	.09	.123	.08
Manifest item	Standardized factor loadings (S.E.)		*p*-value	
Anxiety/Depression symptoms				
Anxiety Symptoms	0.83 (.04)		.000	
Female: Anxiety	0.84 (.05)		.000	
Male: Anxiety	0.84 (.05)		.000	
Depressive Symptoms	0.76 (.04)		.000	
Female: Depressive symptoms	0.74 (.05)		.000	
Male: Depressive symptoms	0.75 (.05)		.000	
Disease activity				
Symptoms	0.87 (.04)		.000	
Female: Symptoms	0.84 (.05)		.000	
Male: Symptoms	0.90 (.04)		.000	
Prescriptions	0.68 (.04)		.000	
Female: Prescriptions	0.67 (.04)		.000	
Male: Prescriptions	0.70 (.04)		.000	

$^{**}p < .001$; $^{*}p < .05$; $^{\dagger}p < .10$.

TLI = .99). Unstandardized estimates, standard errors, effect sizes, and latent factor loadings are in Table 3 for men and women. Results of the multigroup analysis and tests of indirect paths indicate that for men, only family is linked to health through A/D. For women, all indirect paths were significant except for romantic relationships on health through AlRisk (Table 4). Finally, we ran a series of Wald chi-square difference, results indicate that only two indirect paths were significantly different for men and women: Family → A/D → Health and Family → AlRisk → Health (Table 5). These results indicate that while women's family strain has a significantly greater impact on their health compared to men; though, men's family strain is also influential. However, romantic relationship appears to be similarly influential on health from men and women.

Discussion

The purpose of this study was to examine gender as a moderating variable of the hypothesized pathways of the biopsychosocial model, the BBFM. Though this model has been tested multiple times with diverse samples (eg Priest et al., 2015; Priest & Woods, 2015; Wood et al., 2008; Woods & Denton, 2014), the impact of gender on the model's pathways remained unknown. Further, the current literature points to discrepancies in the role of gender on the association between close relationships and health (eg Finkel et al., 2016; Williams & Umberson, 2004). This study addresses these limitations by a SEM with two conceptualizations of family emotional climate (family of origin and romantic relationship) for men and woman using the MIDUS II dataset.

Three patterns emerge from these results. The first pattern is that though Hypothesis 1 is partially supported with a significant link between family emotional climate and anxiety/depression, there is a lack of association between family emotional climate variables and allostatic load, in opposition of Hypothesis 1. With the exception of women, there were no associations found between either family or romantic relationship emotional climate variable and allostatic load. This finding differs from previous research testing the BBFM with the same data. Specifically, when Priest et al. (2015) tested the BBFM using MIDUS data, they found an association between family emotional climate variables and allostatic load. Critically, however, Priest et al. (2015) used only negative aspects of family emotional climate (ie strain received, strain given) while the present study measures family emotional climate using both positive and negative measures (ie strain and support). Moreover, as discussed above, the present study utilizes a cumulative allostatic load risk score including all seven biomarker systems collected in MIDUS. Priest et al. (2015) only used five of the seven systems to test a latent construct of allostatic load. These different findings using the same theoretical orientation and data suggest negative aspects of family emotional climate may be more closely related to allostatic load, whereas both positive and negative aspects of family emotional climate are associated with anxiety and depression symptoms. It will be useful in future research to examine how specific markers of chronic physiological dysregulation and allostatic load (eg inflammation, HPAA) may uniquely link positive and negative aspects of family emotional climate to disease activity, both in-the-moment and across time.

In the second pattern, consistent with previous (Priest et al., 2015), Hypothesis 2 was substantiated whereby allostatic load and anxiety/depression were both linked with

Table 4. Indirect paths for men and women in the BBFM with family emotional climate (FEC)–romantic relationship (RRs) and family relationships.

Indirect Path	Bootstrap result for unstandardized indirect path
Women	
FEC: RR→ Anxiety/Depression Symptoms → Disease Activity	$B = 0.26$, *S.E.* = .11, 95% CI: .08, .44, $p = .02$
FEC: RR → Allostatic Load Risk → Disease Activity	$B = 0.02$, *S.E.* = .06, 95% CI: −.09, .12, $p = .78$
FEC: Family → Anxiety/Depression Symptoms → Disease Activity	$B = 0.43$, *S.E.* = .20, .95% CI: .11, .76, $p = .03$
FEC: Family → Allostatic Load Risk → Disease Activity	$B = 0.19$, *S.E.* = .09, 95% CI: .04, .34, $p = .03$
Men	
FEC: RR → Anxiety/Depression Symptoms → Disease Activity	$B = 10$, *S.E.* = .06, 95% CI: .01, .20, $p = .07$
FEC: RR → Allostatic Load Risk → Disease Activity	$B = -0.02$, *S.E.* = .04, 95% CI: −.08, .04, $p = .62$
FEC: Family → Anxiety/Depression Symptoms → Disease Activity	$B = 0.16$, *S.E.* = .08, 95% CI: .03, .31, $p = .05$
FEC: Family → Allostatic Load Risk → Disease Activity	$B = 0.05$, *S.E.* = .04, 95% CI: −.02, .12, $p = .26$

Table 5. Wald chi–square tests for multigroup model for family emotional climate: romantic relationships (RR) and family relationships.

Tested path	Wald Test
FEC: RR → Anxiety/Depression Symptoms	Wald $\chi^2(1) = .38$, $p = .53$
FEC: RR → Allostatic Load Risk	Wald $\chi^2(1) = .30$, $p = .58$
FEC: Family → Anxiety/Depression Symptoms	Wald $\chi^2(1) = .65$, $p = .42$
FEC: Family → Allostatic Load Risk	Wald $\chi^2(1) = 1.13$, $p = .29$
Anxiety/Depression Symptoms → Disease Activity	Wald $\chi^2(1) = .1.91$, $p = .17$
Allostatic Load Risk → Disease Activity	Wald $\chi^2(1) = 2.05$, $p = .15$
FEC: RR → Disease Activity	Wald $\chi^2(1) = 2.15$, $p = .14$
FEC: Family → Disease Activity	Wald $\chi^2(1) = 2.96$, $p = .08$
FEC: RR→ Anxiety/Depression Symptoms → Disease Activity	Wald $\chi^2(1) = .83$, $p = .36$
FEC: RR → Allostatic Load Risk → Disease Activity	Wald $\chi^2(1) = 1.76$, $p = .18$
FEC: Family → Anxiety/Depression Symptoms → Disease Activity	Wald $\chi^2(1) = 7.70$, $p = .005$*
FEC: Family → Allostatic Load Risk → Disease Activity	Wald $\chi^2(1) = 4.67$, $p = .03$*

Note: Significant (*$p < .05$) Wald chi-square tests indicate that men and women paths are statistically different.

disease activity. Also, this hypothesis was substantiated for both men and women. While there are pervasive differences in how men and women are socialized, psychological (anxiety/depression) and physiological (allosteric risk) responses to stress similarly impact their health. Therefore, future research must focus on both if and how men and women differently experience stress as a reaction to their social context in order to improve health outcomes.

In the third pattern to emerge, we found unique effects for men in the null findings for Hypothesis 3. When there were gender differences in the model (the indirect paths from Family FEC to disease activity), it was because the association for men was not statistically significant and therefore did not substantiate the hypotheses. These null findings for men are similar to that of previous studies that have shown the difference between men and women, but in dissimilar directions. For example, Repetti, Robles, and Reynolds (2011) found that men had higher cortisol levels when there was less social engagement and women did not, while women had higher cortisol levels when they were more socially engaged and men did not. Additionally, Choi and Ha (2011) found that lower partner support was associated with greater depression for women but not significant for men. The results suggest that, for men, the indirect effect of allostatic load and anxiety/depression does not link to family emotional climate to disease activity. It could be that for men, the family emotional climate interacts differently with their allostatic load risk and anxiety/depression; perhaps this is due to societal gender socialization and this finding would be different (diminished or greater) cross-culturally. Additionally, there could be unmeasured mechanisms through which these variables are associated that are unique to older men in the United States. Future research should continue to examine the mechanisms through which family emotional climate, including both family and romantic relationships, impact adult men.

Furthermore, Hypothesis 3 (*a full mediation of the pathway between family emotional climate and disease activity, through biobehavioural reactivity*) was partially substantiated for women, but for different reasons. For women, there appears to be both direct and indirect effects of family emotional climate on disease activity, which was not true for men. Therefore, it appears that the hypotheses of the BBFM are substantiated less often for men compared to women. Even still, for women, family emotional climate may have a dual effect on physical health outcomes whereby family relationships have a

positive effect on health directly and indirectly through biobehavioural reactivity (expected according to the BBFM); also, there is a direct negative effect on health for romantic relationships (unexpected according to the BBFM). This could indicate that depending on the source of the relationship and the mechanism through which emotional climate influences health, a negative family emotional climate can either improve or diminish individual health depending on individual or contextual factors. This finding may not be novel and may reflect alternate research findings such as a dual effect using the BBFM. A first example includes Roberson and others' study of a couple intervention in a Southern community in which relational aggression (ie Conflict Tactic Scale) was found to have a direct positive effect on healthcare utilization and an indirect negative effect on healthcare utilization through depressive symptoms. Second, recent research examining the applicability of the BBFM longitudinally using MIDUS data found a negative direct effect between romantic partner relationship quality and disease activity and a positive indirect association between partner relationship quality and disease activity through biobehavioural reactivity (measured as depressive symptoms and health behaviours). This dual effect, however, is not well understood and these findings point to the complexity of the impact of family emotional climate on physical health outcomes. It is imperative for future research to examine and understand health through a biopsychosocial lens and the mechanisms of through which family emotional climate impacts health.

Limitations

This study is not without limitations. First, there are several factors that may influence the association between family emotional climate and health including relationship duration, socioeconomic status, race/ethnicity, relationship stability. Further, it is unclear if specific demographic characteristics drive both the quality of close relationships and health outcomes potentiating these found associations to be spurious. Future research must tease apart these effects and close relationship as they can potentially buffer the negative effects of social determinants of health. However, because of these demographic limitations, these results may not be generalizable to the general population until we can understand how these associations function within these specific contexts. Second, these data are cross-sectional therefore the causal direction, despite the theorized model, may be in a different order. Further, there is a potential cohort effect so results may be different with a younger or older population as findings change with a newer cohort of adults (see Fry, 2016). Also, the generalizability of these findings is likely limited to the White and middle to upper-income individuals who were over represented in the MIDUS sample. Third, strain and support in the family emotional climate was measured broadly so it is difficult to know what specific aspects of support (eg physical or emotional) or strain (eg physical aggression, emotional distance, disagreements on parenting) drive the effects of these findings. Further, this study does not examine if strain or support are differently associated with the outcomes (See Walen & Lachman, 2000 for this examination). Finally, this study is a test of the BBFM which hypothesizes the mediation of Biobehavioural Reactivity between Family Emotional Climate and Disease Activity. However, the theory also hypothesizes a feedback loop whereby poor health can negatively impact the family emotional climate. The present study does not

preclude reverse causation and suggests future longitudinal studies should test this feedback loop hypothesis.

Conclusion

Overall, this study addresses some of the limitations of previous research. Specifically, previous research exploring close relationships and health has looked mainly at the marital/intimate partner relationship and has ignored the quality of the family relationship. The results here suggest that when it comes to depression symptoms, anxiety symptoms, and health, family relationship quality may be more salient than marital/intimate partner relationship quality. Additionally, this study used a biopsychosocial model of health (Engel, 1977). The vast majority of research exploring close relationship and health, and specifically the research exploring the role of gender as a moderator, has not incorporated the measure of allostatic load, mental health, and physical health simultaneously. Finally, as with previous research, the results here suggest unclear results regarding the role of gender in close relationships and health. It may be that if instead of using gender as a moderator, it may be important to explore gender-related moderators (Robles et al., 2014). It may be that exploring issues of relational power, control, influence, work outside the home, etc., may provide better and more in-depth understanding how gender may influence close relationships and health.

Disclosure statement

No potential conflict of interest was reported by the authors.

ORCID

Patricia N. E. Roberson http://orcid.org/0000-0001-7746-0548

References

Carr, D., & Springer, K. W. (2010). Advances in families and health research in the 21st century. *Journal of Marriage and Family*, *72*, 743–761.

Choi, N. G., & Ha, J.-H. (2011). Relationship between spouse/partner support and depressive symptoms in older adults: Gender difference. *Aging & Mental Health*, *15*, 307–317.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*(3), 316.

Davila, J., Karney, B. R., Hall, T. W., & Bradbury, T. N. (2003). Depressive symptoms and marital satisfaction: Within-subject associations and the moderating effects of gender and neuroticism. *Journal of Family Psychology*, *17*, 557–570.

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, *196*, 129–136.

Finkel, D., Franz, C. E., Horwitz, B., Christensen, K., Gatz, M., Johnson, W., & Silventoinen, K. (2016). Gender differences in marital status moderation of genetic and environmental influences on subjective health. *Behavioral Genetics*, *46*, 114–123.

Fry, R. (2016, May). *For first time in modern era, living with parents edges out other living arrangements for 18- to 34-year-olds*. Washington, D.C.: Pew Research Center.

Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among Blacks and Whites in the United States. *American Journal of Public Health*, *96*, 826–833.

Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., & Seeman, T. E. (2012). History of socioeconomic disadvantage and allostatic load in later life. *Social Science & Medicine*, *74*(1), 75–83.

Kessler, R. C. (2003). Epidemiology of women and depression. *Journal of Affective Disorders*, *74*(1), 5–13.

Kline, R. B. (2015). *Principles and practice of structural equation modeling*. New York, NY: Guilford.

Love, G. D., Seeman, T. E., Weinstein, M., & Ryff, C. D. (2010). Bioindicators in the MIDUS national study: Protocol, measures, sample, and comparative context. *Journal of Aging and Health*, doi:0898264310374355

Maira, C. A., Cutchinb, M. P., & Peek, M. K. (2011). Allostatic load in an environmental Riskscape: The role of stressors and gender. *Health Place*, *17*(4), 978–987. doi:10.1016/j.healthplace.2011.03.009

McEwen, B. S. (2002). Sex, stress and the hippocampus: Allostasis, allostatic load and the aging process. *Neurobiology of Aging*, *23*, 921–939.

Priest, J. B., & Woods, S. B. (2015). The role of close relationships in the mental and physical health of Latino Americans. *Family Relations*, *64*(2), 319–331.

Priest, J. B., Woods, S. B., Maier, C. A., Parker, E. O., Benoit, J. A., & Roush, T. R. (2015). The Biobehavioral Family Model: Close relationships and allostatic load. *Social Science & Medicine*, *142*, 232–240.

Prince, M., Patel, V., Saxena, S., Maj, M., Maselko, J., Phillips, M. R., & Rahman, A. (2007). No health without mental health. *Lancet*, *370*, 859–877.

Repetti, R. L., Robles, T. F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology*, *23*(03), 921–938.

Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological Bulletin*, *140*, 140–187.

Ryff, C., Almeida, D. M., Ayanian, J., Carr, D. S., Cleary, P. D., Coe, C., & Mroczek, D. K. (2012). National Survey of Midlife Development in the United States (MIDUS II), 2004-2006. ICPSR04652-v6. *Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor]*, 04-18.

Seeman, T. E., Singer, B. H., Ryff, C. D., Love, G. D., & Levy-Storms, L. (2002). Social relationships, gender, and allostatic load across two age cohorts. *Psychosomatic Medicine*, *64*(3), 395–406.

Walen, H. R., & Lachman, M. E. (2000). Social support and strain from partner, family, and friends: Costs and benefits for men and women in adulthood. *Journal of Social and Personal Relationships*, *17*(1), 5–30.

Whisman, M. A., Uebelacker, L. A., Tolejko, N., Chatav, Y., & McKelvie, M. (2006). Marital discord and well-being in older adults: Is the association confounded by personality? *Psychology and Aging*, *21*, 626–631.

Williams, K., & Umberson, D. (2004). Marital status, marital transitions, and health: A gendered life course perspective. *Journal of Health and Social Behavior*, *45*, 81–98.

Wood, B. L. (1993). Beyond the "psychosomatic family": A biobehavioral family model of pediatric illness. *Family Process*, *32*(3), 261–278.

Wood, B. L., Lim, J., Miller, B. D., Cheah, P., Zwetsch, T., Ramesh, S., & Simmens, S. (2008). Testing the biobehavioral family model in pediatric asthma: Pathways of effect. *Family Process*, *47*(1), 21–40.

Wood, B. L., Miller, B. D., & Lehman, H. K. (2015). Review of family relational stress and pediatric asthma: The value of biopsychosocial systemic models. *Family Process*, *54*(2), 376–389.

Woods, S. B., & Denton, W. H. (2014). The biobehavioral family model as a framework for examining the connections between family relationships, mental, and physical health for adult primary care patients. *Families, Systems, & Health*, *32*(2), 235.

Woods, S. B., Priest, J. B., & Roush, T. (2014). The biobehavioral family model: Testing social support as an additional exogenous variable. *Family Process*, *53*(4), 672–685.